Obsessive-compulsive disorder:

Core interventions in the treatment of obsessive-compulsive disorder and body dysmorphic disorder

National Clinical Practice Guideline Number 31

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1. INTRODUCTION

This guideline has been developed to advise on the identification, treatment and management of obsessive-compulsive disorder (OCD) and body dysmorphic disorder (BDD). Although distinct disorders, OCD and BDD share a number of common features and there is a high degree of similarity between the treatments for the two conditions. The guideline recommendations have been developed by a multidisciplinary team of healthcare professionals, people with OCD, a carer and guideline methodologists after careful consideration of the best available evidence. It is intended that the guideline will be useful to clinicians and service commissioners in providing and planning high quality care for those with OCD and BDD while also emphasising the importance of the experience of care for people with OCD, BDD, and carers.

This guideline addresses aspects of service provision, psychological and pharmacological approaches for those with OCD and BDD from the age of 8 upwards. Although the evidence base is rapidly expanding, there are a number of major gaps and future revisions of this guideline will incorporate new scientific evidence as it develops. The guideline makes a number of research recommendations specifically to address these gaps in the evidence base. In the meantime, we hope that the guideline will assist clinicians, people with these disorders and their carers by identifying the merits of particular treatment approaches where the evidence from research and clinical experience exists.

1.1 NATIONAL GUIDELINES

1.1.1 What are clinical practice guidelines?

Clinical practice guidelines are 'systematically developed statements that assist clinicians and patients in making decisions about appropriate treatment for specific conditions' (Mann, 1996). They are derived from the best available research evidence, using predetermined and systematic methods to identify and evaluate the evidence relating to the specific condition in question. Where evidence is lacking, the guidelines incorporate statements and recommendations based upon the consensus statements developed by the Guideline Development Group (GDG).

Clinical guidelines are intended to improve the process and outcomes of healthcare in a number of different ways. Clinical guidelines can:

- Provide up-to-date evidence-based recommendations for the management of conditions and disorders by healthcare professionals
- Be used as the basis to set standards to assess the practice of healthcare professionals
- Form the basis for education and training of healthcare professionals

- Assist patients and carers in making informed decisions about their treatment and care
- Improve communication between healthcare professionals, patients and carers
- Help identify priority areas for further research.

1.1.2 Uses and limitations of clinical guidelines

Guidelines are not a substitute for professional knowledge and clinical judgement. They can be limited in their usefulness and applicability by a number of different factors: the availability of high quality research evidence, the quality of the methodology used in the development of the guideline, the generalisability of research findings and the uniqueness of individuals with OCD.

Although the quality of research in OCD and BDD is variable, the methodology used here reflects current international understanding on the appropriate practice for guideline development (AGREE: Appraisal of Guidelines for Research and Evaluation Instrument; www.agreecollaboration.org), ensuring the collection and selection of the best research evidence available, and the systematic generation of treatment recommendations applicable to the majority of people with these disorders and situations. However, there will always be some people and situations for which clinical guideline recommendations are not readily applicable. This guideline does not, therefore, override the individual responsibility of healthcare professionals to make appropriate decisions in the circumstances of the individual, in consultation with the person with OCD and/or carer.

In addition to the clinical evidence, cost-effectiveness information, where available, is taken into account in the generation of statements and recommendations of the clinical guidelines. While national guidelines are concerned with clinical and cost effectiveness, issues of affordability and implementation costs are to be determined by the NHS.

In using guidelines, it is important to remember that the absence of empirical evidence for the effectiveness of a particular intervention is not the same as evidence for ineffectiveness. In addition, of particular relevance in mental health, evidence-based treatments are often delivered within the context of an overall treatment programme including a range of activities, the purpose of which may be to help engage the person with OCD, and to provide an appropriate context for the delivery of specific interventions. It is important to maintain and enhance the service context in which these interventions are delivered; otherwise the specific benefits of effective interventions will be lost. Indeed, the importance of organising care in order to support and encourage a good therapeutic relationship is at times as important as the specific treatments offered.

1.1.3 Why develop national guidelines?

The National Institute for Health and Clinical Excellence (NICE) was established as a Special Health Authority for England and Wales in 1999, with a remit to provide a single

source of authoritative and reliable guidance for patients, professionals and the public. NICE guidance aims to improve standards of care, to diminish unacceptable variations in the provision and quality of care across the NHS and to ensure that the health service is patient-centred. All guidance is developed in a transparent and collaborative manner using the best available evidence and involving all relevant stakeholders.

NICE generates guidance in a number of different ways, two of which are relevant here. First, national guidance is produced by the Technology Appraisal Committee to give robust advice about a particular treatment, intervention, procedure or other health technology. Second, NICE commissions the production of national clinical practice guidelines focused upon the overall treatment and management of a specific condition. To enable this latter development, NICE has established seven National Collaborating Centres in conjunction with a range of professional organisations involved in healthcare.

1.1.4 The National Collaborating Centre for Mental Health

This guideline has been commissioned by NICE and developed within the National Collaborating Centre for Mental Health (NCCMH). The NCCMH is a collaboration of the professional organisations involved in the field of mental health, national patient and carer organisations, a number of academic institutions and NICE. The NCCMH is funded by NICE and is led by a partnership between the Royal College of Psychiatrists' research unit (College Research and Training Unit – CRTU) and the British Psychological Society's equivalent unit (Centre for Outcomes Research and Effectiveness – CORE).

1.1.5 From national guidelines to local protocols

Once a national guideline has been published and disseminated, local healthcare groups will be expected to produce a plan and identify resources for implementation, along with appropriate timetables. Subsequently, a multidisciplinary group involving commissioners of healthcare, primary care and specialist mental health professionals, patients and carers should undertake the translation of the implementation plan into local protocols taking into account both the recommendations set out in this guideline and the priorities set in the National Service Framework for Mental Health and related documentation. The nature and pace of the local plan will reflect local healthcare needs and the nature of existing services; full implementation may take a considerable time, especially where substantial training needs are identified.

1.1.6 Auditing the implementation of guidelines

This guideline identifies key areas of clinical practice and service delivery for local and national audit. Although the generation of audit standards is an important and

necessary step in the implementation of this guidance, a more broadly based implementation strategy will be developed. Nevertheless, it should be noted that the Healthcare Commission will monitor the extent to which Primary Care Trusts (PCTs), trusts responsible for mental health and social care and Health Authorities have implemented these guidelines.

1.2 THE NATIONAL OBSESSIVE-COMPULSIVE DISORDER GUIDELINE

1.2.1 Who has developed this guideline?

The Guideline Development Group was convened by the NCCMH and supported by funding from NICE. The GDG included people with OCD and a carer, and professionals from psychiatry, clinical psychology, child psychology, nursing, and general practice.

Staff from the NCCMH provided leadership and support throughout the process of guideline development, undertaking systematic searches, information retrieval, appraisal and systematic review of the evidence. Members of the GDG received training in the process of guideline development from NCCMH staff and people with OCD received training and support from the NICE Patient and Public Involvement Programme. The NICE Guidelines Technical Adviser provided advice and assistance regarding aspects of the guideline development process.

All GDG members made formal declarations of interest at the outset, which were updated at every GDG meeting. The GDG met a total of 21 times throughout the process of guideline development. The GDG met as a whole, but key topics were led by a national expert in the relevant topic. The GDG was supported by the NCCMH technical team, with additional expert advice from special advisers where needed. The group oversaw the production and synthesis of research evidence before presentation. All statements and recommendations in this guideline have been generated and agreed by the whole GDG.

1.2.2 For whom is this guideline intended?

This guideline will be relevant for people with a diagnosis of obsessive-compulsive disorder (OCD) or body dysmorphic disorder (BDD) aged 8 years and over.

The guideline covers the care provided by primary, community, secondary, tertiary, and other healthcare professionals who have direct contact with, and make decisions concerning the care of adults, children and young people with OCD and BDD.

The guideline will also be relevant to the work, but will not cover the practice, of those in:

- occupational health services
- social services
- the independent sector.

The experience of OCD or BDD can affect the whole family and often the community. The guideline recognises the role of both in the treatment and support of people with these conditions.

1.2.3 Specific aims of this guideline

The guideline makes recommendations for the identification, treatment and management of OCD and BDD. Specifically, it aims to:

- Evaluate the role of specific psychological interventions in the treatment and management of OCD and BDD.
- Evaluate the physical management and role of specific pharmacological agents in the treatment of OCD and BDD.
- Evaluate the role of other biological interventions in the management of OCD and BDD.
- Integrate the above to provide best practice advice on the care of individuals with a diagnosis of OCD or BDD throughout the course of the disorder.
- Promote the implementation of best clinical practice through the development of recommendations tailored to the requirements of the NHS in England and Wales.

1.2.4 The structure of this guideline

The guideline is divided into chapters, each covering a set of related topics. The first two chapters provide a general introduction to guidelines and to OCD/BDD. The third chapter provides testimonies regarding the experience of people with OCD and BDD and their families and carers. The fourth chapter details the methods used to develop the guideline. Chapters 5 to 8 provide the evidence that underpins the recommendations and Chapter 9 covers the use of health service resources. The final chapter provides a summary of the recommendations.

Each evidence chapter begins with a general introduction to the topic that sets the recommendations in context. Depending on the nature of the evidence, narrative reviews or meta-analyses were conducted. Therefore, the structure of the chapters varies. Where appropriate, details about current practice, the evidence base and any research limitations are provided. Where meta-analyses were conducted, information is given about both the interventions included and the studies considered for review. This is followed by selected clinical evidence statements (a complete list of evidence statements can be found in Appendix 18). Clinical summaries are then used to summarise the evidence presented. Finally, recommendations related to each topic are presented at the end of each chapter. On the CD-ROM, full details about the included studies can be found in Appendix 16. Where meta-analyses were conducted, the data are presented using forest plots in Appendix 17 (see Text Box 1 for details).

Text Box 1: Appendices on CD-ROM

Content	**Appendix**
Included/excluded studies	Appendix 16
Forest plots for psychological interventions	Appendix 17a
Forest plots for pharmacological interventions	Appendix 17b
Forest plots for psychological versus pharmacological interventions, combination therapy, and other medical interventions	Appendix 17c
Clinical evidence statements	Appendix 18

2. OBSESSIVE-COMPULSIVE DISORDER AND BODY DYSMORPHIC DISORDER

This guideline is concerned with the identification, treatment and management of obsessive-compulsive disorder (OCD) and body dysmorphic disorder (BDD) as defined in the 10th edition of the *International Classification of Diseases* (ICD-10) (World Health Organization, 1992). Reference is also made to criteria from the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) for OCD and BDD in this chapter.

2.1 OCD

2.1.1 Symptoms, presentation, and patterns of illness

Obsessive-compulsive disorder is characterised by the presence of either obsessions or compulsions, but commonly both. An obsession is defined as an unwanted intrusive thought, image or urge, which repeatedly enters the person's mind. Obsessions are distressing but are acknowledged as originating in the person's mind, and not imposed by an outside agency. They are usually regarded by the individual as unreasonable or excessive. A minority are regarded as having overvalued ideas (Veale, 2002) and, rarely, delusions. The person usually tries to resist an obsession, but in chronic cases this may be to a very minor degree or not at all. The most common obsessions are listed in Table 1. The percentages refer to the frequency in a survey of 431 individuals with OCD (Foa *et al.*, 1995).

Unwanted intrusive thoughts, images or urges are almost universal in the general population and their content is usually indistinguishable from clinical obsessions (Rachman & de Silva, 1978). Examples include having the urge to push someone under a train or a thought that the cooker has been left on. According to current psychological models, the difference between a normal intrusive thought and an obsessional thought is the meaning that OCD patients attach to the occurrence and/or content of the intrusions. Individuals with OCD tend to believe that intrusive thoughts and urges are dangerous or immoral and that they are able to prevent harm occurring either to their self or a vulnerable person (Salkovskis *et al.*, 1995).

Compulsions are repetitive behaviours or mental acts that the person feels driven to perform. A compulsion can either be overt and observable by others, such as checking that a door is locked, or a covert mental act that cannot be observed as in repeating a certain phrase in the mind. Covert compulsions are generally more difficult to resist or monitor than overt ones as they are can be performed anywhere without others knowing and are easier to perform. A compulsion is not in itself pleasurable which differentiates it from impulsive acts, such as shopping or gambling, that are associated with immediate gratification. The term 'ritual' is synonymous with compulsion but usually refers to behaviours that other people can see.

Table 1: Common obsessions in OCD

Obsession	Percent
Contamination from dirt, germs, viruses (e.g. HIV), bodily fluids or faeces, chemicals, sticky substances, dangerous material (e.g. asbestos)	37.8%
Fear of harm (e.g. door locks are not safe)	23.6%
Excessive concern with order or symmetry	10.0%
Obsessions with the body or physical symptoms	7.2%
Religious, sacrilegious or blasphemous thoughts	5.9%
Sexual thoughts (e.g. being a paedophile or a homosexual)	5.5%
Urge to hoard useless or worn out possessions	4.8%
Thoughts of violence or aggression (e.g. stabbing one's baby)	4.3%

Adapted from Foa, E.B., Kozak, M.J., Goodman, W.K., *et al.* (1995) DSM-IV field trial: Obsessive compulsive disorder. *American Journal of Psychiatry*, *152*, 990–996. *Reprinted with permission from the American Journal of Psychiatry, Copyright 1995. American Psychiatric Association.*

'Rumination', which refers to prolonged thinking that is experienced as uncontrollable around and around the same subject, includes both intrusive thoughts, often in the form of doubts or questions, and repeated attempts to find an answer. In this way it covers both the obsession (the doubts or question) and the accompanying compulsive thinking which attempt to answer the question.

Just as with obsessions, there are many types of compulsions; the most common are listed in Table 2. The most frequent presentations are checking and cleaning and are the most easily recognised as on a continuum with everyday behaviour. Repeating compulsions can be of any type of behaviour and may be motivated by a broad range of concerns or fears. In some cases ordering, symmetry and exactness may appear to have a tic-like character to them, but in other cases they are clearly related to perceived threat. Hoarding consists of the acquisition and/or failure to discard objects. In some cases this may be excessive quantities of particular materials that may have some apparent value related to perceived threat or may result from the inability to dispose of materials because of the threat associated with it. However in many cases, hoarding consists of accumulating material that appears to have little or no value. In the former case it is perhaps more likely that other OCD symptoms may be found. In the latter case, there is debate as to the extent to which it may share the key features of OCD (Steketee & Frost, 2003).

Mental compulsions by definition are not observable by others and people may be less likely to be able to describe these cognitive acts. For example, mental compulsions may consist of complex sequences of counting in one's head or special

Table 2: Common compulsions in OCD

Compulsion	Percent
Checking (e.g. gas taps)	28.8%
Cleaning, washing	26.5%
Repeating acts	11.1%
Mental compulsions (e.g. special words or prayers repeated in a set manner)	10.9%
Ordering, symmetry or exactness	5.9%
Hoarding/collecting	3.5%
Counting	2.1%

Adapted from Foa, E.B., Kozak, M.J., Goodman, W.K., *et al.* (1995) DSM-IV field trial: Obsessive compulsive disorder. *American Journal of Psychiatry*, *152*, 990–996. *Reprinted with permission from the American Journal of Psychiatry, Copyright 1995. American Psychiatric Association.*

thoughts that must be formed according to specific rules. Other terms are also used. 'Neutralising' resembles a mental compulsion but is not identical to it. Both are usually anxiety reducing. However, neutralising is not necessarily as stereotypic as a compulsive urge but has the aim of 'undoing' the perceived harm. Neutralising, for example, could consist of briefly forming a positive image to counter an intrusive negative image.

The term 'safety seeking behaviours' is also used in the cognitive behavioural therapy (CBT) literature to refer to any actions in a feared situation that aim to prevent feared catastrophes and reduce harm (Salkovskis, 1985) and will therefore include both compulsions and neutralising behaviours. Examples of other safety seeking behaviours include various mental activities such as trying to be sure of the accuracy of one's memory or trying to suppress or distract oneself from unacceptable thoughts which may reduce anxiety in the short term but may lead to a paradoxical enhancement of the frequency of the thought in a rebound manner.

The aim of a compulsion or neutralising behaviour is thus to reduce harm or feel 'comfortable' or 'just right' and is an additional criterion used for terminating a compulsion. Someone without OCD finishes hand-washing when they can see that their hands are clean. However, someone with OCD and a fear of contamination finishes not only when they can see that their hands are clean but also when they feel 'clean', 'comfortable' or 'just right'. Although avoidance behaviour is not part of the definition of OCD, it is an integral part of the disorder and is most commonly seen in fears of contamination. Typically an individual may avoid touching a wide range of objects or activities to prevent the obsession and distress from occurring.

2.1.2 Diagnosis

The diagnostic criteria for the two main international classification systems, ICD-10 and DSM-IV, are virtually identical and must include the presence of either obsessions or compulsions. The patient must acknowledge that the obsessional thoughts, impulses, or images are a product of their mind and are not imposed by an outside person or influence. At least one obsession or compulsion must be acknowledged as excessive or unreasonable (although patients holding obsessions with delusional intensity are reported). Furthermore, the obsessions or compulsions must cause marked distress, or significantly interfere with the patient's occupational and/or social functioning, usually by wasting time. The exclusion clause is that the obsessions or compulsions are not best explained by another mental disorder. Traditionally it has been believed that insight (the ability to recognise the senselessness of the obsessions) is a key feature of OCD. However, there is growing recognition that the level of insight is highly variable (Lochner & Stein, 2003). Thus some people with OCD may show stable but low levels of insight, others may show insight when not confronted with the feared situation, but lose this insight when their anxiety is high in situations associated with their obsessive fears. There is some evidence showing that insight into the condition is poor among people with particular forms of the disorder, especially hoarding (Lochner & Stein, 2003).

Although in DSM-IV, OCD is classified as an anxiety disorder, in ICD-10 it is included within the broad category of neurotic, stress-related and somatoform disorders but is a separate subcategory from the corresponding disorders with which it is classified in DSM-IV (the corresponding ICD-10 subcategories are the phobic anxiety disorders, the other anxiety disorders, and reaction to severe stress and adjustment disorder). (Please see Appendix 15 for a comparison of diagnostic criteria from ICD-10 and DSM-IV.) From some perspectives (for example, cognitive behavioural therapy models and treatments), OCD shares many important features with other anxiety disorders; from other perspectives, which include biological models and response to pharmacological treatments, it is considered that OCD has more in common with other groups of disorders. For example, some consider it to be an affective disorder (for example, Montgomery, 1993) or as part of a broader OCD spectrum (for example, Hollander, 1998). In summary, although the core features of OCD reach broad consensus, associated features, putative subgroups and its taxonomic status all generate much discussion.

OCD shares a number of surface similarities with obsessive-compulsive personality disorder or traits. Some theories propose that obsessive-compulsive personality disorder (OCPD) is a precursor of OCD or that there is a specific relationship between such personality features and the disorder. However, there is little evidence to date either for a specific premorbid personality pattern, or for a specific association between OCD and OCPD (see Black & Noyes, 1997; Albert *et al*., 2004).

2.1.3 Physical and social consequences

The severity of OCD differs markedly from one person to another. Individuals may be able to hide their OCD often from their own family. However, the disorder may have a major negative impact on social relationships leading to frequent family and marital discord or dissatisfaction, separation or divorce (Koran, 2000). Most studies have found lower rates of marriage among people with OCD than in the general population. It also interferes with leisure activities (Antony *et al*., 1998) and with a person's ability to study or work, leading to diminished educational and/or occupational attainment and unemployment (Koran, 2000; Leon *et al*., 1995). The social cost, that is the person's inability to fully function within society, has been estimated as $5.9 billion in 1990, or 70.4% of the total economic cost of OCD (Dupont *et al*., 1995). OCD is ranked by the World Health Organization in the top 10 of the most disabling illnesses by lost income and decreased quality of life (Bobes *et al.*, 2001).

OCD can have a severe impact on daily activities and family life, and family members may report distress (Amir *et al*., 2000; Magliano *et al.*, 1996; see also Chapter 3, Section 3.5). It can be particularly difficult for families when the person with OCD has poor insight into the disorder. In these cases the person will have difficulty recognising that their concerns are excessive, that they may have OCD, or indeed that they may need help. There may also be a financial burden on the family (Chakrabarti *et al.*, 1993). Although people with OCD often succeed in not letting their symptoms interfere with family responsibilities, there is some limited evidence that parental OCD can sometimes affect children (for example, Black *et al*., 1998; Black *et al.*, 2003). The mechanisms are not yet known, but in one study, children of a parent with OCD were more likely to have emotional, social, and behavioural problems in comparison with children of parents without OCD (Black *et al*., 2003). When children have OCD, parent-child relationships also are changed and there is some evidence that parents and children may behave differently from children with other disorders, particularly around problem-solving and independence (Barrett *et al.*, 2002). Finally, in some rare cases, the symptoms of a parent with OCD may directly impact on the well-being of family members, for example, when concerns about contamination can occasionally lead to extreme hygiene measures being applied to family members.

2.1.4 Course and prognosis

For some people, the symptom type will remain unchanged, but for others there may be changes over time (Rettew *et al*., 1992; Skoog & Skoog, 1999). For some, the change may remain within the symptom type, for example different types of checking, especially in the short term (Mataix-Cols *et al.*, 2002b). OCD may follow an acute, episodic or chronic course. In one of the largest follow-up studies, Skoog and Skoog (1999) conducted a 40-year prospective study and reported that approximately 60% of people with OCD displayed signs of general improvement within 10 years of illness, increasing to 80% by the end of the study. However, only 20%

achieved full remission even after almost 50 years of illness; 60% continue to experience significant symptoms; 10% displayed no improvement; and 10% had deteriorated. A fifth of those patients, who had displayed an early, sustained improvement subsequently relapsed, even after 20 years without symptoms. This suggests that early recovery does not eliminate the possibility of very late relapse. Intermittent, episodic disorder was more common during the early stage of illness and predicted a more favourable outcome, whereas chronic illness predominated in later years. Worse outcome was predicted by early age of onset, particularly in males, experiencing obsessions and compulsions or magical thinking, poor social adjustment and early chronic course.

A recent meta-analytic review of 16 longer term follow-up studies of children and adolescents concluded that although the persistence of OCD in childhood-onset cases may be lower than previously thought, 60% still showed OCD symptoms, whether they met full criteria or not, at follow-up (mean of 5.7 years) (Stewart *et al.*, 2004). While this study may suggest that the long term outcome of these childhood onset cases may differ from the Skoog and Skoog (1999) study, only four of the studies followed-up all participants for more than 5 years and so relatively little information is available on later relapse after initial improvement. The authors also reported that earlier onset and longer duration of the illness at ascertainment of the disorder was associated with persistence of OCD at follow-up. This would suggest that early diagnosis and intervention predicts better outcome so the general public as well as healthcare professionals need to be alert to childhood onset of OCD.

2.1.5 Epidemiology of OCD

According to some studies, OCD is the fourth most common mental disorder after depression, alcohol and substance misuse, and social phobia with a lifetime prevalence in community surveys of about 2–3% (Robins *et al.*, 1984). However the instruments used have been criticised and may have over-diagnosed OCD so that the true prevalence may be somewhat lower (Stein *et al*., 1997b). There is remarkable consistency in the lifetime and annual prevalence of OCD from studies conducted across the world (Weissman *et al*., 1994). The mean age of onset is in late adolescence for men and early twenties for women, although age of onset covers a wide range of ages. However, it may take individuals between 10–15 years or longer to seek professional help. There is often combordity with a range of disorders, especially depression (e.g. Abramowitz, 2004; Abramowitz *et al.*, 2003; Apter *et al.*, 2003), anxiety (e.g. Biederman *et al*., 2004; LaSalle *et al*., 2004; Nestadt *et al*., 2003; Welkowitz *et al*., 2000), alcohol or substance misuse (e.g. Abram *et al*., 2003; Bakken *et al*., 2003; Fals-Stewart & Angarano, 1994), BDD (e.g. Frare *et al*., 2004), or an eating disorder (e.g. Jordan *et al*., 2003).

2.1.6 OCD in children and adolescents

In this guideline, OCD and its management is reviewed across all ages, from the youngest age at which the diagnosis might be reliably made (arguably 4–5 yrs), through the life-span, into old age. There are two main reasons for this. First, adults with OCD often report that they experienced their first symptoms in childhood (Rasmussen & Eisen, 1994). Second, the disorder is remarkably similar in children, adolescents and adults, and responds to the same treatments. Although there are aspects of the disorder in young people which need special consideration, the main symptoms, clinical understanding of OCD, and key strategies for management have a great deal in common at all ages.

OCD was thought to be uncommon in young people, but it frequently begins in childhood or adolescence and reliable population surveys have revealed a prevalence of about 1% (Heyman *et al.*, 2001; Valleni-Basile *et al.*, 1994). It frequently goes undetected (Flament *et al.*, 1988) or is misdiagnosed (Chowdhury *et al.*, 2004). If left untreated it can not only cause marked psychological distress, but can also disrupt social, educational and emotional development, leading to significant disability (Laidlaw *et al.*, 1999; Leonard *et al.*, 1993); there is also an increased risk of morbidity and comorbidity in adulthood (Rasmussen & Eisen, 1990b). It is important not to confuse a normal stage of child development, which is characterised by some repetitive behaviours, with OCD. These behaviours normally occur between age 3 and 6 years, and are self-limiting and rarely time-consuming or distressing. There is a need for long-term follow-up studies, to establish whether early recognition and assertive treatment of OCD in young people results in improved adult adaptation, and decreased rates of OCD in adult life.

While symptoms of OCD are similar in children and adults, the developmental stage affects the way they present, and the way they are described by the sufferer. (It is very common for children and adolescents to involve family members in their rituals.) However, effective treatments in adults also seem to be effective in children (Heyman, 1997; Rapoport & Inoff-Germain, 2000), but treatment of young people needs to involve their family/carers, and also often needs liaison with school or college. Because the disorder has specific and effective treatments, it is desirable for children's OCD to be detected and treated as soon as possible.

2.1.7 The aetiology and maintenance of OCD

OCD is a very heterogeneous disorder in its manifestations and there have been many attempts to categorise sub-groups based usually on the types of obsessions and compulsions. However, behaviours that may appear to be similar may be conducted for different reasons. For example, Rachman (1994) describes several distinct subgroups of people who conduct washing rituals according to their reported motivations to carry out the ritual. Such heterogeneity makes the study of aetiology difficult. A range of factors have been identified as contributing to the expression of OCD, and it is likely that for any given person a number of factors are involved.

2.1.7.1 Biological factors

As with most mental health problems, the cause of OCD is not known. There is increasing research evidence for the involvement of biological factors in this disorder, although, it is highly responsive to psychological interventions as well as to drug treatment (Stein, 2000; Stein, 2002).

There have been a number of family studies of OCD looking at evidence for genetic patterns. A recent meta-analytic review by Hettema and colleagues (2001) reported that a person with OCD is 4 times more likely to have another family member with OCD than a person who does not have the disorder (odds ratio [OR] = 4.0, 95% confidence interval [CI] = 2.2–7.1). Genetic and family studies have shown that OCD appears to be related to tic disorders and Tourette's syndrome. Among children with OCD, many also have tics (Leonard *et al.*, 1992). About 50% of individuals with Tourette's syndrome also have OCD (Pitman *et al.*, 1987). Some authors have discussed how the recurring themes and stereotyped nature of OCD rituals and intrusive thoughts make them seem like 'tics of the mind' (Rapoport & Inoff-Germain, 2000). However, Pauls and colleagues (1995) reported the following findings from their study: 'Some cases are familial and related to tic disorders, some cases are familial and unrelated to tics, and in other cases there appears to be no family history of either obsessive-compulsive disorder or tics'.

Brain imaging studies have consistently demonstrated differing blood flow patterns among people with OCD compared with controls, and that cortical and basal ganglia regions are most strongly implicated (Saxena *et al*., 1998). However, a recent meta-analysis found that differences between people with OCD and healthy controls were found consistently only in the orbital gyrus and the head of the caudate nucleus (Whiteside *et al.*, 2004). Treatment with either medication or CBT is associated with a reversal of the functional neuroimaging findings to the pattern found in control individuals (Schwartz *et al*., 1996). The neurochemical correlates of these differences are not known, but the specificity of effectiveness of selective serotonin reuptake inhibitors (SSRIs) in the treatment of OCD is widely interpreted as suggesting that serotonin is an important neurotransmitter involved in the aetiology and/or maintenance of OCD.

A further recent finding implicating the basal ganglia as a key brain region in OCD is the discovery that in a sub-group of children with OCD the disorder may have been triggered by infections (Dale & Heyman, 2002; Swedo *et al*., 1998). Streptococcal infections trigger an immune response, which in some individuals generates antibodies that cross-react with basal ganglia. This mechanism may explain the subgroup of children in whom OCD develops after a streptococcal infection, and worsens with recurrent infections. However, a recent study found no link between subsequent infections and exacerbation of symptoms (Luo *et al*., 2004).

There is some suggestion that very early onset OCD, probably before puberty, may be a little different to later onset OCD, although of course there are adults with OCD whose illness started when they were young. Some studies suggest that the juvenile-onset form seems more strongly related to a positive family history for OCD and may be more associated with tic disorders (for example, do Rosario-Campos *et al*., 2005; Geller *et al*., 1998; Hanna *et al*., 2005).

2.1.7.2 Adverse life events and difficulties

Several studies report major life events in the period preceding the onset of OCD (Gothelf *et al.*, 2004; Khanna *et al.*, 1988b). This does not mean that the events are in themselves causal, but rather that among people who may be biologically or psychologically predisposed to OCD, a life event can be a triggering factor. The type of event is probably less important than how it is experienced and even positive events can, under some circumstances, be associated with the onset of OCD. In the same way that life events may contribute to onset of OCD, there may be an increase in OCD symptoms as stress levels rise in response to ongoing life. Finally, in some cases, the content of the obsessions may reflect the themes of the life events in some people. Once again, this does not mean that the event causes OCD; rather it means that for some people, the specific content of their obsessions can be influenced by things that happen in their lives (Rheaume *et al.*, 1998).

2.1.7.3 Family factors

Families get caught up in OCD in different ways and to different degrees (see also Chapter 3, Section 3.5). As with other mental disorders, there is evidence of the impact of OCD on the family in a number of ways such as worry, the burden of care, and distress at their limited ability to help the person with OCD (Shafran *et al*., 1995), but there may be greater impact on family life in OCD than in other anxiety disorders (Lochner *et al.*, 2003). In OCD there is evidence that, in some cases, family members get involved in rituals, often at significant cost to themselves in terms of effort, time, and upset (Calvocoressi *et al.*, 1995; Calvocoressi *et al*., 1999). Family members may think that such involvement, which may also include responding to questions and requests for reassurance, will help the person with OCD or reduce their distress, and indeed it may alleviate distress or help the person function better in the short term. However, it is believed that this type of involvement is ultimately unhelpful. This does not mean that the family member is causing OCD, but rather that they are caught up in maintaining the disorder in some ways.

However, family tension and disruption can be a source of stress that can contribute to onset or exacerbation of the disorder (Chambless *et al*., 2001). Although there is speculation that some types of childhood experience may, in association with other factors such as parental over-protectiveness, predispose an individual to OCD, there is no evidence that families play a direct causal role and it is difficult to disentangle changes in parental behaviour in response to a child's OCD from behaviours that may contribute to its development (Barrett *et al*., 2002; Salkovskis *et al*., 1999; Turgeon *et al.*, 2002; Vogel *et al.*, 1997).

2.1.7.4 Socio-cultural factors

Studies from different cultures reveal similar prevalence rates and a surprising consistency in the content and forms of obsessions and compulsions (Horwath & Weissman, 2000). While the exact symptoms of OCD may reflect socio-cultural factors, there is no consistent evidence that any particular factor has any causal role. Thus, socio-cultural factors may shape the expression of OCD (Fontenelle *et al.*, 2004). In this way obsessions and compulsions of a religious nature will reflect the

religious views of the individual and perhaps of the society, but are likely to be based on a particularly rigid or extreme set of beliefs or practice that is not widely shared by other members of the community (Raphael *et al*., 1996; Tek & Ulug, 2001). Preoccupations with contamination may also reflect the society's view of what is clean and what is not, but it is also possible that the individual's concerns are of a highly idiosyncratic nature. Without understanding the specific socio-cultural context of the individual, it is almost impossible to determine what is typical culturally sanctioned behaviour and what is excessive due to the influence of OCD. Therefore, socio-cultural factors should be taken into account when professionals encounter a person with OCD. Given socio-cultural differences when talking about such themes as hygiene, sexuality, blasphemy and so on, great sensitivity must be exercised to enable full disclosure of what may be perceived by the individual and their family as private, embarrassing, or shameful.

2.1.7.5 Psychological factors

Current psychological models of OCD propose that the way in which people interpret their thoughts is an important maintaining factor (Clark, 2004; Rachman, 1997; Salkovskis *et al*., 1995; Wells, 2000). For example, there is evidence that people with OCD do indeed hold stronger beliefs about the importance and the meaning of their thoughts and responsibility for harm to others than people without OCD (see Frost & Steketee, 2002). Likewise, there is some evidence that some people with OCD may hold more perfectionistic beliefs. However, there is as yet little evidence to suggest that these beliefs play a causal role in the aetiology of OCD although there is accumulating evidence suggesting that a range of beliefs including responsibility, the need to control thoughts, and thought-action fusion may all play a maintaining role (Berle & Starcevic, 2005; Clark, 2004; Frost & Steketee, 2002).

2.1.7.6 Personality

OCD shares a number of surface similarities with obsessive-compulsive personality disorder or traits. Some theories propose that OCPD is a precursor of OCD or that there is a specific relationship between such personality features and the disorder. However, there is little evidence to date either for a specific premorbid personality pattern, or for a specific association between OCD and OCPD (see Black & Noyes, 1997; Albert *et al*., 2004). In a broader sense, long-standing beliefs and related behaviours may be found among people with OCD and may be considered by some as key features of an individual's personality. However, as mentioned above, there is little evidence as yet that these beliefs play a causal role in the aetiology of OCD.

2.2 BDD

2.2.1 Symptoms, presentation, and patterns of illness

Body dysmorphic disorder (BDD) is characterised by a preoccupation with an imagined defect in one's appearance or, in the case of a slight physical anomaly, the

person's concern is markedly excessive. The most common preoccupations concern the skin, hair, nose, eyes, eyelids, mouth, lips, jaw, and chin. However, any part of the body may be involved and the preoccupation is frequently focused on several body parts simultaneously (Phillips *et al*., 1993). Complaints typically involve perceived or slight flaws on the face, asymmetrical or disproportionate body features, thinning hair, acne, wrinkles, scars, vascular markings, and pallor, or ruddiness of complexion. Sometimes the complaint is extremely vague or amounts to no more than a general perception of ugliness. BDD is characterised by time consuming behaviours such as mirror-gazing, comparing particular features with those of others, excessive camouflaging tactics to hide the defect, skin-picking, and reassurance-seeking. There is usually avoidance of social situations and intimacy. Alternatively such situations are endured with the use of alcohol, illegal substances or safety-seeking behaviours similar to social phobia.

2.2.2 Diagnosis

According to DSM-IV, to fulfil diagnostic criteria for BDD, the person must also be significantly distressed or handicapped in his or her occupational and social functioning (American Psychiatric Association, 1994). In ICD-10, BDD is not classified as a separate diagnosis and is subsumed under hypochondriacal disorder. (See Appendix 15 for diagnostic criteria for BDD.) The beliefs about one's appearance (e.g. that 'my skin is wrinkled and puffy') may be held with poor insight (when it is regarded as an overvalued idea) or no insight (when it is termed delusional). DSM-IV classifies BDD on the strength of such beliefs according to whether there is an additional diagnosis of a delusional disorder. In ICD-10, if the beliefs are considered delusional, then a patient would receive an alternative diagnosis of 'other persistent delusional disorder' instead of hypochondriacal disorder. There is frequent comorbidity in BDD especially with depression, social phobia and OCD (Neziroglu *et al.*, 1996; Phillips & Diaz, 1997; Veale *et al*., 1996a).

Amputee identity disorder (AID) is often confused with BDD. It is a term used to describe individuals who desire one or more digits or limbs to be amputated (Furth *et al*., 2000; Smith & Fisher, 2003). Some patients may hasten amputation (for example by a chainsaw wound) or carry out self-amputation (for example on a railway line). Although such individuals are preoccupied with becoming disabled, they do not believe (as in BDD) that their limbs are defective or wish to alter their limb cosmetically. They feel that one or more limbs are not part of their 'self' (a form of reverse 'phantom limb') and that amputation will lead to becoming more able-bodied.

2.2.3 Impairment and disability

People with BDD have high rates of unemployment and social isolation, are sometimes housebound and hospitalised, and make frequent suicide attempts (Phillips *et al*., 1993; Phillips *et al.*, 1995; Veale *et al.*, 1996a). In one study people

with BDD reported lower scores on standard scale of quality of life than other psychiatric outpatients (Phillips, 2000).

2.2.4 Course and prognosis

No systematic research has been done on the course and prognosis in BDD. However, milder symptoms in adolescence may resolve over time but those with moderate to severe symptoms tend to follow a chronic course with increasing comorbidity, unsatisfactory attempts at altering their feature(s) or suicide. For some people, the preoccupation with a particular feature will remain unchanged, but for others there may be changes over time.

2.2.5 Epidemiology of BDD

BDD is rarely included in any catchment area surveys of psychiatric morbidity. The data on the prevalence of BDD in the community is generally lacking. However, one study in primary care in Italy found a prevalence of 0.7% (Faravelli *et al.*, 1997) and one in the community of women aged 37–44 found a prevalence of 0.7% (Otto *et al.*, 2001). De Waal and colleagues (2004) however, found no cases of BDD in a survey of somatoform disorders in 1024 patients. However there was no specific screening for BDD and the study excluded anyone under the age of 25 where the majority of cases of BDD may occur. Bohne and colleagues (2002) found the prevalence of BDD in college students (average age 21) from a screening questionnaire to be 5.3% although the specificity of the instrument relative to clinical interview has not been reported. Surveys of people with BDD attending a psychiatric clinic tend to show an equal sex incidence (Neziroglu & Yaryura-Tobias, 1993a; Phillips *et al.*, 1993; Phillips & Diaz, 1997; Veale *et al.*, 1996a). It is also possible that in the community while more women are affected overall, a greater proportion experience milder symptoms. These surveys also suggest that people with BDD are often single or separated.

2.2.6 BDD in children and adolescents

BDD may also present in children and may lead to symptoms of school refusal and suicidal plans. Albertini and Phillips (1999) describe a series of 33 children and adolescents with BDD and reported that bodily preoccupations most often focused on the skin (61%) and hair (55%). They all exhibited similar behaviours to adults with BDD such as camouflaging, comparing their self with others, and mirror-gazing. Social impairment was almost universal and the majority reported impairment in academic functioning because of BDD. The group displayed significant disturbances with 39% having had psychiatric hospitalisation and 21% reporting suicidal ideation or attempted suicide.

2.2.7 Aetiology and maintenance of BDD

Hypothesised risk factors include genetic factors, temperament and childhood adversity such as teasing or bullying, increased aesthetic sensitivity, a history of dermatological or other physical stigmata (Veale, 2004). However, there is virtually no research that has systematically studied risk factors in BDD with other psychiatric disorders. A cognitive behavioural model has been described for the maintenance of symptoms of BDD (Veale, 2004).

2.3 TREATMENT AND MANAGEMENT IN THE NHS

2.3.1 Adults – OCD

2.3.1.1 Pharmacological treatment

The outlook for OCD was dramatically improved by the discovery of effective drug treatments from the early 1980s (Marks *et al*., 1980; Montgomery, 1980). Intensive pharmacological investigation has consistently demonstrated that OCD responds selectively to drugs that act as potent inhibitors of the synaptic reuptake of serotonin (serotonin reuptake inhibitors, SRIs) (Montgomery *et al.*, 2001; Zohar & Judge, 1996). Currently, this includes the tricyclic drug clomipramine, which stands apart from other tricyclics because of its more potent serotonergic actions, and the more highly selective serotonin reuptake inhibitors (SSRIs) citalopram, fluoxetine, fluvoxamine, paroxetine and sertraline (in alphabetical order).

The finding that these drugs are effective even when depression is rigorously excluded in the reference population implies a specific anti-obsessional effect. Drugs lacking these properties such as the standard tricyclic antidepressants and monoamine-oxidase inhibitors (MAOIs) have been found to be ineffective in randomised controlled trials (RCTs) (Ananth *et al*., 1981; Insel *et al.*, 1983; Jenike *et al.*, 1997; Volavka *et al.*, 1985). Studies looking at benzodiazepines and lithium have also not produced positive findings. Antipsychotics have not been found to be effective on their own, but may have a role as agents of augmentation in cases where the response to an SRI is poor or incomplete. The selectivity of the pharmacological response for serotonergic agents distinguishes OCD from depression and other anxiety disorders where a wider range of treatments appear effective, and implicates serotonin in the treatment effect.

2.3.1.2 Psychological treatment

CBT is the most widely used psychological treatment for OCD in adults (Roth & Fonagy, 2004). The main CBT interventions that have been used in the treatment of OCD are exposure and response prevention (ERP) (for example, Foa & Kozak, 1996; Marks, 1997), different variants of cognitive therapy (Clark, 2004; Freeston *et al*., 1996; Frost & Steketee, 1999; Krochmalik *et al*., 2001; Rachman, 1998; 2002; 2004; Salkovskis, 1999; van Oppen & Arntz, 1994; Wells, 2000), and a combination of ERP and cognitive therapy (see Kobak *et al.*, 1998; Roth & Fonagy, 2004). ERP and

cognitive therapy have different theoretical underpinnings but may be used together in a coherent package. However, it is uncertain whether either treatment is superior to the other, or indeed whether combining these interventions confers any added benefit (Abramowitz, 1997).

Whatever CBT intervention is used, the key principles remain the same. This involves first establishing a good therapeutic alliance based on a working partnership between patient and therapist, and using a credible and clear rationale, a treatment focus on the here and now, explicit agreed and operationally defined treatment strategies and collaboratively therapeutic strategies between client and therapist (see Rachman, 2003; Salkovskis *et al*., 1999; Steketee, 1993).

Since the first studies showing the efficacy of exposure and response prevention procedures in the 1970s, many of the studies that have followed have examined the relative efficacy of variations in the mode of delivery of CBT rather than comparison with control conditions. Variants include the length and intensity of treatment, the use of additional components such as cognitive interventions, the value of involving the family in treatment, and employing different formats such as individual or group treatments. In some cases groups are used in the interest of cost effectiveness, in other cases it is believed that particular features of the group therapy experience can enhance the treatment itself.

Variation in therapist time for treating OCD is considerable, ranging from fewer than 10 hours to over 50 hours over 10–20 sessions. Previous consensus guidelines have suggested between 13–20 weekly sessions (March *et al*., 1997). However, there is little evidence to demonstrate the optimal number of sessions required and further research in this area is necessary. Any improvement in detection is likely to increase demand for treatment. Further, provision of CBT is variable and some mental health services in the UK that offer CBT have long waiting lists, precluding easy access to effective interventions (Lovell & Richards, 2000).

Most research has been conducted on face-to-face therapies and more studies have considered individual rather than group based treatments. However, a variety of modes of treatment delivery have been developed, including a computer programme which can be accessed by a touch-telephone system (Baer & Greist, 1997) that can be accessed 24 hours a day, telephone treatment guided by a therapist (Lovell *et al*., 2000; Taylor *et al*., 2003), and bibliotherapy (using self-help books) (Fritzler *et al.*, 1997) which may be offered with brief support sessions (Lovell *et al*., 2005).

Despite the good response rate of CBT with OCD it is important to remember that a significant proportion either refuse treatment or leave treatment early, often because of the demands of exposure and response prevention. Fear of the consequences of not performing rituals and/or unwillingness or inability to tolerate high levels of anxiety may account for these failures to engage in and complete treatment. In addition, even among those who do complete treatment, a significant proportion of people do not respond. Although some work has been completed on predictors of treatment outcome (Keijsers *et al*., 1994; Lax *et al.*, 1992; Mataix-Cols *et al*., 2002a), the results remain mixed and further work in this area is necessary. Further work is also necessary to determine the optimal interventions for those people who do not initially respond to CBT.

2.3.2 Children and young people – OCD

The same types of psychological and drug treatments are generally thought to be effective in both children and adults. However, the numbers of scientific, controlled studies are fewer in children and young people than in adults. Although it may often be reasonable to look at studies conducted in adults and assume that similar results will be found in children, there are important developmental differences that need considering both for psychological and drug treatments.

2.3.2.1 Pharmacological treatment

Medicines are used in childhood OCD but it is important to bear in mind that the long-term effects of drugs on the immature brain of the child are little understood. Moreover, when prescribing drugs for children with OCD it is essential to use a drug and dose appropriate to the child's age and size, generally starting with a very low dose and increasing gradually. The child or young person with OCD and their parents or carers together with the prescriber need to decide whether the potential benefits outweigh any possible risks. Drugs most commonly used for children and young people with OCD are the same as those used for adults, including the SSRIs, and clomipramine is also used for young people.

Current published evidence suggests that SSRIs are effective in treating children and young people with OCD. However, with depression SSRIs can cause significant adverse reactions, including increased suicidal thoughts and self-harm, although they may be safer when combined with psychological treatments. Regulatory authorities have identified that the use of SSRIs to treat depression in children and young people may be associated with the appearance of suicidal behaviour, self-harm or hostility, particularly at the beginning of treatment. There is no clear evidence of an increased risk of self-harm and suicidal thoughts in young adults of 18 years or over. But individuals mature at different rates and young adults are at a higher background risk of suicidal behaviour than older adults, so as a precautionary measure young adults treated with SSRIs should be closely monitored. The Committee on Safety of Medicine's (CSM) Expert Working Group on SSRIs, at a meeting in February 2005, advised that it could not be ruled out that the risk of suicidal behaviour, hostility and other adverse reactions seen in the paediatric depression trials applies to use in children or adolescents in all indications. Consequently, the recommendations about the use of SSRIs for people with OCD or BDD have taken account of the position of regulatory authorities.

With all treatments, including drug treatments, parents and young people need to be fully appraised of the benefits and risks as we currently understand them. As with many drugs used in paediatrics, a specialist may recommend a medication for the treatment of OCD in young people which is unlicensed, but for which there is clinical trial evidence of safety and efficacy. Explanations should be given to the family about why an unlicensed preparation is suggested.

2.3.2.2 Psychological interventions

The main psychological treatment for OCD is cognitive behavioural therapy, and the principles of treatment are much the same for children and adults, although account needs to be taken of the developmental changes in cognitive and linguistic abilities and functions that occur with increasing age. CBT is a well-validated psychotherapeutic technique in a number of different settings (National Collaborating Centre for Mental Health, 2003; National Collaborating Centre for Mental Health, 2004; National Collaborating Centre for Mental Health, 2005a and b) developed from experimental psychology principles initially with behavioural strategies. More recent developments have included interventions that target the young person's beliefs and the way they interpret situations. Published protocols for use in children, such as that of March and Mulle (1998) are generally based on 'exposure' (facing up to the feared stimulus) and 'response prevention' (resisting the urge to carry out a ritual in these circumstances). Cognitive therapy protocols that tackle underlying beliefs about connections between thoughts and behaviours, are also being evaluated, although these principles form a component of most CBT approaches. It is important to note that although there is an increasing evidence base for CBT, its efficacy has largely been demonstrated in older children. In particular, cognitive strategies have not been systematically tested among younger children with OCD. Recent evidence among normally developing children suggest that many 5- to 11-year-old children can engage in tasks required for cognitive therapy, but this is yet to be demonstrated among children with OCD (Doherr *et al*., 2005).

The therapist working with a child or young person should be aware of the impact of OCD on families, and how families can help with treatment. Although some children can be quite secretive and conceal their rituals, many involve an adult in their rituals. Although this is a little researched area, most practitioners with experience in managing children and young people with OCD find that it is essential to involve families closely in the treatment. Working with children and young people will often require effective liaison with the child's school or other agencies involved in the child's life. If there are comorbid difficulties, current practice would suggest that other forms of psychological work may be used in the overall management of OCD (e.g. family therapy, play therapy, and so on).

2.3.3 Special considerations in the treatment and management of BDD in the NHS

People with BDD generally feel misunderstood and are secretive about their symptoms because they think they will be viewed as vain or narcissistic. They may indeed be stigmatised by healthcare professionals who view only true disfigurement as worthy of their attention or confuse BDD with body dissatisfaction (Carter, 2001). Therefore when they do present to healthcare professionals, they are more likely to complain of depression or social anxiety as

these are less stigmatising, or they may present with drug or alcohol related problems. Insight is often poor and they are more likely to disengage from mental health services and not be followed up vigorously. Sometimes, people with BDD will gain access to mental health services with a diagnosis of hypochondriacal disorder, somatoform disorder or even a psychotic disorder, each likely to lead to inappropriate treatment with antipsychotic agents or ineffective talking therapies.

2.3.4 The relationship of the evidence base for adults to that of children and adolescents with BDD

As there is virtually no evidence base for the treatment of BDD among children and adolescents, general principles of treatment for OCD in these age groups may be relevant as long as the specific issues in point 2.3.3 are kept in mind.

2.4 DETECTION, ASSESSMENT AND DIAGNOSIS

2.4.1 Detection

In primary care, 40% of patients presenting will have significant mental health problems, whilst in 20–25% this will be the main reason for attendance (Department of Health, 2001). However the detection rate in general practice, even for relatively 'high profile' mental health problems such as depression, may be no more than 50% (Freeling *et al.*, 1985; Gilbody *et al*., 2003). Although there are not any data for OCD among adults in the UK, a recent study on other anxiety disorders in primary care in the US concluded that less than one third of patients had received either psychotherapy or pharmacotherapy that met a criterion for quality care (Stein *et al*., 2004). It is likely that a similar situation exists for OCD. A report from Holland found that two-thirds of patients with OCD referred to a university medical centre were receiving no medication, inappropriate medication or ineffective doses of appropriate medication (Denys *et al.*, 2002b). Likewise, half of a consecutive series of young people with OCD referred to specialist centre in the UK had not received evidence-based CBT or pharmacotherapy before referral (Chowdhury *et al.*, 2004).

The reasons for this may be that both doctors and patients may perceive psychological problems as having a lower priority than 'physical' difficulties; patients may down-play psychological distress and doctors working within limited time constraints may focus on physical symptoms. For patients with OCD, who may see their symptoms as stigmatising and potentially shameful, these difficulties may be compounded, limiting the consultation to more 'comfortable' physical symptoms such as skin problems resulting from repeated hand washing, but reducing the chances of disclosure of specific psychological symptoms (Eddy & Walbroehl, 1998). Referrals may then be made to physical health settings, such as dermatology, where undiagnosed OCD may be found (Fineberg *et al.*,

2003). In other cases, lack of insight will prevent the person with OCD reporting difficulties to their doctor, although other family members may suspect or recognise some of the difficulties. Even when a family member reports the problems to the doctor, the person with OCD may still be unwilling to discuss the symptoms because they do not believe that they have problems, do not consider that they wish to receive or require help, or because of the difficulties associated with disclosure.

Despite the increased awareness of mental health in primary care, doctors may not be trained, or may not have the time, to systematically ask the right questions that could lead to rapid detection of OCD. Other healthcare professionals may equally lack the training and experience to consistently detect OCD. Given the difficulties that people may have in disclosure, unless the right questions are asked, detection may be difficult.

When the consultation process is working well, a mutually supportive and trusting relationship between patient and doctor or other healthcare professional will provide a safe context within which the disclosure of difficult information is more likely to occur (Di Blasi *et al*., 2001). This in turn can lead to more effective collaboration and communication, increasing the chances of early and accurate diagnosis and of timely and effective treatment (Stewart, 1995). Ideally this process will be facilitated by continuity of care where unusual patterns of consultations or requests for treatment such as, for example, repeated requests for emollients, may alert clinicians to the underlying diagnosis.

Even when people are referred to secondary care, there is no guarantee that the referral is for OCD rather than for anxiety, depression or other diagnoses. Depending on the point of entry, a person with OCD may not receive a rapid and accurate diagnosis. For example, in a large community study of OCD among adolescents, four of the 20 young people with OCD were receiving counselling, and in three of the cases the professional was unaware of the OCD (although they had sought help for depression and anxiety, Valleni-Basile *et al*., 1994).

2.4.2 Disclosure

It should not be underestimated how difficult it can be for people with OCD to first disclose their symptoms to family and friends and to the medical profession (Newth & Rachman, 2001; see also Chapter 3, Section 3.2). The range of difficulties recognised by service users can include any of the following (although this list is by no means exhaustive):

- A person who has read about or heard of OCD may be able to say to their GP 'I think I may have OCD' but may fear the response or doubt whether the GP will know anything about the condition.
- Many people with OCD start to have symptoms as children when they may lack the right language to express what they are experiencing.
- Some people with OCD may talk about their symptoms in very general terms to their GP and are often diagnosed with depression or other forms of anxiety.

- Many people feel guilt, shame or embarrassment at revealing the nature of their obsessions even to supportive family members and friends, especially if they have fears of killing or harming people. They may fear that they may carry out their obsessional thoughts.

2.4.3 Stigma and potential consequences of diagnosis

Many patients express relief at being diagnosed as suffering from OCD as this offers an explanation for their symptoms, eliminates 'self-diagnosis' of other seemingly more serious complaints and opens up the possibility of treatment and help (see also Chapter 3, Section 3.2). The patient's family may also be more accepting of a recognised medical condition, and feel they may participate in the patient's therapy. However, the converse is also true due to the stigma that can be attached to mental health issues giving rise to embarrassment, shame, guilt or depression (Stengler-Wenzke *et al.*, 2004). People may fear stigmatisation at work, and/or concerns about disclosing the condition to new employers, insurers, friends and family.

Fears about disclosure may contribute to people being reluctant to admit to being ill. People have very real concerns which may result in their not accepting treatment, and, in some cases, not seeking help at all. Once a person knows that they have OCD, and that this has been recorded in their medical records, they are likely to be concerned about being labelled 'mentally ill' and the consequences of this. The fears recognised by patients might include any of the following (although this list is by no means exhaustive):

- Concern that GPs and medical specialists will assume any illness is then likely to be anxiety related rather than physical.
- Fear of telling their employer with the possibility of losing employment or the impact on promotion.
- Fear of applying for new jobs and whether to disclose this in any medical questionnaire relating to work.
- Questions about whether the information should be disclosed on any other official form, such as insurance, which asks for health information.
- Uncertainty about whether this will affect eligibility to adopt or foster children, to serve on juries or to emigrate.
- Concern about the likely course of the illness and whether they will be on medication for the rest of their life.
- The likely impact in the long term on relationships and the ability to care for children.
- For younger people, the impact on their education.
- Concern about OCD being mistaken for a condition that is associated with criminal behaviour such as that of a psychopath or paedophile.
- Concern about general stigma from friends or family who do not understand the nature of anxiety-related conditions.

2.4.4 Assessment

2.4.4.1 Adult

OCD is a relatively common illness that can occur across the lifespan (Fineberg & Roberts, 2001), but is poorly recognised and undertreated (Hollander & Wong, 1998). Although there may be evidence that the time lag between onset of symptoms and correct diagnosis is shortening (Mallery, 1996), individuals with the disorder have been reported to wait on average up to 17 years before correct treatment is initiated (Hollander & Wong, 1998). Given the substantial socioeconomic costs associated with untreated OCD, better recognition and treatment of the disorder has been recognised as a major public health objective (Hollander & Wong, 1998).

Whereas OCD is often underdiagnosed for reasons described above, it is occasionally wrongly diagnosed when people present with repetitive or compulsive behaviours that may resemble compulsions in OCD. These include Tourette's syndrome (Muller *et al.*, 1997), autism and autistic spectrum disorders (McDougle *et al*., 1995c; McDougle *et al*., 2000b), Prader-Willi syndrome (Clarke *et al*., 2002), dementia (Mendez *et al*., 1997; Rosso *et al*., 2001; Stein *et al.*, 1997a), and schizophrenia (Ongur & Goff, in press). In these cases, although comorbidity between these conditions and OCD remains a possibility, a misdiagnosis of OCD may result in unsuitable or ineffective treatment.

Increased awareness is the key to better recognition and treatment of OCD. Although it can take years before finding a healthcare professional in whom a person with OCD will spontaneously confide, direct enquiry by a sympathetic health practitioner is usually successful (Fineberg *et al.*, 2003). Practitioners in areas known to attract large numbers of patients should be encouraged to look for symptoms. Active enquiry, involving questions from the Zohar-Fineberg Obsessive Compulsive Screen (see below), should be incorporated into every mental state examination.

There has been a lack of suitable OCD screening instruments for detecting OCD in the non-specialist setting. In order to be useful, the instrument needs to be brief and user-friendly as well as able to accurately identify individuals with the disorder without missing too many (sensitive) and also able to screen out individuals with out the disorder without being over-inclusive (specific). Positive responses to the screening measure would then be followed up by a more detailed clinical evaluation.

Three instruments have been developed for use in the non-specialist as well as the psychiatric setting. The computerised Symptom Driven Diagnostic System for Primary Care (Weissman *et al.*, 1998) screens for a range of affective and anxiety disorder as well as OCD. It consists of a computerised questionnaire and a diagnostic interview administered by a nurse, or other suitably trained mental health worker, to generate a one-page summary of diagnostic information. Unfortunately, comparison of the results of this test with those from a reliable structure clinical interview (Structured Clinical Interview for DSM-IV [SCID-IV] gave poor overall agreement ($\kappa = 0.28$) and the test cannot therefore be recommended (Taylor *et al.*, 2002).

The second instrument is a computerised telephone-administered version of the Primary Care Evaluation of Mental Disorders (Kobak *et al.*, 1997). It also assesses a range of affective and anxiety disorders and takes about 10 minutes to complete. Individuals answer questions that follow an algorithm directed by branching logic. Compared with the SCID-IV The Primary Care Evaluation of Mental Disorders (PRIME-MD) provided reliability in diagnosing OCD ($\kappa = 0.64$). The screening instrument is disadvantaged by requiring a specialised computer programme and the system is not widely available in the UK.

The third instrument, the Zohar-Fineberg Obsessive Compulsive Screen (ZF-OCS), was devised by J. Zohar for the International Council on OCD in 1995. It consists of five brief questions designed to be administered by a doctor or a nurse and takes less than 1 minute to administer (Fineberg & Roberts, 2001). These comprise: Do you wash or clean a lot? Do you check things a lot? Is there any thought keep bothering you that you'd like to get rid of but can't? Do your daily activities take a long time to finish? Are you concerned about orderliness or symmetry?. It was validated against the Mini International Neuropsychiatric Interview in a relatively small population of UK dermatology outpatients (MINI) (Lecrubier *et al.*, 1997) where it was found to have good patient acceptability as well as satisfactory sensitivity (94.4%) and specificity (85.1%) (Fineberg *et al.*, 2003). Its psychometric properties are undergoing further evaluation in a range of psychiatric and non-psychiatric settings. In view of its brevity and utility, it can be considered as a possible screening tool for further evaluation.

A variety of self-report questionnaires have been developed for OCD that may be useful for detection (Taylor, 1995; Taylor *et al.*, 2002). Self-report versions of the Yale-Brown Obsessive Compulsive Scale (Y-BOCS), both paper and computer-administered, have been developed (Rosenfeld *et al.*, 1992; Steketee *et al.*, 1996) and have equivalent properties to the clinician-administered Y-BOCS. It provides both a symptom checklist and a severity score. A short form of the Obsessive Compulsive Inventory has also been developed with good psychometric properties (Foa *et al.*, 2002b), the longer form of which was developed to address some of the issues with other self-report measures. The longer form may be too long for routine detection, but provides a more comprehensive list for general assessment of symptoms (Foa *et al.*, 1998). Another brief measure is the Clark-Beck Obsessive-Compulsive Inventory (Clark & Beck, 2002). These questionnaires may help identify OCD and also provide a measure of severity and an overview of symptoms.

Although there are not extensive data on patients with OCD, a study on patients in the US Food and Drugs Administration (FDA) drug trials for the anxiety disorders indicated that 'suicide risk among patients with anxiety disorders is higher than in the general population by a factor of ten or more' (Khan *et al.*, 2002); there were no significant differences between OCD and the other four anxiety disorders studied. Further, with the high rate of depression in OCD and the risks that may be associated, it is important to investigate depressive symptoms carefully. Measures of depression, such as the Hamilton Depression Rating Scale (a clinician-rated scale) and the Beck Depression Inventory (a self-report measure), may prove useful. The Clinical Outcomes in Routine Evaluation may also be a useful for

assessment of a broad range of issues, but should normally be supplemented by an OCD-specific measure such as those mentioned above. Given the potential range of impact that OCD can have on a person's life, measures of quality of life may also be important, especially when assessing for change in the person's overall function.

For further information about screening for depression, please see the adult depression guideline (NCCMH, 2005a) or the depression in children guideline as appropriate (NCCMH, 2005b).

2.4.5 OCD in specific populations

OCD is frequently complicated by depression which affects over two thirds of people with OCD during their lifetime (Rasmussen & Eisen, 1990a). It is often the depression that encourages the individual to seek help from their primary care physician, and at this point the OCD can be missed if the doctor fails to enquire about the condition. However, surveys have shown that only a small proportion of individuals with OCD actually present to their GP for treatment. For example, a recent survey by de Waal and colleagues (2004) found a point prevalence of 0.5% in a large cohort in general practice. Therefore, the pick-up rate for OCD without formal screening in primary care is not likely to be high.

Higher numbers of individuals with OCD have been identified by a small number of surveys in a range of hospital (secondary care) settings such as 20% of UK dermatology outpatients (Fineberg *et al.*, 2003) and 32% of people presenting to rheumatologists and dermatologists with systemic lupus erythematosis (Slattery *et al.*, 2004). While by no means conclusive, these surveys suggest that screening may be more profitably employed in specific secondary care settings such as those described.

2.4.6 Children and young people

In young people as in adults, there can be a long delay before receiving a diagnosis and starting treatment. Even in young people, OCD can cause significant impairment for several years before help is obtained. The reasons for this are not fully understood, but there may be some commonalities between children and adults. For example, children with OCD can often feel embarrassed about their symptoms, and try to keep them a secret even from their closest family. Interviews should be undertaken with parents or carers as well as with the young person, making sure that the child has an opportunity to be seen alone. Given that symptoms of OCD may be linked to particular activities or contexts, the impact on school, home and leisure activities need to be investigated.

Pre-school children often have some rituals or repetitive behaviours as part of normal development. These can usually be easily distinguished from OCD as they do not cause distress, take up much time, or stop the child doing other things. In the early

stages of OCD, however, a parent may mistake increased ritualising for ordinary childhood behaviour. Even when it becomes apparent that a young person has a problem with intrusive thoughts and ritualistic behaviour, they and their family and friends may not realise that this is OCD, and may be reluctant to seek help. Even when help is sought, healthcare professionals may not be aware of the characteristic symptoms of OCD and the diagnosis may not be made, or an incorrect diagnosis made. Sometimes when a diagnosis has been made, young people may be told to 'wait to see if it goes away', or be referred for an inappropriate treatment.

Once the diagnosis of OCD is suspected, it can be helpful to use standardised rating scales to help the young person reveal specific information regarding symptoms, rate severity, and monitor treatment. These might include disorder specific scales such as the Children's Yale-Brown Obsessive Compulsive Scale (CY-BOCS; Scahill *et al.*, 1997) or the Child Obsessive Compulsive Inventory (Shafran *et al.*, 2003). A general emotional and behavioural symptom checklist, such as the Strength and Difficulties Questionnaire (SDQ; Goodman, 2001) will reduce the possibility that comorbid conditions are missed.

Once the diagnosis of OCD has been made in a young person, it is important that they receive the correct treatment, and that therapists are not distracted by focusing on additional problems, such as anxiety disorders, school or separation problems, and mood difficulties; these are often secondary to the OCD, and resolve with treatment for the primary disorder. If they do not, it is important to target comorbidities with an appropriate intervention. Attention needs to be given to educating the child and their family about OCD, how anxiety operates to maintain symptoms, and the range of treatment choices.

2.4.7 BDD

2.4.7.1 Detection

BDD is relatively easy to detect but is often overlooked. There is a low level of awareness about BDD amongst healthcare professionals. Few practitioners ask simple questions for the diagnosis. However individuals with BDD are often too ashamed to reveal the true nature of their problem without direct questioning (for example, Grant *et al.*, 2002). Furthermore, when individuals with BDD do seek help they are more likely to consult a dermatologist or cosmetic surgeon. When BDD patients finally seek help from a family doctor or mental healthcare professional, they are often too ashamed to reveal their main symptoms and present with symptoms of depression, social phobia, or OCD.

2.4.7.2 Assessment and diagnosis

First and colleagues (1997) suggest the following questions to screen for patients with BDD: 'Some people worry a lot about their appearance. Do you worry a lot about the way you look and wish you could think about it less?' In those who answer positively, then one might ask (a) 'What specific concerns do you have about your appearance? Do you think about them a lot and is it hard to stop thinking about them? On a typical day, how many hours a day is it on your mind? (More than an hour a day is considered

excessive) How much does it bother you? What effect does it have on your life? Does it make it hard to do your work or be with friends?'

Dufresne and colleagues (2001) validated a brief self-report questionnaire to screen for BDD in cosmetic dermatology settings using a reliable clinician-administered diagnostic interview for BDD for comparison. The self-report questionnaire had a sensitivity of 100% and a specificity of 93%. The interrater reliability of the defect rating scale was .88. These results suggest that this questionnaire is an effective screening tool for BDD in a dermatology setting.

2.4.7.3 BDD among those with other psychiatric problems

There are a number of groups of people with psychiatric disorders who are known to be at higher risk of BDD. Grant and colleagues (2002) screened 122 consecutive inpatients and 16 (13.1%) were diagnosed as having BDD by a structured interview but none of the participants had been diagnosed as having BDD by their treating physician. Similarly, Zimmermann and Mattia (1998) found that none of 500 referrals to a psychiatric outpatient clinic in the USA had been diagnosed as having BDD through routine unstructured clinical interview, but 3.2% (n = 16) of a second group of 500 were diagnosed as having BDD when a structured diagnostic interview was introduced (SCID-IV). Of 350 outpatients with major depression who entered an antidepressant treatment study, 23 (6.6%) were diagnosed with current BDD by structured diagnostic interview (Nierenberg *et al.*, 2002). The rate was higher in a typical depression (14.4% compared with 5.1%).

In two surveys of patients attending a clinic for anxiety disorders, the rates of BDD were highest amongst those diagnosed with social phobia (13% in Brawman-Mintzer *et al.*, 1995; 12% in Wilhelm *et al.*, 1997). An additional comorbid diagnosis of social phobia in BDD can only be made when there is a broader fear of negative evaluation by others and not just of one's appearance. The diagnosis of OCD is given only when the obsessions and compulsions are not restricted to concerns about appearance. Sometimes the symptoms overlap; for example a patient may believe that their skin is both ugly and contaminated. A similar situation exists in patients preoccupied with order and symmetry in which one symptom might be focused on one's hair and another on a body part being symmetrical and feeling 'equal'. In a study of 165 patients seeking treatment for an anxiety disorder, 7.7% of OCD patients had a current diagnosis of BDD (Wilhelm *et al.*, 1997).

A common diagnostic dilemma for BDD is with eating disorders. People with BDD and an eating disorder share a distorted body image and many other symptoms such as a low self-esteem. DSM-IV states that a diagnosis of BDD should not be used if symptoms are best accounted for by a diagnosis of an eating disorder. If therefore the preoccupation is predominantly focused on being 'too fat' or overweight, it does not meet criteria for BDD. There is a grey area between individuals with disordered eating who do not fulfil the criteria for an eating disorder. True comorbidity of BDD and an eating disorder occurs when a patient is preoccupied by imagined defects in their appearance that are unrelated to weight and shape. Grant and colleagues (2002) reported that 16 (39%) of 41 patients with anorexia nervosa were diagnosed with comorbid BDD unrelated to weight concerns. The most

common preoccupations in the study by Grant and colleagues were (in descending order) with the nose, skin, hair, chin, lips and eyes. The patients with anorexia nervosa and BDD had significantly lower overall functioning and higher levels of delusionality than the anorexic patients without BDD, suggesting that the former had a more severe form of illness.

2.4.8 BDD and risk of suicide

There is a significant risk of suicide associated with BDD. According to Phillips and Diaz (1997), 22% of their BDD patients had attempted suicide at some time and Veale and colleagues (1996b) reported that in a series of 50 BDD patients in the UK, 24% had attempted suicide. Among individuals with BDD and depression, a sense of hopelessness is more likely to occur when a person with BDD believes that they are trapped and has exhausted all their abilities to camouflage or alter their appearance. An individual who has just had cosmetic surgery and realises that all their hopes have been dashed may be at particular risk.

2.4.9 Cosmetic and dermatological procedures and BDD

Several studies have established that people with BDD may also be found in medical settings that deal with cosmetic or dermatological procedures. There have been six surveys of BDD in a cosmetic surgery setting. BDD is relatively common in such settings with a prevalence of between 3–18% (Aouizerate *et al.*, 2003; Castle *et al.*, 2004; Ishigooka *et al.*, 1998; Sarwer *et al.*, 1998; Vindigni *et al.*, 2002). There have been two surveys in a dermatological setting. Phillips and colleagues (2000) found 12% of 367 of dermatological patients in the USA and Uzun and colleagues (2003) found 8.8% of 159 dermatological outpatients in Turkey had BDD. The prevalence of BDD depends on the procedures offered at each clinic, the sex ratio, and the diagnostic threshold and instrument used to measure BDD. For example, some procedures like rhinoplasty may attract more people with BDD. There may be difficulties in the diagnosis of BDD in a cosmetic or dermatological setting when someone has a minor physical anomaly and when the concern becomes 'markedly excessive'. Also aesthetic cosmetic procedures are often concerned with enhancing a 'normal' appearance and the diagnosis of BDD centres on the degree of preoccupation, the distress and psychosocial handicap.

There are three retrospective studies of cosmetic surgery in BDD patients attending a psychiatric clinic. Phillips and Diaz (1997) reported in a sample of 188 BDD patients that 131 patients sought and 109 received surgical, dermatological or other medical treatments. However 83% reported an exacerbation of or no change in BDD symptoms. The most common outcome following surgery was no change in overall BDD severity (58%) and no change in the concern with the treated body part (48.3%). More patients worsened in overall BDD severity (24.3%) than improved (17.4%). However, in terms of the treated body part, more patients reported a

decrease (34.5%) than an increase (17.2%) in concern. No data is provided on satisfaction for the procedures.

Veale (2000) reported on 25 BDD patients in the UK after cosmetic surgery, who had had a total of 46 procedures and 76% were dissatisfied post-operatively. Three patients claimed that they were not preoccupied by their appearance prior to the surgery and that their symptoms of BDD developed only after surgery, which they believed, had been done badly. Some operations, such as rhinoplasty appear to be associated with higher degrees of dissatisfaction. Most of the patients in the study had multiple concerns about their appearance and reported that after 50% of the procedures the preoccupation transferred to another area of their body. When patients were dissatisfied with their operation, they often felt guilty or angry with themselves or the surgeon at having made their appearance worse, thus further fuelling their depression and a failure to achieve their ideal.

These studies have limitations. The data are retrospective and there is a selection bias of patients in favour of treatment failures. Mental health professionals may not see BDD patients who are satisfied with their cosmetic surgery and overcome their symptoms of BDD. People with milder BDD may also be satisfied with the outcome of surgery and may not immediately return to psychiatric treatment. Further, there is no control group of non-psychiatric or psychiatric patients who have undergone cosmetic surgery but do not have BDD. However, the majority of individuals who do not have BDD appear satisfied with cosmetic surgery and their self-esteem and other psychological measures tend to improve (Harris & Carr, 2001; Klassen *et al.*, 1996). Cosmetic surgeons usually try to determine whether the patient's expectations of change are realistic. In general, patients will have good psychological outcome if they can clearly describe the problem that concerns them and their desired outcome. The surgeon will then discuss with the patient the likely result when surgically altering their appearance and outline the consequences and risks. Among people with BDD, expectations for major psychosocial changes (for example getting a better relationship) are often unrealistic.

2.5 STEPPED CARE

Given the complexity of need and healthcare organisation, the way that psychological treatments, particularly CBT, are delivered has become an increasing focus of interest, such as in the US model of stepped care. Stepped care argues that the least intrusive intervention (for example, education or self-help) should be used first, only moving to more intense therapy when less intensive treatment has proved to be insufficiently effective (Haaga, 2000).

This guideline suggests that such a model could prove useful if applied to UK settings to encourage access to intensive treatment when severity or risk indicate less intensive treatment would be inappropriate. The Stepped Care Model (Figure 1) provides a model for the most effective but least intrusive treatments appropriate to a person's needs. It assumes monitoring of the course of a person's difficulties and referral to the appropriate level of care. Each step introduces additional interventions; the higher steps normally assume interventions in the previous step have been offered

and/or attempted, but there are situations where an individual may be referred to any appropriate level.

It is suggested that the awareness, recognition and treatment of OCD and BDD proceed across six phases, depending upon need and the characteristics of a person's OCD/BDD. The model also provides a framework to organise services to support the public, patients, carers, and healthcare professionals in identifying and accessing the most effective interventions.

At all stages of assessment and treatment, families and carers should be involved as appropriate. This is particularly important in the treatment of children and young people with OCD where it may also be helpful to involve others in their network, for example teachers, school health advisers, educational psychologists, and educational social workers.

2.6 CLINICAL PRACTICE RECOMMENDATIONS

2.6.1 Understanding

2.6.1.1 People with OCD or BDD are often ashamed and embarrassed by their condition and may find it very difficult to discuss their symptoms with healthcare professionals, friends, family or carers. Healthcare professionals should help patients, and their families or carers where appropriate, to understand the involuntary nature of the symptoms by providing accurate information in an appropriate format on current understanding of the disorders from psychological and/or biological perspectives. **[GPP]**

2.6.1.2 When assessing people with OCD or BDD, healthcare professionals should sensitively explore the hidden distress and disability commonly associated with the disorders, providing explanation and information wherever necessary. In particular, people with OCD who are distressed by their obsessive thoughts should be informed that such thoughts are occasionally experienced by almost everybody, and when frequent and distressing are a typical feature of OCD. **[GPP]**

2.6.2 Continuity of care

2.6.2.1 OCD and BDD are frequently recurring or chronic conditions that often affect some of the most intimate aspects of a person's life. Healthcare professionals should therefore ensure continuity of care and minimise the need for multiple assessments by different healthcare professionals. **[GPP]**

2.6.2.2 Because OCD and BDD may occur across a person's lifespan, particular care should be given to the provision of appropriate care at all ages and a seamless transition between services aimed at specific ages, such as the transition from services for young people to services for adults. **[GPP]**

Figure 1: The stepped care model

Who is responsible for care?	What is the focus?	What do they do?
Step 6 Inpatient care or intensive treatment programmes CAMHS Tier 4	OCD or BDD with risk to life, severe self-neglect or severe distress or disability	Reassess, discuss options, care coordination, SSRI or clomipramine, CBT (including ERP), or combination of SSRI or clomipramine and CBT (including ERP), augmentation strategies, consider admission or special living arrangements
Step 5 Multidisciplinary care with expertise in OCD/BDD CAMHS Tier 3/4	OCD or BDD with significant comorbidity, or more severely impaired functioning and/or treatment resistance, partial response or relapse	Reassess, discuss options. **For adults:** SSRI or clomipramine, CBT (including ERP), or combination of SSRI or clomipramine and CBT (including ERP); consider care coordination, augmentation strategies, admission, social care. **For children and young people:** CBT (including ERP), then consider combined treatments of CBT (including ERP) with SSRI, alternative SSRI or clomipramine. For young people consider referral to specialist services outside CAMHS if appropriate
Step 4 Multidisciplinary care in primary or secondary care CAMHS Tier 2/3	OCD or BDD with comorbidity or poor response to initial treatment	Assess and review, discuss options. **For adults:** CBT (including ERP), SSRI, alternative SSRI or clomipramine, combined treatments. **For children and young people:** CBT (including ERP), then consider combined treatments of CBT (including ERP) with SSRI, alternative SSRI or clomipramine
Step 3 GP, primary care team, primary care mental health worker, family support team CAMHS Tier 1/2	Management and initial treatment of OCD or BDD	Assess and review, discuss options. **For adults according to impairment:** Brief individual CBT (including ERP) with self-help materials (for OCD), individual or group CBT (including ERP), SSRI, or consider combined treatments; consider involving the family/carers in ERP. **For children and young people:** Guided self-help (for OCD), CBT (including ERP), involve family or carers and consider involving school
Step 2 GP, practice nurses, school health advisers, health visitors, general health settings (including hospitals) CAMHS Tier 1	Recognition and assessment	Detect, educate, discuss treatment options, signpost voluntary support organisations, provide support to individuals/families/work/schools, or refer to any of the appropriate levels
Step 1 Individuals, public organisations, NHS	Awareness and recognition	Provide, seek and share information about OCD or BDD and its impact on individualsand families/carers

2.6.2.3 Careful consideration should be given to the effective integration and coordination of care of people with OCD and BDD across both primary and secondary care. There should be clear, written agreement among individual healthcare professionals about the responsibility for monitoring and treating people with OCD and BDD. A written copy of this agreement should be given to the patient. This should be in collaboration with the individual and, where appropriate:

- the Care Programme Approach (CPA) should be used
- the patient's family and carers should be involved
- healthcare professionals should liaise with other professionals involved in providing care and support to the patient. **[GPP]**

2.6.2.4 OCD and BDD can have a fluctuating or episodic course, or relapse may occur after successful treatment. Therefore, people who have been successfully treated and discharged should be seen as soon as possible if re-referred with further occurrences of OCD or BDD, rather than placed on a routine waiting list. For those in whom there has been no response to treatment, care coordination (or other suitable processes) should be used at the end of any specific treatment programme to identify any need for continuing support and appropriate services to address it. **[GPP]**

2.6.3 Religion and culture

2.6.3.1 Obsessive-compulsive symptoms may sometimes involve a person's religion, such as religious obsessions and scrupulosity, or cultural practices. When the boundary between religious or cultural practice and obsessive-compulsive symptoms is unclear, healthcare professionals should, with the patient's consent, consider seeking the advice and support of an appropriate religious or community leader to support the therapeutic process. **[GPP]**

2.6.4 Awareness, recognition, and training

Although the more common forms of OCD are likely to be recognised when people report symptoms, less common forms of OCD and many cases of BDD may remain unrecognised, sometimes for many years. Relatively few mental health professionals or GPs have expertise in the recognition, assessment, diagnosis and treatment of the less common forms of OCD and BDD. Less common forms of OCD include an obsession that one will steal something or 'cheat' someone; an obsession that as a car driver, he or she may knock over a cyclist; a need to remember certain things or not make a mistake; obsessions about sacrilege and blasphemy or sinfulness; thoughts involving personally unacceptable sexual behaviour; compulsive hoarding of bodily waste or animals. Less common forms of BDD include a preoccupation with one's genitalia or muscle shape and size or BDD by proxy.

2.6.4.1 Each PCT, mental healthcare trust, and children's trust that provides mental health services should have access to a specialist OCD/BDD multidisciplinary healthcare team offering age-appropriate care. This team would perform the following functions: increase the skills of mental health professionals in the assessment and evidence-based treatment of people with OCD or BDD, provide high-quality advice, understand family and developmental needs, and, when appropriate, conduct expert assessment and specialist cognitive-behavioural and pharmacological treatment. **[GPP]**

2.6.4.2 Specialist mental health professionals in OCD or BDD should collaborate with local and national voluntary organisations to increase awareness and understanding of the disorders and improve access to high-quality information about them. Such information should also be made available to primary and secondary healthcare professionals, and to professionals from other public services who may come into contact with people of any age with OCD or BDD. **[GPP]**

2.6.4.3 Specialist OCD/BDD teams should collaborate with people with OCD or BDD and their families or carers to provide training for all mental health professionals, cosmetic surgeons and dermatology professionals. **[GPP]**

2.6.5 Recognition and assessment

Given that people with OCD and BDD may have difficulty in disclosing their symptoms, people with disorders known to be commonly associated with OCD or BDD should be specifically assessed for these conditions and the possibility of comorbidity, especially those with depression and anxiety. Screening questions provide a rapid way of identifying those who may have the disorder. As most screening questions result in a proportion of false positives when some features may overlap with other disorders (that is, sensitivity is usually better than specificity), it is important to complete the differential diagnosis. People who are identified as having OCD or BDD, especially those with comorbid depression, should be assessed for the risk of self-harm and suicide and other comorbid conditions that may affect decisions about treatment and care.

OCD

2.6.5.1 For people known to be at higher risk of OCD (such as individuals with symptoms of depression, anxiety, alcohol or substance misuse, BDD or an eating disorder), or for people attending dermatology clinics, healthcare professionals should routinely consider and explore the possibility of comorbid OCD by asking direct questions about possible symptoms such as the following:

- Do you wash or clean a lot?
- Do you check things a lot?

- Is there any thought that keeps bothering you that you would like to get rid of but can not?
- Do your daily activities take a long time to finish?
- Are you concerned about putting things in a special order or are you very upset by mess?
- Do these problems trouble you? **[C]**

2.6.5.2 In people who have been diagnosed with OCD, healthcare professionals should assess the risk of self-harm and suicide, especially if they have also been diagnosed with depression. Part of the risk assessment should include the impact of their compulsive behaviours on themselves or others. Other comorbid conditions and psychosocial factors that may contribute to risk should also be considered. **[GPP]**

2.6.5.3 If healthcare professionals are uncertain about the risks associated with intrusive sexual, aggressive or death-related thoughts reported by a person with OCD, they should consult mental health professionals with specific expertise in the assessment and management of OCD. These themes are common in people with OCD at any age, and are often misinterpreted as indicating risk. **[GPP]**

BDD

2.6.5.4 For people known to be at higher risk of BDD (such individuals with symptoms of depression, social phobia, alcohol or substance misuse, OCD or an eating disorder), or for people with mild disfigurements or blemishes who are seeking a cosmetic or dermatological procedure, healthcare professionals should routinely consider and explore the possibility of BDD. **[GPP]**

2.6.5.5 In the assessment of people at higher risk of BDD, the following five questions should be asked to help identify individuals with BDD:

- Do you worry a lot about the way you look and wish you could think about it less?
- What specific concerns do you have about your appearance?
- On a typical day, how many hours a day is your appearance on your mind? (More than 1 hour a day is considered excessive)
- What effect does it have on your life?
- Does it make it hard to do your work or be with friends? **[GPP]**

2.6.5.6 People with suspected or diagnosed BDD seeking cosmetic surgery or dermatological treatment should be assessed by a mental health professional with specific expertise in the management of BDD. **[GPP]**

2.6.5.7 In people who have been diagnosed with BDD, healthcare professionals should assess the risk of self-harm and suicide, especially if they have also been diagnosed with depression. Other comorbid conditions and psychosocial factors that may contribute to risk should also be considered. **[GPP]**

2.6.5.8 All children and young people who have been diagnosed with BDD should be assessed for suicidal ideation and a full risk assessment should be carried out before treatment is undertaken. If risks are identified, all professionals involved in primary and secondary care should be informed and appropriate risk management strategies put into place. **[GPP]**

2.6.5.9 Specialist mental health professionals in BDD should work in partnership with cosmetic surgeons and dermatologists to ensure that an agreed screening system is in place to accurately identify people with BDD and that agreed referral criteria have been established. They should help provide training opportunities for cosmetic surgeons and dermatologists to aid in the recognition of BDD. **[GPP]**

3. THE EXPERIENCE OF PEOPLE WITH OCD AND BDD AND THEIR FAMILIES AND CARERS

This chapter consists of personal testimonies that illustrate the experience of a number of people with OCD and one with BDD and also of those involved as family members and/or carers of people with OCD. The testimonies were chosen to demonstrate something of the range of experience of sufferers and carers and should not be taken as representative. These narratives express the experience of OCD and BDD over the lifetime, the effect on family and carers, and the process of obtaining appropriate treatment and the response to such treatments. The testimonies draw from experiences of OCD and BDD over the last 40 years.

3.1 PERSONAL TESTIMONIES FROM PEOPLE WITH OCD AND BDD

3.1.1 Daniel

I've just arrived home from work. Tired and tense, I'm convinced my hands are contaminated with some hazardous substance and my primary concern now is to ensure that I don't spread that contamination to anything that I, or others, may subsequently touch. I will wash my hands, but first I will need to put a hand in my pocket to get my door keys, contaminating these, the pocket's other contents, and everything else I touch on my way to the sink. It will be late evening before I will have completed the whole decontamination ritual. Tomorrow I will inadvertently touch another contaminant, and a similarly exhausting process will have to be performed.

That's how it was 40 plus years ago when, in my early twenties, my OCD became firmly established. Fear of contamination was the main manifestation, primarily, I suspect, because my work brought me close to genuinely hazardous materials: taking precautions was the expected norm. However, 'checking' had also become a major preoccupation, not least feeling compelled to ensure that I hadn't been responsible for causing harm to others. Frequently, for example, I would retrace a car journey (often over a very long distance) to make sure I hadn't accidentally hit a pedestrian.

Despite the fact that these compulsions were distressing in themselves and were wasting inordinate amounts of time, I did not seek help. I believed my behaviour to be simply that of a responsible citizen. I struggled on as best I could until, at the age of 27, the distress, exacerbated by an upheaval in my domestic arrangements, increased to such a level that I was admitted to hospital. At that time (1966) OCD was

not widely recognised and I was diagnosed with 'anxiety neurosis'. Hospital provided some respite from both the unsatisfactory domestic/accommodation situation and my OCD triggers, but effective treatment for the disorder was not forthcoming (at one stage a lobotomy was discussed, but fortunately not pursued) and, after 12 weeks, I was discharged.

For the next 15 years or so I ploughed on, always managing to work and support myself, but not having much of a life as the condition, which I had come to regard as unalterable, ebbed and flowed. I did seek treatment from time to time and received prescriptions for medication such as nitrazepam and, later, diazepam. The manifestations of the disorder now included the fear of an extended range of contaminants (my cleaning compulsions demanded the removal of every last molecule of 'dirt', so washing or bathing could take hours). Also, I felt compelled to carry out endless checks to make sure things were safe (doors locked, gas off, and so on) and that no mistakes had been made. The latter could be taken to extraordinary extremes: I would imagine that a decision I had taken as part of my work as an engineer could lead to another engineer somewhere relying on my erroneous decision in his calculations, and so on, and so on, until an aeroplane fell out of the sky. And it would all be my fault, so I felt compelled to reassure myself that all was well. Hours could be spent on such exhausting exercises.

Up until this point I had lived alone and so had been able to indulge my compulsions without anyone really noticing. In the early 1980s things changed: I married, became a father, and got some help. This involved more medication and regular sessions with a psychiatrist who diagnosed/confirmed OCD. I was a private patient and, over a period spanning many years, chose to discontinue and re-start the sessions as I felt necessary. The sessions were useful, although in retrospect I can see their value was limited since, instead of finding the courage to confront my fears, I used the meetings for the comfort and relief that an understanding ear can provide. During this period I was still managing to work, but the OCD was certainly restricting my life. I wouldn't travel on certain bus routes because I believed one of the vehicles used on that route was contaminated. I would cross the road to avoid road sweepers and their contaminated brushes. I wouldn't join in outings because of the risk of getting dirt on my limited range of clean clothing.

But now I was a father, and particularly when we had our second child, matching the children's needs for a normal childhood with my own desires for cleanliness and order was not easy. I coped, after a fashion, but the constant anxiety made my experience of their childhood years pretty joyless.

And then about 5 years ago things took a turn for the worse. Following a heart attack and a modification to my diet, I became extremely depressed and the OCD got much worse. I became quite desperate. I vividly recall one occasion at that time when, weeping, I tried to explain the distress I felt over a compulsion to wash my hands that I had resisted. 'Can you imagine what it feels like to believe you've poisoned your own children?' I asked.

Once again I sought help. SSRIs were tried, but soon stopped because I couldn't tolerate the side effects. Then I was given two courses of CBT. Each of these consisted of 1-hour sessions every 2 weeks for a total of around 10 weeks. As I recall,

the sessions were biased towards the cognitive aspects of CBT, with very little in the way of monitored behavioural tasks. Overall, the sessions were quite helpful in that I was able to discuss my concerns, however, it was a further 9 months of weekly behavioural therapy sessions provided by a support group that really helped me to practise ERP. This technique has brought about significant improvements in my condition.

Now, as I approach the age of 65, I strive to maintain the improvement by means of self-administered ERP. The mood stabilisation effects of medication (carbamazepine) newly prescribed to treat my recently diagnosed epileptic absences also seem to be making a positive contribution. So, although by no means '100%', I am a great deal better. But it has been a long 40 years.

3.1.2 Ruth

I experienced what I now realise were the first symptoms of OCD when I was about 12 years old. I started to feel compelled to cancel out any distressing thought I had, such as failing an exam or the possibility of a family member dying, by repeating whatever I was doing when I had the thought and replacing the 'bad thought' with a 'good thought'. This included getting undressed and dressed again, retracing words and sentences I'd written, re-reading pages of a book or walking back over a stretch of ground. I would carry out these actions repeatedly until I had managed to neutralise the thought. This could take a long time and would also cause embarrassing situations when I would make excuses to go back to places or pretend there was something I had to do.

Nothing happened to trigger the start of this way of thinking. Nothing had changed at home or at school and I didn't have any major physical illnesses. I do remember that the thoughts and rituals began slowly but increased over time. At first I would have two or three thoughts a day that needed to be neutralised, but over a period of months this became dozens of unwelcome thoughts each day.

These symptoms caused me great distress, which I felt unable to express to people although I often used to cry at home and at school. The compelling need to repeat certain actions also wasted time and sometimes stopped me going out with friends or to new places for the fear of leaving a 'bad thought' in a place that I couldn't easily return to. If I was unable to undo these thoughts I would feel anxious and uncomfortable and found it difficult to concentrate on other things. On occasions I would make excuses to return to someone's house or a shop several days or even weeks later in order to try and neutralise a thought I had had on the previous visit. I could sometimes remember leaving 'bad' thoughts behind for many months. Throughout my teenage years I kept my thoughts and bizarre actions to myself, concealing them from family and friends and hiding away to carry out my compulsions.

Despite this, I managed to work hard at school and go to university at the age of 19. By this time the distressing thoughts were becoming increasingly sinister and even more painful to me – I started to be scared that I might cause an accident, or I would think about murder, or about hurting or molesting children, although I knew

I had no desire to carry out any of these things. I was terrified of the type of thoughts I was having and hated myself for having them but the harder I tried to shake them off the stronger they seemed to become. I didn't feel I could confide in my parents or even friends or healthcare workers because I thought the nature of these thoughts was so shocking that I would be considered dangerous and perhaps locked up. At this point I became seriously depressed and suicidal, losing a lot of weight and being constantly on the verge of tears. Over the years I saw a number of GPs who diagnosed depression without exploring the nature of my depression in any way. Antidepressants and some sessions with a counsellor offered some temporary relief. However the counselling sessions didn't touch on the real problems I was experiencing; the counsellor didn't ask me the right questions and I didn't feel I could open up about the type of thoughts going through my head.

Over the next few years the disturbing thoughts continued. There would be bad periods when the thoughts seemed to be with me much of the time and then sometimes for no obvious reason they would fade a little, although they never went away completely. Oddly enough when I was very focused on something like preparing for exams, a time which people often think of as stressful, the thoughts would actually ease off a little.

However, in my early 20s the thoughts were joined by a compelling need to check to ensure that I hadn't caused an accident and constant hand washing to avoid passing on contamination. This included regular episodes of going back over journeys on public transport or walking back down a street to check that I hadn't accidentally brushed past someone and hurt or killed them. My fear of contamination was about the possibility that I might have been in touch with a poisonous substance. Germs didn't really bother me and I even remember reading an article at that time about a woman having treatment for her obsessive hand washing due to a fear of exposure to germs and I didn't associate it with what I was experiencing.

Eventually OCD was diagnosed by a GP when I was 26 and I was referred to a psychiatrist. Had I been questioned in any way about the nature of my thoughts and anxiety, I feel that OCD would have been diagnosed much earlier. I was desperate to confide in someone but no one ever asked the right questions. A long course of tricyclic antidepressants (clomipramine) and some behavioural therapy helped get my OCD back to a manageable level and relieved the depression.

There was only a very small amount of relief in having an official diagnosis for what I was going through. By this time I honestly thought it was too late to get my life back to normal. In addition, referral to a psychiatrist meant having a medical label that I would have to carry with me for the rest of my life. I was also frightened that I might have to physically confront my fears in some way. The fact that the medication very quickly dampened some of the worst thoughts was a relief. Clomipramine caused unpleasant side effects including severe sweating, constipation and a dry mouth but they were a small price to pay and I believe that having it prescribed at that time saved me from possible suicide.

However, I continued to have relapses over the next 10 years and it was only when I received a course of CBT along with a different type of antidepressant (an SSRI) that I felt I made significant steps to dealing with my OCD and learning skills

that I could draw on during future relapses. Unlike clomipramine, the SSRIs didn't cause any major side effects and the CBT, although very difficult to do, provided me with a new angle for looking at my obsessions and the skills to stop carrying out the compulsions.

I also tried an OCD support group and I initially enjoyed meeting other people with OCD. After many years of hiding my bizarre rituals and painful thoughts, it seemed quite incredible to meet so many people who had such similar experiences and stories to tell. At the same time I found a tendency amongst some people to want to talk only about how terrible OCD was and after a while I found I didn't gain anything from these groups.

At the age of 41 I still have OCD but I also cope well on a day-to-day basis and have done for a number of years. I hold down a management position at work, am in a long-term relationship and enjoy travelling overseas and socialising with friends – things that didn't seemed possible earlier in my life. I continue to take a maintenance dose of an SSRI (currently fluoxetine). I am aware that if I don't keep on top of my compulsions by not giving in to them then they have a tendency to creep back. I find that having the bad thought and not trying to neutralise it in any way ultimately helps to make the thoughts seem less important, although I have to live with the initial anxiety. The more I try to neutralise the thoughts the more they start to take over. Sometimes I worry that taking antidepressants on such a long-term basis may be damaging for my health but equally I feel that they do work and help me keep on top of my OCD. I would be equally concerned if I was forced to stop taking them.

In looking back, I feel strongly that had my illness been recognised and correctly treated much earlier then I would have been able to achieve far more in life and would certainly not have wasted so much time carrying out senseless compulsions or avoiding situations because of my obsessive thoughts. I came close to suicide on a number of occasions; found relationships difficult because people would pick up on my sometimes odd behaviour and missed out on many opportunities, including overseas travel, flat sharing with friends, promotion at work and marriage and children, because my obsessions prevented me taking these opportunities. OCD came close to completely ruining my life. I don't know where I would be now if I hadn't received CBT from knowledgeable therapists and been prescribed appropriate antidepressants. I do believe that you need to take a certain element of responsibility for your own recovery by being prepared to try appropriate medication and having a go at CBT if it is offered but it is very difficult to do this without sympathetic professional help and guidance.

3.1.3 Estelle

I am 25 years old and OCD first took over my life almost 10 years ago (although I did not know what it was until about 18 months after it all began). I suppose that when I look back I can see 'signs' of OCD back into my childhood but the full-blown illness was triggered suddenly after the suicide of a boy in my year group at school. I believe

it was my attempt to make everything 'certain' and 'safe' again after this horrifying event blew apart my sheltered middle-class life. I began to feel I was being taken over by some force in my brain that I could not control, until eventually my days and nights became ruled by its orders:

'If you don't wash your hands twenty times, you will have to kill yourself … Oh no, I don't think you got the soap underneath every fingernail on that nineteenth wash, so it's all negated, you must start the washes again … you must stop yourself committing suicide … no, while you had that thought you didn't wash between those fingers, start again! … but it doesn't make any sense, other people don't do this! But just what if, what IF you don't do it, then you might not be able to stop yourself killing yourself!'

This may sound like a psychotic experience but in fact I could very well see that what I was doing made no rational sense. When eventually I had satisfied the requirements of the washing ritual I would attempt to get myself out of the bathroom. Walking over the threshold of any doorway was another tortuous experience:

'What did you think about when you stepped over that line? Was it anything related to suicide or death or the boy? You thought about Corn Flakes! But CF were the initials of the boy! Go back and do it again, that connects you to him! Make your mind totally blank!'

In the end I could connect anything I thought of to the awful events, just words sounding slightly similar to 'bad words' was enough, and I had to go back and back and back until my mother came and dragged me away, or I made her say it would be OK, or I fell in a heap from sheer exhaustion. The rituals and thoughts were incessant everywhere I was: getting up from a chair, swallowing food, or writing a word in my schoolbook. If I thought bad thoughts as I did the action, the action had to be repeated. Certain clothes, objects, food, TV programmes, were 'bad' and had to be avoided. I would cry at the end of the day just because I wanted the rituals to leave me alone but they would not, and the consequences seemed so important and so dire that I had to keep doing them to protect myself and others from them.

These experiences, and the fact that it felt as though I no longer had control of my mind, caused me to believe that I had gone insane. I think my parents perhaps thought the same, because they did not want me to see any doctors for fear I would be locked up. They tried to reason with me, to 'rationalise away' the OCD; this did not work and could not work, and so they became more and more involved in my rituals as I asked for constant reassurance that 'bad things' weren't going to happen.

Although I had been to see my GP several times with somatic complaints a bit like glandular fever (unsurprisingly I was exhausted the whole time), she had not probed further to find psychological causes, and I had been too frightened to mention the OCD to her unprompted, because it felt so much like madness. Describing the symptoms to healthcare professionals can be one of the worst things for a person with OCD; the awareness that their thoughts and behaviours are abnormal and bizarre can make them they feel embarrassed and also fearful of being laughed at or worse being told they are mad.

It was a friend's mother, another GP, who mercifully rescued me from this hell, or at least told me that there was a way out of it, when I broke down and told her about it.

She arranged for me to see a psychiatrist and explained to me about OCD; she even gave me a video that explained it. (I was also diagnosed with agoraphobia and social anxiety.) I cannot express the relief of being told that I was not 'mad' in any classical sense, and that something could be done to make the uncontrollable thoughts and rituals less uncontrollable. I was firstly given a tricyclic antidepressant (clomipramine) and although I came off that after 6 years because of side effects such as dyskinesia [involuntary movements], I have since tried the full range of SSRIs and SNRIs, and none has been as effective as clomipramine. It is certainly not a cure all, but for me it was more than I could have hoped for, and allowed me to suppress the OCD enough to get myself through high school final exams and into university with good grades. The psychiatrist tried family therapy with us, which I think is probably essential for any child and young person with severe OCD. One of the most potent perpetuating factors in OCD is family members taking part in rituals or offering reassurance; this may feel like helping but it only reinforces the importance of carrying on this bizarre behaviour in the sufferer's mind. But I was also given fairly ineffective strategies aimed at 'stopping' intrusive thoughts – such as snapping an elastic band on my wrist and saying 'stop' every time one popped up. Unfortunately this just added more focus (and more pain) to the thoughts and became a ritual in itself.

Over the years since then the symptoms have waxed and waned. Each time the OCD hits me again with full force, it seems to be in a different form, and each fixation can last several years. After protecting myself from suicide, the next focus for thoughts and rituals was catching AIDS, which involved much washing and disinfecting and avoiding public places where I believed surfaces harboured the dreaded virus. It made no difference to be told 'you can't catch AIDS from surfaces' – the well-worn 'yes, but I might be the exception' or 'what IF . . . ' or 'better to be safe than sorry' revolved around my mind. My current problem is the fear that I will suddenly lose sight of any sense of morals and enter a state of madness where I will start harming other people in horrible ways. The same types of thoughts still pop into my mind as they did 10 years ago, but these days they have less power over me because of treatment I have received.

Although CBT is always put forward as the treatment of choice for OCD, the initial severity of my illness meant that such a treatment would have been ineffective before medication allowed me to function again to a certain extent. The other therapy that helped me understand better why I might suffer from this disorder was psychodynamic therapy, which I received weekly for 2 years whilst at university. Because OCD is so often accompanied by a range of other anxiety and mood problems, not to mention self-esteem issues, this therapy enabled me for the first time to find some value in myself as a person, and to mitigate the hatred I had long felt for myself partly because of the OCD. I had had problems forming friendships and relationships, but as a result of my therapist's acceptance of me I realised for the first time that I did not have to be a victim of my illnesses and defined by them, and could be liked by other people for the person I am.

Over the last year I have finally had the opportunity to have some CBT in a specialist group for OCD sufferers, but had I not first had the psychodynamic therapy

I don't believe I would have thought enough of myself or have had the confidence to do it. Nor would I have understood that my OCD is my way of trying to have control over uncertainties in life. CBT is very hard work, and requires a lot of thought outside the sessions, but it does equip the sufferer with tools to allow them to realise the difference between thoughts and actions; these two are often fused in the OCD sufferer's mind.

I don't think I will ever be free of all the symptoms of OCD and I may always need medication to make it behave itself, but I think I have been lucky to receive good treatment and have now almost completed a masters degree. It is perfectly possible to lead a satisfying life with OCD but the main problem is to get over the hurdle of explaining the thoughts and rituals to a doctor because of the fear of madness. Voluntary work with sufferers has made me realise too that there is widespread ignorance of the signs and symptoms, not to mention the horrifying nature, of this disorder even amongst GPs. In my own experience, some members of the psychological and psychiatric professions tend to steer clear of patients with OCD as they believe it is a 'difficult' illness to treat.

3.1.4 Jane

Only now do I realise that I actually had all the signs of BDD at an early age. I was always sensitive and self-conscious and felt that I was different from the other girls. My confidence increased slightly when I reached my mid teens and I was able to camouflage my appearance with make up and straighten and control my curly hair. With the arrival of a few boyfriends and my marriage at 18 I felt a little 'more normal' but by this time the obsessive behaviours had also set in. I would spend hours grooming my hair and putting on make up and I would not allow myself or anyone else to see me in my 'natural' state (no make-up and hair left to dry naturally) for fear that they would discover the real ugly and disgusting me. From this time on my whole life revolved around trying to keep up this façade. Having carried out the camouflage rituals I would avoid going out in the rain, swim, anything that could affect my image. I visited countless hairdressers, bought endless amounts of hair and make up products and resulted in cutting my own hair, trying desperately to find a miracle that would make me look acceptable but this never happened. This was to continue until I finally found out that I had BDD at the age of 45 and received the right treatment.

At 21 my marriage broke down and I became severely depressed and the illness took over my life. I was so repulsed and disgusted by my appearance that I thought that no one would ever want me again. My parents took me to the GP who treated me for depression and prescribed Valium. The anxiety and depression became so unbearable that I took an overdose and was then referred to a psychiatrist. The next 7 years of my life were spent in and out of hospital trying countless different types of medication but the symptoms persisted and I took more overdoses. I can't remember now what it was that I was prescribed but I do remember that at times I felt completely 'spaced out' and unable to function and once experienced hallucinations. I also had the feeling that I was being experimented on with endless different drugs

that didn't have any effect. I explained to the doctors that I felt distressed because of the way that I looked but this was dismissed. This increased my feelings of embarrassment and shame and I felt that I was also a 'sinner' for worrying so much about the way that I looked. The illness continued and my life revolved around the level of satisfaction I could achieve with the never-ending cycle of camouflage.

During this time I was unable to work and during one spell in hospital I had a relationship with another patient. This seemed to give me some reassurance that I was not completely undesirable and I went to live with him. This turned out to be a disaster as he had severe mental health problems. I got pregnant and ended up homeless with a 3-month-old child.

When I reached 28 I met my second husband and the following years were much better with the distraction of my home, children, and career. However, social activities were still affected because it was difficult to be around others who were attractive and therefore 'adequate', unlike myself.

At 45 things took a serious turn for the worse with the failure of my second marriage and the BDD became severe. I was constantly checking my reflection in the mirror for up to 4 hours at a time. I felt repulsive and hideous and didn't feel that I looked human or deserved to live. I would constantly compare myself with others and look at old photographs, always focusing on what I considered to be my worse features, for several hours at a time. I thought about my ugly appearance every moment and again became suicidal. I couldn't talk to family and friends about my feelings because I was frightened that they would think that I was vain or mad. I couldn't understand why anyone could bear to look at me and not recoil in horror. The rituals around my make-up became worse and I would only remove it to immediately replace it. I styled my hair several times during the day and at times after washing and styling it would have to start all over again. I spent countless amounts of money on cosmetics, hair products, magazines, and salon treatments but the obsessive thoughts got worse. I felt that I could not get on and do anything if I could not get an acceptable image in the mirror and the more that I tried the more hideous I seemed to look and the more distressed I became.

My emotional state also caused physical problems such as irritable bowel syndrome, chest pains, weight loss and muscle pain. I was being treated at this time by a psychiatrist who tried different medication, including antipsychotics (which didn't help), and referred me to two different psychologists. Most of the sessions were spent going over my past, which was not helpful, and I was discharged because I was making no progress. No one seemed able to help me. At one stage it was suggested that I put post-it notes in my diary with affirmations like 'I am beautiful' but this had no effect because I didn't believe it – it just made me very angry. The last psychologist that I saw told me, in so many words, that I was a hopeless case because I had received so much help (from psychiatrists, psychologists, community psychiatric nurses) and was not getting anywhere – this added to my feelings of shame and hopelessness.

I was convinced that unless I could change my appearance I would have to take my own life. Over a 3-year period I spent £20,000 on numerous cosmetic surgery procedures but it made me more ill, this time believing that it was my own fault rather

than nature. I had two face lifts, a brow lift, chin tuck, laser treatment, upper and lower eye surgery, human tissue inserted under lip/nasal lines and many injectable treatments to plump out laugh lines. I put myself through hell but it seemed my only option, as I had nowhere else to turn. The feeling as I was going under the anaesthetic was wonderful as I felt that there was the chance that this operation might just work and I would be happy again. In reality this never happened. As the surgery healed the anxiety increased as I still saw the ugliness, made worse now by the guilt and shame of what I had done to myself. Even so, I still felt compelled to have more surgery because 'this time it might work and I have no other chance of life'.

Then one day as I was recovering from my last bout of surgery in 2000 I saw a programme on the TV about a girl with BDD. It seemed incredible that other people felt like me and that there was a name for my condition. I then found a BDD specialist who immediately took me into hospital for treatment. To be able to talk to someone who understood my condition after suffering from BDD for 25 years was overwhelming and the hope that this gave me was such a relief. After 6 months of SSRI medication and CBT I began to feel that I was improving and after a further 12 months of treatment I returned to work and felt back in control of my life. I did a lot of exposure therapy, which was difficult, but in time paid off. I also did a lot of cognitive work and after a while found that I was beginning to think in a more positive way. I eventually found that I was starting to believe that I am a worthwhile person and although I still hate the way that I look I realise that I can lead a normal life. I use the CBT skills on a daily basis to help prevent a relapse and I still take the SSRI.

I believe that if BDD had been recognised earlier and treatment had been available my life would have been happier and more fulfilling. It also had a terrible affect on my family. When I was in my twenties my father often said that I should 'pull myself together' and Mum sometimes remarked that I was being vain. When I was diagnosed it was difficult for them to admit that I had a mental health problem, I think that they may have felt partly to blame, that they had done something wrong as I was growing up, but they were good and loving parents. So much time and money has been wasted on this illness and up until 4 years ago it has ruled my life. I am grateful to have now received appropriate treatment. It is an ongoing battle but I am finally in control of the BDD.

3.2 THE PERSPECTIVE OF PEOPLE WITH OCD AND BDD

As the testimonies demonstrate, OCD often develops slowly with no obvious 'cause', although it may also be triggered or exacerbated by a particular event. It may start with a few intrusive thoughts that lead to rituals and compulsions (e.g. needing to check that a door is locked or over-zealous hand washing). In BDD the obsessive thought is related to the person's physical appearance and might mean that the person feels compelled to keep checking his or her appearance in a mirror. Over time these actions or compulsions become more entrenched and part of everyday life and the thoughts that accompany them become more intrusive and obsessive. The range of

rituals may also increase; some may fade away completely, but be replaced by new or more complex ones.

The testimonies reveal that the actions or rituals become absolutely necessary to a person with OCD who believes that they serve various purposes. They can protect the person from possible sources of danger and contamination and make him or her feel 'safe'; but they also shield others from the person with OCD, who may consider that his or her actions may be dangerous or contaminating. The actions and rituals can also, at first, impose some kind of order on the world. If the rituals, whether actions or deliberate thought processes, do not take place, the person with OCD will experience overwhelming anxiety and fear. For someone with OCD his or her compulsions may become the only means of neutralising anxiety and preventing harm, although at the same time they can alienate him or her from other people as the obsessions and compulsions become all consuming. For people with BDD, compulsions, such as keeping the face 'masked' with make up or covering up or disguising a perceived physical flaw, are necessary to be able to confront other people.

As shown in the testimonies, OCD, if left untreated, may become more severe. The symptoms may take on a more 'sinister' and troubling aspect (such as the thought of killing someone), leading the person to feel depressed and suicidal, and can take up a huge amount of time. While the actions are about retaining control, they also become controlling in themselves: people with OCD may feel that they are being 'taken over' by their obsessive thoughts. A person with these conditions may know that the thoughts are not entirely 'rational' but will nevertheless be compelled by them. OCD might also be exacerbated by significant life events, such as starting a new school or university, moving house, marriage breakdown, or health problems. However other people with OCD may find that periods of stress at school or at work may temporarily take their minds off the obsessive thoughts.

The testimonies also vividly express how OCD can significantly affect a person's daily life and his or her relationships with other people. Although some people can disguise their OCD this comes at a great cost: they might decline social invitations that interfere with their compulsions or completely withdraw from the people closest to them. Other people with OCD might 'involve' other people in their compulsions (see the testimonies from carers below): carers and other family members might feel that they are 'controlled' by a person with OCD to the extent that they subscribe to his or her rituals. This puts considerable pressure on personal relationships. Some people with OCD can find that their lives are severely restricted and may confine themselves to a small part of the house or a single room that is deemed to be 'safe' or 'clean'. With BDD, a person may feel unable to be around other people who are perceived to be more attractive.

A person with OCD might be reluctant to seek professional help due to feelings of embarrassment and shame and be worried about talking to someone about what might appear to be 'dangerous' thoughts. It is not uncommon for people with OCD to visit their GPs and not talk specifically about their obsessions and compulsions, but more generally about being anxious or depressed. If a GP diagnoses depression in such instances it is important that the reasons for the depression are explored

otherwise diagnosis of OCD may be missed. Diagnosis of OCD may come as a relief for people who have not realised they suffer from a recognised and treatable illness although a diagnosis and a medical 'label' can in themselves bring problems.

Once the condition has been diagnosed, it is essential that the person with OCD works with a healthcare professional who has appropriate training in the treatment of OCD, and with whom the person can build up a relationship of trust and understanding and find the courage to confront their fears and anxiety. Particular types of antidepressants (SRIs) can reduce some of the anxiety and depression associated with OCD and may reduce the obsessions and compulsions to some extent. This can help the person to feel calm enough to benefit from psychological therapy. It is important that specialists listen and sympathise but are also clear, direct and positive about the treatment process. A sense of humour is usually helpful too. The OCD sufferer is often asked to do the things they most fear, therefore support, encouragement and understanding from the therapist, and family if appropriate, are vital. (See Section 3.5 for family and carer support.)

Some people with OCD report finding group therapy sessions particularly helpful because they can learn from seeing how other people react to treatment. In the same context, two therapists can be useful because one maybe able to reinforce what the other is saying. OCD sufferers are often keen to argue about why their fears may be justified and it is harder to argue against two therapists both offering the same advice.

Once psychological treatment is underway and the sufferer has made some steps towards recovery, the sufferer has to learn to take some responsibility for their treatment, such as taking opportunities to confront on-going obsessions. Home-based tasks or 'homework' will usually be assigned to continue the treatment outside the therapy session. If the person with OCD learns how to tackle their fears and anxieties effectively, this can provide him or her with skills to cope with future relapses. A hierarchy of 'tasks' can help the most difficult seem less daunting once the person has some success tackling more moderate difficulties.

It is also vital that all aspects of OCD and BDD are treated. The nature of the conditions can vary over time and patient may suffer from a number of different types of compulsions. Any one compulsion should not be treated to the exclusion of others, unless the patient is shown clearly how the same principles can be used for different compulsions. It can also be helpful if the patient is treated in the area where the problem exists; this could mean that the contaminated articles from home are brought into therapy sessions or the therapist visits the patient's home.

OCD can be successfully managed, but both sufferers and the medical profession should bear in mind that the condition can last a lifetime and that even sufferers who have responded well to treatment may have periodic relapses. It can be helpful for a patient to know where to return for treatment during a relapse and to be aware of any maintenance treatment that is available. This would help to prevent the patient having to go through the process of waiting for or starting any specialist treatment from scratch.

Some sufferers find support groups beneficial. When meeting others with OCD, people can feel relief that they are no longer isolated. Some people with OCD report

finding the most benefit from well managed support groups that focus on dealing with different aspects of the illness. This may include sharing experiences of treatment and useful self-help strategies. This type of group experience can also encourage people to seek or return to CBT. Moreover it can help people to keep up their efforts to confront anxiety provoking situations without resorting to compulsions or, in the case of BDD, camouflaging. Some sufferers say they have found support groups less beneficial when members of the group use the sessions primarily to complain about lack of treatment for OCD or suggest it is impossible to ever recover from OCD.

Although people with OCD might live with the condition all their lives, it is possible to return to fulltime education or work, conduct fulfilling relationships and regain a sense of equilibrium. The very idea of 'recovery' can in itself improve a person's outlook.

3.3 SUMMARY OF THE NEEDS OF PEOPLE WITH OCD AND BDD

The testimonies suggest that the following are useful for people suffering from OCD and BDD:

- Early recognition and diagnosis of OCD and BDD, particularly in people who may be presenting with depression, anxiety or somatic complaints
- Respect and understanding from healthcare professionals
- Awareness and understanding from public sector services including educational establishments, local authorities, police and emergency services
- That healthcare professionals have adequate awareness of the condition and effective treatment for people with OCD and BDD
- Full information about the nature of OCD and treatment options
- Psychological treatment that directly addresses the OCD
- Group therapy sessions and the possibility of working with more than one therapist
- That all aspects of the OCD are treated
- Information about what to do in case of relapse
- Information about support groups.

3.4 PERSONAL TESTIMONIES FROM FAMILY MEMBERS/CARERS OF PEOPLE WITH OCD

3.4.1 Sophia's mother

My daughter Sophia was diagnosed with OCD a year ago when she was 16, which is when her behaviour was of particular concern. But in retrospect, it is quite possible to recognise some early signs of OCD and anxiety in Sophia that we did not then identify as a problem. For instance, after the death of the Princess of Wales she and her twin brother Tom both seemed more anxious about the possibility of something

happening to my husband and me. They would both need to be at the door as I left the house and sometimes Sophia would make me look at a piece of paper or take something with me. She had a set of things she said, like 'take care' or 'be safe'. She would often say good-bye many times. Tom did the same to some extent, but not as much, and with him it lessened over time. On another occasion when we were on holiday, Sophia became hysterical when we did not walk in single file along a roadside. The traffic was so incredibly sparse that her concern seemed laughable to us and we gently mocked her. I am sure that there were other signs that we passed over without realising.

Sophia's friendships never seemed easy at school; she was ultra-sensitive, becoming hysterically upset about perceived slights from teachers or friends, and was often reluctant to invite friends home or accept social invitations. She was a perfectionist, not confident in her own abilities and a private person about some of her deeper feelings. In 2003 I noticed that she found revision for exams difficult because she had an odd and laborious routine of writing out her notes ('I have to do it this way', she would say) that would never allow her to fit in all of her revision. This clearly began to cause her worries too, although she passed her exams that summer reasonably well.

However, 2 weeks after starting the autumn term, Sophia took an overdose of paracetamol and aspirin. She told me afterwards that she wasn't trying to kill herself, but that she felt so bad about life in general she wanted someone to notice; she also felt that by taking an overdose and making herself sick she could give some form to her inner pain (and that maybe she could get a day off school!). I took her to accident and emergency where she was treated with great sensitivity by the staff and admitted to the children's ward (she was still 15) to be monitored. Before she was discharged the following day a psychiatrist talked to her, and then interviewed my husband and me. I later realised that this was the cause of a major and continuing trauma for Sophia. She felt that the psychiatrist was hostile, was trying to 'catch her out', or suspected she wanted to 'lock me up because I'm mad'. I don't know if the psychiatrist was in fact insensitive and handled the interview poorly, or if Sophia was paranoid about the experience and reading catastrophe into the scenario, which is something I now recognise she does as part of her OCD. Whatever the truth of the matter, it made her hugely reluctant to accept further counselling, medical or psychiatric advice.

The first thing we did, partly on the advice of the psychiatrist, was to immediately arrange a meeting with Sophia's school counsellor. Sophia was very reluctant but we all attended an hour's session as a family the day after the overdose. The counsellor was, we thought, brilliant, unpatronising in tone, and helpful to us all in untangling the reasons for the overdose. Sophia seemed relieved.

I instinctively felt that the best thing was for Sophia to continue with life in as normal way as possible although we all felt that the world had somehow cracked in two. We naturally felt unbelievably stupid not to have noticed how bad she had been feeling about life. The hospital, acting very promptly, had sent a report to our GP who phoned me the day after the overdose. She told us, as had the counsellor, that 'life as normal' was the best thing. Her call was reassuring, sensitive and useful to us.

We had a follow-up counselling session the week after the overdose. This was with the same counsellor we saw at Sophia's school, who was also a practice counsellor at our surgery. Our GP encouraged us to take up the option of private appointments with her because these were longer than those available on the NHS. Furthermore, she and our GP exchanged information about Sophia's condition, which was really useful.

Despite this support from the healthcare professionals, Sophia started to demonstrate other problems, including panic and anxiety attacks, which we found very frightening and deeply upsetting. Sometimes Sophia managed to get in for only an hour of school. Her sleep was now down to about 3 hours a night because she had nightmares and needed to keep the light on. She worried about chairs stored in a cupboard and had to keep checking them 'to make sure they were OK'. During this very difficult period I was often up with her late into the night so she could talk through what was in her head. It was incredibly exhausting and I saw the counsellor on my own to talk about Sophia's behaviour and to gather some strength to deal with things myself.

Our GP continued to be very helpful by supplying a letter to Sophia's school when her GCSE coursework deadlines could not be met. She saw Sophia regularly and offered her a referral to the Child and Adolescent Mental Health Services clinic. By this time Sophia had passed her 16th birthday and her treatment entered the adult phase. It was no longer up to us as parents to accept or not. Sophia initially rejected the offer of the referral but after a few weeks passed, there came some sort of watershed when she realised she wasn't getting better and certainly wasn't coping. She agreed to the referral. We were then told we were on a waiting list, but given no indication how long we might have to wait. This depressed Sophia hugely, not to mention us. We had felt the lifeline we had been offered had been roughly withdrawn. She feared that the NHS thought she was 'making everything up' and didn't believe her.

During this period Sophia's behaviour was still a great concern and in some ways it worsened. I wrote to our GP with a plea for help. I felt that if Sophia was to have any chance of living a reasonably normal 16-year-old life, attending school and taking her exams, she needed help immediately. I wasn't sure that as a family we would have been able to cope with much more in any case and I told our GP how stressed we all were. I became very tired, tearful and often short-tempered, and there were times when my husband and I were sick with worry. As I was Sophia's only home confidante, I also had to relay to my husband and to Tom what I felt was going on in her head to help them understand. I believe Tom thought that her behaviour was occasionally manipulative and attention seeking.

As a result of my letter, our GP asked for the referral to be expedited. She also obtained permission from a consultant to prescribe citalopram (10 mg per day later increased to 20 mg). The referral (with a community psychiatric nurse (CPN) at a family centre) was scheduled to take place within a week of the prescription being made to ensure that any side effects were monitored (there were a few initially – sleeplessness and feeling sick and 'spaced out'). But I was also aware after the first week that a marked improvement had taken place, with significantly reduced

levels of anxiety. (A little later this allowed Sophia to attend sessions of CBT with some hope of benefit and also helped her tackle school and exams.)

Our first appointment with the CPN was for the whole family. Subsequent sessions were for Sophia alone, but we also had a further three sessions as a family during 2004 (which Sophia found very stressful). The sessions were, I think, set up mainly to encourage Sophia to talk to us openly about her feelings and anxieties. I think the CPN took the view that if Sophia felt freer to talk, her emotions would be less likely to 'implode' as they had before with the overdose. CBT was recommended and appointments were set up to see a trainee psychologist working with the clinic.

It was while Sophia was receiving treatment at the family centre that she was first diagnosed with OCD. The trainee psychologist explained it to Sophia and gave her an information sheet, but this was not an adequate method of explaining OCD to a family member or patient and the effect it can have on the whole family. At the next family session we asked specifically about OCD and for advice and strategies for helping Sophia deal with certain situations we had as a family experienced. But we were not offered much advice. At no point was the subject of 'enabling' (or not 'enabling') raised, or how we might help her when her anxiety compromised us in carrying out our ordinary lives safely or efficiently. The fact that Sophia's tendency to catastrophise comes from her OCD was never made clear. It would have been helpful to be told.

I have learned more about the condition from an OCD online forum than from anything else I have read. It gave me reassurance that there were so many others suffering in the same way. I think we were all a bit frightened by the diagnosis at first. It meant she really did 'have something'. But when we thought about it we saw it was actually positive to have a diagnosis as we knew much more about what we were dealing with.

Sophia had about 10 sessions of CBT in all to help her with some of her checking rituals (we never realised just how many of these she had as she had kept them well hidden). I think Sophia found these beneficial and I am not aware of her checking cupboard doors as often as she did. However, Sophia's obsessive thoughts are very deep and are hard for her to live with. I don't think this has been addressed through the treatment to date. I don't think she even recognises that the thoughts are part of the OCD. She seems to think they are separate.

The CPN has also continued to see Sophia regularly and has been very good about fitting in extra sessions if Sophia has been going through a particularly bad patch, as happened before exams in the summer.

Our understanding is that OCD may not be curable, but must be lived with. We see that if Sophia is in control of her own life she manages better. However, she is still subject to some decisions that we make, and while we try to avoid making decisions based on her OCD if we know something is going to cause an anxiety attack that will make her and us stressed for hours, it's sometimes tempting to take the easy option and not make the decision.

I have found the year immensely wearing and have been unable to work since April because I find it virtually impossible to concentrate. I have become deeply

involved with my daughter on a level that may not be good for either of us. However, given that it's clearly dangerous for her not to talk I feel I must be there for her at the moment. I have continued to insist as hard as I can that she continue to see a counsellor. I need her to see someone else because she simply must talk to someone that isn't me.

Tom has also found it hard. He and Sophia often have strident arguments, with Sophia ratcheting up the tension on purpose at times. He then feels he is to blame for causing a scene. He has shown more signs of stress recently, looking sad and withdrawn.

Nevertheless I feel reasonably confident that Sophia has a chance of living a 'normal' life. Although she does not see it that way herself, thinking she is unpopular, incompetent and unattractive, when I look at her with friends she is clearly one of the more mature girls amongst her peers and they look to her for opinions and support. I do worry that her abilities have already been severely hampered by OCD and that her academic results will never reflect her actual ability. But she has had a part-time job that she enjoys and she is clearly valued by her employer.

3.4.2 Peter's mother

My friendly, socially aware, intelligent, considerate 18-year-old son has OCD. The period leading up to Peter's diagnosis 3 years ago was the worst of my life. By the time OCD had escalated into an easily identifiable condition, our family was already exhausted from years of tantrums, which Peter had had since he was 2 years old. OCD was different though, and took us down an enormous slope that we found terrifying.

At its worst, Peter would return from school and then spend 2 hours in the bathroom trying to change out of his uniform. He would check his palms were clean, and then look at the backs of his hands. However, while looking at the backs of his hands the palms may have got dirty, so had to be checked again, then the backs of his hands again, and so on, and so on. His hands would be red raw from unnecessary washing. He was unable to meet friends, play the piano, and go in the garden. He was unable to get onto a bus until he'd checked everyone getting on, to see he wasn't being followed. He thought any conversation we had inside a car could be heard by people on the pavement. He believed that his sister and I could get pregnant just by walking into his bedroom. If I got into his bedroom and straightened his bed cover he would think it was contaminated because it had touched a different part of the wall.

Before this, Peter got top marks at school, but this changed over a few months. First his writing changed from being very neat to scrawled and careless. Then he stopped being able to start work, or if he started he would be unable to complete it or would just lose it. He got through his GCSEs on the basis of his earlier work, but 6th form was a problem. Peter is currently re-taking the lower 6th and will complete his A levels next year. While we had once assumed he would go to university, and felt he would love the experience of living away from home, now we know that university at the moment would be pointless, and that it would be positively negligent to suggest

he moved out. I feel that his academic and work potential is minimal until he can find a way of overcoming the block that OCD places on his being proactive in making progress on set projects. The more important and interesting the project, the more OCD kicks in to block it. There is great sadness when the person you knew as a child with fantastic potential has to delete a computer file just when he's actually managed to do some work, and walks away from AS levels with one grade 'E' when he has the intelligence and skills to do much better.

There are several aspects of Peter's care worth commenting on. After promising an urgent referral to a child and adolescent psychiatric clinic our GP forgot to send the letter. I found this out 5 weeks later. By this point we were desperate. Peter was moaning and crying with frustration. He sometimes climbed on the roof of our house, and often threatened to jump out of an upstairs window because he saw no point in living. He ran away from home once and we had to get the police involved. One weekend we went to the out of hours GP who was the first to suggest OCD and the first to give any medication (buspirone).

When we eventually met the child and adolescent psychiatrist things started to get better. She was honest, calm and realistic (saying for example that when we went on holiday we would also be taking OCD with us, and that we needed to be practical), and engaged very well with Peter and the family, explaining to us clearly about the contribution we could make in helping Peter resist giving way to his compulsions. She emphasised that, however much we wanted to go along with the compulsions just to get some short-term peace, they would just continue, so in the long run the only way was to be calm but firm in resisting the urge to get involved in fulfilling Peter's compulsions. She was also positive in separating the OCD from the person, and saying that Peter's health came above his exam results (she wrote in advance to the examining board but I don't know if that had any effect).

Peter's psychiatrist also prescribed an SSRI (Seroxat) and described how it was believed to work. Although life is still not completely quiet in Peter's head, Seroxat has helped enormously by completely stopping the tantrums and reducing the effects of the OCD by cutting down the time spent trying to get out of a cycle of compulsions.

Peter also started having sessions of cognitive behavioural therapy. Although he found these sessions mildly interesting, the first person who worked with Peter was not inspiring, and did not involve the other family members. The strong implication was that this was Peter's private work and we shouldn't ask too much. We therefore had no idea how we were supposed to support Peter between visits. We asked to see someone else but the same thing happened. Occasionally Peter mentioned that he was trying to use a strategy he'd been taught, and we were very pleased, but this never lasted very long. The therapist did not appreciate that Peter was simply so exhausted by coping with OCD that he couldn't muster the strength of mind to confront it. I know many people are helped by CBT, but at this stage in Peter's life it was not the right thing for him.

Crucial to our survival as people and as a family is to have hope of some kind, but with OCD we have found that the focus of our hope has kept changing, and generally

becoming narrower and lower. We imagine what study Peter could do, then revise it; we imagine what travel he might want to do, then find it's not possible; we suggest employment opportunities, then realise that we're too optimistic. There is a constant need for hope, but a limited number of options to focus on. On bad days, when hope almost disappears I feel that I can't keep living. (I am also taking antidepressants.)

Peter's younger sister has also been very badly affected. She was often asked by him to help in the compulsions, and while she really wanted to respond positively, she knew from the psychiatrist that helping in the short term would not be helping in the long term and could entrench the compulsions. Peter's sister found this emotional battle very difficult and at one point she asked our GP for a course of antidepressants, which she has now stopped.

The situation at present is that Peter says he has no hope of winning against OCD, he feels he just has to learn to live with it. We feel however that Seroxat has had a positive impact; I cannot imagine how our family would still be together, or still be functioning at work or school if Peter wasn't taking it (and he would be very unwilling to stop taking it). Because of it the visible signs of OCD are now less obvious to us, allowing Peter the time and energy to take part in activities inside and outside the house. However, Peter is still struggling internally with the compulsions – we need to remain aware of this battle and somehow keep up our mental and emotional energy to support him and ourselves through the bad patches in the future.

3.4.3 Archie's father

Our son Archie was the light of our lives, he was growing normally, he was well behaved, intelligent and someone we were and still are very proud of. But from the age of about 5 or 6, it was clear that Archie was somehow different from other children, although at this time we had never heard of OCD.

From an early age he would ask challenging questions. All children ask 'why', but Archie wanted more detailed, more scientific answers and usually could not be fobbed off with a fairy-tale answer. At school he got by doing the minimum to get a good mark and generally school was easy for him. He had a clear sense of order and things to him would be in 'black or white', or right or wrong, with nothing in between.

When he was slightly older, if he appeared not to like something then there would be no compromise and he would only do things he did not like with great reluctance. It was not just a dislike, but an obsession. For instance, Archie would not like his hands to be dirty or sticky, and he would frequently wash his hands. This developed into a full-blown contamination obsession resulting in ritual hand washing to the point where his skin was in a dreadful state. Art classes therefore posed a 'danger' because he would get paint or clay on his hands. Football and sports were also a problem as this involved getting muddy and dirty. There were also other obsessions as yet unknown to us.

Archie became a target for ridicule and bullying. Pupils would spit at him and as soon as he got home he would strip off and have a long hot bath. Sadly Archie kept most of this to himself. Both the bullying and OCD affected his schoolwork, but despite this he did obtain some good exam results, if not as good as expected.

It was only when he was in his late teens and when Archie's obsessions had taken over and destroyed his life and ours that we first found out about OCD on a television programme. Had we known about OCD from the outset then maybe we could have done something to help him cope with it. Sadly, despite many trips to our GP both before and after the TV programme Archie was not diagnosed at this time. The only 'advice' we got from the GP was that he would get over it and that it was a teenager thing. All he did was to prescribe some hand cream for Archie's skin problem and then only in hopelessly small quantities. The GP would not even consider referring Archie to someone who might be able to recognise an underlying cause for his obsessions and do something about it. We had taken Archie to the GP on numerous occasions between the ages of 14 and 17 where he had ample opportunity to make a diagnosis or at least try to do something. We foolishly trusted the GP and accepted his advice, something we will regret to our dying days, but it was only after talking to the GP about Archie after he had left home some years later at the age of 22 and had been formally diagnosed (by another GP), that the GP admitted that he had never heard of OCD.

After leaving school Archie started a course in computer science at college, something he excelled in. However, Archie's rituals and contamination fears worsened and often he could not get to college on time for lectures or exams. It took him all day to get out of the house, showering until long after all of the hot water had gone and rituals were completed uninterrupted. Also there were problems leaving the house as this involved touching door handles and so on that were contaminated nor would he go out of the door if there was anyone outside that might see him. In addition there was his constant striving for perfection that meant he was never satisfied with his work so it was never finished or not handed in on time, although the work was of a much higher standard than required. The result was that he never finished the course although he continued to go to the college to unofficially use the facilities and for somewhere to go to get out of the house and to avoid us, his parents, because some of his obsessions and 'bad thoughts' were about us.

It was during the 'unofficial' time at college when Archie was finally diagnosed as having OCD (although we did not know about this at the time). With the help of a friend he went to a new GP and Prozac was prescribed. So at last, years after we had recognised that there was a problem, something could be done, or so we thought.

However, we now entered a very difficult period, which we describe as a 'living bereavement', when we lost our son to OCD. The most destructive and distressing factor was that Archie considered us to be dirty and contaminated. The effect on our relationship with him was unbearable although still we sought an answer. A major blow came when he left home because of his obsessions about us. He tried to cut all links with us although we realised that it was not what he wanted0 – it was what his OCD told him he wanted.

Archie made rules about our relationship with him, some of which were that we were not to contact him, go anywhere near where he lived or ask him questions about his condition. The rules were all very one-way and on his terms, but we feared what would happen if we broke the rules. We did not know what was happening to him or how he was coping. Worst of all was the thought that he may commit suicide (something that we now know he was seriously considering). But with no income he was still dependant on us financially, so a very difficult link was maintained under considerable duress via the occasional phone call from him using a disguised voice to protect him from his obsessions surrounding us and, we think, to protect us from his harmful thoughts.

Archie had some very bad experiences, including his flat being flooded during the floods of Easter 1998. Archie has an obsession about faeces and to find his home full of raw sewage must have been unbelievably traumatic. Because of my job at the time I was the person with overall responsibility for the management of rescue, recovery and restoration of the affected area. In Archie's mind I was contaminated and he had to avoid me. He would make the lengthiest, time-consuming detours to miss routes or places my wife or I might use. In the end he left the area altogether and moved to another town. Because of the move and the difficulties the local authority and all local services were experiencing due to the flood, all contact was lost between Archie and the health and social services.

After several months Archie moved back to the town and contact with the social services was initially restored, but they were still overstretched and limited resources could be devoted to him. Because he no longer had contact with the health services he had no medication. He was living in absolute squalor because he would not throw household rubbish away and it accumulated in the flat where it smelled and was a cause for complaint from his neighbours. But worse still because of his obsession about faeces he would defecate into a plastic bag, which would also be kept in the flat (part of his obsession involved reserving the lavatory for urinating, not for disposing of faeces). Some of his neighbours perceived him to be 'abnormal' or 'mad' and ridiculed him; some were aggressive and subjected him to verbal abuse, threats of violence and carried out acts of vandalism such as breaking windows, throwing eggs at his door and putting dirt and dog faeces through his letter box. Because of this Archie would not respond to callers or letters, and tended to sleep during the day. He also incurred considerable debts. He had no income and his OCD dictated a very extravagant lifestyle. For example, underwear could not be washed because he considered it to be contaminated, so it had to be discarded and new undergarments worn every day. He also faced eviction.

These circumstances combined with the chronic situation at social services again resulted in a complete loss of contact with services. We thought at the time, and have since been proved right, that he was becoming suicidal. At this point I went to the social services, although this was against his 'rules'. Fortunately they were sympathetic and took action. Archie was allocated a GP and seen by a psychiatrist. His medication was restarted and he was given limited practical help, although no psychological treatment was offered. The assessment was that he was not ready for a therapy such as CBT. That judgement was probably correct at the

time. (However, that was in 1998 and I doubt if any further assessment has been carried out.)

There was no significant improvement over the next 4 years but Archie then developed pneumonia and was admitted to hospital and this forced him to allow social workers to make contact with us. This helped enormously with being able to communicate with him and opened a communication channel with his social worker. From then on communication and most importantly his trust in us improved. We found out that his most pressing worry was the substantial debt he had built up. Fortunately we were able to reduce the debt to a manageable level and build the trust between us by 'obeying' his new rules.

This situation however was most frustrating and heartbreaking. We knew that we could do so much to help if only he would allow us to. But if we did this, other than pander to his demands, would it trigger an adverse response leading to his situation deteriorating or even worse? However, we thought it was the only way to proceed and the only way that we could show him that we put his well-being first and to show him that he could trust us to do what was best, or under the restrictions imposed, what we thought would be best for him.

Over time trust did improve and we were able to talk openly and honestly with him about his situation. Gradually we got our son back, that is, he no longer tried to separate and distance himself from us and now we have a good, open parents/son relationship, something we have missed for many years.

There have been other small improvements more due to his own efforts than those of the medical services. He is still on medication and sees a psychiatrist periodically but sessions usually start with the psychiatrist saying that he would like to spend longer with Archie but he is running late so only has a few minutes, which is of no value. There is no suggestion of any further treatment or other sort of action or ways to progress. I have attended the last two sessions and have suggested various treatments or options be considered but this was met with a rather contemptuous attitude by the psychiatrist.

Archie is 30 now, is still unable to work and has a very restricted life. However, we recently went out together for a meal to celebrate his birthday, something a short while ago we thought would be impossible. We are loving parents, supporting him the best we can, and have been able to improve the quality of his life recently although there is far more to do. We cannot become complacent because if we do we feel that he will become comfortable with his OCD and the restrictions it places on him.

If only Archie had been diagnosed when we first went to the GP. If only the GP was aware of OCD and had referred him to a specialist. If only the GP had done something then maybe Archie's life would have been so much better and we would not have suffered so much distress.

Having a family member with OCD has been a dreadful experience and it continues to have a severe and detrimental effect on our lives and health. We have encountered so much ignorance and lack of awareness amongst professionals and society as a whole that it has depressed us. We have suffered more than can be expressed in a few words, although that is so little compared with the torment

sufferers endure – Archie has missed the normal life of a teenager and of a young man due to OCD.

3.4.4 Graham's wife

My husband's OCD shows itself particularly in extreme compulsive hoarding. Graham is 63 years old and in many ways is a wonderful husband – loyal, honest, conscientious and hard working – but he does not really acknowledge the problem either for himself or for the effect it has on me.

We were married in 1975, when Graham was 34 years old. He had been living with his parents in quite a cluttered house, and his mother tended to keep things like yoghurt pots and carrier bags. She was one of the Second World War generation and had the attitude that these things 'might come in useful one day and nothing should be wasted'. Although this was not the way I had been brought up, I did not at that time consider the situation to be particularly abnormal. After we were married I had begun to notice that Graham would also not discard empty packaging, tins and so on, or allow me to get rid of them. Odd bits of wood and other items began to pile up on the landing of our house, causing a fire hazard. He would also keep old newspapers, junk mail, and old letters and cards. By 1982, I was concerned enough about Graham's hoarding tendencies to write a letter to our GP at the time. The matter was never pursued.

When we moved house in 1985 everything came with us 'to be sorted when we get there'. Very little has, but a lot more has been added over the years. I have a constant battle to try and get Graham to dispose of anything. Our parents and an aunt have all died and left complete households for us to clear. Graham will not even let me throw away anything belonging to my own family. According to him, everything is supposed to be sold and the proceeds shared with other family members, but there are many items that are of no value. We live in a cluttered, chaotic junkyard – several rooms in our very large house are completely inaccessible and have been for many years. He cannot find anything when he needs it and can become angry if he finds that I have moved or disposed of something. Maintenance is often impossible because the problems are inaccessible. Often, he will not trust anybody to do the work. He has to do it himself to ensure that it is up to his standard. He is such a perfectionist that things are achieved at a painfully slow speed, or not at all. If he does start on a job, he will focus on it to the exclusion of everything else, so other things get out of control.

In 1989, our younger son was referred to the child and family clinic because of his difficult behaviour at school. One of the three psychiatrists whom we saw in the course of that year about my son's behaviour told me that Graham was suffering from OCD. There was no further explanation, and nothing was said to him. No further reference was made to it, and there was no suggestion that it should be followed up. I appealed for help from our GP several times over the years, but I was told that nothing could be done unless my husband himself requested help, and Graham didn't think that he needed help at this time and

has never acknowledged the need for help. He felt that I was the one who needed help.

During this time I felt that any needs I might have were neither considered nor understood until it was too late. I often felt desperate, helpless and depressed about the situation at home. I was ashamed and embarrassed if anyone had to come into the house, and my social life was also affected. I hated living like this and I broke down completely in 2000. Then all my GP could suggest was for me to take antidepressants, which I refused to do on the grounds that it would not go any way towards solving the underlying problems. Following a visit to an occupational psychologist, I had to ask to be referred to a psychiatrist for myself. I had not been made aware of what services were available, or offered any other help. One psychiatrist whom I saw said that I could not have further treatment as it was my husband who needed help. She said that she would phone my GP that evening, so I made an appointment for a few days later, and wrote to my GP to explain why. I told my husband that if he did not come with me, I would have to leave him. He did come, but the psychiatrist had not contacted the GP. She only realised what was happening because of my letter. That was how my husband was referred to a psychologist.

My husband was then referred to a trainee clinical psychologist in 2002, who diagnosed OCD, and clearly explained the reasons for the diagnosis in understandable terms. Graham was seen at home, which I thought was very helpful, and I was invited to join in with the sessions. The psychologist worked in practical terms, setting targets for each visit. We labelled everything in a certain area with 'Keep (if so, where?)', 'Sort (for a specific reason)' or 'Discard (if so, how and where?)'. She drew up a written contract in which we agreed to work together towards the targets, and which we all signed. The psychologist followed up the progress at each visit. She was very helpful, and Graham had begun to make progress, but when her training placement finished, her supervisor took over. Progress has now more or less ceased. It seems to me that this psychologist has not followed the same practice as his student, has ignored the contract and does not work in the same systematic way on the practical issues. He has not really managed to build any sort of rapport, and seems to have almost given up the attempt. He gives the impression of being extremely frustrated with Graham and spends a lot of time talking to him about 'normal people'. I told him that Graham neither knows nor cares what 'normal people' do, and he acknowledged that that was probably true. He is supposed to be liaising with my psychologist, but I do not believe that this happens with any regularity.

When my own psychiatrist retired, communication broke down regarding my treatment, and it took a letter to my MP to get the matter resolved. I was eventually referred to a psychologist, but she sees me alone at the clinic, and displays very little understanding of the issues involved. She has never met Graham, nor seen the situation at home. She has told me that the situation will not change, and I have to learn to live with it. She has also told me that American 'experts' on hoarding now say that the recognised treatments do not work. I find this thoroughly unhelpful, depressing and untrue. I have learned from my own experience and experiments that it is possible to change the situation gradually; and even the tiniest step is helpful in keeping away my depression.

Because I felt so let down by the NHS, Graham and I are now having private psychological treatment with a GP who is understanding and supportive. We have separate appointments on alternate weeks. It is costing us a fortune, but, from my point of view, is worth every penny – I feel as if I am gaining more control over the situation. It is also helping me to understand my husband's thought processes, so that I can see things from his point of view, and try to respond accordingly. I feel that this has also had an effect on my husband's behaviour.

I am learning to take control of my own emotional states, thus forestalling depressive episodes, and am now more aware of the possibility of learning from experience, and trying new strategies, instead of feeling defeated. This gives me hope for the future.

3.5 UNDERSTANDING THE IMPACT OF OCD ON FAMILY MEMBERS AND CARERS

3.5.1 The role of family members and carers

The time consuming and disruptive rituals resulting from OCD can affect a patient's whole family and individual relationships and can cause stress, frustration, anxiety, and anger in family members and carers. Many carers and family members often rebuke themselves for not having noticed the symptoms of OCD in their relative or friend before it is established. But it is quite common for people with OCD to hide their rituals and obsessions from the family, partly because of the embarrassment of carrying out apparently bizarre acts. Some people with OCD might deny that there is anything wrong if asked about their rituals or compulsions. In some other families, a family member may realise that something is wrong and seek explanation for it in their local library or on the Internet and in this way 'diagnose' the condition. The family member may encourage the sufferer to seek help, although some sufferers may not accept that there is a problem.

People with OCD may also actively 'involve' the carer/family member in his or her rituals and avoidance behaviour, sometimes surreptitiously. Carers and family members may become involved because of the distress and interference in activities, but compliance with this can 'entrench' the rituals or compulsions still further. Even when they are aware that involvement may be making things worse, carers and family members might feel that submitting to the patient's wishes is the only option. This can be frustrating for carers who feel that they have no other means of 'helping'. Others might go along with the rituals and compulsions in order to 'keep the peace'. The rituals can severely restrict and disrupt the lives of family members who might have to engage in, for instance, decontamination activities, leading to extra work in the home and/or extra expense incurred as a result of the ritual. Some family members can become the focus for the obsessive thoughts, which can be enormously upsetting. Others may try to ignore the condition because they do not understand it or think it will go away, while some may find OCD easier to understand if it is seen as a physical illness instead of a mental illness.

Whatever the circumstances however, family members and carers are inevitably drawn into the illness and the resulting environment. Many will want to help in whatever way they can and be included where appropriate in the treatment process. Indeed, people with OCD are likely to benefit from having a supporter who understands the condition and helps the person confront their fears, and who can continue to offer support after treatment to help maintain any improvements achieved. This might be a family member, but in cases where this is not appropriate a friend may be more suitable (occasionally, some people with OCD may lead such isolated lives that there is no one who can help).

In order to participate in the patient's care and the treatment process, family members and carers should be given full and ongoing information (where confidentiality permits) about how best to support the person with OCD and to cope with the condition (see Section 3.5.2). Where appropriate, it may be helpful if they can be part of any decision making process.

Inclusion in the care and treatment process should take into account the carers' circumstances and environment to ensure that an undue burden is not placed on them. It is important to achieve a proper balance between sensitivity to the patient's concerns and avoiding compliance or involvement with the obsessive fears and compulsions. Attention should also be paid to additional problems affecting family members that the patient may not acknowledge.

Carers can feel as lonely and cut off as the person with OCD. This can be exacerbated by the stigma associated with mental health problems, especially where there is an unwillingness to talk about such problems with family members and others. Carers may themselves need support from the professional services including their GP and practice counsellors as a result of stress, anxiety and frustration of living with someone with OCD and providing long-term care. In very severe cases, some carers of patients with long-term OCD, particularly older and lone carers, may need respite care.

3.5.2 Information for carers

Carers and family members should be provided with clear information from healthcare professionals about OCD in a way that they can readily understand and so that they can provide care in the best way.

Carers have requested that the following type of information is made available:

- How best to help people with OCD
- How to deal with the rituals and obsessions of people with OCD
- Recommended treatments for OCD
- Possible side effects of treatment
- The likely prognosis for people with OCD
- How to identify suicidal intent.[1]

[1]These points were identified in a survey of carers of people with OCD conducted by Cliff Snelling, the representative for carers on the GDG. The survey can be found at www.ocdaction.org.uk.

3.5.3 Summary of carer needs

- Respect, understanding and sensitivity from all healthcare professionals, public sector services including educational establishments, local authorities, police and emergency services for people with OCD and their families and carers
- Recognition that OCD can be severe and can have a devastating effect on the lives of family members and carers
- Adequate information about the nature, course and treatment of OCD
- Information and advice about how not to become involved in a family member's rituals and compulsions
- Carers and family members may need support and treatment for anxiety and depression
- Information about support groups and voluntary organisations.

3.6 SPECIFIC ISSUES FOR CHILDREN AND FAMILIES

There is little evidence that life-events *cause* OCD, but in the individual vulnerable to the condition, times of life-stress may be when symptoms worsen or relapses occur. In childhood this can particularly be around events that affect the family, school transitions, examination times or during difficulties with friendships or other relationships. Children experiencing learning problems, which have perhaps been undetected or where their needs have not been adequately met, may be vulnerable to exacerbations of OCD.

The transition from adolescence to adult life, with increasing independent living demands, can be an especially challenging time, particularly for the anxious individual. Young people with OCD have often been more than usually dependent on their parents, more cautious about exploring new experiences out of the home or with friends, or may have particular symptoms that make aspects of life difficult (for example, sharing a rented house/bathroom).

In the UK, mental health services for young people usually stop at age 16 or 18, and transfer occurs to general adult mental health services. For the young person with OCD, who is at a vulnerable stage of their development, continuity of services at this stage is essential. Child and adolescent mental health services should endeavour to link with the appropriate adult service well before discharge from child services needs to occur, to enable the young person to meet the new team, have joint appointments if necessary and so on.

Whatever the age of the person with OCD, clinicians working with the patient need to give time and attention to family members and carers. The younger the child, the more responsibility and decision making will rest with the adults, but even in young children, the child needs to feel involved in the treatment, able to express preferences and to take charge of aspects of their therapy. Young people need to develop autonomy and so for older children and teenagers, the therapist should assess sensitively and collaboratively the degree to which parents need to be involved and negotiating at each stage what information to share. Parents can be invaluable in

ensuring therapy is successful, and indeed involvement is essential when they are closely involved in their child's rituals.

Many parents feel guilty about their child having OCD, and therapists need to take active steps to remove any sense of blame the parent might hold. It can be helpful for parents to understand that parents do not cause OCD but can inadvertently become involved in OCD. Most important, they need to understand that that they can be very helpful in the recovery process, and in maintaining good mental health in the future.

Parents of children with OCD can sometimes be anxious themselves, perhaps because of their own nature, but also because their child is distressed, has difficulty coping in important life areas, or has changed markedly. The issues raised by parental anxiety need to be understood by therapists and dealt with sensitively and actively. Treatment for OCD always involves understanding anxiety, and often helping the child confront and deal with anxiety, rather than ritualising or 'running away'. The anxious parent may find it very challenging to help their child learn to deal with anxiety in this way, and may need help themselves to learn effective strategies.

3.7 SOURCES OF USER AND CARER ADVICE

In addition to professional healthcare services, users and carers may consider the other following sources of information. It is important that users and carers remain aware that the quality of information can be variable, and it may be important to rely on several rather than single sources.

- Books and videos aimed at sufferers, which may include practical advice and guidance on self-help for dealing with different types of obsessions and compulsions. There are also books written by people with OCD and carers of people with OCD.
- National and local support groups and self-help groups.
- National and international charities. There are currently a number of charities supporting those with OCD and other anxiety-related conditions, details of which can be found on the Internet. Some of these have information especially for carers and family members. Membership of such charities may offer newsletters, details of self-help groups and practical advice on getting specialist treatment.
- Conferences aimed at people with OCD and carers.

3.8 CLINICAL PRACTICE RECOMMENDATIONS

3.8.1.1 Treatment and care should take into account the individual needs and preferences of people with OCD or BDD. Patients should have the opportunity to make informed decisions about their care and treatment. Where patients do not have the capacity to make decisions, or children or young people are nor old enough to do so, healthcare professionals should follow the Department of Health guidelines (*Reference guide to*

consent for examination or treatment [2001], available from www.dh.gov.uk). **[GPP]**

3.8.1.2 Good communication between healthcare professionals and people with OCD or BDD is essential. Provision of information, treatment and care should be tailored to the needs of the individual, culturally appropriate, and provided in a form that is accessible to people who have additional needs, such as learning difficulties, physical or sensory disabilities, or limited competence in speaking or reading English. **[GPP]**

3.8.1.3 Healthcare professionals should consider informing people with OCD or BDD and their family or carers about local self-help and support groups, and encourage them to participate in such groups where appropriate. **[GPP]**

3.8.1.4 Because OCD and BDD often have an impact on families and carers, healthcare professionals should promote a collaborative approach with the person with OCD or BDD and their family or carers, wherever this is appropriate and possible. **[GPP]**

3.8.1.5 In the treatment and care of people with OCD or BDD, family members or carers should be provided with good information (both verbal and written) about the disorder, its likely causes, its course and its treatment. **[GPP]**

3.8.1.6 Assessment and treatment plans for people with OCD or BDD should, where appropriate, involve relevant family members or carers. In some cases, particularly with children and young people, when the symptoms of OCD or BDD interfere with academic or workplace performance, it may be appropriate to liaise with professionals from these organisations. Assessment should include the impact of rituals and compulsions on others (in particular on dependent children) and the degree to which carers are involved in supporting or carrying out behaviours related to the disorder. **[GPP]**

3.8.1.7 If dependent children are considered to be at risk of emotional, social or mental health problems as a result of the behaviour of a parent with OCD or BDD and/or the child's involvement in related activity, independent assessment of the child should be requested. If this is carried out, the parent should be kept informed at every stage of the assessment. **[GPP]**

3.8.1.8 In the treatment of people with OCD or BDD, especially when the disorder is moderate to severe or chronic, an assessment of their carer's social, occupational and mental health needs should be offered. **[GPP]**

4. METHODS USED TO DEVELOP THIS GUIDELINE

4.1 OVERVIEW

The development of this guideline drew upon methods outlined by NICE (Eccles & Mason, 2001; NICE, 2002). A team of experts, professionals, people with OCD and a carer, known as the Guideline Development Group (GDG), with support from the NCCMH staff, undertook the development of a patient-centred, evidence-based guideline. There are six basic steps in the process of developing a guideline:

- Define the scope, which sets the parameters of the guideline and provides a focus and steer for the development work
- Define clinical questions considered important for practitioners and patients
- Develop criteria for evidence searching and search for evidence
- Design validated protocols for systematic review and apply to evidence recovered by search
- Synthesise and (meta-) analyse data retrieved, guided by the clinical questions, and produce evidence statements
- Answer clinical questions with evidence-based recommendations for clinical practice.

The clinical practice recommendations made by the GDG are therefore derived from the most up-to-date and robust evidence base for the clinical and cost effectiveness of the treatments and services used in the management of OCD. In addition, to ensure a patient and carer focus, the concerns of people with OCD and carers regarding clinical practice have been highlighted and addressed by good practice points and recommendations agreed by the whole GDG. The evidence-based recommendations and good practice points (GPPs) are the core of this guideline.

4.2 THE GUIDELINE DEVELOPMENT GROUP

The GDG consisted of professionals in psychiatry, clinical psychology, nursing and general practice; academic experts in psychiatry and psychology; and people with OCD and a carer. The guideline development process was supported by staff from the NCCMH, who undertook the clinical and health economics literature searches, reviewed and presented the evidence to the GDG, managed the process, and contributed to the drafting of the guideline.

4.2.1 Guideline Development Group meetings

Twenty-one GDG meetings were held between June 2003 and May 2005. During each day-long GDG meeting, in a plenary session, clinical questions and clinical evidence were reviewed and assessed, statements developed and recommendations formulated. At each meeting, all GDG members declared any potential conflict of interests, and patient and carer concerns were routinely discussed as part of a standing agenda.

4.2.2 Topic leads

The GDG divided its workload along clinically relevant lines to simplify the guideline development process, and individual GDG members took responsibility for advising on guideline work for particular areas of clinical practice (psychological interventions, pharmacological interventions, BDD, children and young people).

4.2.3 People with OCD and carers

Individuals with direct experience of services gave an integral patient and carer focus to the GDG and the guideline. The GDG included two people with OCD and a carer. They contributed as full GDG members to writing the clinical questions, helping to ensure that the evidence addressed their views and preferences, highlighting sensitive issues and terminology associated with OCD, and bringing OCD patient research to the attention of the GDG. In drafting the guideline, they contributed to writing and editing the chapter on the experience of people with OCD and BDD, editing the first draft of the guideline's introduction, and identifying good practice points from the patient and carer perspective.

4.2.4 Special advisers

Special advisers, with specific expertise in one or more aspects of treatment and management relevant to the guideline, assisted the GDG, commenting on specific aspects of the developing guideline and making presentations to the GDG. The names of those who agreed to act as special advisers are listed on the Acknowledgements page.

4.2.5 National and international experts

National and international experts in the area under review were identified through the literature search and through the experience of the GDG members. These experts were contacted to recommend unpublished or soon-to-be published studies in order to ensure up-to-date evidence was included in the development of the guideline. They

informed the group about completed trials at the pre-publication stage, systematic reviews in the process of being published, studies relating to the cost effectiveness of treatment, and trial data if the GDG could be provided with full access to the complete trial report. Appendix 4 lists researchers who were contacted.

4.3 CLINICAL QUESTIONS

Clinical questions, developed from the scope, were used to guide the identification and interrogation of the evidence-base relevant to the topic of the guideline. The questions were initially drafted by the review team and the GDG chair, then refined or developed further by the GDG using informal consensus. The PICO (patient, intervention, comparison and outcome) framework was used to help formulate questions about interventions. This structured approach divides each question into four components: the patients (the population under study); the interventions (what is being done); the comparisons (other main treatment options); and the outcomes (the measures of how effective the interventions have been). Appendix 5 lists the clinical questions.

4.4 SYSTEMATIC CLINICAL LITERATURE REVIEW

The aim of the clinical literature review was to systematically identify and synthesise relevant evidence from the literature in order to answer the specific clinical questions developed by the GDG. Thus, clinical practice recommendations are evidence-based, where possible, and if evidence was not available, informal consensus methods were used (see Section 4.4.6.1) and the need for future research was specified.

4.4.1 Methodology

A stepwise, hierarchical approach was taken to locating and presenting evidence to the GDG. The NCCMH developed this process based on advice from NICE's National Guidelines Support and Research Unit and after considering recommendations from a range of other sources. These included:

- Centre for Clinical Policy and Practice of the New South Wales Health Department (Australia)
- Clinical Evidence Online
- Cochrane Collaboration
- New Zealand Guideline Group
- NHS Centre for Reviews and Dissemination
- Oxford Centre for Evidence-Based Medicine
- Oxford Systematic Review Development Programme
- Scottish Intercollegiate Guidelines Network (SIGN)
- United States Agency for Health Research and Quality.

4.4.2 The review process

A brief search of the major bibliographic databases for recent systematic reviews and existing guidelines was first conducted to help inform the development of the scope. After the scope was finalised, a more extensive search for systematic reviews was undertaken. At this point, the review team, in conjunction with the GDG, developed an evidence map that detailed all comparisons necessary to answer the clinical questions. The initial approach that was taken in order to locate primary-level studies depended on the type of clinical question and availability of evidence.

After consulting the GDG, the review team decided which questions were likely to have a good evidence base and which questions were likely to have little or no directly relevant evidence. For questions in the latter category, a brief descriptive review was initially undertaken by a member of the GDG (see Section 4.4.6.1). For questions with a good evidence base, the review process depended on the type of clinical question.

4.4.2.1 The search process for questions concerning interventions

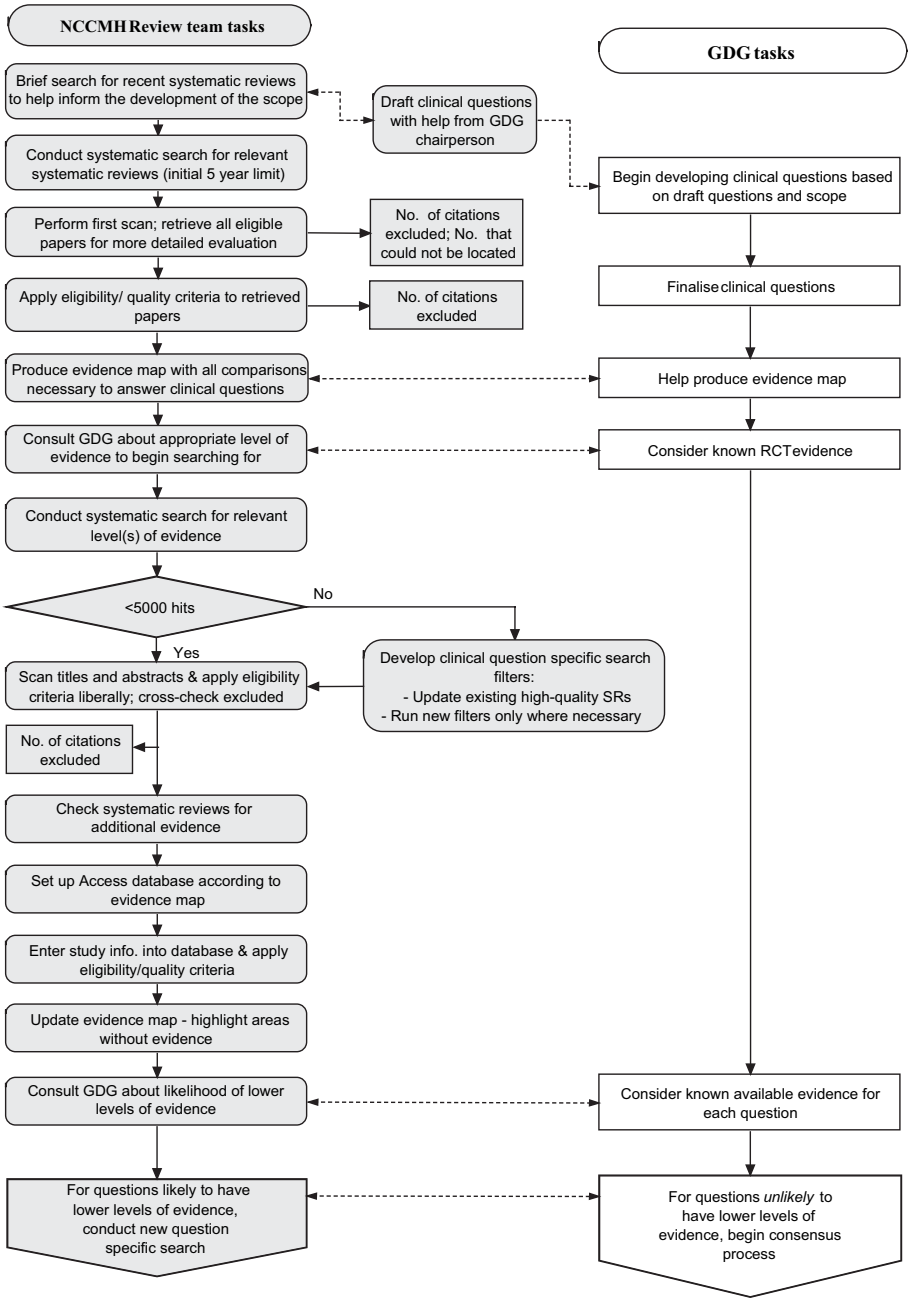
For questions related to interventions, the initial evidence base was formed from well-conducted RCTs that addressed at least one of the clinical questions. Although there are a number of difficulties with the use of RCTs in the evaluation of interventions in mental health, the RCT remains the most important method for establishing treatment efficacy. The initial search for RCTs involved searching the standard mental health bibliographic databases (Embase, Medline, PsycInfo, Cochrane Library) for all RCTs potentially relevant to the guideline. If the number of citations generated from this search was large (>5000), question-specific search filters were developed to restrict the search while minimising loss of sensitivity.

After the initial search results were scanned liberally to exclude irrelevant papers, the review team used a purpose built 'study information' database to manage both the included and the excluded studies (eligibility criteria were developed after consultation with the GDG). For questions without good quality evidence (after the initial search), a decision was made by the GDG about whether to: (a) repeat the search using subject-specific databases (for example, CINAHL, AMED, SIGLE or PILOTS); (b) conduct a new search for lower levels of evidence; or (c) adopt a consensus process (see Section 4.4.6.1). Future guidelines will be able to update and extend the usable evidence base starting from the evidence collected, synthesised and analysed for this guideline.

Recent high-quality English-language systematic reviews were used primarily as a source of RCTs (see Appendix 7 for quality criteria). However, where existing datasets were available from appropriate reviews, they were cross-checked for accuracy before use. New RCTs that met the inclusion criteria set by the GDG were incorporated into the existing reviews and fresh analyses performed. The review process is illustrated in Flowchart 1.

Additional searches were made of the reference lists of all eligible systematic reviews and RCTs, and the list of evidence submitted by stakeholders. Known experts in the field (see Appendix 4), based both on the references identified in early steps

Flowchart 1: Guideline review process



and on advice from GDG members, were sent letters requesting systematic reviews or RCTs that were in the process of being published[2]. In addition, the tables of contents of appropriate journals were periodically checked for relevant studies.

4.4.2.2 Unpublished evidence

The GDG used a number of criteria when deciding whether or not to accept unpublished data. First, the evidence must be accompanied by a full trial report or sufficient detail to properly assess the quality of the data. Second, the evidence must be submitted with the understanding that it will be published in the full guideline. For example, the GDG did not accept evidence submitted as *commercial in confidence*. However, the GDG recognised that unpublished evidence submitted by investigators, might later be retracted by those investigators if the inclusion of such data would jeopardise publication of their study.

4.4.2.3 Search filters

Search filters developed by the review team consisted of a combination of subject heading and free-text phrases. Specific filters were developed for the guideline topic, and where necessary, for each clinical question. In addition, the review team used filters developed for systematic reviews, RCTs and other appropriate research designs (Appendix 6).

4.4.2.4 Study selection

All primary-level studies included after the first scan of citations were acquired in full and re-evaluated for eligibility at the time they were being entered into the study information database. More specific eligibility criteria were developed for each clinical question (see appropriate chapter). All eligible papers were then critically appraised for methodological quality (see Appendix 8). The eligibility of each study was confirmed by at least one member of the group.

4.4.3 Synthesising the evidence

Where possible, outcome data were extracted directly from all eligible studies, which met the quality criteria, into Review Manager 4.2.7 (Cochrane Collaboration, 2004). Meta-analysis was then used, where appropriate, to synthesise the evidence using Review Manager. If necessary, reanalyses of the data or sensitivity analyses were used to answer clinical questions not addressed in the original studies or reviews. For continuous outcomes, where more than 50% of the total number randomised in a particular study were not accounted for, the data were excluded from the analysis because of the risk of bias. In the case of dichotomous outcomes (except for the outcome of leaving the study early), the effects of high attrition rates were examined with sensitivity analyses.

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[2]Unpublished full trial reports were also accepted where sufficient information was available to judge eligibility and quality.

Evidence tables, generated automatically from the study information database, were used to summarise general information about each study (see Appendix 16). Where meta-analysis was not appropriate and/or possible, the reported results from each primary-level study were also presented in the evidence tables.

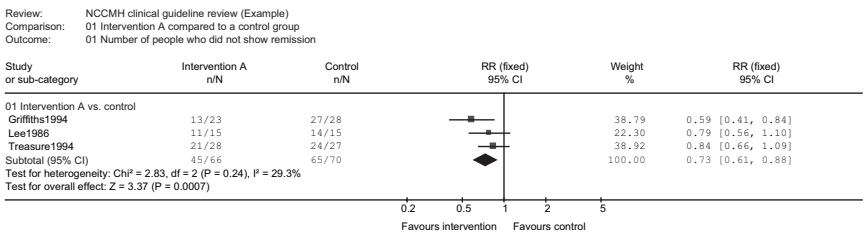
Consultation was used to overcome difficulties with coding. Data from studies included in existing systematic reviews were extracted independently by one reviewer directly into Review Manager and cross-checked with the existing data-set. Two independent reviewers extracted data from new studies, and disagreements were resolved with discussion. Where consensus could not be reached, a third reviewer resolved the disagreement. Masked assessment (that is, blind to the journal from which the article comes, the authors, the institution, and the magnitude of the effect) was not used since it is unclear that doing so reduces bias (Berlin, 2001; Jadad *et al.*, 1996).

4.4.4 Presenting the data to the GDG

Where possible, the GDG were given a graphical presentation of the results as forest plots generated with the Review Manager software. Each forest plot displayed the effect size and confidence interval (CI) for each study as well as the overall summary statistic. The graphs were organised so that the display of data in the area to the left of the 'line of no effect' indicated a 'favourable' outcome for the treatment in question. Dichotomous outcomes were presented as relative risks (RR) with the associated 95% CI (for an example, see Figure 2). A relative risk (or risk ratio) is the ratio of the treatment event rate to the control event rate. An RR of 1 indicates no difference between treatment and control. In Figure 2, the overall RR of 0.73 indicates that the event rate (that is, non-remission rate) associated with intervention A is about three-quarters of that with the control intervention, or in other words, intervention A reduces non-remission rates by 27%. In addition, the CI around the RR does not cross the 'line of no effect' indicating that this is a statistically significant effect. The CI shows with 95% certainty the range within which the true treatment effect should lie.

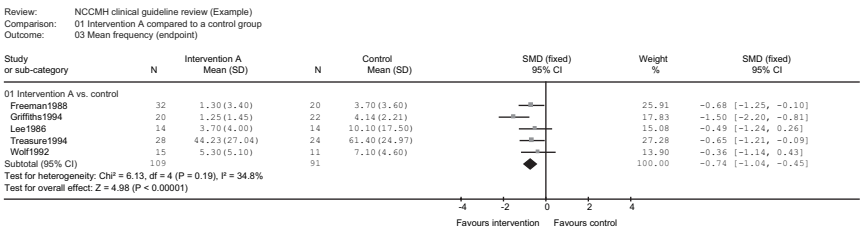
Efficacy outcomes were calculated on an intention-to-treat basis (that is, a 'once-randomised-always-analyse' basis). This assumes that those participants who

Figure 2: Example of a forest plot displaying dichotomous data



ceased to engage in the study – from whatever group – had an unfavourable outcome. Continuous outcomes were analysed as standardised mean differences (SMD) (e.g. see Figure 3).

Figure 3: Example of a forest plot displaying continuous data



To check for heterogeneity between studies, both the I^2 test of heterogeneity and the chi-squared test of heterogeneity ($p < .10$), as well as visual inspection of the forest plots were used. The I^2 statistic describes the proportion of total variation in study estimates that is due to heterogeneity (Higgins & Thompson, 2002). An I^2 of less than 30% was taken to indicate mild heterogeneity and a fixed effects model was used to synthesise the results. An I^2 of more than 50% was taken as notable heterogeneity. In this case, an attempt was made to explain the variation. If studies with heterogeneous results were found to be comparable, a random effects model was used to summarise the results (DerSimonian & Laird, 1986). In the random effects analysis, heterogeneity is accounted for both in the width of CIs and in the estimate of the treatment effect. With decreasing heterogeneity the random effects approach moves asymptotically towards a fixed effects model. An I^2 of 30% to 50% was taken to indicate moderate heterogeneity. In this case, both the chi-squared test of heterogeneity and a visual inspection of the forest plot were used to decide between a fixed and random effects model.

4.4.5 Forming and grading the statements and recommendations

The evidence tables and forest plots formed the basis for developing clinical statements and recommendations.

4.4.5.1 Intervention studies

For intervention studies, all evidence was classified according to an accepted hierarchy. Recommendations were then graded A to C based on the level of associated evidence, as a good practice point (GPP) (see Text box 1).

In order to facilitate consistency in generating and drafting the clinical statements the GDG utilised a statement decision tree (see Flowchart 2). The flowchart was designed to assist with, but not replace clinical judgement.

Text Box 1: Hierarchy of evidence and recommendations grading scheme

Level	Type of evidence	Grade	Evidence
I	Evidence obtained from a single randomised controlled trial or a meta-analysis of randomised controlled trials	**A**	At least one randomised controlled trial as part of a body of literature of overall good quality and consistency addressing the specific recommendation (evidence level I) without extrapolation
IIa	Evidence obtained from at least one well-designed controlled study without randomisation	**B**	Well-conducted clinical studies but no randomised clinical trials on the topic of recommendation (evidence levels II or III); or extrapolated from level-I evidence
IIb	Evidence obtained from at least one other well-designed quasi-experimental study		
III	Evidence obtained from well-designed non-experimental descriptive studies, such as comparative studies, correlation studies and case studies		
IV	Evidence obtained from expert committee reports or opinions and/or clinical experiences of respected authorities	**C**	Expert committee reports or opinions and/or clinical experiences of respected authorities (evidence level IV) or extrapolated from level I- or II-evidence. This grading indicates that directly applicable clinical studies of good quality are absent or not readily available
		GPP	Recommended good practice based on the clinical experience of the GDG

Adapted from Eccles, M. and Mason, J. (2001) How to develop cost-conscious guidelines. *Health Technology Assessment* 5, 16; Mann, T. (1996) *Clinical Guidelines: Using Clinical Guidelines to Improve Patient Care Within the NHS*. London: Department of Health.

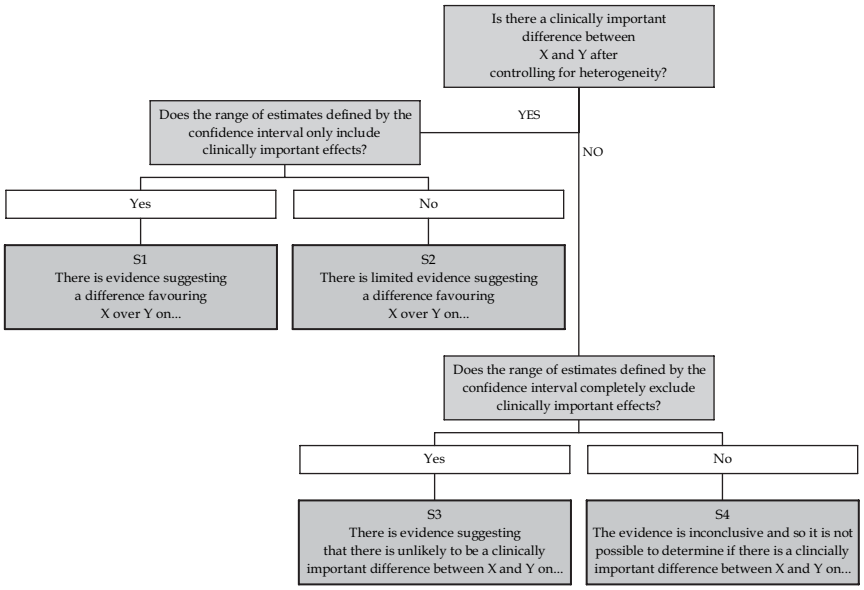
Using the decision tree (Flowchart 2), the GDG classified each effect size as clinically important or not (that is, whether or not the treatment is likely to benefit patients), taking into account several factors including statistical significance, the magnitude and precision of the effect, the trial population and the nature of the outcome. The starting point for determining whether or not the magnitude of the effect was likely to be clinically significant was a RR of 0.80 or less and a SMD of –0.50 or less.

Where heterogeneity between studies was judged problematic, either a random effects model was used or sub-analyses were conducted to examine the possibility of moderators.

In cases where an effect was judged clinically important, a further consideration was made about the strength of the evidence by examining the range of estimates defined by the CI. For level-I evidence, where the effect size was judged clinically important for the full range of plausible estimates, the result was characterised as *evidence suggesting a difference favouring x over y on* . . . (S1). For non-level-I evidence or in situations where the CI included clinically unimportant effects, the result was characterised as *limited evidence suggesting a difference favouring x over y on* . . . (S2). Where an effect size was judged as *not* clinically important and the CI did not include any clinically important effects, the result was characterised as *unlikely to be clinically important* . . . (S3).

Alternatively, if the range of estimates defined by the CI included clinically important benefits as well as no effect or harmful effects, the result was characterised as *inconclusive* (S4).

Flowchart 2: Guideline statement decision tree



Once all evidence statements relating to a particular clinical question were finalised and agreed by the GDG, the associated recommendations were produced and graded. Grading allowed the GDG to distinguish between the level of evidence and the strength of the associated recommendation. It is possible that a statement of evidence would cover only one part of an area in which a recommendation was to be made or would cover it in a way that would conflict with other evidence. In order to produce more comprehensive recommendations suitable for people in England and Wales, there were times when the GDG had to extrapolate from the available evidence. This led to a weaker level of recommendation (that is, B, as data were based upon level-I evidence). In addition, it is possible to have methodologically sound (level-I) evidence about an area of practice that is of little direct clinical relevance or has such a small effect that it is of little practical importance. In this case, the evidence would attract a lower strength of recommendation (that is, there would be necessity for extrapolation). It is important to note that the grading of the recommendation is not a reflection of its clinical importance or relevance.

The process also allowed the GDG to moderate recommendations based on factors other than the strength of evidence. Such considerations include the applicability of the evidence to the people in question, economic considerations, values of the development group and society, or the group's awareness of practical issues (Eccles *et al.*, 1998).

4.4.6 Method used to answer a clinical question in the absence of appropriately designed, high-quality research

In the absence of level-I evidence (or a level that is appropriate to the question), or where the GDG decided (on the basis of previous searches or their knowledge of the literature) that there was unlikely to be such evidence, either an informal or formal consensus process was adopted. This process focused on those questions that the GDG considered a priority.

4.4.6.1 Informal consensus

The starting point for this process was that a member of the GDG identified, with help from the systematic reviewer, a narrative review that most directly addressed the clinical question. Where this was not possible, a brief review of the recent literature was initiated.

This existing narrative review or new review was used as a basis for beginning an iterative process to identify lower levels of evidence relevant to the clinical question and to lead to written statements for the guideline. The process involved a number of steps:

- A description of what is known about the issues concerning the clinical question was written by one of the GDG members.
- Evidence from the existing review or new review was then presented in narrative form to the GDG and further comments were sought about the evidence and its perceived relevance to the clinical question.

- Based on this feedback, additional information was sought and added to the information collected. This may include studies that did not directly address the clinical question but were thought to contain relevant data.
- If, during the course of preparing the report, a significant body of primary-level studies (of appropriate design to answer the question) were identified, a full systematic review was done.
- Following this, on occasions and as deemed appropriate by the GDG, the report was then sent to appointed experts outside of the group for peer review and comment. The information from this process was then fed back to the GDG for further discussion of the statements.
- Recommendations were then developed and could also be sent for further external peer review.
- After this final stage of comment, the recommendations were again reviewed and agreed upon by the GDG.

4.5 HEALTH ECONOMICS REVIEW STRATEGIES

The aim of the health economics review was to contribute to the guideline development process. Data on the economic burden of OCD and evidence of cost effectiveness of the different treatment options for OCD were collected and assessed to help the decision-making process. See Chapter 9 for the detailed health economic review strategies.

4.6 STAKEHOLDER CONTRIBUTIONS

Professionals, people with OCD, and companies have contributed to and commented on the guideline at key stages in its development. (See Appendix 3.) Stakeholders for this guideline include:

- Patient/carer stakeholders: the national patient and carer organisations that represent people whose care is described in this guideline
- Professional stakeholders: the national organisations that represent healthcare professionals who are providing services to people with OCD
- Commercial stakeholders: the companies that manufacture medicines used in the treatment of OCD
- Primary Care Trusts
- Department of Health and Welsh Assembly Government.

Stakeholders have been involved in the guideline's development at the following points:

- Commenting on the initial scope of the guideline and attending a briefing meeting held by NICE
- Contributing lists of evidence to the GDG
- Commenting on the first and second drafts of the guideline.

4.7 VALIDATION OF THIS GUIDELINE

This guideline has been validated through two consultation exercises. The first consultation draft was submitted to the NICE Guidelines Advisory Committee, and circulated to stakeholders and other reviewers nominated by GDG members.

The GDG reviewed comments from stakeholders, the NICE Guidelines Advisory Committee, a number of health authority and trust representatives and a wide range of national and international experts from the first round of consultation. The GDG then responded to all comments and prepared a second consultation draft which was submitted to NICE, circulated to all stakeholders for final comments and posted on the NICE website for public consultation. Based on review of their comments, a final draft was then submitted to the NICE Guidelines Advisory Committee for review prior to publication.

5. PSYCHOLOGICAL INTERVENTIONS

5.1 INTRODUCTION

Psychological interventions have been described for obsessive-compulsive disorder since the time of Freud. However, despite extensive writing about the disorder, OCD was generally considered to be virtually untreatable for over 50 years. In 1966 Victor Meyer described the successful treatment of two people with OCD by what would now be considered as the forerunner of modern day CBT treatments by changing cognitions and blocking compulsive rituals (Meyer, 1966). Following on from this, staff at the Maudsley Hospital developed behaviour therapy (BT) techniques in the early 1970s that offered hope for the first time and demonstrated efficacy in a series of small quasi-experimental studies (Marks *et al.*, 1975; Rachman *et al.*, 1971; Rachman *et al.*, 1973). Other researchers in the UK, Europe and North America rapidly experimented with a range of behavioural techniques (Emmelkamp & Kraanen, 1977; Foa & Goldstein, 1978; Rabavilas *et al.*, 1979).

By the early 1980s the common elements of several procedures that had been developed at different centres evolved into what is now known as exposure and response prevention (ERP) (see Steketee, 1994 for a review). Given the absence of effective treatments until the seventies, the early studies were so convincing that most researchers explored different ways of delivering the treatment components in trials looking at the differential efficacy of treatment formats rather than conducting randomised controlled trials to establish efficacy against non-treatment or attention controls. In fact, with one or two exceptions, most of the controlled trials date from after 1990.

With the rise of cognitive therapy in the eighties (Salkovskis, 1985), a variety of cognitive approaches have also been developed, mostly in combination with behavioural techniques (Freeston *et al.*, 1996; Salkovskis, 1999; Salkovskis & Warwick, 1986; van Oppen & Arntz, 1994). While many therapists have continued to offer a variety of psychological approaches, there has been relatively little written about other approaches and even less research.

5.2 BEHAVIOUR AND COGNITIVE THERAPIES

5.2.1 Introduction

More than 30 years of published research and a large number of authoritative accounts have led to a widely held consensus that behaviour therapy is an effective treatment for OCD. Indeed, the successful treatment of OCD was one of the early success stories for behaviour therapy. The early experimentation with a diverse range of behaviourally based procedures has evolved into a therapy with a central

technique, ERP, that can be used in a variety of formats, including book and computer-based self-help, group therapy, and individual therapy that ranges from minimal therapist contact or telephone contact through to intensive outpatient and inpatient regimes (Foa & Franklin, 2000; Himle *et al.*, 2003; Lovell *et al.*, 2000; Marks, 1997). Cognitive therapies have emerged more recently with the hope that they would improve the efficacy of behaviour therapy and provide an alternative to those who have difficulty in engaging in ERP (Salkovskis & Warwick, 1986; Wilhelm, 2000). Many contemporary treatment approaches combine behavioural and cognitive approaches, but there are proponents of purer forms of both. It is important to note that in general, published treatment studies almost certainly do not cover all symptom presentations equally. Washing/cleaning and checking are probably well represented, but obsessions without overt compulsions and hoarding are most likely to be underrepresented except in studies that specifically target these forms. It is difficult to know the extent to which other less frequent forms are included in treatment studies, or their response to treatment (see also Mataix-Cols *et al.*, 2002a).

5.2.2 Current practice

During the rapid development of behaviour therapy in seventies, Professor Isaac Marks established a training programme at the Maudsley Hospital in 1972 to develop behaviour therapy skills among psychiatric nurses. This programme, and others that followed, established a strong core of skilled behaviour nurse therapists working in the NHS (Gournay *et al.*, 2000). The work at the Maudsley and other centres also influenced professional training for psychologists and psychiatrists, among others, and so emerged a strong multidisciplinary tradition for behaviour therapy in centres throughout the UK. Multidisciplinary training in cognitive therapy developed in the early nineties and in 2004 there were over twenty post-qualification courses across the UK offering training in cognitive and behavioural therapies (www.babcp.org). There are, however still gaps in the provision of training and although most are accredited with universities, accreditation by the British Association of Cognitive and Behavioural Therapies is not yet widespread. Almost all basic professional training in psychiatry, psychology and nursing includes some training in these therapies. Thus, there is a large body of clinicians with knowledge of these approaches, although there are relatively few with specific expertise and experience in the application of cognitive and behavioural therapies to the treatment of OCD.

Therapists with the necessary expertise have traditionally been found in secondary and tertiary care settings. There is an unequal distribution of accredited therapists across the UK (Shapiro *et al.*, 2003) and the picture is likely to be similar with trained but non-accredited therapists. However, there are increasing numbers of clinicians with CBT training in primary care and there are a number of recent training programmes to enable professionals in primary care with little CBT experience to provide assisted self-help to people with anxiety disorders (for example, Lovell *et al.*, 2003), including OCD.

A recent report from the Department of Health (2004), addresses issues related to current provision of psychological therapies, training, supervision, and competence. While not limited to CBT, the report makes a number of recommendations on these issues and provides guidance on the organisation and provision of training, continuing professional development, and clinical supervision.

5.2.3 Interventions included in the review

The following interventions were included:

- Behaviour therapy
- Cognitive therapy
- Cognitive behavioural therapy
- Rational-emotive therapy.

5.2.4 Studies considered for review[3]

The review team conducted a new systematic search for RCTs that assessed the efficacy and tolerability of behavioural and cognitive therapies among adults with OCD. Thirty-seven studies were identified, of which 20 did not meet the inclusion criteria of the GDG. The 17 included studies provided efficacy data from 820 participants and tolerability data from 734 participants.

Of the included studies, two compared ERP with systematic relaxation or anxiety management (GREIST2002; LINDSAY1997), and two compared CBT with waitlist controls (CORDIOLI2003; FREESTON1997). Four studies compared ERP with cognitive therapy (COTTRAUX2001; MCLEAN2001; VANOPPEN1995; WHITTAL2005), one study with CBT (VOGEL2004), two with rational-emotive therapy (EMMELKAMP1988; EMMELKAMP1991), and seven with other variants of behaviour therapy (DEARAUJO1995; EMMELKAMP1983; GREIST2002; HISS1994; KENWRIGHT2005; LOVELL1994; MEHTA1990).

All included studies were between 3 and 44 weeks long (mean length = 12 weeks). Patients were treated in an outpatient setting in eight studies; the setting was unclear in the remaining nine studies. In one study (FREESTON1997), the patients had obsessive symptoms only. Another study (LOVELL1994) was concerned with patients with rituals only. Four studies were conducted in the US, four in the Netherlands, three in the UK, and one each in Australia, Brazil, Canada, France, India and Norway. The average age of the participants was 35 years and the average duration of illness was 12.26 years.

Full details of the studies included in the guideline and the reasons for excluding are given in Appendix 16.

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[3]Here and elsewhere in the guideline, each study considered for review is referred to by a study ID (primary author and date of study publication in capital letters, except where a study is *in press* or only submitted for publication, then a date is not used).

5.2.5 Psychological interventions versus control (systematic relaxation, anxiety management or wait list)

5.2.5.1 Behaviour therapy versus control (systematic relaxation, anxiety management or waitlist)

Clinical evidence statements[4]

Efficacy[5]	***Included studies***
There is evidence suggesting a difference favouring ERP over anxiety management control on reducing obsessive-compulsive symptoms as measured by the clinician-rated Y-BOCS (K = 1; N = 18; SMD = –2.89; 95% CI, –4.30 to –1.48). **I**	LINDSAY1997
There is evidence suggesting a difference favouring clinician-guided ERP over systematic relaxation control on reducing obsessive-compulsive symptoms as measured by the self-reported Y-BOCS (K = 1; N = 121; SMD = –1.10; 95% CI, –1.49 to –0.72). **I**	GREIST2002
There is limited evidence suggesting a difference favouring computer-guided behaviour therapy over systematic relaxation control on reducing obsessive-compulsive symptoms as measured by the self-reported Y-BOCS (K = 1; N = 121; SMD = –0.68; 95% CI, –1.05 to –0.31). **I**	GREIST2002
There is limited evidence suggesting a difference favouring ERP over controls on reducing obsessive-compulsive symptoms as measured by the Padua Inventory (K = 1; N = 18; SMD = –1.28; 95% CI, –2.32 to –0.24). **I**	LINDSAY1997
There is limited evidence suggesting a difference favouring ERP over anxiety management control on reducing the impact of OCD on life and activities as measured on an interference rating scale (K = 1; N = 18; SMD = –3.16; 95% CI, –4.64 to –1.67). **I**	LINDSAY1997
There is limited evidence suggesting a difference favouring clinician-guided ERP over systematic relaxation	GREIST2002

[4]The full list of all evidence statements generated from meta-analyses (and the associated forest plots) are on the CD-ROM that accompanies the guideline.

[5]In the case of SMD or WMD, negative effect sizes favour the treatment group.

control on improving functioning as measured by the patient-rated Work and Social Adjustment Scale (K = 1; N = 121; SMD = –0.60; 95% CI, –0.96 to –0.23). **I**

There is limited evidence suggesting a difference favouring computer-guided ERP over systematic relaxation control on improving functioning as measured by the patient-rated Work and Social Adjustment Scale (K = 1; N = 121; SMD = –0.40; 95% CI, –0.76 to –0.04). **I** — GREIST2002

There is evidence suggesting a difference favouring clinician-guided ERP over systematic relaxation control on the likelihood of treatment response, defined as 'much improved' or 'very much improved' on the Clinical Global Impressions (CGI) scale (K = 1; N = 125; RR = 0.51; 95% CI, 0.38 to 0.69). **I** — GREIST2002

There is limited evidence suggesting a difference favouring computer-guided ERP over systematic relaxation control on the likelihood of treatment response, defined as CGI 'much improved' or 'very much improved' (K = 1; N = 123; RR = 0.73; 95% CI, 0.59 to 0.91). **I** — GREIST2002

Tolerability

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and controls on leaving the study early (K = 2; N = 125; RR = 4.47; 95% CI, 0.51 to 38.92). **I** — GREIST2002 LINDSAY1997

5.2.5.2 CBT versus waitlist control

Clinical evidence statements

Efficacy — ***Included studies***

There is limited evidence suggesting a difference favouring group CBT over waitlist control on the likelihood of treatment response, defined as a 35% or greater reduction on the clinician-rated Y-BOCS (K = 1; N = 47; RR = 0.32; 95% CI, 0.17 to 0.59). **I** — CORDIOLI2003

There is limited evidence suggesting a difference favouring CBT over waitlist on reducing obsessive-compulsive symptoms as measured on the clinician-rated Y-BOCS in patients with obsessive symptoms only (K = 1; N = 29; SMD = –1.18; 95% CI, –1.98 to –0.38). **I** — FREESTON1997

There is evidence suggesting a difference favouring group CBT over waitlist on reducing obsessive-compulsive symptoms as measured on the clinician-rated Y-BOCS (K = 1; N = 47; SMD = –1.18; 95% CI, –1.81 to –0.56). **I** — CORDIOLI2003

There is limited evidence suggesting a difference favouring CBT over waitlist control on reducing obsessive-compulsive symptoms as measured by the Padua Inventory (K = 1; N = 29; SMD = –0.83; 95% CI, –1.59 to –0.07). **I** — FREESTON1997

There is limited evidence suggesting a difference favouring group CBT over waitlist control on improving psychological quality of life as measured by the WHOQOL-BREF psychological subscale (K = 1; N = 47; SMD = –0.59; 95% CI, –1.18 to –0.01). **I** — CORDIOLI2003

There is limited evidence suggesting a difference favouring group CBT over waitlist control on improving environmental quality of life as measured by the WHOQOL-BREF environmental subscale (K = 1; N = 47; SMD = –1.05; 95% CI, –1.66 to –0.44). **I** — CORDIOLI2003

There is limited evidence suggesting a difference favouring CBT over waitlist control on reducing anxiety symptoms as measured by the Beck Anxiety Inventory in patients with obsessive symptoms only (K = 1; N = 29; SMD = –0.87; 95% CI, –1.64 to –0.10). **I** — FREESTON1997

Tolerability

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBT and waitlist on the likelihood of leaving the study early (K = 2; N = 76; RR = 0.77; 95% CI, 0.24 to 2.49). **I** — FREESTON1997 CORDIOLI2003

5.2.6 Psychological interventions versus other psychological interventions

5.2.6.1 Behaviour therapy versus cognitive therapy

Clinical evidence statements

Efficacy — ***Included studies***

There is limited evidence suggesting a difference favouring group behaviour therapy over group cognitive therapy on the likelihood of recovering at 12 months' follow-up, defined as a reliable change on the clinician-rated Y-BOCS — MCLEAN2001

and a clinician-rated Y-BOCS score less than 13 (K = 1; N = 93; RR = 0.74; 95% CI, 0.6 to 0.92). **I**

Tolerability

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and cognitive therapy on the likelihood of leaving the study early (K = 4; N = 305; RR = 0.97; 95% CI, 0.63 to 1.47). **I**

COTTRAUX2001
VANOPPEN1995
MCLEAN2001
WHITTAL2005

5.2.6.2 Behaviour therapy versus CBT

Clinical evidence statements

Efficacy

There is limited evidence suggesting a difference favouring CBT over behaviour therapy on reducing obsessive-compulsive symptoms at 6 months' follow-up as measured on the clinician-rated Y-BOCS (K = 1; N = 35; SMD = 0.86; 95% CI, 0.16 to 1.56). **I**

Included studies

VOGEL2004

Tolerability

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and CBT on the likelihood of leaving the study early (K = 1; N = 35; RR = 6.74; 95% CI, 0.94 to 48.29). **I**

VOGEL2004

5.2.6.3 Behaviour therapy versus rational-emotive therapy

Clinical evidence statements

Efficacy

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between behaviour therapy and rational-emotive therapy on the efficacy of treatment. **I**

Included studies

EMMELKAMP1988
EMMELKAMP1991

5.2.6.4 Self-exposure versus partner-assisted exposure therapy

Clinical evidence statements

Efficacy

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between self-exposure and partner-assisted exposure on the efficacy of treatment. **I**

Included studies

EMMELKAMP1983

5.2.6.5 Imaginal plus live ERP versus live ERP

Clinical evidence statements

Efficacy	***Included studies***
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between imaginal plus live ERP and live ERP on the efficacy of treatment. **I**	DEARAUJO1995

5.2.6.6 Exposure plus relapse prevention versus exposure plus associative therapy

Clinical evidence statements

Efficacy	***Included studies***
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between exposure plus relapse prevention and exposure plus associative therapy on the efficacy of treatment. **I**	HISS1994

5.2.6.7 Audiotaped exposure to anxiogenic thoughts versus audiotaped exposure to neutral thoughts

Clinical evidence statements

Efficacy	***Included studies***
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between audiotaped exposure to anxiogenic thoughts and audiotaped exposure to neutral thoughts on the efficacy of treatment. **I**	LOVELL1994

5.2.6.8 Computerised ERP (BTSTEPS) plus scheduled support versus computerised ERP (BTSTEPS) plus requested support

Clinical evidence statements

Efficacy	***Included studies***
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between BTSTEPS plus scheduled support and BTSTEPS plus requested support on reducing obsessive-compulsive symptoms as measured by the Y-BOCS (K = 1; N = 36; SMD = –0.55; 95% CI, –1.22 to 0.12). **I**	KENWRIGHT2005
Tolerability	
There is evidence suggesting a clinically significant effect of fewer patients being likely to leave the study early in BTSTEPS plus scheduled support when compared with BTSTEPS plus requested support (K = 1; N = 44; RR = 0.23; 95% CI, 0.08 to 0.70). **I**	KENWRIGHT2005

5.2.6.9 Family-based behaviour therapy versus patient-based behaviour therapy

Clinical evidence statements

Efficacy	***Included studies***
There is limited evidence suggesting a difference favouring family-based behaviour management over patient-based behaviour management on reducing obsessive-compulsive symptoms as measured by the Maudsley Obsessive-Compulsive Inventory (K = 1; N = 30; SMD = –0.89; 95% CI, –1.65 to –0.14). **I**	MEHTA1990
There is evidence suggesting a difference favouring family-based behaviour management over patient-based behaviour management on reducing obsessive-compulsive symptoms at 6 months' follow-up as measured by the Maudsley Obsessive-Compulsive Inventory (K = 1; N = 30; SMD = –1.44; 95% CI, –2.25 to –0.62). **I**	MEHTA1990
There is evidence suggesting a difference favouring family-based behaviour management over patient-based behaviour management on reducing depression as measured by the Zung Self-rating Depression scale (K = 1; N = 30; SMD = –1.38; 95% CI, –2.19 to –0.58). **I**	MEHTA1990
There is evidence suggesting a difference favouring family-based behaviour management over patient-based behaviour management on reducing depression at 6 months' follow-up as measured by the Zung Self-rating Depression scale (K = 1; N = 30; SMD = –1.81; 95% CI, –2.67 to –0.94). **I**	MEHTA1990
There is limited evidence suggesting a difference favouring family-based behaviour management over patient-based behaviour management on improving social adjustment at work as measured by the Global Assessment of Severity (K = 1; N = 30; SMD = –0.91; 95% CI, –1.67 to –0.16). **I**	MEHTA1990
There is evidence suggesting a difference favouring family-based behaviour management over patient-based behaviour management on improving social adjustment at work at 6 months' follow-up as measured by the Global Assessment of Severity (K = 1; N = 30; SMD = –1.34; 95% CI, –2.15 to –0.54). **I**	MEHTA1990

There is limited evidence suggesting a difference favouring family-based behaviour management over patient-based behaviour management on improving family adjustment as measured by the Global Assessment of Severity (K = 1; N = 30; SMD = –0.78; 95% CI, –1.52 to –0.03). **I** MEHTA1990

5.2.7 Therapist time in psychological interventions

5.2.7.1 Introduction

Although the efficacy of CBT (including ERP) is generally widely accepted, there is great variability in exactly how treatment is delivered. Group and individual treatments for OCD usually show a high degree of overlap in that planning for and carrying out ERP exercises between sessions is believed to be one of the most important components. Likewise, ERP may be delivered through guided self-help using books or computer-based packages with minimal support from therapists. Indeed, guided self-help may be conceptualised as an alternative delivery format of ERP rather than a distinct form of intervention. Finally, in individual formats, therapist input can vary from extremely brief (for example, 15 minutes per week) to highly intensive (e.g. 2 hours per day, 5 days per week, for 3 weeks). Consequently, it is difficult to determine what an adequate 'dose' of CBT may be. The aim of this review is thus to examine treatment intensity. Although intensity can be defined in a number of ways such as frequency and duration of sessions, from a resource viewpoint, the therapist time per patient may be considered a proxy. As a result, a course of group CBT, over and beyond any advantages that may arise from the interaction of group members, may be considered a lower intensity treatment if the number of therapist hours per patient is below that of an equivalent course of individual CBT.

5.2.7.2 Current practice

ERP for OCD was developed in the 1970s in specialist units in the UK, often in an inpatient setting (see Marks, 1997), as well as elsewhere in Europe and the US. Since that time psychological treatment for OCD has been delivered mainly in secondary and tertiary care. But because there remain difficulties in accessing trained behavioural and cognitive therapists in a timely manner and because provision varies from one region to another (Shapiro *et al.*, 2003), there may be a role for forms of CBT requiring relatively little therapist input that can be delivered by a broader range of healthcare professionals. These professionals may not have been trained extensively in CBT, but if they have been trained to deliver the key components of the therapy in an effective way, access and availability could improve (Lovell & Richards, 2000). Brief CBT-based therapies have become increasingly available in primary care settings over the last 5 years for a variety of disorders and there is evidence of expansion. However, although the essential features of ERP are easy to grasp, its application in OCD can be complicated. Thus, even though low intensity treatments may be effective for some people, there is likely to be a role for traditional individual

CBT for those who have not adequately responded to treatment with lower intensity treatment. Furthermore, given the heterogeneity of clinical presentation found in OCD, there may be a proportion of people with OCD for whom low intensity treatments may not be suitable due to the need to substantially adapt approaches.

5.2.7.3 Method

The aim of this review was to determine whether the number of hours spent by a therapist per client in session predicted the efficacy of psychological interventions in patients with OCD. Due to time constraints, the review considered only studies on adult patients with OCD. For inclusion, studies had to report pre- and post-treatment scores of an outcome measure, such as the Y-BOCS, and the number of therapist hours per client. The latter was calculated as [the number of hours per session X total number of sessions] multiplied by the number of therapists per session. If the mode of treatment delivery was group therapy, then the number of therapist hours was divided by the number of patients per group.

Based on the distribution of the number of therapist hours per client across the studies, the studies were categorised into high, medium and low intensity groups. Interventions in which the number of therapist hours per client was less than 10 were classified as low treatment intensity interventions. Interventions in which the number of therapist hours per client was 10 or more and less than 30 were classified as medium intensity interventions. Interventions with more than 30 therapist hours per client were classified as high intensity interventions.

5.2.7.4 Studies considered

A new systematic search for studies of psychological interventions in adults with OCD was conducted. The review considered studies that included exposure and/or response prevention and/or cognitive therapy as part of the intervention. The search identified 1,107 studies of which 1,037 were excluded as being irrelevant. Of the 69 studies that could potentially be included, 19 studies were excluded because the patients were children with OCD (K = 14) or had BDD (K = 5). Other reasons for exclusion were that the data was not extractable (K = 10), the number of therapist-hours per client could not be extracted (K = 5), the study had less than 5 participants in the treatment group (K = 1), the report was a case study (K = 2), the report was a review of the literature (K = 1), the study tested a pharmacological intervention (K = 1), the study was in German (K = 1).

The number of studies included in the review was 29 (BOUVARD2002; CHAMBLESS1999; CORDIOLI2003; DEARAUJO1995; EMMELKAMP1983; EMMELKAMP1988; EMMELKAMP1991; ENRIGHT1991; FOA1984; FOA1985; FOA2004; FREESTON1997; FRITZLER1997; GREIST2002; HISS1994; HUGHES2004; LINDSAY1997; MCKAY1996; MCLEAN2001; WHITTAL2005; NEZIROGLU2001; OCONNOR1999; ROSQVIST2001; ROTHBAUM2000; TAYLOR2003; VANOPPEN1995; VANNOPPEN1997; VANNOPPEN1998; VOGEL2004).

5.2.7.5 Sub-group analysis

Based on the 10 and 30 cut-off scores of the number of therapist hours per client, 11 interventions from 8 studies were classified as low intensity (CORDIOLI2003; EMMELKAMP1983_COUPLES; EMMELKAMP1983_PATIENT; ENRIGHT1991; FRITZLER1997; HUGHES2004; MCLEAN2001_BT; MCLEAN2001_CT; TAYLOR2003_DELAYED; TAYLOR2003_IMMEDIATE; VANNOPPEN1998), 22 interventions from 14 studies were classified as medium intensity (BOUVARD2002; CHAMBLESS1999; DEARAUJO1995_EXV; DEARAUJO1995_EXI; EMMELKAMP1988_BT; EMMELKAMP1988_CT; EMMELKAMP1991_BT; EMMELKAMP1991_CT; HISS1994_AT; HISS1994_RP; GREIST2002_CLINICIAN BT; LINDSAY1997; OCONNOR1999; ROTHBAUM2000; VANNOPPEN1997_GROUP BT; VANNOPPEN1997_MFBT; VANOPPEN1995_BT; VANOPPEN1995_CT; VOGEL2004_BT; VOGEL2004_CBT; WHITTAL2005_BT; WHITTAL2005_CT) and 10 interventions from 7 studies were classified as high intensity (FOA1984_ERP; FOA1984_EX; FOA1984_RP; FOA1985_EXI; FOA1985_EXV; FOA2004; FREESTON1997; MCKAY1996; NEZIROGLU2001; ROSQVIST2001). The pre- and post-treatment scores on the study's key measure of efficacy were entered into the Review Manager software, which was used to estimate heterogeneity across all studies and to produce an effect size for each sub-group calculated as the SMD between the pre- and post-treatment scores.

Across all studies, there was statistically significant heterogeneity ($\chi^2 = 174.46$; df = 44; $p < 0.00001$; $I^2 = 74.8\%$).

The effect size for each sub-group was:

- Low intensity group: N = 261; SMD = –0.93; 95% CI, –1.11 to –0.75
- Medium intensity group: N = 461; SMD = –1.44; 95% CI, –1.59 to –1.29
- High intensity group: N = 157; SMD = –1.65; 95% CI, –1.91 to –1.38.

There was significant heterogeneity within the medium intensity sub-group ($\chi^2 = 38.3$; df = 19; $p = 0.005$). Therefore, sensitivity analysis was used to examine the effect of removing outliers. By excluding Lindsay, 1997, heterogeneity was reduced ($\chi^2 = 29.9$; df = 18; $p = 0.04$), while the effect size remained similar (N = 452; SMD = –1.42; 95% CI, –1.57 to –1.27).

To examine whether number of therapist hours predicted treatment efficacy, a meta-regression analysis was performed controlling for the year of publication, study design (RCT or non-RCT), and treatment modality (individual or group). The number of therapist hours per client significantly predicted change in efficacy scores following treatment, z = –2.09; $p = 0.04$, after controlling for publication date, $z_{date} = 0.29$, $p = 0.77$, study design, $z_{study\ design} = –1.30$, $p = 0.19$, and treatment modality, $z_{treatment\ modality} = 0.93$, $p = 0.35$. When a sensitivity analysis was conducted by removing outliers (Lindsay, 1997), therapist hours still significantly predicted change in efficacy scores, z = –2.24, $p = 0.03$, after controlling for publication date, $z_{date} = 0.24$, $p = 0.81$, study design, $z_{study\ design} = –1.17$, $p = 0.24$, and treatment modality, $z_{treatment\ modality} = 0.82$, $p = 0.41$.

5.2.7.6 Limitations

There are important limitations to this review as (1) therapist time is only a proxy for treatment intensity, (2) therapist time is confounded with treatment format (group versus individual), (3) these studies were not designed to address this particular question, (4) although all studies included some degree of ERP, the exact content was unknown, and (5) while mean symptom severity in these studies is typically in the moderate range, it is not possible to link response to initial severity. Furthermore, as in almost all studies of CBT, there is generally insufficient control over the degree of patient adherence and particularly of the quantity of work conducted between sessions. Likewise therapist training, competence and adherence for each of the interventions are not adequately controlled. Little is known, as yet, about the effects of sequential CBT treatments that would arise in the proposed stepped care model. Properly designed prospective studies that examine all these factors are needed; in particular, the severity and complexity of OCD needs to be taken into account.

5.2.8 Clinical summary

As noted in the introduction, despite 35 years of research into the cognitive behavioural treatment of OCD, there are a limited number of RCTs that compare active treatments with controls. Those that exist indicate that clinician-guided ERP is an effective treatment for OCD. One study found that family-based behaviour therapy including ERP was superior to individual therapy. This study was conducted in India and these findings may not necessarily apply widely in the UK, although experts would certainly consider involving the family in many cases.

There is also some evidence for computerised ERP some indication that there should be scheduled support as fewer people left the study early when it was delivered with scheduled brief support sessions rather than support on request.

There is as yet little evidence for either cognitive therapy or CBT from RCTs against control conditions although the effects observed from head to head trials suggest that effects post-treatment are of a similar order to ERP. There is support for CBT (including ERP) for obsessive thoughts. Likewise, group CBT has been shown to be effective compared with waitlist control.

Currently there is no evidence showing that cognitive therapy is more or less effective than ERP alone. One study did find that adding cognitive therapy to ERP midway through treatment resulted in a better outcome at 6 months' follow-up. However, a second study found that group ERP was superior to group cognitive therapy at 12 months' follow-up. This question may never be answered in a convincing way as it is difficult to deliver two distinct, non overlapping-treatments. Many modern exposure-based treatments do include a high informational content and the strategies used to engage people in ERP can resemble cognitive therapy. Likewise, most current cognitive therapy explicitly seeks behaviour change but is not operating within a habituation paradigm. Although there is as yet no research on those who refuse, fail to engage with, or do not respond to ERP, cognitive therapy may yet have a role to play for these individuals, either as a new modality, or as a means of ultimately engaging them in ERP.

The review of therapist hours as an indicator of intensity revealed that the effect sizes (between pre- and post-treatment) for all three treatment intensity bands were large or greater (>0.8 SMD) although these are not controlled comparisons. Despite such effects, a proportion of patients in all three bands will not have benefited from treatment, and others will have shown a limited response and have residual symptoms that require further treatment, perhaps more likely in the low intensity band because of the smaller treatment effect. However, patients receiving more therapist hours of cognitive and behavioural intervention per patient were more likely to improve on OCD symptom severity compared with patients receiving fewer therapist hours. This effect was strongest when comparing patients receiving fewer than 10 therapist hours per patient with patients receiving more than 10 therapist hours per patient of psychological intervention. These findings together suggest that there may be benefit in more intensive forms of therapy over less intensive forms of therapy when calculated in terms of therapist hours per client.

Despite the preliminary nature of these findings and the limitations noted above, there are important implications for stepped care as less intensive therapies have a role to play, particularly in primary care and there may be benefits for some people receiving care in this setting rather than in secondary care or specialist services. There is also clearly a role for more intensive treatment, usually individual therapy, which may be found in increasingly specialist settings, especially for those for whom initial lower intensity interventions have proved inadequate.

5.3 PSYCHOANALYSIS

5.3.1 Introduction

Until the 1960s psychoanalysis was widely viewed as the treatment of choice for neurosis and so for all of the anxiety disorders including OCD. Psychoanalysis for OCD was heavily influenced by the work of Freud's 14 papers on the subject, including his classic case history of the 'Rat Man' (Freud, 1909). Freud's conceptualisation of obsessional phenomena focuses on anxiety derived from unresolved Oedipal conflicts resulting in anal-sadistic regression, which the ego fends off through defence mechanisms such as reaction formation, intellectualisation, undoing and isolation (Barth, 1990; Freud, 1909). Although some authors have expanded on, or offered other psychoanalytic formulations of OCD (Esman, 2001; Wells, 1990), the Freudian conceptualisation remains powerful today. As late as 2001 Burgy wrote: 'Attention is focused on the intrapsychological structure and conflicts, so that Freud's theory of an internal dependence on the superego instead of the external dependence on people around continues to prevail in obsessive compulsive neurosis' (Burgy, 2001).

Psychoanalysis focuses on the identification, clarification and alteration of the defence mechanisms that maintain the anxiety (Salzman, 1997). Treatment emphasises the relationship between therapist and patient and involves transference, counter-transference and interpretation (Salzman, 1983). Traditional psychoanalysis involves a highly trained practitioner who provides up to four sessions a week over a

period of up to several years although less frequent sessions and shorter courses of treatment may be offered.

Psychoanalysis for OCD remains a treatment option in parts of England and Wales where such services are available. However, over the last 3 decades psychoanalytic therapy has become less frequent as a treatment for OCD. This may be because the emergence and widespread acceptance of treatments such as specific pharmacological therapies and CBT for OCD have provided a range of treatment options for which there is an evidence base.

5.3.2 Studies considered for review

No systematic reviews or meta-analyses of the effectiveness of psychoanalysis for OCD were found therefore a narrative review was undertaken. Only papers written in English were considered and a total of 64 papers were reviewed (dating from 1912 to 2002). None of the papers considered for review was an RCT or cohort study and so the evidence reviewed consists of single case reports, a few case series, and theoretical reviews.

5.3.3 Descriptive review

Many of the articles describe single case reports detailing the nature of the analysis in both adults (Boehm, 2002; Cela, 1995; Finell & McDougall, 1985) and children (Fingert Chused, 1999; Karush, 1998). A few articles were found citing case series, though the maximum number of patients in these studies was three (Fingert Chused, 1999; Lang, 1997). Moreover, Lang (1997) used these cases to illustrate unconscious determinants rather than to report outcome. Some authors described group analytic treatment (Schwartz, 1972; Wells, 1990), but these case reports do not report outcome in any systematic way. A number of the single cases provided a description of ongoing analysis with a patient (Boehm, 2002; Deri, 1990; Willick, 1995), but did not provide any measure of outcome.

The single case reports that reported successful outcome (Chatterji, 1963; Parfitt, 1999) did not report clinical process or any measure of outcome. More importantly, the individualised nature of psychoanalytic interventions makes it almost impossible to replicate. Several of the articles and case reports acknowledge the limitations of psychoanalysis for OCD in terms of its ineffectiveness, and argue against its utility in integrating it with other interventions (Fingert Chused, 1999; Gabbard, 2001; Kay, 1996). For example, psychoanalysis has been used to help engage people with OCD to undertake other forms of treatment such as CBT (McCarter, 1997), and in conjunction with pharmacotherapy and behavioural treatments in both adults (Leib, 2001) and children (Gold-Steinberg & Logan, 1999). However, such combination treatments tell us little about the effect of each individual intervention, and the absence of outcome measures precludes any conclusions regarding treatment efficacy.

Despite generally poor outcomes many of the case reports reported intense analytic therapy ranging from one to four sessions per week over periods extending from 6 months to 19 years (Boehm, 2002; Cela, 1995; Juni, 1987). Esman (2001) sums up the evidence base: 'In our series of 21 patients who were collectively the recipients of more than one century of psychodynamic treatments, we had no reason to be … optimistic'.

5.3.4 Clinical summary

There is no evidence of efficacy or effectiveness for psychoanalysis in the treatment of OCD. Given the lack of evidence and the resources required for such intensive treatment, there is doubt as to whether it has a place in mental health services for OCD.

5.4 OTHER PSYCHOLOGICAL INTERVENTIONS

5.4.1 Introduction

Although the efficacy and effectiveness of CBT has been demonstrated, there are limits to its utility. Emmelkamp and Foa (1983) cited that 30% of OCD sufferers declined behaviour therapy while Kozak and colleagues (2000) reported during the course of their study that 40% of those who commenced behaviour therapy did not complete treatment. ERP can be unpleasant and distressing to clients and may lead to discontinuation of therapy. When taking into account those who refuse or drop out of treatment and those who do not benefit immediately or relapse, researchers have estimated that those treated successfully with behaviour therapy is around 55% (Stanley & Turner, 1995).

Although there is a strong evidence base for cognitive and behavioural therapies, people with OCD who are seeking help frequently indicate that they would like to be informed about a range of other treatments, their efficacy and availability. If first-line treatments are unavailable, have been unsuccessful, or discontinued due to distress or intolerable side effects, it may be useful for clinicians and people with OCD to know about other psychological interventions that could be beneficial.

5.4.2 Current practice

Current practice in the treatment of OCD using other psychological interventions and alternative/complimentary therapies is difficult to determine, as there is a paucity of literature. To date there has been one published RCT on an alternative therapy (yogic meditation) in the treatment of OCD in adults. No RCTs have been published on any of the other psychological interventions that have been used with OCD. Furthermore, no well-designed single case studies have been published on either other psychological interventions or alternative/complementary therapies. The literature is limited

and restricted to clinical case reports of one or more adults with OCD. Subsequently, the number of OCD clients treated using other psychological or alternative/complementary therapies is small, with minimal replication for any given approach.

Although CBT is available in many places in the NHS in England and Wales, people with OCD continue to be offered a range of other psychological treatments. There are a number of factors why this is likely to be the case. First, many psychological therapists are trained in modalities other than CBT and so would tend to offer the therapy in which they are trained. Second, some psychological therapists have a level of knowledge of a range of therapeutic modalities and choose an eclectic position. They would use elements of different therapies based on the way that they understand the person's particular problems and their view of what may be effective elements. Third, referrers to psychological services may choose to refer to particular therapeutic modalities based on their own training and understanding of the person with OCD. Finally, if choice is available, people with OCD may choose particular therapeutic modalities that either conform to their own understanding of their difficulties or because other options may appear too daunting or anxiety provoking.

5.4.3 Interventions included in the review

A number of commonly used treatments were considered for review (although the list is not exhaustive):

- Yogic meditation
- Hypnosis
- Homeopathy
- Marital therapy
- Transactional analysis
- Systemic therapy
- Integrated psychological approach
- Paradoxical intention
- Gestalt therapy
- Counselling
- Morita therapy
- Group cognitive analytic therapy
- Virtual reality therapy.

5.4.4 Studies considered for review

A systematic review of all relevant literature identified one RCT on yogic meditation (Shannahoff-Khalsa *et al.*, 1999) and 26 articles of clinical case reports without comparative treatments that described other psychological and alternative/complementary therapies in the treatment of OCD in adults (Becker & North, 1998; Churchill, 1986; Dormaar, 1987; Erickson, 1973; Fields, 1998; Gomez de Setien, 1982; Gomibuchi *et al.*, 2000; Hafner, 1982; Harvey & Green, 1990; Johnson &

Hallenbeck, 1985; Keiley, 2002; Mitzman & Duigan, 1993; Moore & Burrows, 1991; Morphy, 1980; Norland, 1988; Pelton, 1987; Pollard, 2001; Reichenberg & Ullman, 1998a; Reichenberg & Ullman, 1998b; Reichenberg & Ullman, 1999; Reichenberg & Ullman, 2000; Scrignar, 1981; Sheinberg, 1988; Stern, 1973; Walker, 1981; Yoder, 1994). The systematic review did not identify any literature for acupuncture, neuro-linguistic processing, reflexology or aromatherapy in the treatment of OCD in adults.

5.4.5 RCT of yogic meditation

Shannahoff-Khalsa and colleagues (1999) compared the efficacy of two yogic meditation protocols in the treatment of OCD: kundalini yoga meditation and relaxation response plus mindfulness meditation. The study was in two phases; the first a 3-month RCT, the second involving the groups merging and the efficacious protocol being employed for a further 12 months. Inclusion and exclusion criteria were determined prior to enrolment and a DSM-III-R diagnosis of OCD was established. The primary outcome measure used was the Y-BOCS. Groups were matched for age, sex and medication status before randomisation of group to therapy. Fourteen adults completed phase one, seven in each group. The authors reported that kundalini yoga meditation was an effective treatment for OCD when compared with baseline measures and also when compared with the alternative protocol. However, these published results should be viewed with caution as subsequent reanalysis with Review Manager (Cochrane Collaboration, 2004) failed to support these assertions apart from a scale measuring a construct labelled Purpose in Life.

5.4.6 Clinical case reports

5.4.6.1 Hypnosis

There were eight published articles on hypnosis; all were case studies reporting on one or more adults (Churchill, 1986; Dormaar, 1987; Erickson, 1973; Harvey & Green, 1990; Johnson & Hallenbeck, 1985; Moore & Burrows, 1991; Scrignar, 1981; Walker, 1981). Three of these case reports were not reviewed further because in two (Erickson, 1973; Walker, 1981) the diagnosis was obsessional personality and the third (Dormaar, 1987) did not include a DSM/ICD diagnosis or equivalent detailed clinical description of OCD. Three of the remaining case reports reported using a multifaceted approach combining hypnosis with one or more other treatment modalities, namely: conjoint family therapy (Churchill, 1986); the behavioural technique of flooding (Scrignar, 1981); relaxation, cognitive and behavioural strategies and pharmacotherapy (Moore & Burrows, 1991). Five of the clinical case reports involved only one client (Churchill, 1986; Harvey & Green, 1990; Johnson & Hallenbeck, 1985; Moore & Burrows, 1991; Walker, 1981), one of which involved the family in treatment (Churchill, 1986). Another article (Scrignar, 1981) reported on the treatment of two people.

Although these case reports generally reported improvement, their validity is severely restricted by the fact that they are uncontrolled case reports and have numerous methodological deficiencies such as a lack of standardised diagnoses, combined treatment modalities, and lack of recognised outcome measures.

5.4.6.2 Homeopathy

Five published articles on homeopathy were identified; three were clinical case reports of one adult (Norland, 1988; Reichenberg & Ullman, 1999; Reichenberg & Ullman, 1998b) and two were clinical case reports of one or more young persons (Reichenberg & Ullman, 1998a; Reichenberg & Ullman, 2000). One of the articles (Reichenberg & Ullman, 1999) also reported on the cases of three young persons. All of the adult case reports reported improvement in symptoms. However, it is difficult to draw meaningful conclusions as the numbers were small and the use of standardised diagnostic or outcome measures was not reported.

5.4.6.3 Marital/couple therapy

Two articles were identified on marital therapy (Hafner, 1982; Stern, 1973) and one on couple therapy (Keiley, 2002). All were clinical case reports of one or more adults. Hafner (1982) reported on the treatment of five inpatients using a multi-modal treatment programme: behaviour therapy, individual and group psychotherapy, conjoint marital and family therapy, social skills training and pharmacotherapy. Improvement in symptoms was reported during inpatient stay, however all clients relapsed on returning home. Stern and Marks (1973) reported on the treatment of a single case of obsessive compulsive neurosis with marital discord using contract therapy and Keiley (2002) described the use of affect regulation and attachment focused treatment with one OCD client and their partner. Again a variety of factors such as small numbers, other interventions, and lack of specified outcome measures of OCD symptoms hamper interpretation of these reports.

5.4.6.4 Transactional analysis

Two articles on transactional analysis were identified (Gomez de Setien, 1982; Pelton, 1987); both were primarily a description of the treatment technique. There was insufficient information on diagnosis, assessment and outcome of treatment to allow any conclusions about treatment effects.

5.4.6.5 Other therapies

Other case reports have appeared in the literature for psychological and alternative/complimentary therapeutic strategies, but no single technique has more than one case report. Those identified were systemic therapy (Sheinberg, 1988), an integrated psychological approach (Fields, 1998), paradoxical intention (Yoder, 1994), gestalt therapy (Morphy, 1980), counselling (Pollard, 2001), morita therapy (Gomibuchi *et al.*, 2000) and group cognitive analytic therapy (Mitzman & Duigan, 1993). Many of the difficulties encountered in the above reviews were found in these case reports thereby limiting any conclusions that could be drawn.

5.4.6.6 Virtual reality therapy

Becker and North (1998) described the development of the virtual reality therapy system (VRT-2002) for the treatment of various psychological disorders including OCD through exposure methods. Virtual reality therapy consists of using computer technology to support ERP by exposing the patient to a virtual representation of the environment that contains the feared situation rather than taking the patient into the actual environment or having the patient imagine the stimulus.

A brief description of one scene for OCD was provided and a second was said to be under development. Evaluation of the effectiveness and efficiency of VRT-2002 in the treatment of OCD has yet to be reported. Further, given the often idiosyncratic nature of concerns in OCD, it may be difficult to develop the required stimuli for more than a few subgroups of patients who have common concerns.

5.4.7 Clinical summary

The literature revealed very few clinical case reports of other psychological therapies in the treatment of OCD in adults. Those that were identified were primarily small in sample size, often including only one client. No clinical case reports used standardised outcome measures, although one report, (Fields, 1998), used a recognised validated measure at baseline. The largest number of clinical case reports identified for any one intervention was hypnosis: eight articles were identified and five reviewed. Although improvement was reported in these cases, in three cases other concurrent treatment was provided, leading to difficulty attributing causality. No comparative clinical case reports were identified.

There is insufficient evidence to support the use of other psychological therapies, hypnosis, or homeopathy therapies as routine treatments for the core features of OCD. This lack of evidence is in contrast with a much larger evidence base for cognitive and/or behavioural therapies although there are important limitations to the latter. Based on current evidence, ensuring access to adequate cognitive and/or behavioural therapies would currently appear to provide people with OCD with the best chance of improvement through psychological therapies.

5.5 PSYCHOLOGICAL INTERVENTIONS FOR CHILDREN AND YOUNG PEOPLE WITH OCD

5.5.1 Introduction

Several interventions have been used in the treatment of children and young people with OCD. These include behavioural interventions such as exposure, response prevention, flooding, extinction, shaping and operant techniques. Cognitive behavioural protocols have been developed that include behavioural techniques, as well as incorporating anxiety management, cognitive restructuring and parental involvement (March *et al.*, 1994). Finally, there have been several uncontrolled studies that have

used other therapeutic approaches, almost always in conjunction with behavioural techniques, that include systemic and psychodynamic approaches (Crago, 1995; O'Connor, 1983), individual psychotherapy (Warneke, 1985), insight-orientated therapy (Friedmann & Silvers, 1977; Willmuth, 1988), family therapy (Dalton, 1983; Goodman, 1988), hypnotic induction (Kellerman, 1981), social skills training (Hallam, 1974), and unspecified milieu therapy (Apter *et al.*, 1984).

Management and treatment is often complicated in children and young people with OCD as they frequently have other comorbid problems (Last & Strauss, 1989). For instance, in a survey of 70 children and young people with OCD, only 26% had OCD as their sole disorder (Swedo *et al.*, 1989). Frequently children and young people also present with secondary anxiety disorders, including generalised anxiety disorder, separation anxiety in younger children, and social anxiety in young people. Furthermore, a major complication of OCD in young people is the development of social avoidance and withdrawal from family and friends. Aggressive behaviour and temper tantrums can also be a management problem, and frequently occur when rituals are interrupted. Therefore the careful assessment and consideration of treating comorbid problems is necessary in clinical practice.

Although OCD in young people is similar to that found in adults, there are various developmental differences that are important to consider in the management and treatment of the young. Young children's obsessional thoughts are more likely to be characterised by 'magical' or superstitious thinking (e.g. 'If I don't count up to 20 my parents will die'). Treatment needs to take account of the child's developmental stage in order to engage them in a collaborative working relationship. Age-appropriate delivery may also include the use of metaphors in order to explain difficult psychological processes, for example the use of the 'white bear' experiment to explain the persistence of intrusive thoughts (Shafran, 1997). Researchers have also utilised game formats of ERP interventions in order to appeal to younger children (Moritz, 1998). Young children are also more orientated in the present than adults and may be less motivated to engage in difficult activities in order to achieve future positive changes.

Furthermore, the child's age-appropriate dependence on his or her family is a key difference between the presentation of children/young people and adults. As outlined in a study by Bolton and colleagues (1983), members of the family are almost always involved in the young person's rituals, although the nature and extent of involvement often varies. Involvement can range from the relatively mild, such as occasionally providing the child with reassurance, to the extreme where the parent is highly immersed in all of the child's rituals. The degree to which families can become involved with the child or young person's OCD can lead to difficulties in management and can affect treatment compliance. Frequently, one parent is more involved than the other and the parents may not work together as a team (Bolton *et al.*, 1983; Dalton, 1983). Some families react to their child's presentation of OCD by becoming critical and rejecting, alienating the child and adding to management difficulties. Parental behaviours may inadvertently reinforce and maintain the child's difficulties with OCD, and are often a source of family upset and discomfort. These factors have led to the frequent inclusion of family members in most of the CBT treatment protocols.

Family members are often provided with psychoeducation and may be encouraged to participate in some processes of therapy, such as ERP.

5.5.2 Current practice

Current practice for the treatment of young people with OCD varies widely according to professional orientation, training, and availability of resources. Multidisciplinary teams may offer a range of therapeutic interventions, often combining psychological and pharmacological treatments. CBT protocols for children and young people have been developed that can guide practitioners on the practice of cognitive and behavioural strategies with young people (March & Mulle, 1998). Most young people are treated on an outpatient basis unless the extent of their symptoms, distress and interference in their daily levels of functioning warrants an inpatient admission.

The aim of psychological treatment for children and young people with OCD is to reduce symptoms, distress and interference in daily functioning. A positive outcome would also include improved social, educational and family functioning. Treatment is further aimed at improving the young person's coping skills and teaching strategies to prevent future relapse.

OCD in children and young people has received relatively little empirical study compared with adult OCD, and many questions currently remain unanswered by the literature. To date there have been only two published randomised controlled trials of the psychological treatment of children and young people with OCD, and no systematic replication studies. Caution is needed when interpreting the results of the published studies as many have significant limitations that reduce the confidence that can be placed in their results. Although most studies report a large percentage of responders to treatment, this is often measured by different criteria across studies, and may not always represent clinically meaningful change. The participants often have a range of comorbid difficulties, may be receiving concurrent pharmacological treatment, or are receiving components of two or more treatment approaches. Furthermore, most studies are of adolescents, thus making it difficult to generalise to younger children. The methodological weaknesses found in most studies of childhood OCD indicate caution must be exercised in making statements about treatment efficacy.

5.5.3 Interventions included in the review

The contemporary psychological treatment approaches identified by the GDG and included in the review are:

- Behaviour therapy
- CBT
- Cognitive therapy
- Family therapies
- Psychoanalysis, psychoanalytic/psychodynamic/supportive/insight-orientated psychotherapy.

5.5.4 Studies considered for review

The review team conducted a new systematic search for RCTs that assessed the efficacy and tolerability of behavioural and cognitive therapies among children with OCD. The search identified one study (BARRETT2004).

The study compared individual cognitive behavioural family therapy (CBFT) with group CBFT and waitlist control. The duration of treatment was 14 weeks long, with 3 and 6 months' follow-up. The mean age of the participants was 12 years.

5.5.4.1 Individual CBFT versus waitlist control

Clinical evidence statements

Efficacy	***Included studies***
There is limited evidence suggesting a difference favouring individual CBFT over waitlist on reducing obsessive-compulsive symptoms as measured on the clinician-rated CY-BOCS (K = 1; N = 46; SMD = –2.73; 95% CI, –3.55 to –1.91). **I**	BARRETT2004
There is limited evidence suggesting a difference favouring individual CBFT over waitlist on improving family functioning as measured on the FAD mother's rating scale (K = 1; N = 32; SMD = –0.93; 95% CI, –1.67 to –0.19). **I**	BARRETT2004

5.5.4.2 Group cognitive behavioural family therapy versus waitlist control

Clinical evidence statements

Efficacy	***Included studies***
There is limited evidence suggesting a difference favouring group CBFT over waitlist on reducing obsessive-compulsive symptoms as measured on the CY-BOCS (K = 1; N = 53; SMD = –2.54; 95% CI, –3.28 to –1.81). **I**	BARRETT2004
There is limited evidence suggesting a difference favouring group CBFT over waitlist on reducing depressive symptoms as measured on the Childrens Depression Inventory (K = 1; N = 38; SMD = –0.78; 95% CI, –1.46 to –0.11). **I**	BARRETT2004
There is limited evidence suggesting a difference favouring group CBFT over waitlist on improving family functioning as measured on the FAD mother's rating scale (K = 1; N = 40; SMD = –0.78; 95% CI, –1.45 to –0.11). **I**	BARRETT2004

5.5.4.3 Individual CBFT versus group CBFT

Clinical evidence statements

Efficacy	***Included studies***
There is limited evidence suggesting a difference favouring group CBFT over individual CBFT on reducing anxiety as measured by the Multidimensional Anxiety Scale in Children (MASC) (K = 1; N = 42; SMD = 0.66; 95% CI, 0.03 to 1.28). **I**	BARRETT2004

5.5.5 Clinical summary

The only study to date suggests that CBT (including ERP) involving the family is effective in reducing OCD symptoms in both individual and group formats. There is some evidence to suggest that these treatments also improve family function. There is also evidence to suggest that group therapy is somewhat more effective than individual therapy in reducing the young person's anxiety.

5.5.6 Descriptive review

Sixty-nine articles were identified that described or investigated the psychological treatment of OCD in one or more children or young people. Of the 69 articles, 53 were direct clinical investigations.

Fifteen papers were clinical review articles or chapters of the general psychological treatment of OCD in children and young people (AACP, 1998; Albano & DiBartolo, 1997; Franklin *et al.*, 2003; Geffken *et al.*, 1999; King & Scahill, 1999; King *et al.*, 1998; March, 1995; March & Mulle, 1996; March & Mulle, 1998; March *et al.*, 2001; Piacentini, 1999; Rapoport *et al.*, 1993; Tolin & Franklin, 2002; Wolff & Wolff, 1991; Wolff & Rapoport, 1998).

Ten were open clinical trials involving 10 to 42 children. A protocol driven CBT manual based on the work of March and Mulle (1998) was used for several of the open clinical trials (Barrett *et al.*, 2003; March *et al.*, 1994), with one using a group format (Thienemann *et al.*, 2001). The protocol incorporated psychoeducation, anxiety management training (AMT), stimulus hierarchies, graded ERP, family participation and cognitive training such as thought stopping, constructive self-talk and cognitive restructuring. One study used a protocol developed by Piacentini and colleagues (2002), which involved ERP, behavioural rewards, cognitive restructuring and parental involvement. Two other studies have concentrated upon graded ERP parent sessions (Franklin *et al.*, 1998; Scahill *et al.*, 1996). One open clinical trial used an adolescent group format (Fischer *et al.*, 1998) based on a behavioural protocol developed by Krone and colleagues (1991). One used treatment-naïve children and adolescents (Benazon *et al.*, 2002). Finally, one open clinical trial compared CBT with medication (Wever & Rey, 1997). Methodologically, it is difficult to draw conclusions from these studies as to which components of the treatment package were the most effective ingredients of change. The results of the open trial studies

could also be affected by bias as none used assessors that were blind to the treatment conditions.

Three studies were case series of consecutively referred young people, involving six to ten cases, and standardised protocols. The first study (N = 10) investigated pharmacological and psychological treatment that included ERP and cognitive methods for coping with anxiety and challenging appraisals (Williams & Allsopp, 1999). The second (N = 7) investigated individual CBT based on the protocol developed by March and Mulle (1998) and a parallel parent skills training module (Waters *et al.*, 2001). The third (N = 6) investigated cognitive treatment, which included reappraising notions of responsibility (Williams *et al.*, 2002).

Nine were experimental single case designs involving one or more individuals, defined as any report that provides a quantitative baseline assessment plus either assessment across multiple symptom domains (multiple baseline design) or treatments (such as ABAB design) (Detweiler & Albano, 2001; Francis, 1988; Freeston, 2001; Green, 1980; Harris & Wiebe, 1992; Kearney & Silverman, 1990; Knox *et al.*, 1996; March & Mulle, 1995; Moritz, 1998).

Thirty-two papers were clinical case reports of one or more children. These are defined as having insubstantial descriptions of assessment, treatment, and/or outcome with little or no accompanying quantitative data. None of the studies measured multiple symptom domains (multiple baseline design) or treatments (such as ABAB design). Many of these studies combine two or more treatments, making it difficult to assess which approach was more effective. Two papers described consecutive cases of young people (eight and fifteen cases respectively) referred for treatment (Apter *et al.*, 1984; Bolton *et al.*, 1983), with one including a long-term follow-up (Bolton *et al.*, 1996). The following described one or more cases: (Bolton & Turner, 1984; Clark *et al.*, 1982; Crago, 1995; Dalton, 1983; Desmarais & Lavallee, 1988; Fine, 1973; Franklin *et al.*, 2001; Frare & Lebel, 1996; Friedmann & Silvers, 1977; Goodman, 1988; Hafner *et al.*, 1981; Hallam, 1974; Hand, 1988; Harbin, 1979; Kellerman, 1981; Morelli, 1983; O'Connor, 1983; Ong & Leng, 1979; Owens & Piacentini, 1998; Ownby, 1983; Piacentini *et al.*, 1994; Querioz *et al.*, 1981; Stanley, 1980; Tolin, 2001; Warneke, 1985; Weiner, 1967; Willmuth, 1988; Yamagami, 1978; Zikis, 1983).

5.5.6.1 Evidence for psychological interventions

What interventions have the best outcome as measured by reduction of symptoms?

Behaviour therapy

Almost all studies have used behavioural therapy interventions, even if they are combined with other psychological therapies. The most commonly used is graded ERP. Studies have also investigated flooding, extinction, operant techniques, modelling, shaping and pacing.

ERP

ERP is the most researched therapeutic intervention in child and adolescent OCD studies and appears to be the most promising. Out of the 53 studies analysed, more

than 30 reported using either exposure or response prevention, or both, indicating that ERP appears to be the treatment of choice. All of the open trials, two of the consecutive case series (Waters *et al.*, 2001; Williams & Allsopp, 1999), and all but two of the single case design studies (Francis, 1988; Franklin *et al.*, 2001) used ERP as part of their intervention packages. The CBT studies that have incorporated ERP have shown a range of outcomes from 87% of children rated as improved post-therapy (Bolton *et al.*, 1983) to more modest results of 25% mean reduction in symptoms on the CY-BOCS (Thienemann *et al.*, 2001). Two open clinical trials concentrated upon more of a behavioural invention, focusing on ERP with parental involvement. Franklin and colleagues (1998) reported a mean reduction on the CY-BOCS score of 67% at post-treatment and 62% at follow-up (Franklin *et al.*, 2003). Scahill and colleagues (1996) reported a mean post-treatment reduction of 61% on the CY-BOCS in a pilot study of behavioural therapy, ERP, and pharmacology, with a reduction of 51% at 3 months' follow-up. There has been one open clinical trial of group behaviour therapy, consisting of therapist assisted ERP and behavioural homework, which reported clinically significant improvement post-treatment and at 6 months' follow-up (Fischer *et al.*, 1998). There have been no open trials that have only used individual ERP. Taken together, the data from these studies is encouraging and points to the efficacy of ERP.

Extinction

Extinction is used to describe the techniques involved when the child or young person's OCD behaviours are being maintained by the verbal responses of others in the environment. There are seven case studies that have included extinction in their intervention. It has been mostly used in cases of compulsive questioning and reassurance seeking, where parents have been instructed not to provide reassurance. There has been one single case ABAB design study that used an extinction procedure to treat reassurance seeking behaviour. The study indicated that during the extinction phase reassurance-seeking behaviour remitted, and returned on the non-extinction phase. The results indicated that the child had completely stopped asking for reassurance at one month's follow-up, once extinction was re-employed (Francis, 1988). Similar positive results have been reported in a case report of extinction for reassurance seeking in a 5 year-old child (Tolin, 2001). Other case reports have used extinction together with other therapeutic interventions, making it difficult to assess the effectiveness of the extinction component. It has been used as part of family therapy interventions (Dalton, 1983; Fine, 1973; Morelli, 1983), with insight-orientated psychotherapy (Willmuth, 1988), and social skills approaches (Hallam, 1974). The studies have all reported positive treatment gains, but due to the lack of controlled studies and methodological weaknesses the use of extinction in child and adolescent OCD still remains unsubstantiated.

Other behavioural interventions

There are several case reports that have reported positive effects with other behavioural strategies. Three case reports have reported positive effects with modelling, shaping and pacing (Clark *et al.*, 1982; March & Mulle, 1998; Ong & Leng, 1979;

Warneke, 1985), mostly in the treatment of obsessional slowness in eating, grooming and washing. Only one case report used flooding, and this was in conjunction with graded ERP (Harris & Wiebe, 1992). A larger number of studies have acknowledged that operant techniques and behavioural rewards may play a positive role indirectly in creating change by helping the child attempt exposure, but again this is always used in combination with other treatment strategies and so is an adjunct to enhance other strategies rather than a strategy in its own right (Bolton *et al.*, 1983; Bolton & Turner, 1984; Dalton, 1983; Fine, 1973; Green, 1980; Ong & Leng, 1979; Owens & Piacentini, 1998; Piacentini *et al.*, 1994; Piacentini *et al.*, 2002; Querioz *et al.*, 1981; Warneke, 1985; Yamagami, 1978).

CBT

Out of the 53 intervention studies, 19 reported using a CBT intervention (Benazon *et al.*, 2002; Bolton & Turner, 1984; Detweiler & Albano, 2001; Franklin *et al.*, 2001; Freeston, 2001; Kearney & Silverman, 1990; Kellerman, 1981; March *et al.*, 1994; March & Mulle, 1995; Ownby, 1983; Piacentini *et al.*, 1994; Piacentini *et al.*, 2002; Scahill *et al.*, 1996; Thienemann *et al.*, 2001; Tolin, 2001; Waters *et al.*, 2001; Weiner, 1967; Wever & Rey, 1997; Williams & Allsopp, 1999). The open clinical trials that have used CBT protocols have incorporated sessions that include psychoeducation, anxiety management, cognitive training, behavioural rewards, ERP, as well as parental involvement. The cognitive element frequently included training in consecutive self-talk and positive coping strategies (March *et al.*, 1994). The CBT open clinical trials and case series studies have shown a range of outcomes with mean symptom reduction rates ranging from 25% (Thienemann *et al.*, 2001) to 79% (Piacentini *et al.*, 2002). The majority of studies have reported a mean symptom reduction on the CY-BOCS between 45 to 70% at post-treatment and at follow-up (Benazon *et al.*, 2002; Franklin *et al.*, 1998; March *et al.*, 1994; Scahill *et al.*, 1996; Waters *et al.*, 2001; Wever & Rey, 1997). Furthermore results from a small number of single case design studies have also yielded positive results for CBT (Detweiler & Albano, 2001; Freeston, 2001; Kearney & Silverman, 1990; March & Mulle, 1995).

The CBT protocols frequently incorporate anxiety management training (AMT), which often includes progressive muscle relaxation, diaphragmatic breathing and coping imagery. As this is often presented together with other behavioural strategies including ERP, it is difficult to judge whether AMT is effective or necessary in the treatment of child and adolescent OCD. There have been no studies that have just used AMT to treat OCD. One open trial has examined whether a simplified manualised CBT approach to ERP without AMT is effective (Franklin *et al.*, 1998). The results showed a mean CY-BOCS reduction of 67% at post-treatment and 62% at follow-up. From these results the authors argue that AMT is not a necessary component of CBT and may only serve to make ERP more accessible to younger people (the main active ingredient in treatment being ERP). Some clinicians have gone as far as arguing against the use of AMT as part of a first-line intervention for OCD (Tolin & Franklin, 2002), because intentionally eliciting anxiety through exposure work, whilst also learning strategies to minimise anxiety (through anxiety management), is

likely to confuse the young person, and at a theoretical level, may interfere with habituation to anxiety, the putative mechanism for therapeutic change in ERP. They acknowledge that AMT may be a useful adjunct to ERP if children are so anxious at baseline that they are unable to tolerate ERP (Tolin & Franklin, 2002). There is no evidence that AMT alone is effective in the treatment of children and young people with OCD.

Cognitive therapy

There has been little research that has specifically investigated cognitive therapy in children and young people with OCD. As cognitive therapy strategies are often combined with other treatment approaches in CBT protocols, it is difficult to judge how effective the cognitive components are, compared with ERP, in effecting change. Cognitive restructuring is aimed at challenging the child's thought processes by questioning the reality of his or her obsessions and the necessity of the compulsive behaviours, and it has been used in two studies that reported positive results (Kearney & Silverman, 1990; March & Mulle, 1995). Finally, several uncontrolled case studies specify teaching children positive self-statements (for example, 'I'm not afraid of germs, I can do this') to repeat during exposure (Willmuth, 1988; Zikis, 1983). Only one study has focused on utilising a more cognitive approach to treat a consecutive case series of six young people with OCD. The treatment protocol focused upon normalising intrusive thoughts, reappraising notions of responsibility, helping the young person re-evaluate the basis of their fears, and conducting behavioural experiments (Williams *et al.*, 2002). The results showed good clinical outcomes for the cases, with cognitive changes in responsibility appraisals being associated with clinical improvement. One single case study has used an alternating design of response prevention and cognitive therapy, with the cognitive element including identifying obsessional thinking, examining more realistic probabilities, and conducting behavioural experiments (Kearney & Silverman, 1990). The results indicated that the total average improvement in symptoms for both procedures was similar, and that the combination of the two treatments was found to be effective in eliminating OCD. Research into cognitive therapy with children and young people is still in its infancy, therefore the specific efficacy of cognitive therapy for the treatment of OCD in children and young people has yet to be proven.

Strategies for obsessive thoughts

In some of the open clinical trials, treatment protocols include other thought stopping and satiation. These interventions have also been used in various case reports. Thought stopping is intended to interrupt the occurrence of obsessive thoughts, and positive treatment effects have been reported by the few studies that have specifically implemented thought stopping in the treatment of OCD (Frare & Lebel, 1996; Friedmann & Silvers, 1977; Kellerman, 1981; Ownby, 1983), although three of these studies are more than 20 years old. Finally, satiation is produced by repeating obsessional thoughts, or replaying an audiotape of obsessions. Five investigations of OCD have reported positive effects when satiation techniques have been incorporated (Friedmann & Silvers, 1977; Green, 1980; Kellerman, 1981; O'Connor, 1983; Taylor,

1985). These approaches would now be considered a variant of exposure that may or may not have been accompanied with response prevention.

Therapies that target family function

There have been nine case reports that have incorporated family therapy into their treatment protocols with one or more children and young people. These have mostly described strategies aimed at altering the family system, and increasing communication and emotional expression within the young person's family. There have been two reports that have included procedures designed to alter family dynamics directly (Bolton *et al.*, 1983; Dalton, 1983). However most of the family therapy studies also include either acknowledged or unacknowledged behavioural components, including exposure, extinction and operant reinforcement (Dalton, 1983; Fine, 1973; Hafner *et al.*, 1981; Harbin, 1979; O'Connor, 1983). Two studies adopted systemic family therapy approaches where OCD was represented as a metaphor for family dysfunction (Dalton, 1983; O'Connor, 1983). These investigations encouraged ERP as a 'paradoxical intervention', making it impossible to determine how or whether the family intervention added to conventional, if implicit, cognitive behavioural approaches. Only two case reports focused more specifically on strategic therapy (Goodman, 1988) and marital and family therapy (Hand, 1988) specifically. One other case report described positive gains for a child with OCD by using a cognitive intervention aimed at decreasing angry cognitions in the mother (Morelli, 1983). Although the results of these reports all outline improvements in symptoms, they have methodological flaws; either in the specification of the treatment or in the measurement of OCD symptoms and/or family function. Consequently, the specific efficacy of these approaches has yet to be proven.

Psychoanalysis, psychoanalytic/psychodynamic/supportive/individual psychotherapy

There are seven reports of different forms of individual psychotherapy for children and young people with OCD that appear in the recent literature (Apter *et al.*, 1984; Bolton *et al.*, 1983; Crago, 1995; Friedmann & Silvers, 1977; O'Connor, 1983; Warneke, 1985; Willmuth, 1988). All of the studies used a theoretically eclectic combination of treatment approaches. Three of these reported unspecified 'milieu therapy' as an additional feature (Apter *et al.*, 1984; Bolton *et al.*, 1983; Friedmann & Silvers, 1977). Several included individual work, parent work, and group activities, frequently with behavioural interventions or narrative approaches (Crago, 1995). The studies all reported symptom reduction, but this is unsubstantiated as none uses standardised measures of outcome. To date the specific efficacy of these approaches for the treatment of OCD in children and young people has yet to be proven.

Are there developmental differences in the treatments most likely to achieve improvements in the identified outcomes for children (aged 8 –11 years) and young people (12–18 years)?

Most of the intervention studies have concentrated upon the adolescent age group (12–18 years). There have only been 13 studies describing children with one or more children aged 11 years and under (Desmarais & Lavallee, 1988; Fine, 1973; Francis,

1988; Frare & Lebel, 1996; Goodman, 1988; Knox *et al.*, 1996; March, 1995; Moritz, 1998; O'Connor, 1983; Querioz *et al.*, 1981; Stanley, 1980; Tolin, 2001; Waters *et al.*, 2001). These have highlighted the usefulness of CBT protocols, ERP and extinction with younger children. Several open clinical trials have used a range of ages, from 7–17 years, but analyses have not been conducted to ascertain whether there is a difference in treatment outcome dependent upon age. One study involved five children under the age of 11 years, and seven young people over the age of 11 years, and reported that 11 out of 12 did not meet criteria for OCD post-therapy (Barrett *et al.*, 2003), indicating that age did not appear to affect outcome. One study used a single subject cross-over design with four children aged 6–11 years. The researcher used a manualised game program to developmentally present psychoeducation and behavioural interventions. The results showed that OCD symptom severity decreased during treatment (Moritz, 1998). Preliminary evidence therefore suggests that CBT protocols may be equally accessible to younger children, but further research is needed.

What should the duration and intensity of the specified treatment be?

Most of the treatment trials have used weekly sessions, with CBT treatment protocols ranging from 12 to 22 sessions. There has been only one study that has compared intensive CBT sessions (18 sessions over 1 month) with weekly CBT sessions (16 sessions over 4 months). Children with daily sessions did not show superior outcomes to those with weekly sessions (Franklin *et al.*, 1998). However, methodological limitations, including lack of random assignment, restrict the confidence that can be placed in this finding. It is likely that the more severely affected children received daily ERP, whereas less severely ill children were given weekly treatment. One participant was written up as a case report, and the authors reported markedly reduced OCD symptoms after two evaluation sessions and 11 daily sessions (Franklin *et al.*, 2001), with the child falling in the sub-clinical range at post-therapy. A further study investigating a combined behavioural and pharmacological protocol offered on an intensive basis (10 daily sessions of CBT) showed a 68% remission rate and a 60% decrease in symptoms at 4 weeks (Wever & Rey, 1997). There are currently insufficient comparative studies to draw conclusions about the effectiveness of more intensive treatment approaches.

What is the most effective format for treating children and young people with OCD?

Very few studies in the literature have taken a purely individual format of treatment. The large majority of studies combine individual therapy with some family sessions aimed at parent skills training. All of the open case trials and CBT protocols include parent sessions in order to provide psychoeducation, build problem-solving skills, teach strategies to reduce family involvement in the OCD, and encourage family support. All reported positive effects by involving parents in therapy. Parents are often involved by assisting in the between-session exposure sessions and with ensuring treatment adherence. Only one study to date has attempted to empirically investigate the role of involving parents in CBT

protocols. Knox and colleagues (1996) used a staggered baseline design to assess whether the addition of active parental participation to ERP would improve the effectiveness of the treatment. The results indicate that children reported less distress associated with their rituals (decreased SUDS ratings) when their parents were involved in therapy and were taught to ignore their compulsions (Knox *et al.*, 1996). One single case design study (Francis, 1988), and one case report (Tolin, 2001), found that extinction, practiced by the parents, was effective in decreasing compulsive reassurance seeking.

One RCT investigated group formats of CBT and showed no difference between individual and group formats (Barrett *et al.*, 2003). Two open clinical trials investigated group formats of CBT treatment with young people (Fischer *et al.*, 1998; Thienemann *et al.*, 2001). The first study was an open trial of behavioural group therapy with 15 young people, concentrating on ERP. Fischer and colleagues reported that all participants showed significant improvements on the CY-BOCS at post-treatment and at 6 months' follow-up. The second study investigated a CBT group that incorporated ERP with cognitive therapy with 18 children. Thienemann and colleagues reported a mean CY-BOCS reduction of 25%.

Preliminary results indicate that group formats of treatment may be an effective format of treatment, but both studies also incorporated parent sessions. From the other studies it is difficult to judge the treatment effects of group format of treatment, compared with the content of treatment, but involving parents in the child's therapy seems to be the treatment of choice.

5.5.7 Clinical summary

- Research evidence from one RCT, open clinical trials, case series, single case studies and case reports all point to the efficacy of CBT, which incorporates ERP, in the treatment of OCD in children and young people.
- In terms of format of treatment, there is evidence to suggest that involving parents in the treatment of their children, especially in CBT protocols incorporating ERP, is linked to good outcome.
- Limited research indicates that there is no difference between intensive and weekly sessions, but further research is needed to substantiate these findings.
- Although cognitive therapy may have some utility, to date the lack of outcome studies makes it difficult to draw definite conclusions about its effectiveness.
- Anxiety management training is often included in CBT protocols, but there is little evidence to point to its direct treatment effect for OCD for young people.
- For compulsive questioning and reassurance seeking, some studies suggest that extinction may be beneficial, but its use for other forms of OCD remains unsubstantiated.
- There is no evidence for the use of modelling, shaping and pacing in the treatment of OCD.
- Many studies include the use of operant rewards as an adjunct to therapy, alongside ERP interventions, to increase the child's motivation in therapy.

- There is currently no evidence that treatments aimed at changing family functioning can in themselves bring about an improvement in OCD symptoms among children with OCD.
- There is no evidence to suggest that psychotherapy approaches (psychodynamic, insight-oriented) are effective in the treatment of OCD.
- In terms of developmental factors, lack of research makes it difficult to ascertain whether there are differences in treatment outcomes for children under the age of 11 years, compared with young people aged over 11 years of age. However, the incorporation of younger children in several open clinical trials indicates that CBT protocols appear to be equally accessible when adapted to younger children.

5.6 PSYCHOLOGICAL INTERVENTIONS FOR PEOPLE WITH BDD

5.6.1 Introduction

The cognitive and behavioural manifestations of BDD resemble, at least superficially, those found in other disorders as do the social anxiety and avoidance that are commonly associated with BDD. Consequently, as for other disorders with these types of features, a variety of psychological approaches have been attempted or developed.

5.6.2 Current practice

There are no surveys on what psychological interventions are used for BDD in the UK or what proportion of patients with BDD receives a psychological treatment. Many individuals with BDD have difficulty accepting a psychological or pharmacological intervention and prefer to either avoid or camouflage their appearance, alternatively they seek a cosmetic or dermatological procedure. Few mental health professionals have clinical experience in treating many patients with BDD.

Current practice is not underpinned by a strong evidence base. There are few studies upon which to base clinical decisions and doubts about the generalisability of research findings to people encountered in practice who may refuse to participate in therapy.

5.6.3 Interventions included in the review

The following interventions were included:
- CBT
- Behaviour therapy
- Cognitive therapy.

5.6.4 Studies considered for the review

The review team conducted a new systematic search for studies that examined psychological interventions in BDD. Two RCTs were identified.

Rosen and colleagues (1995) conducted an RCT of group CBT in 54 participants with BDD. Results indicated that 81.5% of the 27 patients were clinically improved after treatment. Treatment involved a small group format for an 8-week period. Therapy sessions consisted of education about causation and treatment of BDD, constructing a hierarchy of distressing aspects of their appearance, homework assignments involving exposure to anxiety provoking situations and preventing body checking behaviours, as well as keeping a body image diary. The participants in this study were different from those described in other centres, as they were less severely impaired by BDD, they were all female and the most common preoccupation was their weight and shape. However they did not have a diagnosable eating disorder.

Veale and colleagues (1996b) conducted an RCT of CBT in 19 participants who were predominantly female but more severely impaired than those in the Rosen and colleagues study. There was a 50% reduction in symptoms on the main outcome measure (Y-BOCS, modified for BDD). The emphasis in the therapy was helping the individual to have a good psychological understanding of the factors that maintained the symptoms, behavioural experiments to test out an alternative theory, exposure to situations avoided and dropping of excessive safety behaviours and rituals.

5.6.5 Descriptive review

Older RCTs and case series on body image therapy were excluded from the meta-analysis or narrative review as these were for body dissatisfaction and not body dysmorphic disorder or dysmorphophobia (Butters & Cash, 1987; Rosen *et al.*, 1989).

Neziroglu and Yaryura-Tobias (1993b) reported on the use of exposure and response prevention and cognitive therapy in five individuals with BDD and OCD. Participants were not on any medication and received either weekly or daily 90-minute sessions for 4 to 12 weeks. One individual dropped out and the other four showed significant improvement on observer rated measures. Results suggest that intensive sessions, more than once a week, seem to provide the greatest gains.

McKay and colleagues (1997) evaluated a maintenance follow-up programme for individuals with BDD after CBT. Individuals were contacted bi-weekly for assessment with all measures for a total of 6 months. All subjects were assessed at the follow-up and all had remained symptom free. Patients in the maintenance group, however, had continued to improve on measures of anxiety and depression and showed significantly lower levels of anxiety and depression at follow-up. McKay (1999) followed up these patients 2 years' later and noted treatment gains were maintained.

Wilhelm and colleagues (1999) evaluated group CBT in BDD. It led to significant improvement in both BDD and depressive symptoms. Participants received weekly 90-minute group CBT including psychoeducation, self-monitoring, cognitive restructuring, ERP, and scheduling of pleasant events and achievement oriented activities.

Psychological interventions

Geremia and Neziroglu (2001) investigated the role of cognitive restructuring. Four individuals with BDD were treated in a single-subject multiple baseline design in which each patient served as his/her own control. Treatment consisted of 7 weeks of 75-minute sessions twice a week for cognitive treatment followed by 3 weeks of follow-up data gathering. Results indicated that cognitive therapy resulted in statistically significant reductions in BDD symptoms, depression and anxiety in three out of the four patients. Minimal improvement was seen with overvalued ideation. In this study, no behavioural assignments were given but the authors suggest that this may enhance treatment efficacy.

There are also several older case reports or case series on the successful use of behaviour therapy by Munjack (1978), Solyom and colleagues (1985), Campisi (1995), Watts (1990), Marks and Mishan (1988) and Gomez-Perez and colleagues (1994). Some of the cases in the latter two were also being treated with medication. There also case reports of CBT by Schmidt and Harrington (1995) and Neziroglu and colleagues (1996) and descriptions of the addition of reverse role-play to behaviour therapy by Newell and Shrubb (1994) and Cromarty and Marks (1995). Vitiello and DeLeon (1990) reported one unsuccessful case after many years of psychoanalysis and then behaviour therapy with medication. Eye movement desensitisation and reprocessing (EMDR) resulted in improvement in six out of seven cases (Brown *et al.*, 1997). There is one case report describing the use of psychodynamic psychotherapy (Bloch & Glue, 1988). A review and summary of the literature in cognitive behavioural treatments for BDD is provided by Neziroglu and Khemlani-Patel (2002).

Children and young people with BDD

There are no RCTs of any psychological interventions in children and young people with BDD. There is one successful case report of behaviour therapy (Braddock, 1982); one successful case report of behaviour therapy combined with doxepine (Sobanski & Schmidt, 2000); one of multiple treatment modalities (psychodynamic therapy, CBT, family therapy and medication) (Horowitz *et al.*, 2002) and one of psychodynamic therapy (Philippopulis, 1979).

5.6.6 Clinical summary

There is some evidence from two RCTs and several case series on the benefit of CBT in adults with BDD. Little is known about the optimum frequency, type or duration of the therapy or the rate of relapse in the long term. One case report and expert opinion suggest that optimal therapy may need more intensive sessions (e.g. more than once a week especially in the early stages). The average duration of outpatient therapy may need to be slightly longer than other disorders (e.g. 20 to 25 sessions). Some evidence exists on the benefit of group CBT but this has not been compared with individual CBT or a combination of the two. There is virtually no evidence on psychological interventions in young people with BDD.

5.7 CLINICAL PRACTICE RECOMMENDATIONS

5.7.1 Interventions for people with OCD or BDD

The treatments for OCD and BDD that are effective should be offered at all levels of the healthcare system. The difference in the treatments at the higher levels will reflect increasing experience and expertise in the implementation of a limited range of therapeutic options. For many people, initial treatment may be best provided in primary care settings. However, people with more impaired functioning, higher levels of comorbidity, or poor response to initial treatment will require care from teams with greater levels of expertise and experience in the management of OCD or BDD. Regardless of the level of care or the level of expertise, all professionals offering psychological treatments should have received appropriate training in the interventions they are offering. Furthermore, to ensure safe practice it is essential that they receive ongoing clinical supervision. The organisation and provision of training and clinical supervision should follow the recommendations found in Organising and Delivering Psychological Therapies (Doh, 2004).

5.7.1.1 All healthcare professionals offering psychological treatments to people of all ages with OCD or BDD should receive appropriate training in the interventions they are offering and receive ongoing clinical supervision in line with the recommendations in Organising and Delivering Psychological Therapies (Doh, 2004). **[GPP]**

Initial treatment options

Effective treatments for OCD and BDD should be offered at all levels of the healthcare system. The difference in the treatments at the higher levels will reflect increasing experience and expertise in the implementation of a limited range of therapeutic options. For many people, initial treatment may be best provided in primary care settings. However, people with more impaired functioning, higher levels of comorbidity, or poor response to initial treatment will require care from teams with greater levels of expertise and experience in the management of OCD or BDD.

Irrespective of the level of care, the following recommendations should be taken into account when selecting initial treatments for people with OCD or BDD. The specific recommendations as to how to provide these treatments follow in the subsequent sections.

Regulatory authorities (including the Medicines and Healthcare products Regulatory Agency[6]) have identified that the use of SSRIs to treat depression in children and young people may be associated with the appearance of suicidal behaviour, self-harm or hostility, particularly at the beginning of treatment. There is no clear evidence of an increased risk of self-harm and suicidal thoughts in

[6]www.mhra.gov.uk/.

young adults aged 18 years or older. But individuals mature at different rates and young adults are at a higher background risk of suicidal behaviour than older adults. Hence, young adults treated with SSRIs should be closely monitored as a precautionary measure. The Committee on Safety of Medicine's Expert Working Group on SSRIs, at a meeting in February 2005, advised that it could not be ruled out that the risk of suicidal behaviour, hostility and other adverse reactions seen in the paediatric depression trials applies to use in children or young people in all indications. Consequently, the recommendations about the use of SSRIs for people with OCD or BDD have taken account of the position of regulatory authorities.

Adults

In the current regulatory context, offer adults with OCD with milder impairments low intensity CBT first, reserving higher intensity CBT and specific drug treatments for those with greater impairment. The intensity of psychological treatment has been defined as the hours of therapist input per patient. By this definition most group treatments meet the definition of a low intensity treatment (<10 hours therapist input per patient), although each patient may be receiving a much greater number of hours of therapy.

5.7.1.2 In the initial treatment of adults with OCD, low intensity psychological treatments (including ERP) (up to 10 therapist hours per patient) should be offered if the patient's degree of functional impairment is mild and/or the patient expresses a preference for a low intensity approach. Low intensity treatments include:

- brief individual CBT (including ERP) using structured self-help materials **[C]**
- brief individual CBT (including ERP) by telephone **[C]**
- group CBT (including ERP) (note, the patient may be receiving more than 10 hours of therapy in this format). **[C]**

5.7.1.3 Adults with BDD with mild functional impairment should be offered a course of CBT (including ERP) that addresses key features of BDD in individual or group formats. The most appropriate format should be jointly decided by the patient and the healthcare professional. **[B]**

Children and young people

In the current regulatory context regarding prescribing SSRIs (see above under 'initial treatment options'), offer children and young people with OCD or BDD psychological treatments first. However, if a child/young person and/or their family are unable to engage in psychological treatment or it is declined, an SSRI may be cautiously considered with specific arrangements for careful monitoring of adverse events.

5.7.1.4 For children and young people with OCD with mild functional impairment, guided self-help may be considered in conjunction with support and information for the family or carers. **[C]**

5.7.1.5 Children and young people with OCD with moderate to severe functional impairment, and those with OCD with mild functional impairment for whom guided self-help has been ineffective or refused, should be offered CBT (including ERP) that involves the family or carers and is adapted to suit the developmental age of the child as the treatment of choice. Group or individual formats should be offered depending upon the preference of the child or young person and their family or carers. **[B]**

5.7.1.6 All children and young people with BDD should be offered CBT (including ERP) that involves the family or carers and is adapted to suit the developmental age of the child or young person as first-line treatment. **[C]**

5.7.1.7 The co-existence of comorbid conditions, learning disorders, persisting psychosocial risk factors such as family discord, or the presence of parental mental health problems, may be factors if the child or young person's OCD or BDD is not responding to any treatment. Additional or alternative interventions for these aspects should be considered. The child or young person will still require evidence-based treatments for his or her OCD or BDD. **[C]**

How to use psychological interventions for adults

Cognitive behavioural treatments involving ERP are effective treatments for OCD and BDD. The format and delivery of such therapy should take into account specific features of problems experienced by the person with OCD or BDD and the interventions should be adapted accordingly.

5.7.1.8 For adults with obsessive thoughts who do not have overt compulsions, CBT (including exposure to obsessive thoughts and response prevention of mental rituals and neutralising strategies) should be considered. **[B]**

5.7.1.9 For adults with OCD, cognitive therapy adapted for OCD may be considered as an addition to ERP to enhance long-term symptom reduction. **[C]**

5.7.1.10 For adults with OCD living with their family or carers, involving a family member or carer as a co-therapist in ERP should be considered where this is appropriate and acceptable to those involved. **[B]**

5.7.1.11 For adults with OCD with more severe functional impairment who are housebound, unable or reluctant to attend a clinic, or have significant problems with hoarding, a period of home based treatment may be considered. **[C]**

5.7.1.12 For adults with OCD with more severe functional impairment who are housebound and unable to undertake home-based treatment because of the nature of their symptoms (such as contamination concerns or hoarding that prevents therapists' access to the patient's home), a period of CBT by telephone may be considered. **[C]**

5.7.1.13 For adults with OCD who refuse or cannot engage with treatments that include ERP, individual cognitive therapy specifically adapted for OCD may be considered. **[C]**

5.7.1.14 When family members or carers of people with OCD or BDD have become involved in compulsive behaviours, avoidance or reassurance seeking, treatment plans should help them reduce their involvement in these behaviours in a sensitive and supportive manner. **[GPP]**

5.7.1.15 Adults with OCD or BDD with significant functional impairment may need access to appropriate support for travel and transport to allow them to attend for their treatment. **[GPP]**

5.7.1.16 Towards the end of treatment, healthcare professionals should inform adults with OCD or BDD about how the principles learned can be applied to the same or other symptoms if they occur in the future. **[GPP]**

5.7.1.17 When adults with OCD request forms of psychological therapy other than cognitive and/or behavioural therapies as a specific treatment for OCD (such as psychoanalysis, transactional analysis, hypnosis, marital/couple therapy) they should be informed that there is as yet no convincing evidence for a clinically important effect of these treatments. **[C]**

How to use psychological interventions for children and young people

Psychological treatments for children and young people should be collaborative and engage the family. Always consider the wider context and the other professionals involved with the child. Rewards to encourage the child can be helpful. When working with young people, the recommendations on the use of psychological interventions for adults may also be considered when appropriate.

5.7.1.18 In the cognitive-behavioural treatment of children and young people with OCD or BDD, particular attention should be given to:

- developing and maintaining a good therapeutic alliance with the child or young person as well as their family or carers
- maintaining optimism in both the child or young person and their family or carers
- collaboratively identifying initial and subsequent treatment targets with the child or young person
- actively engaging the family or carers in planning treatment and in the treatment process, especially in ERP where, if appropriate and acceptable, they may be asked to assist the child or young person
- encouraging the use of ERP if new or different symptoms emerge after successful treatment
- liaising with other professionals involved in the child or young person's life, including teachers, social workers and other healthcare professionals, especially when compulsive activity interferes with the ordinary functioning of the child or young person
- offering one or more additional sessions if needed at review appointments after completion of CBT. **[GPP]**

5.7.1.19 In the psychological treatment of children and young people with OCD or BDD, healthcare professionals should consider including rewards in order to enhance their motivation and reinforce desired behaviour changes. **[C]**

6. PHARMACOLOGICAL INTERVENTIONS

6.1 CURRENT PRACTICE

The guidelines of the Expert Consensus Panel for OCD present specific judgements on a comprehensive range of issues relating to pharmacological and psychological treatments (March *et al.*, 1997). Although the guidelines did not distinguish between clomipramine and SSRIs, improved tolerability of the latter was acknowledged. Combined CBT and medication was preferred by experts in terms of speed, efficacy, durability, tolerability and acceptability, and was thought the best approach for most patients. More recently a smaller group of members of the World Council on Anxiety met to agree recommendations for long-term treatment. They emphasised the importance of continuing treatment long-term from the outset and recommended 1–2 years continuation in treatment-responsive individuals (Greist *et al.*, 2003).

OCD responds to drug treatment in a characteristically slow, gradual way and improvements can take many weeks and months to develop. Patients often need to be encouraged to persevere in the early stages when progress can seem frustratingly slow. Dose titration is usually recommended, with patients remaining at the lowest effective dose levels for several weeks and reassessed before gradually increasing up to the maximum licensed doses according to observed efficacy and tolerability.

There remains a paucity of data on long-term outcome, but the studies that have been performed suggest that the SRIs remain effective for as long as they are continued, and continuation protects against relapse. There is no convincing evidence supporting dose-reduction in the longer-term, and the adage ‘the dose that gets you well keeps you well’ probably applies.

6.2 SSRIs

6.2.1 Treatments included

The following treatments were included:

- Citalopram
- Fluoxetine
- Fluvoxamine
- Paroxetine
- Sertraline

All the above compounds with the exception of citalopram have Marketing Authorisation for the treatment of OCD in the UK.

6.2.2 Studies considered[7]

The review team conducted a new systematic search for RCTs that assessed the efficacy and tolerability of SSRIs among adults with OCD. Fifty-five studies were identified, of which 20 did not meet the inclusion criteria of the GDG. The 33 published trials plus one unpublished trial (Burnham) provided efficacy data from 4102 participants and tolerability data from 4907 participants.

Of the included studies, 18 compared SSRIs with placebo (BEASLEY1992; BURNHAM, CHOUINARD1990; GOODMAN1989; GOODMAN1996; GREIST1995A; HOLLANDER2003B; HOLLANDER2003D; JENIKE1990A; JENIKE1990B; JENIKE1997; KAMIJIMA2004; KRONIG1999; MALLYA1992; MONTGOMERY1993; MONTGOMERY2001; PERSE1987; ZOHAR1996A), with two of these studies (BEASLEY1992; MONTGOMERY1993) featuring an extension phase involving participants classified as responders on completion of the acute phase of treatment.

Six studies examined the effects of different doses of SSRI (BEASLEY1992; BOGETTO2002; GREIST1995A; HOLLANDER2003D; MONTGOMERY1993; MONTGOMERY2001), with two of these studies (BEASLEY1992; MONTGOMERY1993) featuring an extension phase involving participants classified as responders on completion of the acute phase of treatment.

Two studies compared SSRIs with other SSRIs (BERGERON2002; MUNDO1997a), 10 with clomipramine (ASKIN1999; BISSERBE1997; BURNHAM, FREEMAN1994; KORAN1996A; LOPEZ-IBOR1996; MILANFRANCHI1997; MUNDO2001; SMERALDI1992; ZOHAR1996A) and four with other drugs including desipramine (GOODMAN1990a; HOEHN-SARIC2000), phenelzine (JENIKE1997) and venlafaxine (DENYS2003a).

All 32 acute phase studies (ASKIN1999; BEASLEY1992; BERGERON2002; BISSERBE1997; BOGETTO2002; BURNHAM, CHOUINARD1990; DENYS2003A; FREEMAN1994; GOODMAN1989; GOODMAN1990A; GOODMAN1996; GREIST1995A; HOEHN-SARIC2000; HOLLANDER2003B; HOLLANDER2003D; JENIKE1990A; JENIKE1990B; JENIKE1997; KAMIJIMA2004; KORAN1996A; KRONIG1999; LOPEZ-IBOR1996; MALLYA1992; MILANFRANCHI1997; MONTGOMERY1993; MONTGOMERY2001; MUNDO1997A; MUNDO2001; PERSE1987; SMERALDI1992; ZOHAR1996A) were between 8 and 16 weeks long (mean length = 10.96 weeks). Where the treatment duration was greater than 16 weeks (BERGERON2002), efficacy data was extracted at the 12-week time-point. Participants were classified as outpatient in 18 studies, inpatient in one study and unclear in 13 studies. The average age of the patients in the acute phase studies was 36 years. The average duration of illness in 20 studies was 13.72 years. Although ten studies included patients with comorbid depression, the remaining two-thirds of the studies excluded individuals with comorbid depression, so the trials were testing the effect specifically for obsessive-compulsive symptoms. Ten studies were multi-centre trials, of which three were conducted in the US and six in Europe. Fourteen other studies were conducted in the US, three in Italy, one each in Austria, Canada, Japan, the Netherlands and the UK.

[7]Here and elsewhere in the guideline, each study considered for review is referred to by a study ID (primary author and date of study publication in capital letters, except where a study is *in press* or unpublished, then a date is not used).

Full details of the studies included in the guideline and the reasons for excluding studies are given in Appendix 16.

6.2.3 SSRIs versus placebo

6.2.3.1 Clinical evidence statements[8]

Efficacy[9]	***Included studies***
There is evidence suggesting a difference favouring SSRIs over placebo on the likelihood of treatment response, defined as a 25% + or 35% + reduction on the clinician-rated Y-BOCS and/or CGI 'much improved' or 'very much improved' (K = 10; N = 2588; RR = 0.77; 95% CI, 0.73 to 0.82). **I**	BEASLEY1992 BURNHAM GREIST1995A HOLLANDER2003B HOLLANDER2003D KAMIJIMA2004 MALLYA1992 MONTGOMERY1993 MONTGOMERY2001 ZOHAR1996A
There is limited evidence suggesting a difference favouring fluvoxamine over placebo on the likelihood of remission, defined as a score of 8 or less on the clinician-rated Y-BOCS (K = 1; N = 253; RR = 0.90; 95% CI, 0.82 to 0.98). **I**	HOLLANDER2003B
There is limited evidence suggesting a difference favouring SSRIs over placebo on reducing obsessive-compulsive symptoms as measured by the clinician-rated Y-BOCS (K = 12; N = 1629; SMD = –0.42; 95% CI, –0.53 to –0.31). **I**	BEASLEY1992 BURNHAM GOODMAN1989 GOODMAN1996 HOLLANDER2003B JENIKE1990A JENIKE1990B JENIKE1997 KAMIJIMA2004 MONTGOMERY1993 MONTGOMERY2001 ZOHAR1996A

[8]The full list of all evidence statements generated from meta-analyses (and the associated forest plots) are on the CD-ROM that accompanies the guideline.

[9]In the case of SMDs, negative effect sizes favour the treatment group.

There is limited evidence suggesting a difference favouring SSRIs over placebo on reducing depressive symptoms as measured by the Montgomery-Asberg Depression Rating Scale (K = 3; N = 608; SMD = –0.28; 95% CI, –0.44 to –0.11). **I**	MONTGOMERY1993 MONTGOMERY2001 ZOHAR1996A
There is limited evidence suggesting a difference favouring paroxetine over placebo on reducing the severity of illness as measured by the CGI Severity of Illness subscale (K = 1; N = 293; SMD = –0.36; 95% CI, –0.61 to –0.06). **I**	ZOHAR1996A
There is limited evidence suggesting a difference favouring citalopram over placebo on reducing impairment of family life as measured by the Sheehan Disability Scale family subscale (K = 1; N = 203; SMD = –0.33; 95% CI, –0.61 to –0.06). **I**	MONTGOMERY2001
There is limited evidence suggesting a difference favouring citalopram over placebo on reducing impairment of social life as measured by the Sheehan Disability Scale social subscale (K = 1; N = 203; SMD = –0.33; 95% CI, –0.61 to –0.05). **I**	MONTGOMERY2001
There is limited evidence suggesting a difference favouring citalopram over placebo on reducing impairment of work as measured by the Sheehan Disability Scale work subscale (K = 1; N = 203; SMD = –0.35; 95% CI, –0.63 to –0.08). **I**	MONTGOMERY2001
There is limited evidence suggesting a difference favouring SSRIs over placebo on the likelihood of treatment response, defined as a 25% + or 35% + reduction on the clinician-rated Y-BOCS and/or CGI 'much improved' or 'very much improved' in patients with comorbid depression (K = 3; N = 763; SMD = –0.42; 95% CI, –0.53 to –0.31). **I**	BEASLEY1992 KAMIJIMA2004 MONTGOMERY1993
There is limited evidence suggesting a difference favouring SSRIs over placebo on reducing obsessive-compulsive symptoms as measured by the clinician-rated Y-BOCS in patients with comorbid depression (K = 4; N = 489; SMD = –0.58; 95% CI, –0.76 to –0.4). **I**	BEASLEY1992 GOODMAN1989 KAMIJIMA2004 MONTGOMERY1993

Tolerability	BURNHAM
There is limited evidence suggesting that SSRIs when compared with placebo increase the risk of reporting adverse effects (K = 10; N = 1786; RR = 1.16; 95% CI, 1.1 to 1.23). **I**	GOODMAN1996 GREIST1995A JENIKE1990A JENIKE1990B KAMIJIMA2004 KRONIG1999 MALLYA1992 MONTGOMERY2001 ZOHAR1996A
There is evidence suggesting that there is unlikely to be a clinically important difference between SSRIs and placebo on the likelihood of leaving the study early (K = 16; N = 2623; RR = 0.98; 95% CI, 0.85 to 1.13). **I**	BURNHAM CHOUINARD1990 GOODMAN1989 GOODMAN1996 GREIST1995A HOLLANDER2003B HOLLANDER2003D JENIKE1990A JENIKE1990B JENIKE1997 KRONIG1999 MALLYA1992 MONTGOMERY1993 MONTGOMERY2001 PERSE1987 ZOHAR1996A
There is evidence suggesting that SSRIs when compared with placebo increase the risk of leaving the study early due to adverse effects (K = 13; N = 3009; RR = 2.15; 95% CI, 1.62 to 2.86). **I**	BEASLEY1992 BURNHAM CHOUINARD1990 GOODMAN1989 GOODMAN1996 GREIST1995A HOLLANDER2003B HOLLANDER2003D KAMIJIMA2004 KRONIG1999 MONTGOMERY1993 MONTGOMERY2001 ZOHAR1996A

6.2.4 Different doses of SSRIs

6.2.4.1 Clinical evidence statements

Efficacy	***Included studies***
There is limited evidence suggesting a difference favouring 20 mg of an SSRI (citalopram/fluoxetine/paroxetine) over placebo on treatment response (K = 4; N = 666; RR = 0.82; 95% CI, 0.75 to 0.91). **I**	BEASLEY1992 HOLLANDER2003D MONTGOMERY1993 MONTGOMERY2001
There is limited evidence suggesting a difference favouring 40 mg of an SSRI (citalopram/fluoxetine/paroxetine) over placebo on treatment response (K = 4; N = 661; RR = 0.79; 95% CI, 0.71 to 0.87). **I**	BEASLEY1992 HOLLANDER2003D MONTGOMERY1993 MONTGOMERY2001
There is evidence suggesting a difference favouring 60 mg of an SSRI (citalopram/fluoxetine/paroxetine) over placebo on treatment response (K = 4; N = 666; RR = 0.71; 95% CI, 0.64 to 0.8). **I**	BEASLEY1992 HOLLANDER2003D MONTGOMERY1993 MONTGOMERY2001
There is evidence suggesting there is unlikely to be a clinically important difference between 40 mg of an SSRI (citalopram/fluoxetine/paroxetine) and 20 mg of an SSRI on treatment response (K = 4; N = 655; RR = 0.96; 95% CI, 0.85 to 1.08). **I**	BEASLEY1992 HOLLANDER2003D MONTGOMERY1993 MONTGOMERY2001
There is limited evidence suggesting a difference favouring 60 mg of an SSRI (citalopram/fluoxetine/paroxetine) over 20 mg of an SSRI on treatment response (K = 4; N = 660; RR = 0.87; 95% CI, 0.76 to 0.98). **I**	BEASLEY1992 HOLLANDER2003D MONTGOMERY1993 MONTGOMERY2001
There is evidence suggesting there is unlikely to be a clinically important difference between 60 mg of an SSRI (citalopram/fluoxetine/paroxetine) and 40 mg of an SSRI on treatment response (K = 4; N = 655; RR = 0.90; 95% CI, 0.8 to 1.03). **I**	BEASLEY1992 HOLLANDER2003D MONTGOMERY1993 MONTGOMERY2001
Tolerability	
There is evidence suggesting that 20 mg of an SSRI (citalopram/fluoxetine/paroxetine) when compared with placebo increases the risk of leaving the study early because of adverse events (K = 4; N = 666; RR = 3.69; 95% CI, 2.03 to 6.7). **I**	BEASLEY1992 HOLLANDER2003D MONTGOMERY1993 MONTGOMERY2001

There is limited evidence suggesting that 40 mg of an SSRI (citalopram/fluoxetine/paroxetine) when compared with placebo increases the risk of leaving the study early because of adverse events (K = 4; N = 661; RR = 2.22; 95% CI, 1.17 to 4.22). **I**	BEASLEY1992 HOLLANDER2003D MONTGOMERY1993 MONTGOMERY2001
There is limited evidence suggesting that 60 mg of an SSRI (citalopram/fluoxetine/paroxetine) when compared with placebo increases the risk of leaving the study early because of adverse events (K = 4; N = 666; RR = 2.67; 95% CI, 1.44 to 4.98). **I**	BEASLEY1992 HOLLANDER2003D MONTGOMERY1993 MONTGOMERY2001
There is limited evidence suggesting a difference favouring 40 mg of an SSRI (citalopram/fluoxetine/paroxetine) over 20 mg of an SSRI on leaving the study early because of adverse events (K = 4; N = 655; RR = 0.60; 95% CI, 0.39 to 0.92). **I**	BEASLEY1992 HOLLANDER2003D MONTGOMERY1993 MONTGOMERY2001
There is limited evidence suggesting a difference favouring 60 mg of an SSRI (citalopram/fluoxetine/paroxetine) over 20 mg of an SSRI on leaving the study early because of adverse events (K = 4; N = 660; RR = 0.72; 95% CI, 0.48 to 1.07). **I**	BEASLEY1992 HOLLANDER2003D MONTGOMERY1993 MONTGOMERY2001
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between 60 mg of an SSRI (citalopram/fluoxetine/paroxetine) and 40 mg of an SSRI on leaving the study early because of adverse events (K = 4; N = 655; RR = 1.21; 95% CI, 0.75 to 1.93). **I**	BEASLEY1992 HOLLANDER2003D MONTGOMERY1993 MONTGOMERY2001

6.2.5 SSRIs versus other SSRIs

6.2.5.1 Clinical evidence statements

Efficacy	***Included studies***
There is limited evidence suggesting a difference favouring sertraline over fluoxetine on reducing obsessive-compulsive symptoms as measured by the clinician-rated Y-BOCS at 12 weeks (K = 1; N = 148; SMD = 0.39; 95% CI, 0.07 to 0.72). **I**	BERGERON2002
There is limited evidence suggesting a difference favouring fluvoxamine over citalopram on reducing obsessive-compulsive symptoms as measured by the National Institute of Mental Health Obsessive-Compulsive Global Scale (NIMH-OC) (K = 1; N = 21; SMD = 21.22; 95% CI, –2.17 to –0.27). **I**	MUNDO1997a
Tolerability	
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between different SSRIs on the tolerability of treatment.	BERGERON2002 MUNDO1997a

6.2.6 SSRIs versus clomipramine

6.2.6.1 Clinical evidence statements

Efficacy	***Included studies***
There is evidence suggesting there that is unlikely to be a clinically important difference between SSRIs and clomipramine on treatment response (OCD) (K = 8; N = 1019; RR = 1.02; 95% CI, 0.89 to 1.17). **I**	BISSERBE1997 BURNHAM LOPEZ-IBOR1996 KORAN1996A MILAN FRANCHI1997 MUNDO2001 ZOHAR1996A
There is evidence suggesting that there is unlikely to be a clinically important difference between SSRIs and clomipramine on OCD symptoms (Y-BOCS) (K = 7; N = 739; SMD = 0.14; 95% CI, –0.01 to 0.29). **I**	LOPEZ-IBOR1996 KORAN1996A MILAN FRANCHI1997 MUNDO2001 SMERALDI1992 ZOHAR1996A

There is evidence suggesting that there is unlikely to be a clinically important difference between SSRIs and clomipramine on OCD symptoms (NIMH-OC) (K = 3; N = 666; SMD = 0.08; 95% CI, –0.08 to 0.23). **I**	BURNHAM MUNDO2001 ZOHAR1996A
There is evidence suggesting that there is unlikely to be a clinically important difference between SSRIs and clomipramine on OCD symptoms (Y-BOCS) in patients with comorbid depression (K = 3; N = 192; SMD = 0.22; 95% CI, –0.06 to 0.51). **I**	LOPEZ-IBOR1996 MUNDO2001 SMERALDI1992

Tolerability

There is evidence suggesting that there is unlikely to be a clinically important difference between SSRIs and clomipramine on adverse effects (K = 7; N = 1037; RR = 0.95; 95% CI, 0.89 to 1). **I**	ASKIN1999 BISSERBE1997 BURNHAM FREEMAN1994 KORAN1996A MUNDO2001 ZOHAR1996A
There is limited evidence suggesting that clomipramine when compared with SSRIs increases the risk of leaving the study early (K = 10; N = 1139; RR = 0.72; 95% CI, 0.59 to 0.88). **I**	ASKIN1999 BISSERBE1997 BURNHAM FREEMAN1994 KORAN1996A LOPEZ-IBOR1996 MILANFRANCHI1997 MUNDO2001 SMERALDI1992 ZOHAR1996A
There is limited evidence suggesting that clomipramine when compared with SSRIs increase the risk of leaving the study early due to adverse effects (K = 8; N = 1095; RR = 0.62; 95% CI, 0.46 to 0.84). **I**	BISSERBE1997 BURNHAM FREEMAN1994 KORAN1996A LOPEZ-IBOR1996 MILANFRANCHI1997 MUNDO2001 SMERALDI1992 ZOHAR1996A

6.2.7 SSRIs versus placebo or clomipramine: continuation treatment

6.2.7.1 Descriptive review

Five trials examined the continuation of treatment in patients with OCD (ANSSEAU; LOPEZ-IBOR1996; MONTGOMERY1993; BEASLEY1992; GREIST1995A).

In MONTGOMERY1993, treatment responders (N = 173) continued their blinded treatment for an additional 16 weeks (after an 8-week, double-blind, placebo-controlled trial of three fixed doses of fluoxetine; total N = 217).

Response was maintained during the extension phase. The mean improvement of Y-BOCS scores was fluoxetine 20 mg (–1.3, N = 18); fluoxetine 40 mg (–1.6, N = 20); fluoxetine 60 mg (–1.7, N = 23); placebo (–1.8, N = 12). However, there was no significant difference between fluoxetine and placebo on mean improvement on the Y-BOCS.

ANSSEAU was an unpublished 30-week extension phase trial of a 12-week acute-phase RCT (ZOHAR1996a) that compared the maintenance of efficacy in patients who had responded to paroxetine, clomipramine or placebo. Patients (N = 83) continued on the drug they received during the acute phase.

Ninety per cent of patients in the paroxetine group had maintained a response to treatment (defined as a reduction of at least 25% in the total Y-BOCS scores), compared with 89.5% of patients in the clomipramine group and 75% of patients in the placebo group. However, there was no significant difference between the continuation of paroxetine and placebo (N = 63; SMD = –0.51; 95% CI, –1.15 to 0.12) or between the continuation of paroxetine and clomipramine (N = 71; SMD = –0.01; 95% CI, –0.53 to 0.5) on reducing obsessive-compulsive symptoms as measured by the Y-BOCS. There was also no significant difference between treatment groups on the likelihood of reporting adverse events, paroxetine versus placebo (82% versus 66%, N = 63; RR = 1.24; 95% CI, 0.81 to 1.88), paroxetine versus clomipramine (82% versus 75%, N = 71; RR = 1.1; 95% CI, 0.83 to 1.46).

In a continuation study of BEASLEY1992 (Tollefson *et al.*, 1994), treatment responders (N = 76) continued their blinded treatment for an additional 6 months after a 13-week, double-blind, placebo-controlled trial of three fixed doses of fluoxetine (N = 355).

Sixty-seven per cent of placebo-treated patients improved on the Y-BOCS at the end of the responder extension phase, as did 69.6% of fluoxetine 20 mg-treated patients, 76.2% of fluoxetine 40 mg-treated patients, and 76.9% of fluoxetine 60 mg-treated patients. Combining the fluoxetine-treated groups, 74.3% of patients experienced improvement beyond that seen during the 13-week acute phase. In terms of the number of patients who achieved ≥25% improvement in their Y-BOCS score, there was no difference between any dose of fluoxetine and placebo and between different doses of fluoxetine, 40 mg versus 20 mg (RR = 0.94; 95% CI; 0.57 to 1.54), 60 mg versus 20 mg (RR = 0.7; 95% CI, 0.4 to 1.21) and 60 mg versus 40 mg (RR = 0.74, 95% CI, 0.41 to 1.32).

In GREIST1995a, responders to a 12-week randomised trial of one of three fixed doses of sertraline (50, 100 or 200 mg) or placebo were continued on their treatment. Responders to the acute phase, defined as ‘marked’ or ‘moderate’ improvement on

the CGI Efficacy Index were offered an additional 40 weeks of double-blind treatment at their assigned doses. Three hundred and twenty-five patients entered the acute phase, of which 125 patients were classified as responders. One-hundred-and - eighteen patients entered the continuation phase.

Overall, the pooled sertraline group exhibited greater improvement on measures of efficacy over the 48-week treatment period, Y-BOCS mean change scores (SDs): sertraline –5.7 (7.4) versus placebo –2.8 (5.8), F (1,289) = 7.06, $p = 0.001$. When changes from week 12 to endpoint were examined on efficacy, no differences were seen among any of the individual treatment groups or between the pooled sertraline group and placebo. Patients in the sertraline groups were more likely to report adverse events compared with patients in the placebo group, sertraline 50 mg 94%, sertraline 100 mg 91%, sertraline 200 mg 96%, placebo 79%. Two patients in the sertraline group and none in the placebo group attempted suicide.

LOPEZ-IBOR1996 continued patients for 12 weeks on the same double-blind fluoxetine versus clomipramine treatment received during an 8-week acute phase. Responders (fluoxetine, n = 11; clomipramine, n = 13) received a lower dose of the drug (20 mg fluoxetine and 100 mg clomipramine), while non-responders (fluoxetine, n = 14; clomipramine, n = 8) received a higher dose of the same acute-phase drug (60 mg fluoxetine and 200 mg clomipramine).

No formal statistical analysis took place due to the small size of the treatment arms. However, among responders to fluoxetine the mean endpoint Y-BOCS score was lower (8.8, SD 5.79) compared with baseline (9.6, SD 5.35), whereas among responders to clomipramine the mean endpoint score was higher (13.7, SD 11.79) compared with baseline (11.4, SD 6.13). Among non-responders, there was a decrease in the mean Y-BOCS score from baseline in patients receiving fluoxetine (mean baseline score 26.7, SD 6.38, mean endpoint score 24, SD 7.54), and similarly in patients receiving clomipramine (mean baseline score 21.5, SD 8.62, mean endpoint score 15, SD 9.32).

6.2.8 SSRIs versus placebo or clomipramine: relapse prevention

6.2.8.1 Descriptive review

Five trials were included in the review that examined relapse prevention in patients with OCD (ANSSEAU, BAILER, HOLLANDER2003; KORAN2002; ROMANO2001). A further trial (RAVIZZA1996A) was conducted open-label and therefore excluded from the review.

In BAILER, following a 6-month acute phase RCT of paroxetine versus placebo (BURNHAM) and a 6-month open label phase with flexible dosing of paroxetine (N = 154), non-responders (N = 44) were randomised to a 6-month double-blind relapse prevention phase of fixed dose paroxetine versus placebo.

For the percentage of patients with partial relapses (defined as a Y-BOCS score increase from the acute phase baseline score and an increase of one or more points on the CGI Severity of Illness subscale from the last open label score), the treatment effect was not significant ($p = 0.22$), although the percentage of partial

relapses in the placebo group (63.6%) was greater than the percentage of partial relapses in the paroxetine group (42.1%). There was a significant difference favouring paroxetine over placebo on the Y-BOCS (N = 20; SMD = –1.17; 95% CI, –2.15 to –0.19).

In HOLLANDER2003, treatment responders (N = 105) were randomised to 6-month double-blind, fixed dose, parallel paroxetine/placebo treatment (after completing both a 12-week RCT of paroxetine/placebo [total N = 348] and 6 months of open-label paroxetine treatment [total N = 263].

The results indicated that more of the patients randomised to receive placebo (N = 52) experienced a relapse than those who continued to receive paroxetine (N = 53) when the criteria included an increase of one point or more on the CGI Severity of Illness subscale (38% versus 60%; RR = 0.63; 95% CI, 0.42 to 0.96). With a stricter criterion of a return to baseline of patients' Y-BOCS scores, the effect was stronger, but the difference was not statistically significant (9% versus 23%; RR = 0.41; 95% CI, 0.15 to 1.08). The mean time it took for patients continuing with paroxetine to relapse was 62.9 days compared with 28.5 days for those who relapsed after switching to placebo. Statistically significant differences favouring paroxetine over placebo on mean Y-BOCS scores were found as early as week 2 (F = 5.25; df = 1.68; $p = 0.02$) and up until 5 months. However, no difference was found between the groups at the end of 6 months. Significantly more patients receiving placebo left the study early because of adverse events compared with those receiving paroxetine (38% versus 5%; N = 105; RR = 0.15; 95% CI, 0.05 to 0.47).

In ANSSEAU, following an acute phase RCT of paroxetine versus clomipramine versus placebo (ZOHAR1996a) and an 8-week maintenance phase, patients were re-randomised within group to the drug or placebo (paroxetine/paroxetine versus paroxetine/placebo, clomipramine/clomipramine versus clomipramine/placebo, and placebo).

There was no significant difference between paroxetine and placebo on the rate of partial relapse (defined as an increase on the Y-BOCS from the baseline score or an increase in CGI severity by one or more points from the last observation), paroxetine 1/10 (10%) versus placebo 2/8 (25%). Based on a 25% response criterion on the Y-BOCS, the rate of response was lower in paroxetine (18.2%) compared with placebo (33.3%), but this was not statistically significant ($p = 0.45$). There was also no significant difference between paroxetine and placebo on the likelihood of reporting adverse effects, 57% versus 50%, respectively.

In ROMANO2001, treatment responders (N = 71) were randomised to 52 weeks double-blind, fixed dose fluoxetine or placebo treatment (after completing a 20-week single-blind treatment of fluoxetine [total N = 130]). People who continued to receive fluoxetine had a lower estimated 1-year relapse rate compared with those randomised to placebo, though this difference was not statistically different (20.6% versus 31.9%, $p = 0.14$). Among patients who responded to acute phase treatment with fluoxetine 60 mg/day, a subset of those who continued to receive fluoxetine, the 1-year estimated rate of relapse was lower compared with patients randomised to placebo (17.5% versus 38%, $p = 0.04$). This estimate was not significantly different for people who responded to acute phase treatment with fluoxetine 20 or 40 mg/day and who continued to receive fluoxetine for a year compared with patients randomised to

placebo (28.6% versus 21.3%, $p = 0.79$). Change on the Y-BOCS from randomisation to endpoint was not statistically different between fluoxetine and placebo ($p = 0.54$).

In KORAN2002, treatment responders (N = 223) were randomised to 28 weeks double-blind sertraline or placebo treatment (after completing a 52-week single-blind treatment of sertraline [total N initially enrolled = 649]). Patients assigned to sertraline continued on the same dose of sertraline as during the 52-week phase, while patients assigned to placebo were blindly discontinued from sertraline. Mean Y-BOCS scores increased in both groups (mean change at end-point, –1.3 and –3.1 in sertraline and placebo groups respectively). However, patients continuing with sertraline had a lower mean score at endpoint than those receiving placebo (N = 221; SMD = –0.37; 95% CI, –0.63 to –0.10). When a strict criteria for relapse (increase in Y-BOCS >=5, Y-BOCS score >=20 and CGI Global Improvement >=1 continuing for a month) was applied, there was no difference between sertraline and placebo (3.6% versus 5.2%; N = 223; RR = 0.7; 95% CI, 0.2 to 2.4). However, more patients receiving placebo experienced a relapse or insufficient clinical response compared with sertraline (RR = 0.39; 95% CI, 0.20 to 0.76) and more patients experienced an acute exacerbation of OCD (RR = 0.34; 95% CI, 0.19 to 0.60).

6.2.9 Clinical summary

There is evidence from ten studies involving 2,588 patients for the efficacy of SSRIs (citalopram, fluoxetine, fluvoxamine, paroxetine, sertraline) for adults with OCD from placebo-controlled trials. The tolerability data indicate that they are generally well tolerated as the likelihood of leaving the study is unlikely to be greater for active treatment than for placebo. However there is limited evidence for more adverse events reported in the active arms and a greater chance of leaving the study because of adverse effects. In addition, there is some evidence that higher doses may be more efficacious than lower doses for citalopram, fluoxetine and paroxetine, and that higher doses may be associated with relatively fewer premature withdrawals because of adverse effects than lower doses. This may be due to the greater response at higher doses encouraging people to stay in the study whether they experience adverse effects or not. There is yet very little evidence suggesting that any one SSRI is more effective than another.

There is evidence from eight studies involving 1,019 patients that there is unlikely to be any clinically important differences between SSRIs and clomipramine either for efficacy or for adverse effects, but there is a greater likelihood of people discontinuing clomipramine based on those leaving the studies early and leaving early due to adverse effects. Thus, although SSRIs and clomipramine are both efficacious treatments (see also 6.4 below), SSRIs may be better tolerated. The continuation studies have investigated long-term treatment with SRIs for up to 48 weeks and the results suggest that patients who have responded to acute-phase treatment continue to respond in the longer term. The relapse prevention studies have used survival analysis to investigate patients for up to 12 months. The results suggest that SSRI treatment continued over the longer term protects against relapse of OCD.

6.3 CLOMIPRAMINE

6.3.1 Introduction

Clomipramine was the first pharmacotherapeutic agent found to have efficacy in OCD (Marks *et al.*, 1980; Montgomery, 1980). The drug shares the pharmacological properties of the tricyclic group of antidepressants from which it is derived, but can be distinguished from other tricyclics by its potent effects at inhibiting the synaptic reuptake of the neurotransmitter serotonin. However, its effects are not selectively mediated by serotonin mechanisms. As a tricyclic, clomipramine is associated with the adverse effects and toxicity in overdose that typifies this group of drugs. For this reason it is usually considered second-line after SSRIs for patients whose symptoms have failed to respond to SSRIs or who are unable to tolerate them.

The following treatments were included:

- Oral clomipramine
- Intravenous clomipramine.

6.3.2 Studies considered

The review team conducted a new systematic search for RCTs that assessed the efficacy and tolerability of clomipramine. Sixty-four studies were identified, of which 36 did not meet the inclusion criteria of the GDG. The 27 included studies provided efficacy data from 2121 participants and tolerability data from 2208 participants.

Of the included studies, seven compared clomipramine with placebo (BURNHAM; CCSG1991; KATZ1990; MONTGOMERY1990; STEIN1992; THOREN1980A; ZOHAR1996A), five with other tricyclic antidepressants (ANANTH1981; KHANNA1988; THOREN1980A; VOLAVKA1985; ZOHAR1987A), ten with SSRIs[10] (ASKIN1999; BISSERBE1997; BURNHAM; FREEMAN1994; KORAN1996A; LOPEZ-IBOR1996; MILANFRANCHI1997; MUNDO2001; SMERALDI1992; ZOHAR1996A), and five with other drugs (ALBERT2002; HEWLETT1992; INSEL1983B; PATO1991; VALLEJO1992). Other comparisons included intravenous clomipramine versus placebo (FALLON1998) and oral clomipramine (KORAN1997).

All included acute phase studies (N = 25) were between 2 and 16 weeks long (mean length = 9.13 weeks). Patients were classified as outpatient in 11 studies, inpatient in 3 studies and mixed in 3 studies. Eight studies did not report patient setting. The average age of the patients was 33.65 years. The average duration of illness based on 10 studies was 10.94 years. Six studies were multi-centre studies, of which two were conducted in the US, two in Europe, one in France and Spain and the other in France and Belgium. Nine other studies were conducted in the US, three in Italy, three in the UK and one each in Austria, Canada, India and Sweden.

[10]The results of the analysis of these studies are considered in the earlier section on SSRIs versus Clomipramine.

Two cross-over trials comparing clomipramine with SSRIs (KHANNA1988, ZOHAR1987a) are summarised in narrative form, as it was not possible to extract data at the point of cross-over. The other two cross-over trials, INSEL1983b and HEWLETT1992, are considered narratively in the sections on MAOIs and anxiolytics respectively.

Full details of the studies included in the guideline and the reasons for excluding studies are given in Appendix 16.

6.3.3 Clomipramine versus placebo

6.3.3.1 Clinical evidence statements

Efficacy	***Included studies***
There is limited evidence suggesting a difference favouring clomipramine over placebo on the likelihood of treatment response, defined as a 25% or greater reduction on the clinician-rated Y-BOCS, or CGI 'much improved/very much improved' (K = 3; N = 401; RR = 0.81; 95% CI, 0.68 to 0.96). **I**	BURNHAM STEIN1992 ZOHAR1996A
There is evidence suggesting a difference favouring clomipramine over placebo on the likelihood of remission, defined as a score of 1 to 6 on the NIMH-OC scale (K = 1; N = 520; RR = 0.54; 95% CI, 0.48 to 0.61). **I**	CCSG1991
There is evidence suggesting a difference favouring clomipramine over placebo on reducing obsessive-compulsive symptoms as measured by the clinician-rated Y-BOCS (K = 3; N = 694; random effects SMD = –1.04; 95% CI, –1.72 to –0.36). **I**	CCSG1991 (1) CCSG1991 (2) ZOHAR1996A
There is evidence suggesting a difference favouring clomipramine over placebo on reducing obsessive-compulsive symptoms as measured by the Comprehensive Psychopathological Rating Scale (CPRS) or 6-item OCD scale (K = 2; N = 30; SMD = –1.12; 95% CI, –1.92 to –0.32). **I**	MONTGOMERY1990 THOREN1980A
There is evidence suggesting a difference favouring clomipramine over placebo on reducing obsessive-compulsive symptoms as measured by the NIMH-OC	BURNHAM CCSG1991 (1) CCSG1991 (2) ZOHAR1996A

scale (K = 4; N = 847; random effects SMD = –0.87; 95% CI, –1.37 to –0.38). **I**

There is limited evidence suggesting a difference favouring clomipramine over placebo on reducing the severity of illness as measured by the CGI Severity of Illness subscale (K = 1; N = 193; SMD = –0.32; 95% CI, –0.60 to –0.03). **I** — ZOHAR1996a

There is limited evidence suggesting a difference favouring clomipramine over placebo on reducing psychological distress as measured by the Symptom Checklist-90 (K = 1; N = 151; SMD = –0.32; 95% CI, –0.64 to 0.00). **I** — ZOHAR1996a

Tolerability

There is evidence suggesting that clomipramine when compared with placebo increases the risk of reporting adverse effects (K = 3; N = 877; random effects RR = 1.25; 95% CI, 0.96 to 1.62). **I** — BURNHAM CCSG1991 ZOHAR1996A

There is evidence suggesting that clomipramine when compared with placebo increases the risk of leaving the study early due to adverse effects (K = 2; N = 357; RR = 2.35; 95% CI, 1.31 to 4.22). **I** — BURNHAM ZOHAR1996A

6.3.4 Clomipramine versus other TCAs

6.3.4.1 Clinical evidence statements

Efficacy — ***Included studies***

There is limited evidence suggesting a difference favouring clomipramine over imipramine on reducingobsessive-compulsive symptoms as measured by the Self-Rating Obsessional Neurotic Scale (K = 1; N = 16; SMD = –1.17; 95% CI, –2.26 to –0.09). **I** — VOLAVKA1985

There is limited evidence suggesting a difference favouring clomipramine over imipramine on improving global efficacy as measured by the Global Evaluation of Efficacy (K = 1; N = 16; SMD = –1.05; 95% CI, –2.12 to 0.02). **I** — VOLAVKA1985

There is limited evidence suggesting a difference favouring clomipramine over imipramine on reducing depression as measured by the Hamilton Depression Rating Scale (K = 1; N = 16; SMD = –1.04; 95% CI, –2.11 to 0.02). **I**	VOLAVKA1985

Tolerability

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and imipramine on leaving the study early because of adverse effects (K = 1; N = 23; RR = 1.09; 95% CI, 0.18 to 6.48). **I**	VOLAVKA1985

6.3.4.2 Results summary from additional cross-over trials

In KHANNA1988, patients received double-blind clomipramine or nortriptyline in a randomised fashion for 6 weeks and then crossed over to the other drug after a 4-week drug-free interval. Out of the 18 patients who entered the trial, eight completed the full cross-over sequence.

There was no significant difference between pre- and post-trial scores on the Leyton Obsessional Inventory and the Maudsley Obsessive-Compulsive Inventory. There was also no difference between clomipramine and nortriptyline on obsessive-compulsive symptoms.

In ZOHAR1987a, after a 2–4 week placebo-phase, patients were randomly assigned to clomipramine or desipramine. After a 4-week placebo interval, patients were crossed-over to the other drug. The duration of each active treatment was 6 weeks. Fourteen patients entered the trial, of which 10 patients completed the entire cross-over sequence.

The reduction in obsessive-compulsive symptoms as measured by the Comprehensive Psychopathological Rating Scale – Obsessive-Compulsive subscale (CPRS-OC) and the NIMH-OC scale was greater in clomipramine than desipramine (CPRS-OC-5: F1,8 = 8.03, p = 0.02; NIMH-OC: F1,8 = 7, p = 0.03). Using the CPRS-OC subscale, the mean (±SD) improvement during the 6 weeks for clomipramine was 28.4% ± 20.1% compared with 4.2% ± 11.4% for desipramine.

6.3.5 Intravenous clomipramine + placebo pills versus oral clomipramine + placebo IV

6.3.5.1 Clinical evidence statements

Efficacy	***Included studies***
There is limited evidence suggesting a difference favouring intravenous clomipramine + placebo pills over oral clomipramine + placebo IV on the likelihood of treatment response, defined as a 25% or greater reduction on the clinician-rated Y-BOCS from baseline (K = 1; N = 15; RR = 0.16; 95% CI, 0.03 to 1.02). **I**	KORAN1997
There is limited evidence suggesting a difference favouring intravenous clomipramine + placebo pills over oral clomipramine + placebo IV on reducing obsessive-compulsive symptoms as measured by the clinician-rated Y-BOCS (K = 1; N = 15; SMD = –1.26; 95% CI, –2.40 to –0.12). **I**	KORAN1997
Tolerability	
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between intravenous clomipramine + placebo pills over oral clomipramine + placebo IV on the tolerability of treatment.	KORAN1997

6.3.6 Clomipramine versus placebo or SSRIs: continuation or discontinuation of treatment

There were 3 trials that examined the continuation or discontinuation of clomipramine (KATZ1990; LOPEZ-IBOR1996; RAVIZZA1996A). RAVIZZA1996A was an open-label trial and therefore excluded from the review.

In KATZ1990, outpatients received an initial 10-week double-blind treatment of clomipramine or placebo. Patients who responded to the 10-week acute phase (response being defined as response to treatment on at least three occasions during the 10 weeks as judged by the treating physician) entered a double-blind 1-year extension. Two hundred and eighty-six patients entered the study, of which 124 patients with a baseline Hamilton Depression Rating Scale score ≤17 and responding to the acute phase treatment entered the 1-year extension.

Patients from both groups showed improvement over the course of the extension phase compared with baseline as measured by the NIMH-OC scale, clomipramine –4.7 (SD 2.7) and placebo –1.7 (SD 2.5). The improvement was greater in

clomipramine compared with placebo ($p < 0.001$). Ninety-eight percent of the clomipramine patients and 65% of the placebo patients reported one or more adverse events. Twenty-five patients taking clomipramine discontinued the extension trial because of medical problems, whereas no placebo patients discontinued the study because of medical problems. Twelve clomipramine patients had serious adverse events. However, the authors suggest that the small sample size, small entry cohort and discontinuation patterns argue against any general or long-term efficacy of placebo, and also against any conclusive interpretations of the data for this highly selected group.

LOPEZ-IBOR1996 continued patients for 12 weeks on the same double-blind fluoxetine versus clomipramine treatment received during an 8-week acute phase. Responders (fluoxetine, n = 11; clomipramine, n = 13) received a lower dose of the drug (20 mg fluoxetine and 100 mg clomipramine), while non-responders (fluoxetine, n = 14; clomipramine, n = 8) received a higher dose of the same acute-phase drug (60 mg fluoxetine and 200 mg clomipramine).

No formal statistical analysis took place due to the small size of the treatment arms. However, among responders to fluoxetine the mean endpoint Y-BOCS score was lower (8.8, SD 5.79) compared with baseline (9.6, SD 5.35), whereas among responders to clomipramine the mean endpoint score was higher (13.7, SD 11.79) compared with baseline (11.4, SD 6.13). Among non-responders, there was a decrease on the mean Y-BOCS score from baseline in patients receiving fluoxetine (mean baseline score 26.7, SD 6.38, mean endpoint score 24, SD 7.54), and similarly in patients receiving clomipramine (mean baseline score 21.5, SD 8.62, mean endpoint score 15, SD 9.32).

6.3.7 Clinical summary

The results of these studies show that clomipramine is effective in the acute treatment of OCD. There have been no 'dose-finding' studies for clomipramine so we cannot judge the most effective dose for OCD. There is evidence from one study that intravenous clomipramine may be more effective than oral in partially responsive individuals. The evidence supporting the ongoing efficacy in continuation studies is limited by the shortage of studies in this area.

6.4 OTHER TRICYCLIC ANTIDEPRESSANTS

6.4.1 Introduction

Unlike SSRIs and clomipramine, other antidepressants that have been investigated in OCD so far, for example, tricyclic antidepressants (apart from clomipramine), tricyclic-related antidepressants, serotonin and noradrenaline re-uptake inhibitors (SNRIs) and monoamine-oxidase inhibitors (MAOIs) have not been shown to have convincing anti-obsessional efficacy (see below).

6.4.2 Treatments included

The following treatments were included:
- Amitriptyline
- Desipramine
- Imipramine
- Nortriptyline.

6.4.3 Studies considered

The review team conducted a new systematic search for RCTs that assessed the efficacy and tolerability of TCAs other than clomipramine. Twelve studies were identified, of which five did not meet the inclusion criteria of the GDG. The seven included studies provided efficacy data from 258 participants and tolerability data from 259 participants.

Of the included studies, five compared TCAs with clomipramine (ANANTH1981; KHANNA1988; THOREN1980A; VOLAVKA1985; ZOHAR1987A) and two with SSRIs (GOODMAN1990A; HOEHN-SARIC2000). One study featured an additional placebo comparison (THOREN1980a).

All included studies were between 5 and 28 weeks long (mean length = 12.4 weeks). Patients were classified as outpatient in three studies, inpatient in one study and mixed in one study. One study did not report patient setting. The mean age of the patients was 36.6 years and the mean duration of illness in two studies was 18 years. Four studies were conducted in the US and one each in Canada, India and Sweden.

The results of two cross-over studies (KHANNA1988; ZOHAR1987A) are summarised in narrative form, as it was not possible to extract data at the point of cross-over.

Full details of the studies included in the guideline and the reasons for excluding studies are given in Appendix 16.

6.4.4 Tricyclic antidepressants versus placebo

6.4.4.1 Clinical evidence statements

Efficacy	***Included studies***
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between nortriptyline and placebo on the efficacy of treatment in adults with OCD (K = 1; N = 16).	THOREN1980a

6.4.4.2 Results summary from additional cross-over trials

In KHANNA1988, patients received double-blind clomipramine or nortriptyline in a randomised fashion for 6 weeks and then crossed-over to the other drug after a

4-week drug-free interval. Out of the 18 patients who entered the trial, 8 completed the full cross-over sequence.

There was no significant difference between pre- and post-trial scores on the Leyton Obsessional Inventory and the Maudsley Obsessive-Compulsive Inventory. There was also no difference between clomipramine and nortriptyline on obsessive-compulsive symptoms.

In ZOHAR1987A, after a 2–4 week placebo-phase, patients were randomly assigned to clomipramine or desipramine. After a 4-week placebo interval, patients were crossed-over to the other drug. The duration of each active treatment was 6 weeks. Fourteen patients entered the trial, of which 10 patients completed the entire cross-over sequence.

The reduction in obsessive-compulsive symptoms as measured by the CPRS-OC subscale and the NIMH-OC scale was greater in clomipramine than desipramine (CPRS-OC-5: F1,8 = 8.03; $p = 0.02$; NIMH-OC: F1,8 = 7; $p = 0.03$). Using the CPRS-OC subscale, the mean (±SD) improvement during the 6 weeks for clomipramine was 28.4% ± 20.1% compared with 4.2% ± 11.4% for desipramine.

6.4.5 Clinical summary

The results of these studies do not support the efficacy of tricyclic antidepressants (apart from clomipramine) for OCD.

6.5 TRICYCLIC-RELATED ANTIDEPRESSANTS

6.5.1 Treatments included

The following treatments were included:

- Trazodone.

6.5.2 Studies considered

The review team conducted a new systematic search for RCTs that assessed the efficacy and tolerability of tricyclic-related antidepressants. Two studies were identified, one of which did not meet the inclusion criteria of the GDG (ANTONELLI1973). The included study (PIGOTT1992) compared trazodone with placebo in a 10-week outpatient trial that provided efficacy data from 17 participants. Participants were begun on a fixed oral dosage regimen of 50 mg/day and were gradually increased, as tolerated, to a maximum of 300 mg/day.

Full details of the studies included in the guideline and the reasons for excluding studies are given in Appendix 16.

6.5.3 Descriptive review

In PIGOTT1992, patients received 10 weeks of either trazodone or placebo in a randomised fashion. There were no treatment differences between trazodone and placebo in patients completing the 10-week trial. Mean maximum decreases from baseline on the Y-BOCS was 2.7, SD 1.87; $t = -1.44$; $p = 0.18$, in patients receiving trazodone and –2.83; SD 1.19; $t = -2.37$; $p = 0.06$, in patients receiving placebo.

6.5.4 Clinical summary

There is no evidence supporting the efficacy of tricyclic-related drugs in OCD.

6.6 SEROTONIN AND NORADRENALINE REUPTAKE INHIBITORs (SNRIs)

6.6.1 Treatments included

The following treatments were included:

- Venlafaxine.

6.6.2 Studies considered

The review team conducted a new systematic search for RCTs that assessed the efficacy and tolerability of SNRIs. Five studies were identified, of which three did not meet the inclusion criteria of the GDG (DENYS2002; TENNEY2003; YARYURATOBIAS1996). The two included studies provided efficacy data from 218 participants and tolerability data from 223 participants, and compared venlafaxine with clomipramine (ALBERT2002) and paroxetine (DENYS2003A).

Both studies were 12 weeks long. Patients were classified as outpatient in both studies. The duration of OCD was 5.15 and 15 years in ALBERT2002 and DENYS2003A respectively. ALBERT2002 was conducted in Italy and DENYS2003A was conducted in the Netherlands.

Full details of the studies included in the guideline and the reasons for excluding studies are given in Appendix 16.

6.6.3 Venlafaxine versus other antidepressants

6.6.3.1 Clinical evidence statements

Efficacy	***Included studies***
There is evidence suggesting there is unlikely to be a clinically important difference between venlafaxine and other	ALBERT2002 DENYS2003A

SRIs on treatment response (OCD) (K = 2; N = 223; RR = 1.13; 95% CI, 0.91 to 1.40). **I**

There is evidence suggesting there is unlikely to be a clinically important difference between venlafaxine and other SRIs on OCD symptoms (K = 2; N = 218; SMD = 0.11; 95% CI, 20.16 to 0.38). **I** — ALBERT2002 DENYS2003A

Tolerability

There is limited evidence suggesting a difference favouring venlafaxine over clomipramine on the likelihood of reporting adverse effects in adults with OCD (K = 1; N = 73; RR = 0.67; 95% CI, 0.49 to 0.92). **I** — ALBERT2002

6.6.4 Clinical summary

There is no placebo-controlled evidence to support the efficacy of SNRIs in OCD. Although two studies showed similar levels of improvement on venlafaxine that have been found with drugs known to have shown efficacy in previous OCD, the absence of a placebo for comparison in these studies prevents us from concluding that in these studies the drug was actually effective. However, the results are suggestive of efficacy and point to further systematic investigation of SNRIs as a potential treatment for OCD.

6.7 MONOAMINE-OXIDASE INHIBITORs (MAOIs)

6.7.1 Treatments included

The following treatments were included:

- Clorgyline
- Phenelzine.

6.7.2 Studies considered

The review team conducted a new systematic search for RCTs that assessed the efficacy and tolerability of MAOIs. Five studies were identified, of which two did not meet the inclusion criteria of the GDG (INSEL1982; ZAHN1984). The three included studies provided efficacy data from 67 participants and tolerability data from 94 participants.

One study compared phenelzine with placebo (JENIKE1997) and all three studies compared MAOIs with other drugs, including fluoxetine (JENIKE1997) and clomipramine (INSEL1983B; VALLEJO1992).

The included studies were between 6 and 12 weeks long (mean length = 9.3 weeks). Patients were classified as outpatient in two studies and mixed in one study. The average age of the patients was 33 years. The duration of illness was 6.7 and 11 years in INSEL1983b and VALLEJO1992 respectively. Two studies were conducted in the US and one in the UK.

The results of one cross-over study (INSEL1983b) are summarised in narrative form, as it was not possible to extract data at the point of cross-over.

Full details of the studies included in the guideline and the reasons for excluding studies are given in Appendix 16.

6.7.3 MAOIs versus placebo

6.7.3.1 Clinical evidence statements

Efficacy	***Included studies***
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between phenelzine and placebo on the efficacy of treatment in adults with OCD.	JENIKE1997
Tolerability	
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between phenelzine and placebo on the likelihood of leaving the study early (K = 1; N = 41; RR = 1.05; 95% CI, 0.24 to 4.61). **I**	JENIKE1997

6.7.4 MAOIs versus other drugs

6.7.4.1 Clinical evidence statements

Efficacy	***Included studies***
There is limited evidence suggesting a difference favouring phenelzine over clomipramine on reducing anxiety as measured by the Hamilton Anxiety Scale (K = 1; N = 26; SMD = 20.88; 95% CI, 21.69 to –0.07). **I**	VALLEJO1992
Tolerability	
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between MAOIs and other drugs on the tolerability of treatment.	JENIKE1997 VALLEJO1992

6.7.4.2 Results summary from additional cross-over trials

In INSEL1993B, 24 patients with OCD were randomised to placebo-clorgyline-placebo or placebo-clomipramine-placebo sequences and then crossed over to the other treatment over a 6-month period. The duration of each active treatment was 6 weeks. Ten patients completed the entire cross-over sequence.

Neither clorgyline nor clomipramine were effective in reducing symptom severity as measured by the Leyton Interference score, though clomipramine was effective in ameliorating scores on 13 of 19 measures while clorgyline showed no improvement on all 19 measures. When the 10 patients who completed the entire cross-over sequence were compared, improvement with clomipramine surpassed that seen with clorgyline on the CPRS-OC subscale, the Global Obsessive-Compulsive scale and the Obsessive-Compulsive rating scale. Only one patient displayed more improvement with clorgyline than with clomipramine on obsessive-compulsive symptoms. Side effects were prevalent on placebo, clorgyline and clomipramine.

6.7.5 Clinical summary

There is no convincing evidence supporting the efficacy of MAOIs in OCD.

6.8 ANXIOLYTICS

6.8.1 Introduction

OCD is classified in some diagnostic systems, such as DSM-IV, together with the anxiety disorders. However, anxiolytics (excluding SSRIs) are not considered effective for the treatment of the core symptoms of OCD. The dependence producing effects of benzodiazepines argue against their use as long-term treatments.

6.8.2 Treatments included

The following treatments were included:

- Buspirone
- Clonazepam.

6.8.3 Studies considered

The review team conducted a new systematic search for RCTs that assessed the efficacy and tolerability of anxiolytics. Seven studies were identified, of which four did not meet the inclusion criteria of the GDG (KIM1997; LIN1979; ORVIN1967;

WAXMAN1977). The three included studies provided efficacy data from 70 participants and tolerability data from 47 participants.

One study compared clonazepam with placebo (HOLLANDER2003C) and two with other drugs (HEWLETT1992; PATO1991).

The included studies were between 6 and 26 weeks long. Patients were classified as outpatient in one study. The average age of the patients was 35 years. All three studies were conducted in the US.

The results of one cross-over study (HEWLETT1992) are summarised in narrative form, as it was not possible to extract data at the point of cross-over.

Full details of the studies included in the guideline and the reasons for excluding studies are given in Appendix 16.

6.8.4 Anxiolytics versus placebo

6.8.4.1 Clinical evidence statements

Efficacy	***Included studies***
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clonazepam and placebo on the efficacy of treatment.	HOLLANDER2003C
Tolerability	
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clonazepam and placebo on the tolerability of treatment.	HOLLANDER2003C

6.8.5 Anxiolytics versus other drugs

6.8.5.1 Clinical evidence statements

Efficacy	***Included studies***
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between buspirone and clomipramine on the efficacy of treatment in adults with OCD.	PATO1991
Tolerability	
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between buspirone and clomipramine on the tolerability of treatment in adults with OCD.	PATO1991

6.8.5.2 Results summary from additional cross-over trial

In HEWLETT1992, 28 patients were initially randomly assigned to clomipramine, clonazepam, clonidine or diphenhydramine as control and then crossed-over to each of the other treatments. The study took place over 26 months, with 6 weeks in each treatment. Sixteen patients completed the entire cross-over sequence. Eight patients discontinued because of adverse side effects.

Clonazepam and clomipramine produced significantly lower Y-BOCS scores than did diphenhydramine and clonidine. In turn, Y-BOCS scores in diphenhydramine were significantly lower than baseline, whereas there was no difference between ratings for clonidine treatment and baseline. While the response rate for diphenhydramine and clonidine was 27% and 20% respectively, the response rates for clonazepam and clomipramine was higher at 48% and 54% respectively. Patients who responded to clonazepam tended to respond to clomipramine, the cross-response rate being 73% and patients who responded to clomipramine tended to respond to clonazepam, cross-response rate 80%.

6.8.6 Clinical summary

Although there is some evidence suggesting potential efficacy for clonazepam, the dependence-producing effects known to occur with benzodiazepines argues against the routine use of the drug for OCD.

6.9 SSRIs/CLOMIPRAMINE VERSUS NON-SRIs

6.9.1 Introduction

There is a body of opinion that SSRIs and clomipramine are superior to non-SRIs. This is based on the evidence that SRIs have been found to be effective compared with placebo, whereas non-SRIS have not. Thus prescribers may well be more likely to choose an SRI for treating OCD. Head-to-head trials comparing SRIs with non-SRIs may influence the confidence with which treatment preferences are made.

6.9.2 Treatments included

The following treatments were included:

- SSRIs
- Clomipramine
- TCAs
- MAOIs
- Anxiolytics.

Venlafaxine was excluded because it has the properties of both SRIs and TCAs.

6.9.3 Studies considered

Trials which performed head-to-head comparisons of a SSRI or clomipramine with a non-SRI were considered. Six studies were included: GOODMAN1990A; HOEHN-SARIC2000; JENIKE1997; PATO1991; VALLEJO1992; VOLAVKA1985. The studies provided efficacy data on 278 patients and tolerability data on 343 patients. The duration of treatment ranged between 6 and 12 weeks. Patients were classified as outpatient in 4 studies, patient setting was not reported in 2 studies. The average age of the patients was 35 years. Five studies were conducted in the US and one study was conducted in the UK. Two studies included patients with comorbid depression.

6.9.3.1 Clinical evidence statements

Efficacy	***Included studies***
There is limited evidence suggesting a difference favouring SSRIs/clomipramine over non-SRIs on reducing obsessive-compulsive symptoms, as measured by the clinician-rated Y-BOCS in adults with OCD (K = 4; N = 258; SMD = 20.3; 95% CI, 20.54 to –0.05). **I**	GOODMAN1990A HOEHN-SARIC2000 JENIKE1997 PATO1991
Tolerability	
There is limited evidence suggesting a difference favouring SSRIs/clomipramine over non-SRIs on the likelihood of leaving the study early due to adverse events in adults with OCD (K = 5; N = 279; RR = 0.51; 95% CI, 0.28 to 0.95). **I**	GOODMAN1990A HOEHN-SARIC2000 PATO1991 VALLEJO1992 VOLAVKA1985

6.9.4 Clinical summary

There is some evidence that SRIs, not considering venlafaxine, are better than non-SRIs such as TCAs, MAOIs and anxiolytics on efficacy and tolerability, but this is limited by the small number of studies in this comparison. This evidence is supported by the fact that SRIs are better than placebo, whereas TCAs are not better than placebo.

6.10 OTHER PHARMACOLOGICAL INTERVENTIONS

6.10.1 Introduction

In the search for alternative treatment for OCD, a wide range of other pharmacological treatments have been investigated. However, each has been subjected to only very limited study and the results have not supported further systematic exploration. This section considers alternative treatments in patients who were not yet considered as refractory to treatment.

6.10.2 Treatments included

The following treatments were included:

- Inositol
- Pindolol
- Tryptophan
- Gabapentin
- Triptans
- Anti-testosterone
- St. John's wort
- Kava kava
- Ginko biloba
- Amphetamine
- Oxytocin
- Clonidine
- Practolol
- Ondansteron
- Ritanserin
- Anti-androgen
- Cyproterone.

6.10.3 Studies considered

A systematic search was conducted for reports on the above treatments in patients with OCD or BDD. The search yielded 704 records, of which 2 studies (DENBOER1992, FUX1996) were RCTs and 9 studies were non-RCTs. Full details of the search strategy are given in Appendix 6.

6.10.4 Inositol versus placebo

6.10.4.1 Clinical evidence statements

Efficacy	***Included studies***
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between inositol and placebo on the efficacy of treatment.	FUX1996

6.10.5 Oxytocin versus placebo

6.10.5.1 Clinical evidence statements

Tolerability	***Included studies***
There is limited evidence suggesting a difference favouring placebo over oxytocin on the likelihood of reporting adverse effects in adults with OCD (K = 1; N = 12; RR = 6.00; 95% CI, 1.00 to 35.91). **I**	DENBOER1992

6.10.6 Descriptive review

The quality of the evidence was poor, since most studies were case reports (Ansseau *et al.*, 1987; Casas *et al.*, 1986; Eriksson, 2000; Feldman *et al.*, 1988; Knesevich, 1982) or had very small sample sizes (Hewlett *et al.*, 2003; Salzberg & Swedo, 1992; Taylor & Kobak, 2000; Yaryura Tobias & Bhagavan, 1977).

Ansseau and colleagues (1987) reported the treatment of a patient with OCD with intranasal oxytocin or placebo in a double-blind cross-over manner, each randomised period lasting 4 weeks. While no significant changes in obsessive-compulsive symptoms were present during the placebo period, intranasal oxytocin induced a clear improvement on the CPRS-OC. However, the patient also complained of gross memory disturbances during oxytocin therapy and increased psychotic symptoms. In contrast, Salzberg and Swedo (1992) failed to find any discernible effects of oxytocin or vasopressin on obsessive-compulsive symptoms in 3 patients randomised in a blinded fashion to intranasal oxytocin, intranasal vasopressin or normal saline.

Casas and colleagues (1986) studied the anti-obsessional effect of the anti-androgen, Cyproterone acetate. In patients with OCD, during the 10 days of treatment there was a notable improvement in obsessive symptoms and a decrease in compulsive rituals. After the third month of treatment, there was a gradual reappearance of obsessive symptoms, in particular anxiety and the need to perform compulsive rituals. Feldman and colleagues (1988) failed to find improvement in obsessive-compulsive symptoms after treating a patient with OCD with cyproterone acetate for 5 months. Eriksson (2000) studied the effect of a long-acting gonadotropin-releasing hormone analogue, triptorelin. After 4 months of treatment, the patient experienced fewer obsessive-compulsive symptoms and further improvement after 3 more months of treatment.

Hewlett and colleagues (2003) studied the anti-obsessional properties of 5-HT3 receptor antagonist, ondansetron, in 8 patients with OCD. Over the course of the trial, 8 patients experienced an average of 28% reduction on the Y-BOCS from baseline, while 3 patients achieved a clinically significant response (≥35% reduction in Y-BOCS score). Moreover, at 2 weeks' follow-up of the 6 completers, there was a 45% worsening of symptoms associated with discontinuing treatment.

Taylor and colleagues (2000) conducted a 12-week open-label trial of St. John's Wort in 12 patients. Treatment consisted of a fixed dose of 450 mg of 0.3% hypericin twice daily. At the end of treatment, five out of 12 patients were rated as 'much improved' or 'very much improved' on the CGI Global Improvement scale. A significant improvement similar to that found in clinical trials was found on the Y-BOCS, with a mean change of 7.4 points.

Knesevich (1982) reported improvement on compulsions following a 2-week trial with clonidine (0.1 mg t.i.d.) in a patient with OCD. Increasing the dose to 0.1 mg q.i.d. caused ritualistic behaviour to return, but the symptoms diminished after dosage was brought back to 0.1 mg t.i.d.

Yaryura-Tobais and Bhagavan (1977) reported improvement among seven patients with OCD after treatment with L-tryptophan (3 to 9 g daily) for

1 month. The patients' conditions were stabilised after 6 months to 1 year of treatment.

6.10.7 Clinical summary

In conclusion, the paucity of clinical evidence on other pharmacological treatments for OCD limits our confidence in the efficacy of these alternative pharmacological treatments.

6.11 TREATMENT STRATEGIES FOR PATIENTS SHOWING AN INCOMPLETE RESPONSE TO SRIs

6.11.1 Introduction

Although most individuals with OCD experience substantial improvements on first-line treatment with SRI drugs, for many the treatment response is not complete. In about 30% cases residual symptoms remain in spite of prolonged treatment. The clinical management of 'incomplete responders' is an area that has not yet been thoroughly investigated, although there is much interest in the area and treatment-studies indicating promising strategies are already entering the scientific literature.

Research into the area of treatment-resistant OCD has been bedevilled by the lack of universally accepted definitions of treatment-response and treatment-failure. Many of the published studies have used different criteria, making it rather difficult to draw generalised conclusions from them. For example, some studies included only extremely refractory cases whose symptoms had failed to respond to successive treatments with more than one SRI, whereas others included those who had made a partial response to treatment with a single SRI drug. Pallanti and colleagues (2002a) recently proposed criteria based on expert consensus opinion. Specifically, they argued an improvement of ≥35% in baseline Y-BOCS score, or 'much' or 'very much improved' on the CGI Global Improvement, represented a meaningful clinical response, while 'remission' required a total Y-BOCS score of less than 16. Those showing between 25–35% improvement in Y-BOCS scores were considered partial responders. Relapse was defined as a 25% worsening in Y-BOCS (or a CGI score of 6), after a period of remission, and the term 'treatment-refractory' was reserved for those who do not respond to 'all available treatments'. Levels of non-response, according to the numbers of failed treatments, were also defined. It remains to be seen whether these criteria will be universally accepted by the scientific community.

This section reviews research on the pharmacological treatment of individuals who have symptoms failing to completely respond to SRI medication. First there will be a description of current practice, including predictors of treatment failure and definitions of incomplete response. Then the drugs and the studies will be considered,

followed by a narrative review of the studies and a clinical summary encapsulating practice points and areas for further research.

6.11.2 Current practice

In contrast to the large amount of data for first-line treatments for OCD, the evidence base for the treatment of individuals whose symptoms have failed to respond adequately to first-line treatments is rather slim. Indeed, we do not understand clearly which clinical factors might predict a better or worse outcome after pharmacological treatment; few pharmacological studies provide information on response status at outcome and the literature on pharmacological response predictors is sparse and inconsistent. The factor analysis by Mataix-Cols and colleagues (1999) suggested that adults with compulsive rituals, early-onset age, longer duration, chronic course, comorbid tics and personality disorders (especially schizotypal), responded poorly to clomipramine and SSRIs. Additional analyses of large databases for clomipramine and fluoxetine reported better responses for previously SRI-naïve individuals, and poorer responses for those with sub-clinical depression. Those with an earlier age of onset responded poorly to clomipramine, but not to fluoxetine (Ackerman & Greenland, 2002). The more recent analysis of data from a large trial of citalopram also reported that patients with a longer duration of illness or previous SSRI treatment, as well as greater illness severity, were less likely to respond well to drug-treatment (Stein *et al.*, 2001). One small study identified better responses in females (Mundo *et al.*, 1999) but children with comorbid attention deficit hyperactivity disorder, tic disorder and oppositional defiant disorder showed a less favourable response (Geller *et al.*, 2003).

OCD responds to treatment gradually with some patients responding more slowly than others, therefore it is important not to anticipate treatment-failure prematurely. There is no agreed consensus on what constitutes an adequate period of treatment, but published expert consensus guidelines suggest at least 12 weeks (Greist *et al.*, 2003; March *et al.*, 1997) and possibly longer may be required in some cases.

In the UK, a number of strategies are currently employed for individuals who have symptoms that are failing to respond adequately to first-line treatment with SRI drugs. Sometimes the individual is treated for longer on the original drug to see if a delayed response occurs. Alternatively the individual may be switched to an alternative SRI, or the SRI continued with the introduction of a drug of either the same class (that is, two SRIs given together) or a drug of another class added in combination (see below). Sometimes the dose of SRI is increased beyond usual recommended daily-dosage limits, or the mode of delivery is changed, for example to intravenous as opposed to oral delivery. Sometimes the individual is switched to a drug of another class used as a monotherapy. These strategies are usually managed under the guidance of a consultant psychiatrist or his/her team. The evidence for these strategies will be reviewed below. The evidence for those involving only SRIs will be reviewed first, and those involving drugs of other classes will be reviewed later.

6.11.3 Descriptive review of strategies involving SRIs

The studies considered in this section were based on a literature review on the pharmacotherapy of OCD (Fineberg & Gale, 2004).

6.11.3.1 Switching SRI

Given the limitations of data supporting alternative strategies, and the acceptability of switching from one SSRI to another, this remains the preferred option for many clinicians. Sometimes, however, it is appropriate to persist for longer with a particular SRI even in patients who show little sign of improvement, since a delayed response may occur after 6 months or more. A panel of international OCD experts made recommendations on switching (Expert Consensus Guideline, March *et al.*, 1997) and they suggested changing the SRI after 8–12 weeks on the maximal dose if the clinical effect was incomplete. They proposed a 40% chance of responding to a second SRI, and a lesser response to a third and suggested switching to clomipramine after 2–3 failed trials on SSRIs. Since that time, a limited evidence-base supporting switching has accrued, but the data remains sparse and inconclusive. In a placebo-controlled sertraline trial, one third of patients benefited from a switch to a second SSRI (Koran *et al.*, 2002). Denys and colleagues (2004) took 43 non-responders to 12 weeks treatment with either paroxetine or venlafaxine and switched them under double-blind conditions to the alternate treatment, whereupon 18 (43%) showed a clinical response. A single-blind study in 29 cases of SRI-resistant OCD showed encouraging results for venlafaxine (37.5–375 mg) as a monotherapy (Hollander *et al.*, 2003b).

These results intimate that patients whose symptoms have failed to respond to an SSRI may benefit from switching agent, but fail to control for the effect of prolongation of treatment per se. RCTs comparing continuation of the original drug with switching are required to properly evaluate the role of switching in OCD.

6.11.3.2 Increasing dose

Results from uncontrolled case studies suggest that, for some patients, increasing SSRI doses above formulary limits may procure a better effect (Bejerot & Bodlund, 1998; Byerly *et al.*, 1996). Although doses of clomipramine up to 300 mg have been systematically investigated and found to be acceptable, the risk of seizures and cardiotoxicity associated with this drug suggest that doses exceeding this should be generally avoided, and if attempted, ECG and clomipramine plasma-level monitoring should be considered.

6.11.3.3 Changing mode of drug-delivery

Changing mode of administration may be considered though this is not practical in many cases. Intravenous clomipramine has been shown to be more effective than placebo in a single double-blind study investigating refractory OCD, with 6 out of 29 patients randomised to clomipramine classed as responders after 14 daily infusions, compared with none receiving placebo infusions (Fallon *et al.*, 1998). A positive open study showing benefit for 21 days intravenous citalopram has also been reported (Pallanti *et al.*, 2002b) but requires confirmation using a controlled study design.

6.11.4 Intravenous clomipramine versus intravenous placebo (for treatment resistant patients)

6.11.4.1 Clinical evidence statements

Efficacy	***Included studies***
There is limited evidence suggesting a difference favouring intravenous clomipramine over intravenous placebo on the likelihood of treatment response, defined as CGI 'much improved' or 'very much improved' (K = 1; N = 54; RR = 0.79; 95% CI, 0.66 to 0.96). **I**	FALLON1998
Tolerability	
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between intravenous clomipramine and intravenous placebo on the likelihood of leaving the study early (K = 1; N = 54; RR = 0.43; 95% CI, 0.04 to 4.48). **I**	FALLON1998

6.11.4.2 Combining two SRIs

If a patient has symptoms that fail to respond to successive SRI trials, augmented with CBT, the Expert Consensus Guideline (March *et al.*, 1997) recommends adding another agent to the SRI. The evidence is acknowledged to be limited, based on the results of small RCTs and open case series.

Combining clomipramine with an SSRI has been proposed for adults or children unresponsive to, or intolerant of, SRI monotherapy. This strategy should be approached with caution since the pharmacokinetic interactions on the hepatic cytochrome P 450 isoenzymes may lead to a build-up of clomipramine that could be dangerous, and ECG and clomipramine plasma-level monitoring is advisable. Positive results have been reported from small, uncontrolled case series (Szegedi *et al.*, 1996), although the fluoxetine–clomipramine combination resulted in ECG changes in some cases. Pallanti *et al.* (1999) compared 9 treatment-refractory patients given citalopram plus clomipramine with 7 given citalopram alone, in a randomised open-label trial. They reported a significantly larger improvement in Y-BOCS ratings for those given the combination, all of whom experienced decreases ≥35% from baseline. This combination is advantageous in not altering the metabolism of clomipramine, and was well tolerated. No controlled studies of the co-administration of different SSRIs have been published.

6.11.5 Augmentation strategies with drugs of other classes

The following drugs, given in order to improve clinical outcome in individuals who have symptoms that are failing to completely respond to SRI monotherapy, were included:

- Buspirone (added to fluvoxamine)
- Desipramine (added to SSRI)
- Haloperidol (added to fluvoxamine)
- Inositol (added to SRI)
- Lithium (added to fluvoxamine)
- Nortriptyline (added to clomipramine)
- Olanzapine (added to fluoxetine)
- Pindolol (added to paroxetine)
- Quetiapine (added to SRI)
- Risperidone (added to SRI)
- Venlafaxine.

6.11.5.1 Studies considered

The review team conducted a new systematic search to identify studies that used augmentation strategies in the treatment of OCD. The search identified 38 studies that were of interest (Atmaca *et al.*, 2002; Barr *et al.*, 1997; Bejerot & Bodlund, 1998; Blier & Bergeron, 1996; Bogetto *et al.*, 2000; Byerly *et al.*, 1996; Crocq, 2002; D'Amico *et al.*, 2003; Dannon *et al.*, 2000; Denys *et al.*, 2002a; Denys *et al.*, 2004; Francobandiera, 2001; Fux *et al.*, 1999; Grady *et al.*, 1993; Hewlett *et al.*, 1992; Hollander *et al.*, 2003a; Koran *et al.*, 2000; Koran *et al.*, 2001; McDougle & Walsh, 2001; McDougle *et al.*, 1990; McDougle *et al.*, 1991; McDougle *et al.*, 1993; McDougle *et al.*, 1994; McDougle *et al.*, 1995a; McDougle *et al.*, 1995b; McDougle *et al.*, 2000a; Maina *et al.*, 2003; Metin *et al.*, 2003; Mohr *et al.*, 2002; Mundo *et al.*, 1998; Noorbala *et al.*, 1998; Pallanti *et al.*, 1999; Pigott *et al.*, 1991; Pigott *et al.*, 1992a; Pigott *et al.*, 1992b; Sevincok & Topuz, 2003; Shapira *et al.*, 2004; Weiss *et al.*, 1999).

6.11.5.2 Combining SRIs and drugs exerting antidepressant or anxiolytic properties

Controlled studies have found no evidence for the efficacy of lithium augmentation in OCD (McDougle *et al.*, 1991; Pigott *et al.*, 1991). Similarly, three double-blind placebo controlled studies have demonstrated that combining buspirone with an SRI is not helpful (Grady *et al.*, 1993; McDougle *et al.*, 1993; Pigott *et al.*, 1992a). Clonazepam, a benzodiazepine with putative effects on serotonin neurotransmission, failed to impact on the core symptoms of OCD as a monotherapy in one study (Hollander *et al.*, 2003c), but produced some evidence of anti-obsessional effect in another (Hewlett *et al.*, 1992). Pigott and colleagues (1992b) reported limited efficacy for clonazepam given together with fluoxetine or clomipramine in a placebo-controlled study. Pindolol is a beta-blocker which also acts as an antagonist at presynaptic 5HT1A autoreceptors. Dannon and colleagues (2000) demonstrated efficacy for pindolol when combined with paroxetine in a double-blind placebo-controlled study of 14 treatment resistant cases, but another study combining it with fluvoxamine did not (Mundo *et al.*, 1998). Blier and Bergeron (1996) found a beneficial effect for pindolol only when l-tryptophan was openly added to the combination.

The limitations of adding drugs acting on serotonin led investigators to re-examine the role of noradrenergic agents for OCD. While not evidently effective as a monotherapy, nortryptyline (50 mg) administered in combination with 150 mg clomipramine was found to be more effective than clomipramine plus placebo in a small 8-week study looking at 30 individuals with non-refractory OCD (Noorbala *et al.*, 1998). However, Barr and colleagues (1997) investigated the addition of another noradrenergic antidepressant, desipramine, to 20 patients whose symptoms had failed to respond to SSRI monotherapy, in a double-blind placebo-controlled study, and found no added benefit.

6.11.5.3 Combining SRIs and drugs with antipsychotic properties

No positive studies of antipsychotic monotherapy in OCD meet today's standards, and OCD is not recognised as responding to these drugs individually. McDougle and colleagues (1990) reported a benefit from adding open-label pimozide (6.5 mg) in 17 patients unresponsive to fluvoxamine. Patients with comorbid chronic tics or schizotypal disorder were most responsive. A subsequent double-blind placebo-controlled study by the same author demonstrated a significant Y-BOCS improvement for low-dose haloperidol (6.2 mg) added to fluvoxamine. Eleven of 17 patients receiving the active drug achieved 'responder' status by as early as 4 weeks, compared with none on placebo. Again, a preferential response was seen in patients with comorbid tics (McDougle *et al.*, 1994). Antipsychotics such as haloperidol and sulpiride are first-line treatments for Tourette's syndrome, so this finding supports a theoretical link between these disorders. This combination increases the side-effect burden, including extra-pyramidal effects. It is therefore recommended to start treatment with very low doses, and increase cautiously subject to tolerability (for example, 0.25–0.5 mg haloperidol, titrated slowly to 2–4 mg (McDougle & Walsh, 2001).

Newer second-generation antipsychotics that modulate serotonin and dopamine neurotransmission also offer promise and are associated with a lower risk of side effects, although they may cause dysregulation of endocrine and metabolic functions and weight gain. Positive reports from open case series were confirmed by McDougle and colleagues (2000a) in the first reported double-blind placebo-controlled study showing efficacy for risperidone augmentation in 36 patients unresponsive to 12 weeks on an SRI. Risperidone (2.2 mg) was superior to placebo in reducing Y-BOCS scores as well as anxiety and depression, was well-tolerated and there was no difference between those with and without comorbid tics or schizotypy. A smaller double-blind study by Hollander and colleagues (2003a) examined patients who had symptoms that were failing to respond to at least two trials of SRIs. Four of 16 patients randomised to risperidone (0.5–3 mg) turned out to be responders, defined as a CGI Global Improvement score of 1 or 2 and Y-BOCS decrease of >25% at the 8-week end-point, compared with none of the six patients randomised to placebo. However, the results, which were limited by the small sample size, did not reach statistical significance.

Quetiapine has also been the subject of recent controlled investigation. There have been contradictory results from open studies (Denys *et al.*, 2002a; Mohr *et al.*, 2002; Sevincok & Topuz, 2003). However, a placebo-controlled single-blind study looking

at 27 cases of SRI-resistant OCD showed a significant advantage from adding quetiapine in doses up to 200 mg daily to ongoing SRI (Atmaca *et al.*, 2002). Moreover, the recent double-blind placebo-controlled study by Denys and colleagues (2004) showed evidence of efficacy for 8 weeks quetiapine (<300 mg) augmentation in 20 patients whose symptoms had failed to respond to at least two SRIs, showing a mean decrease of 31% on baseline Y-BOCS, compared with 20 placebo-treated controls whose symptoms showed only 7% improvement ($p < 0.001$). Eight out of twenty (40%) patients responded to quetiapine therapy, compared with two out of 20 (10%) on placebo.

Encouraging results from a small number of open-label studies of olanzapine (Bogetto *et al.*, 2000; Crocq, 2002; D'Amico *et al.*, 2003; Francobandiera, 2001; Koran *et al.*, 2000; Weiss *et al.*, 1999) were not, however, supported by the double-blind placebo-controlled study by Shapira and colleagues (2004), which investigated 44 partial or non-responders to 8 weeks of fluoxetine. Both groups improved over the 6-week study period, with no additional advantage for the olanzapine-treated patients compared with extending the duration of fluoxetine monotherapy. Differences in entry criteria between studies may partly explain differences in the results.

There has been a positive open-label study of amisulpride (Metin *et al.*, 2003).

Augmentation with clozapine has not been systematically investigated. The results for clozapine monotherapy in OCD have not been encouraging (McDougle *et al.*, 1995a). Some authors have reported emergent obsessions during treatment with atypical antipsychotics, which may be related to their mixed receptor antagonist properties.

Altogether, these results favour the use of second generation antipsychotics such as risperidone and quetiapine as the first-line strategy for augmentation in resistant OCD. It remains uncertain as to how long patients need to remain on augmented treatment. A small retrospective study by Maina and colleagues (2003) showed that the vast majority of patients who had responded to the addition of an antipsychotic to their SRI, subsequently relapsed when the antipsychotic was withdrawn.

6.11.5.4 Other strategies for refractory OCD

Inositol (18 g/day) is an experimental compound that acts through intracellular messenger systems. It was thought to have mild anti-obsessional efficacy but results from a placebo-controlled augmentation study by Fux and colleagues (1999) refute this. Sumatriptan is a 5HT1D agonist used to treat migraine. A small open case series suggested improvement over 4 weeks of treatment but, in a double-blind placebo-controlled study of 10 patients, 5-day treatment was associated with a worsening of OCD (Koran *et al.*, 2001).

6.11.6 Clinical summary

Treatment-resistant OCD is now receiving systematic evaluation. The first step to managing resistant OCD is to check adherence to the original treatment. In the case of resistance, augmentation with second generation antipsychotic agents appears a promising strategy for which there is some supportive evidence from controlled clinical trials. Other tech-

niques for resistant cases, such as increasing the dose above SPC limits, changing to another SSRI or clomipramine, combining SRIs or changing the mode of drug-delivery are under evaluation. Important questions requiring further investigation include identification of clinically relevant predictors relating to treatment–response and relapse, the clarification of optimal duration of treatment and the evaluation of anti-obsessional treatment in comorbid disorders such as schizophrenia with OCD. Agreed definitions for response, relapse, resistance and refractoriness will improve research in this area.

6.12 PHARMACOLOGICAL INTERVENTIONS FOR CHILDREN AND YOUNG PEOPLE WITH OCD

6.12.1 Treatments included

The following treatments were included:

- SSRIs (fluoxetine, fluvoxamine, sertraline, paroxetine)
- Clomipramine
- Desipramine.

6.12.2 Studies considered

The review team conducted a new systematic search for RCTs that assessed the efficacy of pharmacological interventions among children and young people with OCD. Eighteen published trials were identified, of which four did not meet the inclusion criteria of the GDG. One unpublished trial (Carpenter) was identified. The 14 included studies provided efficacy data from 1034 participants and tolerability data from 1068 participants.

Of the included studies, seven compared acute-phase SSRI treatment with placebo (GELLER2001; GELLER2004; LIEBOWITZ2002; MARCH1998; POTS2004; RIDDLE1992; RIDDLE2001), four compared clomipramine with placebo (DEVEAUGHGEISS1992; FLAMENT1985; MARCH1990; RAPOPORT1980), three compared clomipramine with desipramine (LEONARD1989A; LEONARD1991A; RAPOPORT1980), two examined continuation treatment of SSRIs (CARPENTER; LIEBOWITZ2002), and one study compared continuation of clomipramine treatment with desipramine substitution (LEONARD1991A). Of the seven studies comparing SSRIs with placebo, three examined fluoxetine (GELLER2001; LIEBOWITZ2002; RIDDLE1992), one fluvoxamine (RIDDLE2001), two sertraline (MARCH1998; POTS2004), and one paroxetine (GELLER2004).

All included acute-phase randomised studies were between 8 and 20 weeks long (mean length = 12 weeks). Patients were classified as outpatient in 7 studies, mixed in 3 studies and unclear in four studies. The average age of the patients was 13.2 years. The average duration of illness based on 7 studies was 3.6 years. All studies were conducted in the US, including three studies which were multi-centre studies and one study which was conducted in the US and Canada.

Additional data on suicidal behaviour/ideation was obtained from a review made public by the FDA (Hammad, 2004). The review came about after the FDA commissioned a re-classification of the original patient-level data by experts in suicidality at Columbia University (Iyasu, 2004). The FDA concluded that this blinded classification process identified and corrected many misclassification errors, providing more accurate risk estimates (Hammad, 2004). Four studies were included in the review (GELLER2001; GELLER2004; MARCH1998; RIDDLE2001) providing data on 616 participants.

The results of three cross-over studies (FLAMENT1985; LEONARD1989A; RAPOPORT1980) are summarised in narrative form, as it was not possible to extract data at the point of cross-over.

6.12.3 SSRIs versus placebo

6.12.3.1 Clinical evidence statements

Efficacy	***Included studies***
There is limited evidence suggesting a difference favouring SSRIs over placebo on the likelihood of treatment response, defined as a 25% + 40% + reduction on the Children's Y-BOCS (K = 3; N = 430; RR = 0.70; 95% CI, 0.59 to 0.83). **I**	GELLER2001 GELLER2004 RIDDLE2001
There is limited evidence suggesting a difference favouring SSRIs over placebo on reducing obsessive-compulsive symptoms as measured on the Children's Y-BOCS (K = 7; N = 718; SMD = 20.43; 95% CI, 20.58 to 20.28). **I**	GELLER2001 GELLER2004 LIEBOWITZ2002 MARCH1998 POTS2004 RIDDLE1992 RIDDLE2001
Tolerability	
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between SSRIs and placebo on the risk of suicidal behaviour/ideation (K = 4; N = 616; RR = 1.81; 95% CI, 0.46 to 7.13). **I**[11]	GELLER2001 GELLER2004 MARCH1998 RIDDLE2001

[11]Given the size of the RR and the upper limit of the CI, an increased risk of suicidal behaviour/ideation can not be ruled out.

There is evidence suggesting that SSRIs when compared with placebo increase the risk of leaving the study early due to adverse effects (K = 6; N = 732; RR = 2.74; 95% CI, 1.46 to 5.14). **I**

GELLER2001
GELLER2004
LIEBOWITZ2002
MARCH1998
POTS2004
RIDDLE1992
RIDDLE2001

6.12.4 Clomipramine versus placebo

6.12.4.1 Clinical evidence statements

Efficacy

Included studies

There is limited evidence suggesting a difference favouring clomipramine over placebo on reducing obsessive-compulsive symptoms as measured by the Children's Y-BOCS (K = 1; N = 16; SMD = 20.94; 95% CI, 21.99 to 0.11). **I**

MARCH1990

Tolerability

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine and placebo on the tolerability of treatment.

DEVEAUGHGEISS1992
MARCH1990

6.12.4.2 Results summary from additional cross-over trials

In FLAMENT1985, following a 1-week evaluation period, patients were randomised to 5 weeks of clomipramine or placebo and then crossed-over to 5 weeks of the other treatment. Twenty-three patients entered the drug study and 19 completed the entire cross-over sequence.

Compared with placebo, clomipramine was significantly more effective in relieving obsessional symptoms, as measured on Leyton Obsessional Inventory – Child Version (LOI-CV) symptom (t = 2.19, $p = 0.04$), resistance (t = 2.12, $p = 0.05$) and interference scores (t = 2.24, p = v0.04), the OCR scale (t = 3.05, $p = 0.007$), and the NIMH-OC scale (t = 2.83, $p = 0.02$). Side-effects scores increased markedly during the first week with clomipramine and by week 5 they were significantly higher than with placebo (t = –2.52, $p = 0.02$). One patient experienced a grand mal seizure during clomipramine treatment.

In RAPOPORT1980, patients were randomised to 3–5 week consecutive periods of treatment with clomipramine, desipramine or placebo and then crossed-over to each of the other 2 treatments over 16 weeks. Nine patients entered the study and eight completed the entire cross-over sequence.

At the end of treatment, symptom severity as measured by the Leyton Interference and Resistance subscales was comparable across clomipramine, desipramine and placebo. Side effects did not differ between treatment periods.

6.12.5 Clomipramine versus desipramine substitution

6.12.5.1 Clinical evidence statements

Efficacy	***Included studies***
There is limited evidence suggesting a difference favouring clomipramine continuation over desipramine substitution on the likelihood of relapse as defined by the Physician's Relapse Scale (K = 1; N = 20; RR = 4.89; 95% CI, 1.37 to 17.49). **I**	LEONARD1991A
Tolerability	
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between clomipramine continuation and desipramine substitution on the likelihood of leaving the study early (K = 1; N = 21; RR = 3.27; 95% CI, 0.15 to 72.23). **I**	LEONARD1991A

6.12.5.2 Results summary from additional cross-over trials

In LEONARD1989a, following a 2-week single-blind placebo phase patients were randomised to 5-week consecutive periods of treatment with clomipramine or desipramine and then crossed-over to the other treatment. Forty-nine patients participated in the trial.

At the end of treatment, symptom severity was decreased to a greater extent by clomipramine than desipramine based on the NIMH-OC scale (F = 16.62; $p = 0.002$), Ward OCD Rating scale (F = 21.16; $p = 0.00001$) and CPRS-OC subscale (F = 10.34; $p = 0.003$). There was a significant effect of order of drug, whereby the rate of relapse was greater in the clomipramine-to-desipramine cross-over sequence than the desipramine-to-clomipramine cross-over sequence.

In RAPOPORT1980, patients were randomised to 3–5 week consecutive periods of treatment with clomipramine, desipramine or placebo and then crossed-over to each of the other 2 treatments over 16 weeks. Nine patients entered the study and eight completed the entire cross-over sequence.

At the end of treatment, symptom severity as measured by the Leyton Interference and Resistance subscales was comparable across clomipramine, desipramine and placebo. Side effects did not differ between treatment periods.

6.12.6 SSRI versus placebo: continuation/discontinuation

6.12.6.1 Descriptive review

Two studies examined the continuation of SSRIs in children, CARPENTER and LIEBOWITZ2002.

CARPENTER was an unpublished 32-week two-phase trial of paroxetine. In the first phase, patients received open-label paroxetine for 16 weeks. In the second phase, patients who responded to the first phase were either continued with paroxetine at the final daily dose achieved during the first phase or discontinued from paroxetine onto placebo in a double-blind randomised fashion. The duration of the second phase was 16 weeks. Three hundred and thirty-five patients participated in the phase I trial, of which 194 entered the second phase.

The results indicated that the rate of relapse, defined as (a) an increase in CGI Global Improvement score by 1 point for 2 consecutive visits, (b) an increase in CGI Global Improvement score by 2 or more points at any single visit, and (c) a CGI Global Improvement score of 5 or more points at any time, was not significantly different between patients continuing with paroxetine (34.7%) and patients discontinuing from paroxetine (43.9%). Comparing the response rates between the two groups based on a 25% or greater reduction of CY-BOCS from baseline favoured paroxetine continuation over discontinuation (25% versus 13.3%; RR = 0.86; 95% CI, 0.75 to 0.99). Symptom severity increased in both groups during the continuation phase as measured by the CY-BOCS, though this increase was lower in the paroxetine continuation group than the discontinuation group (SMD = –3.3; 95% CI, –5.77 to –0.83).

In LIEBOWITZ2002, patients who responded to an 8-week acute phase of fluoxetine or placebo were entered into an 8-week maintenance phase. Response was defined as 'much improved' or 'very much improved' on the CGI Global Improvement scale. Patients continued to receive the same drug as during the acute phase and were maintained on the final dose achieved during the acute phase.

Of the 43 patients who entered the acute phase, 18 entered the maintenance phase. At the end of the 8-week maintenance phase, fluoxetine patients had lower CY-BOCS scores than did placebo patients, mean (SD) was 5.64 (5.41) and 11.43 (11.12) respectively, F (1,14) = 9.22; $p = 0.009$. None of the fluoxetine patients relapsed, while one placebo patient relapsed. There were significantly more fluoxetine than placebo responders (57% versus 27%; $\chi^2 = 3.94$; $p = 0.05$). Twenty-seven percent of the fluoxetine patients reported at least one adverse event, compared with none among the placebo patients.

6.12.7 Clinical summary

There is evidence supporting the treatment of OCD in children and young people with SSRIs. The literature is not extensive, but it is consistent in showing beneficial effects in terms of symptom remission and improvements in global functioning. There are always concerns about the use of drugs in young people, particularly medications which act on the developing central nervous system. These potential risks have to be weighed against the known risks of untreated OCD on emotional, educational and social development, and the impact of chronic OCD on adult adaptation. More research is needed in children and young people in the acute phase, in long-term follow-up, and on educational and cognitive progress.

Although the evidence-base is small for psychological treatment, clinical consensus recommends the use of psychological treatment as first-line in young people with OCD. However, in severe or chronic cases, where CBT has been ineffective or is unavailable, or where the patient chooses medication, this is an effective treatment option, either alone or ideally with, CBT.

For OCD, clinical trial evidence, although not extensive, suggests that CBT and SSRI/SRI treatment have similar efficacy. Clinical trial evidence, observational data and yellow card data all show that SSRIs have a range of side effects related to activation syndromes that have not yet been observed or systematically studied in studies of psychological treatments. At the time of writing this guideline, there is concern about the use of SSRIs in children and young people with major depression, because, with the exception of fluoxetine, they show no evidence of clinically significant efficacy whilst being associated with an increased risk of adverse events, particularly suicidal thought and behaviours. In contrast, in children and young people with OCD clinical trials have shown limited evidence for efficacy, although there is insufficient evidence to determine whether or not SSRIs are associated with an increased risk of suicidal thought and behaviours.

6.13 PHARMACOLOGICAL INTERVENTIONS FOR PEOPLE WITH BDD

6.13.1 Introduction

This topic is of interest because of the lack of experience by psychiatrists in treating BDD and the paucity of evidence concerning pharmacotherapy in this area.

6.13.2 Current practice

There are no surveys of how BDD is currently treated or managed in the UK. Our clinical impression is that following an influential case series of pimozide in delusional disorder (Riding & Munro, 1975) many patients are treated with antipsychotic drugs. The popularity of pimozide itself has since dwindled probably because of concerns about its toxicity and the need to perform an ECG prior to its administration. However, this does not seem to have stopped the prescribing of antipsychotic drugs for BDD.

Current practice is not underpinned by a strong evidence base, for example, there are few studies upon which to base clinical decisions, and there are concerns about the generalisability of patient samples.

6.13.3 Treatments included

The following treatments were included:

- Fluoxetine
- Clomipramine
- Desipramine
- Pimozide.

6.13.4 Studies considered

The review team conducted a new systematic search for RCTs that assessed the efficacy of pharmacotherapy in BDD.

Two trials met the eligibility criteria set by the GDG. Phillips and colleagues (2002) entered 74 participants with BDD (including those with beliefs of delusional intensity) into the study. Sixty seven were randomised to either fluoxetine or a placebo for 12 weeks. The range of the dose of fluoxetine was between 40 and 80 mg a day. Fluoxetine was significantly more effective than placebo on the Y-BOCS modified for BDD after 12 weeks of treatment (N = 77; SMD = –0.60; 95% CI, –1.09 to –0.11). The baseline for the fluoxetine group was 31.5 (SD 5.6) reducing to 21.0 (SD 9.8) at 12 weeks, which represented a 33% reduction on the main outcome measure. The rate of response, defined as a 30% or greater decrease from baseline on the Y-BOCS, was greater in fluoxetine than placebo at 12 weeks (N = 77; RR = 0.58; 95% CI, 0.39 to 0.85). Patients with delusional beliefs were as likely as those without delusional beliefs to respond to fluoxetine, and no patients with delusional beliefs responded to the placebo. The effect was independent of comorbid diagnoses of OCD or depression.

There has been one double-blind randomised crossover trial comparing clomipramine with desipramine (Hollander *et al.*, 1999). Forty participants with BDD (including those with beliefs of delusional intensity) were enrolled and 29 were randomised into a 2-week, single-blind run-in, followed by 8 weeks of either clomipramine or 8 weeks of desipramine which was then crossed over. Clomipramine is a potent serotonergic reuptake inhibitor and a tricyclic antidepressant. Desipramine is a potent noradrenergic reuptake inhibitor and another tricyclic. Both clomipramine and desipramine led to a reduction in the severity of obsessive-compulsive symptoms from baseline to endpoint as indicated by the BDD-YBOCS scores, baseline BDD-YBOCS was 25.4 (SD 7.2), BDD-YBOCS following clomipramine treatment was 16.2 (SD 8.5) and following desipramine treatment was 20.7 (SD 7.7). The reduction in symptom severity was significantly greater following clomipramine treatment than following desipramine treatment (F 1, 21 = 11.03; $p = 0.003$). Response rates were 65% for clomipramine and 35% for desipramine, based on 25% improvement on the BDD-YBOCS ($p = 0.09$). Treatment efficacy was not influenced by comorbidity of OCD, depression or social phobia.

There are limitations to this trial, including a lack of a placebo arm, a maintenance phase after the crossover and potential carry over effects, which are inherent in

crossover designs. There is some evidence that the response may have been greater if a higher dose of clomipramine (mean 138 mg/day) was used and for a longer duration (at least 12 to 16 weeks).

6.13.5 SSRI augmentation

Phillips (2005) has conducted an RCT of pimozide augmentation of fluoxetine. Twenty-eight people with BDD (including those with beliefs of delusional intensity) whose symptoms had failed to respond to fluoxetine participated in an 8-week double-blind study of up to 10 mg pimozide or placebo augmentation of fluoxetine. Pimozide was no more effective than placebo; 18.2% of subjects responded to pimozide and 17.6% to placebo. There was no significant effect of baseline delusionality on endpoint BDD severity. Delusionality did not decrease significantly more with pimozide than placebo. Possible explanations of the lack of efficacy include the study's low power and the modest mean pimozide dose. In OCD, augmentation of an SSRI with pimozide and haloperidol has found higher response rates in patients with a tic disorder or schizotypal personality disorder. No BDD subject in this study had either additional diagnosis.

6.13.6 Descriptive review

6.13.6.1 Antidepressants

SRIs other than fluoxetine or clomipramine may be of benefit both theoretically and from the evidence of four case series. There are similar modest benefits from four open label trials. There are two open label case series of fluvoxamine (Perugi *et al.*, 1996; Phillips *et al.*, 1998), one of citalopram (Phillips & Najjar, 2003) and one case series of clomipramine (Hollander *et al.*, 1989).

Phillips and colleagues (1998) entered 30 participants with BDD who received fluvoxamine over 16 weeks. The average dose was 238.8 mg and the range was 50 to 300 mg. The Y-BOCS modified for BDD decreased from 31.1 (SD 5.4) at baseline to 16.9 (SD 11.8) at 16 weeks. This represents a 45% reduction on the main outcome measure. Sixty-three per cent of participants responded based on a criterion of a 30% decrease or more on the Y-BOCS modified for BDD. Fluvoxamine was as effective in participants with an additional diagnosis of delusional disorder as without.

Perugi and colleagues (1996) entered 15 participants with BDD in an open label trial. The duration was for 10 weeks. The average dose was 208 mg and the range of dose was 100 to 300 mg. They did not use the modified Y-BOCS as an outcome measure but reported a 60% reduction over 12 weeks on symptom scores and 10 out of the 15 participants responding on the CGI.

Phillips and Najjar (2003) entered 15 participants with BDD (including those with beliefs of delusional intensity) in an open label study of citalopram over 12 weeks. The average dose was 51.3 mg and the range was 10 to 60 mg. The Y-BOCS modi-

fied for BDD decreased from 30.7 (S.D 4.9) at baseline to 15.3 (SD 10.6) at 12 weeks. This represents a 50% reduction on the main outcome measure. 73.3% of participants responded defined as a 30% decrease or more on the Y-BOCS modified for BDD. Citalopram was as effective in participants with an additional diagnosis of delusional disorder as without.

Hollander and colleagues (1989) reported on a case series of five participants with BDD who all responded to either clomipramine or fluoxetine. Four of the five patients' symptoms had failed to respond previously to drugs that had some serotonergic action including tricyclics, trazodone and lithium.

Phillips (1996b) conducted a retrospective case review of 130 patients who had 316 treatment trials of which 42% of 65 SRI trials had led to an improvement on the CGI, compared with 30% of 23 trials with a monoamine-oxidase inhibitor and 15% of 48 trials with a non-SRI tricyclic drug.

No continuation, maintenance or discontinuation studies of an SRI have been reported. Expert opinion and clinical experience suggest that, like OCD, there may be small further gains with an SRI after 12–16 weeks treatment. Furthermore, like OCD, there is a high rate of relapse on discontinuation on a SRI (Phillips *et al.*, 2001).

Lastly there are case reports on the benefit of sertraline (El-Khatib & Dickey, 1995) and two cases with intravenous clomipramine (Pallanti & Koran, 1996). There are two case reports of the benefit of an MAOI, tranlcypromine (Jenike, 1984) and one with a combination of phenelzine, trimipramine and perphenazine (Phillips, 1991).

6.13.6.2 Antipsychotic drugs as a monotherapy

Antipsychotic drugs were prescribed for BDD following a case series describing the benefit of pimozide in individuals with delusional disorder (Riding & Munro, 1975). However the case series included cases of delusions of infestation, delusions of body odour and dysmorphic delusions.

Phillips and colleagues (1996b), in a retrospective survey of medication trials, reported that only 3% of 83 trials of an antipsychotic were of any benefit in BDD. Grant (2001) has described one case report of olanzapine for BDD without delusional disorder. The individual however fulfilled diagnostic criteria for BDD, alcohol dependence and bipolar II disorder. At the end of 3 weeks, she reported no preoccupation with her appearance and no longer met criteria for BDD.

6.13.6.3 SSRI switching or augmentation studies

In individuals with BDD whose symptoms have failed to respond to an SSRI, or who have a partial response to an SSRI, then switching to another SSRI or augmentation with another drug has been tried.

Phillips and colleagues (2001) reported that in those subjects whose symptoms failed to respond to an adequate SRI trial, 42.9% (n = 6) responded to at least one subsequent SRI trial and 43.5% (n = 10) of subsequent SRI trials received by these subjects were effective.

Phillips and colleagues (2001) reported on an open label series of buspirone (extended from Phillips, 1996a) in a chart review of patients whose symptoms failed to respond to an SRI alone or have had only a partial response. In 12 participants, buspirone was added after an adequate does of an SRI; 33.3% of trials were successful. The mean dose was 56.5 mg (range 30–80 mg daily) and was as effective in delusional as non-delusional cases.

6.13.6.4 Young people with BDD

Albertini and Phillips (1999) reported on 33 children and young people with BDD of whom 10 out 19 (53%) treated with an SRI improved on the CGI. No non-SRI medications in 8 trials were effective.

There is one case report on clomipramine in an adolescent with BDD with delusional disorder (Sondheimer, 1988) and one case report of doxepine and behaviour therapy in an adolescent with BDD (Sobanski & Schmidt, 2000).

6.13.7 Clinical summary

The only placebo-controlled RCT in BDD suggested benefit from fluoxetine or clomipramine in BDD. There are also several case series of other SRIs that support this finding.

No evidence exists on the optimal dose of an SRI in BDD but expert opinion is that SRIs in BDD may have a dose response relationship similar to OCD and that the maximum tolerated dose should be tried.

No evidence exists on the optimal duration of a trial of an SRI but expert opinion suggests that at least 12 weeks is required. An SRI is however associated with a high rate of relapse on discontinuation (similar to the treatment of OCD) although this has not been formally evaluated.

There is no evidence for the efficacy of an antipsychotic in BDD as a monotherapy or as augmentation strategy with an SRI. However, an antipsychotic may still be useful for the symptomatic treatment of individuals with BDD who are highly agitated.

There is no evidence for the benefit of MAOIs, non-SRI tricyclics, or atypical SRIs as a monotherapy for BDD. There is no evidence for the benefit or electroconvulsive therapy (ECT) or psychosurgery in BDD.

No studies have yet compared a SRI with CBT or a combination of the two treatments. Expert opinion suggests that the combination of CBT with an SRI is helpful in moderate to severe BDD.

Limited evidence suggests that SSRIs can be effective in children and young people with BDD with a similar response to adults with BDD and young people with OCD. However no evidence exists for the safety of SSRIs in children and young people with BDD.

6.14 CLINICAL PRACTICE RECOMMENDATIONS

6.14.1 Initial treatment options

Children and young people

6.14.1.1 If psychological treatment is declined by children or young people with OCD or BDD and their families or carers, or they are unable to engage in treatment, an SSRI may be considered with specific arrangements for careful monitoring for adverse events. **[B]**

6.14.2 How to use pharmacological interventions for adults

Current published evidence suggests that SSRIs are effective in treating people with OCD and BDD, although evidence for the latter is limited and less certain. However, SSRIs may increase the risk of suicidal ideas and self-harm in people with depression and in younger people. It is currently unclear whether there is an increased risk for people with OCD or BDD. Regulatory authorities recommend careful monitoring, especially when initiating treatment and around dose changes. Patients should also be warned about, and monitored for, relapse and discontinuation/withdrawal symptoms when stopping or reducing SSRIs.

Starting the treatment

6.14.2.1 Common concerns about taking medication for OCD or BDD should be addressed. Patients should be advised, both verbally and with written material, that:

- craving and tolerance do not occur **[C]**
- there is a risk of discontinuation/withdrawal symptoms on stopping, missing doses, or reducing the dose **[C]**
- there is a range of potential side effects, including worsening anxiety, suicidal thoughts and self-harm, which need to be carefully monitored, especially in the first few weeks of treatment **[C]**
- there is commonly a delay in the onset of effect of up to 12 weeks, although depressive symptoms improve more quickly **[C]**
- taking medication should not be seen as a weakness. **[GPP]**

Monitoring risk

6.14.2.2 Adults with OCD or BDD started on SSRIs who are not considered to be at increased risk of suicide or self-harm should be monitored closely and seen on an appropriate and regular basis. The arrangements for monitoring should be agreed by the patient and the healthcare professional, and recorded in the notes. **[GPP]**

6.14.2.3 Because of the potential increased risk of suicidal thoughts and self-harm associated with the early stages of SSRI treatment, younger adults (younger than age 30 years) with OCD or BDD, or people with OCD or

BDD with comorbid depression, or who are considered to be at an increased risk of suicide, should be carefully and frequently monitored by healthcare professionals. Where appropriate, other carers – as agreed by the patient and the healthcare professional – may also contribute to the monitoring until the risk is no longer considered significant. The arrangements for monitoring should be agreed by the patient and the healthcare professional, and recorded in the notes. **[C]**

6.14.2.4 For adults with OCD or BDD at a high risk of suicide, a limited quantity of medication should be prescribed. **[C]**

6.14.2.5 When adults with OCD or BDD, especially with comorbid depression, are assessed to be at a high risk of suicide, the use of additional support such as more frequent direct contacts with primary care staff or telephone contacts should be considered, particularly during the first weeks of treatment. **[C]**

6.14.2.6 For adults with OCD or BDD, particularly in the initial stages of SSRI treatment, healthcare professionals should actively seek out signs of akathisia or restlessness, suicidal ideation and increased anxiety and agitation. They should also advise patients to seek help promptly if symptoms are at all distressing. **[C]**

6.14.2.7 Adults with OCD or BDD should be monitored around the time of dose changes for any new symptoms or worsening of their condition. **[C]**

Choice of drug treatment

Selective serotonin reuptake inhibitors (SSRIs)

6.14.2.8 For adults with OCD, the initial pharmacological treatment should be one of the following SSRIs: fluoxetine, fluvoxamine, paroxetine, sertraline, or citalopram. **[A]**

6.14.2.9 For adults with BDD (including those with beliefs of delusional intensity), the initial pharmacological treatment should be fluoxetine because there is more evidence for its effectiveness in BDD than there is for other SSRIs. **[B]**

6.14.2.10 In the event that an adult with OCD or BDD develops marked and/or prolonged akathisia, restlessness or agitation while taking an SSRI, the use of the drug should be reviewed. If the patient prefers, the drug should be changed to a different SSRI. **[C]**

6.14.2.11 Healthcare professionals should be aware of the increased risk of drug interactions when prescribing an SSRI to adults with OCD or BDD who are taking other medications. **[GPP]**

6.14.2.12 For adults with OCD or BDD, if there has been no response to a full course of treatment with an SSRI, healthcare professionals should check that the patient has taken the drug regularly and in the prescribed dose and that there is no interference from alcohol or substance use. **[GPP]**

6.14.2.13 For adults with OCD or BDD, if there has not been an adequate response to a standard dose of an SSRI, and there are no significant side effects after 4–6 weeks, a gradual increase in dose should be considered in line with the schedule suggested by the Summary of Product Characteristics. **[C]**

6.14.2.14 For people with OCD or BDD, the rate at which the dose of an SSRI should be increased should take into account therapeutic response, adverse effects and patient preference. Patients should be warned about, and monitored for, the emergence of side effects during dose increases. **[GPP]**

6.14.2.15 If treatment for OCD or BDD with an SSRI is effective, it should be continued for at least 12 months to prevent relapse and allow for further improvements. **[C]**

6.14.2.16 When an adult with OCD or BDD has taken an SSRI for 12 months after remission (symptoms are not clinically significant and the person is fully functioning for at least 12 weeks), healthcare professionals should review with the patient the need for continued treatment. This review should consider the severity and duration of the initial illness, the number of previous episodes, the presence of residual symptoms, and concurrent psychosocial difficulties. **[GPP]**

6.14.2.17 If treatment for OCD or BDD with an SSRI is continued for an extended period beyond 12 months after remission (symptoms are not clinically significant and the person is fully functioning for at least 12 weeks), the need for continuation should be reviewed at regular intervals, agreed between the patient and the prescriber, and written in the notes. **[GPP]**

6.14.2.18 For adults with OCD or BDD, to minimise discontinuation/withdrawal symptoms when reducing or stopping SSRIs, the dose should be tapered gradually over several weeks according to the person's need. The rate of reduction should take into account the starting dose, the drug half-life and particular profiles of adverse effects. **[C]**

6.14.2.19 Healthcare professionals should encourage adults with OCD or BDD who are discontinuing SSRI treatment to seek advice if they experience significant discontinuation/withdrawal symptoms. **[C]**

Other drugs

With the exception of clomipramine, other antidepressants should not normally be used in the treatment of OCD or BDD. Most other drugs have limited or no use in this context.

6.14.2.20 The following drugs should not normally be used to treat OCD or BDD without comorbidity:

- Tricyclic antidepressants other than clomipramine
- Tricyclic-related antidepressants
- Serotonin and noradrenaline reuptake inhibitors (SNRIs), including venlafaxine
- Monoamine-oxidase inhibitors (MAOIs)
- Anxiolytics (except cautiously for short periods to counter the early activation of SSRIs). **[C]**

6.14.2.21 Antipsychotics as a monotherapy should not normally be used for treating OCD. **[C]**

6.14.2.22 Antipsychotics as a monotherapy should not normally be used for treating BDD (including beliefs of delusional intensity). **[C]**

6.14.3 How to use clomipramine for adults

Clomipramine can be offered as a second line drug for OCD/BDD. Always do an ECG and check blood pressure before starting treatment if there is significant risk of cardiovascular disease. Dose changes should be gradual.

6.14.3.1 For adults with OCD or BDD who are at a significant risk of suicide, healthcare professionals should only prescribe small amounts of clomipramine at a time because of its toxicity in overdose. The patient should be monitored regularly until the risk of suicide has subsided. **[GPP]**

6.14.3.2 An electrocardiogram (ECG) should be carried out and a blood pressure measurement taken before prescribing clomipramine for adults with OCD or BDD at significant risk of cardiovascular disease. **[C]**

6.14.3.3 For adults with OCD or BDD, if there has not been an adequate response to the standard dose of clomipramine, and there are no significant side effects, a gradual increase in dose should be considered in line with the schedule suggested by the Summary of Product Characteristics. **[C]**

6.14.3.4 For adults with OCD or BDD, treatment with clomipramine should be continued for at least 12 months if it appears to be effective and because there may be further improvement. **[B]**

6.14.3.5 For adults with OCD or BDD, when discontinuing clomipramine, doses should be reduced gradually in order to minimise potential discontinuation/withdrawal symptoms. **[C]**

6.14.4 How to use pharmacological treatments in children and young people

In adults with OCD treated by medication, there is clinical trial evidence that supports practice on the onset of therapeutic response, dose needed, rate of increase of dose, duration of treatment, likelihood of relapse on discontinuation. Trials of these aspects have not been done in children and/or young people, but the following good practice for prescribing SSRIs or clomipramine is based on adult trials and clinical experience.

How to use SSRIs in children and young people

6.14.4.1 When an SSRI is prescribed to children and young people with OCD or BDD, it should be in combination with concurrent CBT (including ERP). If children and young people are unable to engage with concurrent CBT, specific arrangements should be made for careful monitoring of adverse events and these arrangements should be recorded in the notes. **[C]**

6.14.4.2 Children and young people with OCD or BDD starting treatment with SSRIs should be carefully and frequently monitored and seen at an appropriate and regular basis. This should be agreed by the patient, his or her family or carers and the healthcare professional, and recorded in the notes. **[GPP]**

6.14.4.3 An SSRI should only be prescribed to children and young people with OCD or BDD following assessment and diagnosis by a child and adolescent psychiatrist who should also be involved in decisions about dose changes and discontinuation. **[GPP]**

6.14.4.4 A licensed medication (sertraline or fluvoxamine) should be used when an SSRI is prescribed to children and young people with OCD, except in patients with significant comorbid depression when fluoxetine should be used, because of current regulatory requirements. **[A]**

6.14.4.5 Fluoxetine should be used when an SSRI is prescribed to children or young people with BDD. **[C]**

6.14.4.6 For children and young people with OCD or BDD who also have significant depression, the NICE recommendations for the treatment of childhood depression[12] should be followed and there should be specific monitoring for suicidal thoughts or behaviours. **[GPP]**

6.14.4.7 Children and young people with OCD or BDD starting treatment with SSRIs should be informed about the rationale for the drug treatment, the delay in onset of therapeutic response (up to 12 weeks), the time course of treatment, the possible side effects and the need to take the medication as prescribed. Discussion of these issues should be supplemented by written information appropriate to the needs of the child or young person and their family or carers. **[GPP]**

6.14.4.8 The starting dose of medication for children and young people with OCD or BDD should be low, especially in younger children. A half or quarter of the normal starting dose may be considered for the first week. **[C]**

6.14.4.9 If a lower dose of medication for children and young people with OCD or BDD is ineffective, the dose should be increased until a therapeutic response is obtained, with careful and close monitoring for adverse events. The rate of increase should be gradual and should take into account the delay in therapeutic response (up to 12 weeks) and the age of the patient. Maximum recommended doses for children and young people should not be exceeded. **[C]**

6.14.4.10 Children and young people prescribed an SSRI and their families or carers, should be informed by the prescribing doctor about the possible appearance of suicidal behaviour, self-harm or hostility, particularly at the beginning of treatment. They should be advised that if there is any sign of new symptoms of these kinds, they should make urgent contact with their medical practitioner. **[GPP]**

6.14.4.11 Where children or young people with OCD or BDD respond to treatment with an SSRI, medication should be continued for at least 6 months post-remission (i.e. symptoms are not clinically significant and the child or young person is fully functioning for at least 12 weeks). **[C]**

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[12] 'Depression in children: identification and management of depression in children and young people in primary care and specialist services' (NICE, September 2005).

How to use clomipramine in young people

6.14.4.12 Children and young people with OCD or BDD and their families or carers should be advised about the possible side effects of clomipramine, including toxicity in overdose. **[C]**

6.14.4.13 Before starting treatment with clomipramine in children and young people with OCD or BDD, an ECG should be carried out to exclude cardiac conduction abnormalities. **[C]**

6.14.4.14 For a child or young person with OCD or BDD, if there has not been an adequate response to the standard dose of clomipramine, and there are no significant side effects, a gradual increase in dose may be cautiously considered. **[C]**

6.14.4.15 Treatment of a child or young person with OCD or BDD with clomipramine should be continued for at least 6 months if the treatment appears to be effective, because there may be further improvement in symptoms. **[B]**

Stopping or reducing SSRIs and clomipramine in children and young people

6.14.4.16 In children and young people with OCD or BDD, an attempt should be made to withdraw medication if remission has been achieved (i.e. symptoms are no longer clinically significant and the child or young person is fully functioning) and maintained for at least 6 months, and if this is their wish. Patients and their family or carers should be warned that relapse and/or discontinuation/withdrawal symptoms may occur. They should be advised to contact their medical practitioner should symptoms of discontinuation/withdrawal arise. **[C]**

6.14.4.17 For children and young people with OCD or BDD, to minimise discontinuation/withdrawal reactions on reducing or stopping antidepressants, particularly SSRIs, the dose should be tapered gradually over several weeks according to the individual's need. The rate of reduction should take into account the starting dose, the drug half-life and particular profiles of adverse effects. **[C]**

6.14.4.18 Children and young people with OCD or BDD should continue with psychological treatment throughout the period of drug discontinuation because this may reduce the risk of relapse. **[C]**

Other drugs

6.14.4.19 Tricyclic antidepressants other than clomipramine should not be used to treat OCD or BDD in children and young people. **[C]**

6.14.4.20 Other antidepressants (MAOIs, SNRIs) should not be used to treat OCD or BDD in children and young people. **[C]**

6.14.4.21 Antipsychotics should not be used alone in the routine treatment of OCD or BDD in children or young people, but may be considered as an augmentation strategy. **[C]**

7. COMBINED INTERVENTIONS AND INTENSIVE INTERVENTIONS

7.1 INTRODUCTION

There have been many advances in the treatment of OCD over the past 35 years, with the development of effective pharmacological and psychological treatments for a disorder that was previously considered extremely refractory to treatment (Black, 1974). Despite the overall success of these treatments, the situation is far from ideal (Foa *et al.*, 2000). The acute efficacy of SRI-based pharmacotherapy is still moderate both in terms of the proportion of people who respond and their average response; a significant number report adverse effects, and many relapse on discontinuing medication. Likewise, the acute efficacy of ERP-based treatments is also moderate both in terms of the proportion of people who respond and their average response. The long-term maintenance of gains is unknown, and a significant number refuse treatment or do not complete it. Given the moderate efficacy of both treatment types, their differential and combined effects become a question of obvious interest. From the point of view of the person with OCD, the ability to make informed choices depends on information on the relative efficacies, possible adverse effects, durability of effects and availability of treatments and their combinations. From the point of view of healthcare providers, knowledge of efficacy and effectiveness is required in order to make decisions about which resources to provide.

7.2 PSYCHOLOGICAL VERSUS PHARMACOLOGICAL INTERVENTIONS

7.2.1 Introduction

There have been at least five English language meta-analyses addressing the comparison but some of these have contrasted arms from different studies, in some cases using different measures, in order to address this question (Abramowitz, 1997; Christensen *et al.*, 1987; Cox *et al.*, 1993; Kobak *et al.*, 1998; van Balkom *et al.*, 1994). These reviews have not reached any consistent conclusions. Although there has been a keen debate between proponents of psychological and biological approaches to understanding OCD and its treatment, as yet there is no unified theory that can readily accommodate the key elements of both approaches of OCD. However, a broad biopsychosocial framework is accepted by many, at least for the treatment of OCD. Beyond its academic interest, the question of differential efficacy

has important implications for people with OCD, their families and carers, and health care professionals.

7.2.2 Current practice

With the advent of SSRIs that are generally better tolerated than clomipramine and better known by medical practitioners given their use for a range of disorders, pharmacotherapy has become more widely available. People are often offered pharmacotherapy in primary care, whether or not they are referred on to secondary or tertiary care. Despite the fact that many professionals are trained each year in cognitive behavioural therapies, there is an increasing demand on therapists as CBT becomes indicated for a greater range of disorders and waiting lists for psychological services are common (Lovell & Richards, 2000). Although there is increasing provision of mental health resources in primary care, there are relatively few professionals who have currently received training to provide the focused and structured treatments that are required for OCD. Consequently, many people do not have ready access to CBT of any type. Although some people are at least eventually able to choose when they obtain access to both, in a proportion of cases the treatment received may be determined more by referral patterns and availability of resource rather than by informed choice.

7.2.3 Studies considered[13]

The review team conducted a new systematic search for RCTs that assessed the efficacy of alternative (psychological versus pharmacological) or combination interventions among adults and children/young people with OCD. Six studies were identified, of which two studies (HEMBREE2003; OCONNOR1999) did not meet inclusion criteria. Of the four included studies, two studies were on adults with OCD (FOA2005; MARKS1980), while two studies were on children and young people with OCD (DEHAAN1998; POTS2004). The studies on adults with OCD provided efficacy data on 104 patients and tolerability data on 84 participants (FOA2005). The studies on children and young people with OCD provided efficacy and tolerability data on 79 participants (DEHAAN1998; POTS2004).

The studies involving adults compared ERP with clomipramine (FOA2005) and ERP and placebo with relaxation and clomipramine (MARKS1980). The studies involving children compared CBT with clomipramine (DEHAAN1998) and CBT with sertraline (POTS2004).

[13]Here and elsewhere in the guideline, each study considered for review is referred to by a study ID (primary author and date of study publication in capital letters, except where a study is *in press* or only submitted for publication, then a date is not used).

The included studies were 12 weeks long, except MARKS1980, which was 10 weeks long though data from this study was extracted at the 7-week time-point when all patients receiving relaxation were switched to exposure. In the adult studies, the average age of the participants was 35 years and the mean duration of illness was 14 years. The studies did not report the mean final dosage.

In the studies on children and young people, participants were classified as outpatient. Participants received a maximum dosage of the drug of 200 mg per day and a mean final dosage of the drug of 196 mg per day. The average age of the participants ranged from 12–18 years across studies. Comorbid conditions included anxiety disorder, eating disorder and tic disorder.

Full details of the studies included in the guideline and the reasons for excluding studies are given in Appendix 16.

7.2.4 Behaviour therapy versus clomipramine

7.2.4.1 Clinical evidence statements[14]

Efficacy[15]	***Included studies***
There is limited evidence suggesting a difference favouring ERP over clomipramine on reducing obsessive-compulsive symptoms as measured on the Y-BOCS or the Compulsive Checklist (K = 2; N = 68; SMD = 20.67; 95% CI, 21.16 to 20.17). **I**	FOA2005 MARKS1980
Tolerability	
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP and clomipramine on the likelihood of leaving the study early (K = 1; N = 84; RR = 1.02; 95% CI, 0.62 to 1.67). **I**	FOA2005

7.2.5 CBT versus clomipramine (children and young people)

7.2.5.1 Clinical evidence statements

Efficacy	***Included studies***
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBT and clomipramine on the efficacy of treatment.	DEHAAN1998

[14]The full list of all evidence statements generated from meta-analyses (and the associated forest plots) are available on the CD-ROM that accompanies the guideline. Where a meta-analysis was not possible (or not appropriate), a summary of the results from each study used to generate a statement can be found in Appendix 18.

[15]In the case of SMD or WMD, negative effect sizes favour the treatment group.

Tolerability

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBT and clomipramine on the likelihood of leaving the study early (K = 1; N = 23; RR = 2.36; 95% CI, 0.11 to 52.41). **I**	DEHAAN1998

7.2.6 CBT versus sertraline (children and young people)

7.2.6.1 Clinical evidence statements

Efficacy	***Included studies***
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBT and sertraline on the efficacy of treatment.	POTS2004

Tolerability

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBT and sertraline on the likelihood of leaving the study early (K = 1; N = 56; RR = 1.5; 95% CI, 0.27 to 8.3). **I**	POTS2004

7.2.7 Clinical summary

Based on the two head-to-head comparisons for adults with OCD reviewed here, there is some evidence for greater efficacy of ERP over clomipramine. The results on tolerability were inconclusive. There are no comparisons for SSRIs. These results would argue for ERP being offered to all adults with OCD, although ultimately it will depend on patient preference.

The evidence for children and young people is inconclusive for both efficacy and tolerability based on the two studies reported here. Given concerns about the safety of SSRIs and clomipramine for children and young people, CBT should be offered as initial treatment. When CBT is not available or when the young person and their family prefer, SRIs may be considered.

7.3 COMBINATION INTERVENTIONS

7.3.1 Introduction

Despite numerous studies of both CBT and SRIs, there are relatively few that have investigated the combination of both. This is somewhat surprising given that both are only partially effective, many people relapse on discontinuing SRIs, and some people cannot tolerate CBT, especially the ERP component because of anxiety.

Consequently it is important to investigate whether the combination can increase efficacy and overcome some of the limitations of each treatment.

7.3.2 Current practice

The Expert Consensus Panel for OCD (March *et al.*, 1997) compiled guidelines with recommendations on a comprehensive range of issues relating to pharmacological and psychological treatments. They concluded that CBT should be the first line treatment for mild OCD for adults and young people and for children (regardless of severity). Combined SRI and CBT should be the first line treatment for moderate to severe OCD for both adults and young people, but not children. However, it is not clear at present what the optimal timing should be for introducing each treatment (Foa *et al.*, 2002a).

It is likely that in the UK many people with OCD do indeed receive combined treatment, especially for moderate to severe OCD, but due to availability of CBT this may be offered sequentially and may happen in a relatively unplanned manner rather than by explicit decision and careful planning. In some cases there may be little coordination between the various professionals involved in care who may be in different services with different levels of experience and knowledge of the treatment of OCD, particularly of the other treatment modality. In other cases, especially in integrated multidisciplinary teams who can provide both treatments, the combined treatment is more likely to be explicitly planned and better coordinated.

7.3.3 Studies considered

The review team conducted a new systematic search for RCTs that assessed the efficacy and tolerability of combination interventions among adults and children with OCD. Eighteen studies were identified, of which eleven did not meet the eligibility criteria of the GDG. Five studies had no extractable data (KASVIKIS1988A; MARKS1988; MAWSON1982; OSULLIVAN1991; PETER2000), three studies were based on analyses from other studies (COTTRAUX1993; KASVIKIS1988; LAX1992), one study included patients with phobic neurosis (AMIN1977), one study was not an RCT (HEMBREE2003) and in another study patients were not properly randomised to treatment groups (OCONNOR1999). The seven included studies (COTTRAUX1990; FOA2005; HOHAGEN1998; MARKS1980; NEZIROGLU2000; POTS2004; VANBALKOM1980) provided efficacy data on 470 participants and tolerability data on 469 participants.

All five included studies on adults featured behaviour therapy (BT) and serotonin reuptake inhibitor (SRI) combinations (COTTRAUX1990; HOHAGEN1998; MARKS1980; FOA2005; VANBALKOM1998). One study also featured a cognitive therapy (CT) and fluvoxamine combination (VANBALKOM1998). Of the two studies on children and young people, one study (NEZIROGLU2000) featured a behaviour therapy and fluvoxamine combination intervention, while the other study (POTS2004) featured a CBT and sertraline combination intervention.

Six studies were between 8 and 24 weeks long (mean length = 13 weeks), while one study was 1 year long (NEZIROGLU2000). In four studies participants were classified as outpatient, in one study as inpatient, in one study as mixed and in one study it was unclear. In the adult studies, the mean age of the participants was 35 years. The duration of illness based on four studies on adults was 13 years. The average age of the participants in the studies on children and young people was 13 years. Three studies were conducted in the US, one each in the UK, France, Germany and the Netherlands. Participants receiving fluvoxamine received up to 300 mg per day and participants receiving clomipramine received a mean final dose of 180 mg per day. Participants in the children study received up to 200 mg of the drug per day.

Full details of the studies included in the guideline and the reasons for excluding studies are given in Appendix 16.

7.3.4 BT + SRIs versus BT

7.3.4.1 Clinical evidence statements

Efficacy	***Included studies***
There is limited evidence suggesting a difference favouring multimodal BT + fluvoxamine over multimodal BT + placebo on the likelihood of treatment response, defined as a 35% or greater reduction on the Y-BOCS (K = 1; N = 49; RR = 0.31; 95% CI, 0.10 to 1.00). **I**	HOHAGEN1998
There is limited evidence suggesting a difference favouring BT + SRIs over BT on reducing obsessive-compulsive symptoms as measured by the Y-BOCS (K = 3; N = 126; SMD = 20.37; 95% CI, 20.72 to 20.01). **I**	HOHAGEN1998 FOA2005 VANBALKOM1998
There is limited evidence suggesting a difference favouring BT + SRIs over BT on reducing compulsive symptoms as measured by the Compulsive Activity Checklist (K = 2; N = 51; SMD = 20.55; 95% CI, 21.12 to 0.01). **I**	COTTRAUX1990 MARKS1980
There is limited evidence suggesting a difference favouring BT + SRIs over BT on reducing the time spent performing rituals (K = 2; N = 51; SMD = –0.81; 95% CI, –1.38 to –0.23). **I**	COTTRAUX1990 MARKS1980
There is limited evidence suggesting a difference favouring BT + SRIs over BT on reducing depressive symptoms (K = 4; N = 137; SMD = 20.73; 95% CI, 21.08 to –0.38). **I**	COTTRAUX1990 HOHAGEN1998 MARKS1980 VANBALKOM1998

Tolerability

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between BT + SRI combinations and BT on the tolerability of treatment. — COTTRAUX1990, FOA2005, VANBALKOM1998

7.3.5 BT + clomipramine versus clomipramine

7.3.5.1 Clinical evidence statements

Efficacy — ***Included studies***

There is limited evidence suggesting a difference favouring ERP and clomipramine over clomipramine on reducing obsessive-compulsive symptoms as measured by the Y-BOCS (K = 1; N = 45; SMD = 20.63; 95% CI, –1.23 to –0.03). **I** — FOA2005

There is limited evidence suggesting a difference favouring ERP plus clomipramine over clomipramine on the likelihood of response, defined as CGI 'much improved' or 'very much improved' (K = 1; N = 80; RR = 0.53; 95% CI, 0.33 to 0.87). **I** — FOA2005

Tolerability

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP + clomipramine and clomipramine on the tolerability of treatment. — FOA2005

7.3.6 ERP + fluvoxamine versus anti-ERP + fluvoxamine

7.3.6.1 Clinical evidence statements

Efficacy — ***Included studies***

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP + fluvoxamine and anti-ERP + fluvoxamine on the efficacy of treatment. — COTTRAUX1990

Tolerability

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between ERP + fluvoxamine and anti-ERP + fluvoxamine on the tolerability of treatment. — COTTRAUX1990

7.3.7 CT + fluvoxamine versus CT

7.3.7.1 Clinical evidence statements

Efficacy — ***Included studies***

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CT + fluvoxamine and CT on the efficacy of treatment. — VANBALKOM1998

Tolerability

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CT + fluvoxamine and CT on the likelihood of leaving the study early (K = 1; N = 49; RR = 1.74; 95% CI, 0.75 to 4.03). **I** — VANBALKOM1998

7.3.8 BT + fluvoxamine versus CT + fluvoxamine

7.3.8.1 Clinical evidence statements

Efficacy — ***Included studies***

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between BT + fluvoxamine and CT + fluvoxamine on the efficacy of treatment. — VANBALKOM1998

Tolerability

The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between BT + fluvoxamine and CT + fluvoxamine on the likelihood of leaving the study early (K = 1; N = 52; RR = 0.86; 95% CI, 0.43 to 1.70). **I** — VANBALKOM1998

7.3.9 BT + fluvoxamine versus fluvoxamine (children and young people)

7.3.9.1 Clinical evidence statements

Efficacy — ***Included studies***

There is limited evidence suggesting a difference favouring BT + fluvoxamine over fluvoxamine on reducing obsessive-compulsive symptoms as measured by the Children's Y-BOCS 52 weeks after beginning the treatment (K = 1; N = 10; SMD = 21.50; 95% CI, 23.00 to 0.00). **I** — NEZIROGLU2000

7.3.10 CBT + sertraline versus sertraline (children and young people)

7.3.10.1 Clinical evidence statements

Efficacy	***Included studies***
There is limited evidence suggesting a difference favouring CBT + sertraline over sertraline on the likelihood of relapse, defined as a score of less than or equal to 10 on the Children's Y-BOCS (K = 1; N = 56; RR = 0.59; 95% CI, 0.38 to 0.92). **I**	POTS2004
There is limited evidence suggesting a difference favouring CBT + sertraline over sertraline on reducing the severity of obsessive-compulsive symptoms as measured by the Children's Y-BOCS (K = 1; N = 56; SMD = 20.59; 95% CI, 21.13 to 20.05). **I**	POTS2004
Tolerability	
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBT + sertraline and sertraline on the likelihood of leaving the study early (K = 1; N = 56; RR = 1; 95% CI, 0.22 to 4.54). **I**	POTS2004

7.3.11 CBT + sertraline versus CBT (children and young people)

7.3.11.1 Clinical evidence statements

Efficacy	***Included studies***
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBT + sertraline and CBT on the efficacy of treatment.	POTS2004
Tolerability	
The evidence is inconclusive and so it is not possible to determine if there is a clinically important difference between CBT + sertraline and CBT on the likelihood of leaving the study early (K = 1; N = 56; RR = 1; 95% CI, 0.22 to 4.54). **I**	POTS2004

7.3.12 Clinical summary

The evidence from five studies suggests that there is greater improvement for OCD symptoms from combined SRIs and ERP when compared with ERP alone. There is also evidence from a single study that the combination may be better than SRI alone

(clomipramine in this case). These results suggest that adults with OCD should be offered the possibility of combined treatment. However, the evidence to date is from simultaneous combined treatment and we do not know whether this is the best way of using the two treatments together.

For children and young people, one study found greater improvement in OCD symptoms with the combination of sertraline and CBT over sertraline alone but not over CBT alone. For children and young people, one small study found some superiority for the combination of CBT and fluvoxamine over fluvoxamine alone after 52 weeks. These results suggest for children and young people, especially given the concerns about safety of SRIs in young people, CBT should be offered first, but that combined treatment may also be offered.

7.3.13 Combinations of an SRI and CBT in BDD

No RCTs have been conducted that compare an SRI or any other medication with CBT or a combination of the two. There are a few case reports of combination treatments highlighted in the psychological interventions but they do not assist in guiding clinical practice.

7.4 CLINICAL PRACTICE RECOMMENDATIONS

7.4.1 Initial treatment options

Adults

7.4.1.1 Adults with OCD with mild functional impairment who are unable to engage in low intensity CBT (including ERP), or for whom low intensity treatment has proved to be inadequate, should be offered the choice of either a course of an SSRI or more intensive CBT (including ERP) (more than 10 therapist hours per patient) because these treatments appear to be comparably efficacious. **[C]**

7.4.1.2 Adults with OCD with moderate functional impairment should be offered the choice of either a course of an SSRI or more intensive CBT (including ERP) (more than 10 therapist hours per patient), because these treatments appear to be comparably efficacious. **[B]**

7.4.1.3 Adults with OCD with severe functional impairment should be offered combined treatment with an SSRI and CBT (including ERP). **[C]**

7.4.1.4 Adults with BDD with moderate functional impairment should be offered the choice of either a course of an SSRI or more intensive individual CBT (including ERP) that addresses key features of BDD. **[B]**

7.4.1.5 Adults with BDD with severe functional impairment should be offered combined treatment with an SSRI and CBT (including ERP) that addresses key features of BDD. **[C]**

7.4.2 Poor response to treatment

Poor response to initial treatment for adults

If initial treatment does not result in a clinically significant improvement in both symptoms and functioning, other treatment options should be considered. When additional treatment options also fail to produce an adequate response, multidisciplinary teams with specific expertise in OCD/BDD should become involved, including supporting and collaborating with those professionals already involved in an individual's care.

7.4.2.1 For adults with OCD or BDD, if there has not been an adequate response to treatment with an SSRI alone (within 12 weeks) or CBT (including ERP) alone (more than 10 therapist hours per patient), a multidisciplinary review should be carried out. **[GPP]**

7.4.2.2 Following multidisciplinary review, for adults with OCD or BDD, if there has not been an adequate response to treatment with an SSRI alone (within 12 weeks) or CBT (including ERP) alone (more than 10 therapist hours per patient), combined treatment with CBT (including ERP) and an SSRI should be offered. **[C]**

7.4.2.3 For adults with OCD or BDD, if there has not been an adequate response after 12 weeks of combined treatment with CBT (including ERP) and an SSRI, or there has been no response to an SSRI alone, or the patient has not engaged with CBT, a different SSRI or clomipramine should be offered. **[C]**

7.4.2.4 Clomipramine should be considered in the treatment of adults with OCD or BDD after an adequate trial of at least one SSRI has been ineffective or poorly tolerated, if the patient prefers clomipramine or has had a previous good response to it. **[C]**

7.4.2.5 For adults with OCD or BDD, if there has been no response to a full trial of at least one SSRI alone, a full trial of combined treatment with CBT (including ERP) and an SSRI, and a full trial of clomipramine alone, the patient should be referred to a multidisciplinary team with specific expertise in the treatment of OCD/BDD for assessment and further treatment planning. **[GPP]**

7.4.2.6 The assessment of adults with OCD and BDD referred to multidisciplinary teams with specific expertise in OCD/BDD should include a comprehensive assessment of their symptom profile, previous pharmacological and psychological treatment history, adherence to prescribed medication, history of side effects, comorbid conditions such as depression, suicide risk, psychosocial stressors, relationship with family and/or carers and personality factors. **[GPP]**

7.4.2.7 Following multidisciplinary review, for adults with OCD if there has been no response to a full trial of at least one SSRI alone, a full trial of combined treatment with CBT (including ERP) and an SSRI, and a full trial of clomipramine alone, the following treatment options should also be considered (note, there is no evidence of the optimal sequence of the options listed below):

- additional CBT (including ERP) or cognitive therapy **[C]**
- adding an antipsychotic to an SSRI or clomipramine **[C]**
- combining clomipramine and citalopram. **[C]**

7.4.2.8 Following multidisciplinary review, for adults with BDD, if there has been no response to a full trial of at least one SSRI alone, a full trial of combined treatment with CBT (including ERP) and an SSRI, and a full trial of clomipramine alone, the following treatment options should also be considered (note, there is no evidence of the optimal sequence of the options listed below):

- additional CBT or cognitive therapy by a different multidisciplinary team with expertise in BDD **[GPP]**
- adding buspirone to an SSRI. **[C]**

7.4.2.9 For adults with BDD, if there has been no response to treatment, or the patient is not receiving appropriate treatment, more intensive monitoring is needed because the rate of suicide is high in people with BDD. **[GPP]**

7.4.2.10 Treatments such as combined antidepressants and antipsychotic augmentation should not be routinely initiated in primary care. **[GPP]**

Poor response to initial treatment in children and young people

If CBT (including ERP) involving the family has not produced an adequate response in terms of a clinically significant reduction in symptoms and increase in functioning within 12 sessions, then review and consider further options according to the age of the child as described below.

Current published evidence suggests that SSRIs are effective in treating children and young people with OCD. The only SSRIs licensed for use in children and young people with OCD are fluvoxamine and sertraline. However, with depression SSRIs can cause significant adverse reactions, including increased suicidal thoughts and self-harm, although they may be safer when combined with psychological treatments. The UK regulatory authority has contraindicated all SSRIs in paediatric depressive illness, except fluoxetine. Although the risk associated with the use of SSRIs in children and young people with OCD is unclear, appropriate caution should be observed, especially in the presence of comorbid depression.

7.4.2.11 For a child or young person with OCD or BDD, if there has not been an adequate response within 12 weeks to a full trial of CBT (including ERP) involving the family or carers, a multidisciplinary review should be carried out. **[GPP]**

7.4.2.12 Following multidisciplinary review, for a child (aged 8 to 11 years) with OCD or BDD with moderate to severe functional impairment, if there has not been an adequate response to CBT (including ERP) involving the family or carers, the addition of an SSRI to ongoing psychological treatment may be considered. Careful monitoring should be undertaken, particularly at the beginning of treatment. **[C]**

7.4.2.13 Following multidisciplinary review, for a young person (aged 12 to 18 years) with OCD or BDD with moderate to severe functional impairment

if there has not been an adequate response to CBT (including ERP) involving the family or carers, the addition of an SSRI to ongoing psychological treatment should be offered. Careful monitoring should be undertaken, particularly at the beginning of treatment. **[B]**

7.4.2.14 For a child or a young person with OCD or BDD, if treatment with an SSRI in combination with CBT (including ERP) involving the family or carers, is unsuccessful or is not tolerated because of side effects, the use of another SSRI or clomipramine with careful monitoring may be considered, especially if the child or young person has had a positive response to these alternatives in the past. This should also be in combination with CBT (including ERP). **[C]**

7.4.3 Intensive treatment services and inpatient services for people with OCD or BDD

OCD and BDD can usually be treated and managed in the community and in primary care. However, people with severe and/or chronic problems that have not responded adequately to treatment should be referred to multidisciplinary teams with specialist expertise in the treatment of OCD/BDD. Occasionally inpatient treatment may be needed for children, young people or adults who are at particular risk or whose ability to function is severely impaired. Special support may be needed, especially for young adults with impaired autonomy and personal functioning as a result of severe OCD with onset in childhood or adolescence.

7.4.3.1 People with severe, chronic, treatment-refractory OCD or BDD should have continuing access to specialist treatment services staffed by multidisciplinary teams of healthcare professionals with expertise in the management of the disorders. **[C]**

7.4.3.2 Inpatient services, with specific expertise in OCD and BDD, are appropriate for a small proportion of people with these disorders, and may be considered when:

- there is risk to life
- there is severe self-neglect
- there is extreme distress or functional impairment
- there has been no response to adequate trials of pharmacological/psychological/combined treatments over long periods of time in other settings
- a person has additional diagnoses, such as severe depression, anorexia nervosa or schizophrenia, that make outpatient treatment more complex
- a person has a reversal of normal night/day patterns that make attendance at any daytime therapy impossible
- the compulsions and avoidance behaviour are so severe or habitual that they cannot undertake normal activities of daily living. **[GPP]**

7.4.3.3 A small minority of adults with long-standing and disabling obsessive-compulsive symptoms that interfere with daily living and have prevented

them from developing a normal level of autonomy may, in addition to treatment, need suitable accommodation in a supportive environment that will enable them to develop life skills for independent living. **[GPP]**

7.4.3.4 For children and young people with severe OCD or BDD with high levels of distress and/or functional impairment, if there has been no response to adequate treatment in outpatient settings, or there is significant self-neglect or risk of suicide assessment for intensive inpatient treatment in units where specialist treatment for children or young people with OCD or BDD is available should be offered. **[GPP]**

7.4.4 Discharge after recovery

After full recovery, children, young people and adults with OCD or BDD should be followed up for a year. After discharge, those re-referred should be seen quickly and should not be placed on a routine waiting list.

7.4.4.1 When a person of any age with OCD or BDD is in remission (symptoms that are not clinically significant and the person is fully functioning for 12 weeks), he or she should be reviewed regularly for 12 months by a mental health professional. The exact frequency of contact should be agreed between the professional and the person with OCD or BDD and/or the family and/or carer and recorded in the notes. At the end of the 12-month period if recovery is maintained the person can be discharged to primary care. **[C]**

8. OTHER MEDICAL INTERVENTIONS

8.1 INTRODUCTION

A minority of people suffering from OCD remain refractory to all standard pharmacological and psychological treatments. It is largely, but not exclusively, this group that have been considered for treatment with other medical interventions. The medical interventions reviewed for OCD were electroconvulsive therapy (ECT), transcranial magnetic stimulation (TMS), ablative neurosurgical procedures, and two non-ablative procedures, namely, deep brain stimulation (DBS), and vagus nerve stimulation. It should be noted that because of the nature of these interventions, many of which may involve invasive and ablative procedures, a high proportion of patients who have undergone these procedures would have met strict referral criteria, especially in the more recent studies. These criteria will include previous and often repeated trials of pharmacotherapy and psychological therapies such as behaviour therapy. Consequently, the studies reviewed below are based on a limited group of patients and so represent a select sample with particular characteristics. Most studies are necessarily small in nature and reflect particular practices conducted at specific sites, and often by a small group of individuals (see also Freeman *et al.*, 2000, for a further discussion of these and related issues).

In the case of invasive procedures, evaluation of the evidence is further complicated by the difficulties in performing controlled trials, particularly randomised control trials with credible sham procedures. Overall there were insufficient data to complete a systematic review and hence a narrative review was undertaken. It should be noted that the review addresses OCD symptoms only in the context of refractory OCD rather than other potential co-morbid disorders. This decision, although perhaps arbitrarily limiting access to some potentially useful data on mixed samples, follows the strategy used elsewhere in this guideline.

8.2 ELECTROCONVULSIVE THERAPY

8.2.1 Introduction

Historically, ECT has occasionally been used for the treatment of intractable OCD. Although there are relatively few reports specifically for OCD, severe and intractable OCD is often associated with severe depression for which ECT may be indicated. It is entirely possible that significant numbers of people with OCD have received ECT, although the primary indication for the treatment would be the severe depression that has not responded to other treatment approaches. Some protocols for neurosurgery for intractable OCD suggest ECT may be tried before considering ablative procedures,

especially if the patient is depressed, within the context of a specialist service for patients who have not responded to adequate trials of psychological and pharmacological approaches (e.g. Matthews & Eljamel, 2003). Consequently, it is important to consider the evidence base for ECT for OCD.

8.2.2 Current practice

People with severe OCD may occasionally receive ECT and it has been recommended by the Expert Consensus Guideline for OCD for treatment refractory patients who may also be depressed only if they have not responded to three or more trials of SRIs nor to CBT (March *et al.*, 1997). There are no current recommendations for ECT for OCD in the UK. The practice of ECT for other conditions, namely depressive illness, schizophrenia, mania, and catatonia, is discussed in detail in the NICE Technology Appraisal No. 59.

8.2.3 Studies considered

A total of nine papers were found specifically addressing ECT for OCD from 1973–2003: one descriptive paper, five single case reports (two of which are letters), one letter describing three cases, one open trial, and one retrospective review of 32 cases treated over a 20-year period.

8.2.4 Descriptive review

There are four case reports describing a successful outcome for ECT as a treatment for OCD (Casey & Davis, 1994; Husain *et al.*, 1993; Mellman & Gorman, 1984; Thomas & Kellner, 2003). The case reports are generally of poor quality and lack methodological rigour. A further case report described the onset of mania following the use of ECT with OCD and treatment was discontinued (Chung *et al.*, 2001). Three cases of successful outcome following ECT for OCD are described in a letter (Beale *et al.*, 1995) although the absence of outcome measures precludes any firm conclusions regarding outcome.

Khanna and colleagues (1988a) conducted an open trial with nine subjects, all of whom met DSM-III criteria for OCD (American Psychiatric Association, 1980). Measures of OCD symptoms were administered at pre-treatment, during and post-ECT. Monthly follow-up assessments were conducted for 6 months. The authors reported that all subjects returned to pre-trial state within 6 months. The largest study (Maletzky *et al.*, 1994) is a retrospective review of 32 patients with OCD (19 of whom were described as non-depressed). All subjects had previously received trials of CBT and pharmacotherapy with little or no effect. Subjects were evaluated on the Maudsley Obsessive Compulsive Inventory (MOCI) and two depression scales at pre- and post-treatment and at 6- and 12-month follow-ups. The results indicated that the non-depressed group improved on measures of OCD symptoms at 12-month follow-up,

but depression scores deteriorated substantially. Overall, 18 of 32 (56%) maintained some improvement at 12-month follow-up. These results have to be treated with extreme caution as all of the subjects also received active treatment such as medication during the 1-year follow-up so any improvement cannot be attributed solely to ECT.

8.2.4.1 Electroconvulsive therapy in BDD

There are eight published case reports of ECT, six of which were unsuccessful (Phillips, 1991) and one case report noting it to be successful (Carroll, 1994). Phillips (1996b) notes that in her retrospective chart review none of the eight ECT trials was successful.

8.2.5 Clinical summary

There is a paucity of work undertaken regarding the efficacy of ECT and OCD. Most of the literature comprises single case reports without standardised measures, with one open trial and one retrospective review. Given the serious methodological weaknesses and the lack of convincing evidence for sustained improvement in the above studies, it is concluded that there is insufficient evidence on which to base a recommendation for the use of ECT in the treatment of OCD, especially given potential associated risks with ECT (NICE, 2003).

8.3 TRANSCRANIAL MAGNETIC STIMULATION

8.3.1 Introduction

TMS involves the use of a pulsed magnetic field to induce changes in function in cortical structures. It was developed in 1985 (Barker *et al.*, 1985) to investigate cerebral cortical activity and more recently has been used therapeutically for some mental disorders, namely, depression and OCD. In OCD this treatment aims to modify prefrontal cortical activity in order to influence obsessive-compulsive symptoms.

8.3.2 Current practice

Although TMS is not currently available for OCD in the UK, it has been used in two trials for depression at the Maudsley Hospital in London.

8.3.3 Studies considered

Three studies, one open trial and two randomised trials were identified. In addition, a recent systematic review (Martin *et al.*, 2003) identified the three papers and concluded that there was insufficient evidence from randomised controlled trials to determine efficacy of this technique.

8.3.4 Descriptive review

The interest in TMS for OCD arose from an open trial of 12 subjects who received repetitive transcranial magnetic stimulation (rTMS) (Greenberg *et al.*, 1997). Each patient received TMS to right or left lateral prefrontal areas or to a control (midoccipital) region. There was a short-lived significant increase in mood and temporary reduction in OCD symptoms with right prefrontal stimulation. Two patients reported headache and one reported visual distortion. The authors concluded that TMS might be a useful probe for studying cortical mechanisms in OCD.

Others have explored the therapeutic potential of TMS. One double-blind placebo-controlled study has been undertaken for OCD (Alonso *et al.*, 2001) where patients were randomly allocated to either rTMS or to sham rTMS. Ten patients were allocated to the experimental arm and eight to the sham arm. All but five were receiving medication. There were no significant differences post-intervention between the two conditions on either OCD (Y-BOCS) or depression (Hamilton) symptoms. One patient reported mild headache. Finally, Sachdev and colleagues (2001) randomised 12 patients with resistant OCD to either right or left prefrontal rTMS. There were no significant differences between right and left rTMS. A significant linear trend from pre-treatment to post-treatment was reported for the two groups combined, indicating improvement in obsessive-compulsive symptoms. However, there was no sham arm so the treatment effect is uncontrolled. An examination of individual response indicated that four of the 12 participants showed a 40% or greater symptom reduction at post intervention, though one had relapsed at 1-month follow-up. Three patients reported headaches.

8.3.5 Clinical summary

The evidence for transcranial magnetic stimulation as a treatment for OCD is as yet inconclusive. Although its possible interest lies in the fact that it is a non-invasive procedure, there is insufficient evidence upon which to base a recommendation for the use of transcranial magnetic stimulation in the treatment of OCD.

8.4 NEUROSURGERY

8.4.1 Introduction and current practice

Neurosurgery for mental disorders has changed significantly since its initial introduction in the 1930s (see Freeman *et al.*, 2000, for a historical overview) and although there remains interest worldwide, the number of centres in the UK offering neurosurgery has decreased over the last 10 years. Freeman and colleagues (2000) suggest there is greater specificity as procedures have evolved, but it is less clear whether surgical innovation is due to theoretical advances or to pragmatic considerations. Neurosurgery has been recommended by the Expert Consensus Guideline for

treatment refractory OCD in the case of non-response to three or more trials of SRIs (including clomipramine) and to CBT as an 'infrequently needed, but sometimes life saving intervention' (March *et al.*, 1997). Although the number of neurosurgical interventions conducted in the UK for OCD has decreased in recent times (see Freeman *et al.*, 2000), there are currently two centres (Cardiff and Dundee) where patients with OCD from England and Wales may be referred for assessment for possible neurosurgical intervention (Matthews & Eljamel, 2003).

It is important to review the evidence for ablative neurosurgery for OCD given that earlier reviews have reported promising results. For example, Kiloh and colleagues (1988) reported that among 478 patients from 24 studies between 1961 and 1980, 58% showed marked improvement. Over half the operations in this review were non-stereotactically guided. A review of 12 studies from 1961 to 1988 by Waziri (1990) reported that 67% of the 300 patients fell into the 'symptom free' or 'minor symptoms' categories; all but three studies used stereotactically guided procedures. Finally, Freeman and colleagues (2000) reviewed five studies involving 198 patients, and found an identical result of 67% fell into these categories.

Although a variety of different procedures are used, all involve the ablation or disconnection, of ventral and medial prefrontal cortical areas. Four main ablative procedures have been used in OCD:

- Anterior capsulotomy
- Anterior cingulotomy
- Subcaudate tractotomy
- Limbic leucotomy.

In addition, there is one non-ablative procedure, deep brain stimulation, which has been investigated with OCD. A second procedure, vagal nerve stimulation, has been discussed as a potential treatment for OCD. Although it is used for other conditions including depression (George, 2000), it has not, to our knowledge, been investigated in the treatment of OCD and will not be reviewed.

8.4.2 Studies considered

Forty-nine studies were identified describing neurosurgery for OCD (including ablative and non-ablative procedures). No randomised control trials were found that compared ablative neurosurgical procedures with a placebo or credible treatment control. One double-blind RCT and one systematic review were found for the more recent non-ablative procedures.

8.4.3 Ablative procedures

8.4.3.1 Subcaudate tractotomy

This procedure was developed by Geoffrey Knight in the UK (1969). Although it was used extensively for affective disorders for several years, the operation is no longer performed. Radioactive 90-Yttrium rods were inserted into the target area, a region

called the substantia innominata, found below the head of the caudate nucleus. Much of the literature on this technique describes the treatment of depression and there are limited reports on its use for OCD (Bartlett & Bridges, 1977; Cosyns *et al.*, 1994; Goktepe *et al.*, 1975; Hodgkiss *et al.*, 1995; Strom-Olsen & Carlisle, 1971). In an early study (Strom-Olsen & Carlisle, 1971), 20 patients with OCD received this procedure; ten improved, but four relapsed during the follow-up period. In a further study (Goktepe *et al.*, 1975), 50% of 18 patients were reported to have shown significant improvement or better. These early studies reported global ratings only rather than specific measures of OCD symptoms. Although this intervention as described above is no longer practiced as such, lesions in the same area of the brain are part of the limbic leucotomy described below.

8.4.3.2 Anterior capsulotomy

Two main procedures for making lesions in the anterior capsule have been described, namely, radiofrequency thermo-capsulotomy and radiosurgical gamma knife capsulotomy (Rasmussen *et al.*, 2000). There are a number of earlier reports between 1961 and 1982 but these used global rather than OCD-specific scales to evaluate thermocapsulotomy (see Freeman *et al.*, 2000 for a review).

One prospective series from the Karolinska Hospital in Stockholm, Sweden, has been reported on extensively (Lippitz *et al.*, 1997; Lippitz *et al.*, 1999; Mindus *et al.*, 1994; Mindus *et al.*, 1999; Nyman *et al.*, 2001). Twenty-four patients from 1979–1990 underwent thermocapsulotomy and 19 were included in a study on personality characteristics (Mindus *et al.*, 1999). Patients were assessed pre-intervention and a mean of 8 years later. Five of the patients received a second intervention during the follow-up period. The authors reported that five patients were unchanged or deteriorated on the CPRS-OC, six were less than 50% improved and eight were more than 50% improved. On 15 self-report scales designed to measure personality (Karolinska Scales of Personality), there was a decrease at 8-year follow-up (average) in anxiety proneness dimensions, but no evidence of changes for other aspects of personality, except one person who had haemorrhaged showed increases in psychopathic traits.

The patients who had received thermocapsulotomy between 1978 and 1999 (n = 21) were compared at follow-up with a group who had been assessed but had not yet received neurosurgery (n = 8) (Nyman *et al.*, 2001). No significant differences were found on a battery of neuropsychological tests but the study would be, in all likelihood, insufficiently powered to detect changes on these tests given the small numbers of patients involved. The authors conclude that 'patients with OCD by capsulotomy generally perform in the lower region of the normal range or show mild impairment on standardized tests 2 to 15 years after the operation'.

Lippitz and colleagues (1999) reported again on this series but also included those who had received the gamma knife intervention. They conducted a retrospective study to define potential common topographic denominators among the lesions from the patients reported earlier (Mindus *et al.*, 1999) who had been successfully treated with gamma knife capsulotomy (n = 10). The 19 patients receiving thermocapsulotomy reported in this study are the same as those reported in the Mindus and colleagues

(1999) study. Magnetic Resonance Imaging indicated the location and size of the lesion. Clinical outcome was reported, mostly using the CPRS-OC although later patients may have been rated on the Y-BOCS. Results are presented as percentage improvement on psychiatric rating scales and showed that 9/19 of the patients who received thermocapsulotomy and 7/10 who received gamma knife capsulotomy showed at least a 50% improvement on either CPRS or Y-BOCS.

Christensen and colleagues (2002) reported on two cases of severe OCD in 'younger' (18-yrs) and 'older' (64-yrs) patients and who received capsulotomy. They reported a successful outcome based on a reduction in OCD symptoms on the Y-BOCS.

Oliver and colleagues (2003) reported on a series of 15 patients who underwent thermo-capsulotomy. Three patients received a second capsulotomy. Forty-six per cent reported a 50% or greater reduction on the Y-BOCS. Two patients reported transient adverse effects (one case each of hallucinations and seizure) and one 'postoperative bifrontal swelling with permanent behaviour impairment'. Finally, a review by Greenberg and colleagues (2003) reported preliminary results of an unpublished study of 15 patients who had undergone gamma knife capsulotomy. The authors reported that single bilateral lesions in the anterior capsule were ineffective. However, following placement of a second set of bilateral lesions, four of the 15 were judged as showing a 35% decrease on the Y-BOCS and at least a 15-point improvement on the Global Assessment Scale at 5-year follow-up. They also reported a second study of 16 patients who received two pairs of bilateral lesions. At 3-year follow-up, ten met the same improvement criteria. Although the second series would seem to suggest a more positive outcome than the first series, results from neither series have yet been subject to peer review and should be treated cautiously.

In summary, anterior capsulotomy has been reported to be effective based mainly on retrospective trials using global ratings and, more recently, on prospective case series or open trials using OCD specific measures. Although the data are reported in increasing detail on a broader range of measures, conclusions are ultimately limited by the design and the small number of patients involved in the more recent series. The serious persistent adverse effects (1/19 in the Swedish series and 1/15 in the Oliver study) would suggest caution.

8.4.3.3 Anterior cingulotomy

In an early study, Fodstad and colleagues (1982) randomised two patients to either stereotactic anterior capsulotomy or cingulotomy. Follow-up to 12 to 24 months indicated that all four remained improved or much improved at the last follow-up. However, those who had received cingulotomy were judged as having a lesser response compared with those who had received capsulotomy.

A case series of five patients underwent what is labelled a 'modified leucotomy' but describe lesions to the cingulated gyrus and so would correspond to cingulotomy (Tippin & Henn, 1982). The authors reported improvement on a five-point clinician-rated global outcome measure, with four out of five classed as marked improvement or symptom free at follow-ups at 1 to 6.6 years. No specific measures of OCD were used.

There are two series, one retrospective and one prospective, from Massachusetts General Hospital in Boston that have each been described in several reports.

The retrospective series described in most detail by Jenike and colleagues (1991) followed-up 35 patients with OCD who had received cingulotomies from 1962 until the late eighties. It was determined that 33 would have met criteria for OCD according to DSM-III-R; 26 of these also met criteria for depression. Of the 33, 23 had received additional interventions, second cingulotomies in 16 cases, second and third cingulotomies in four cases, and other interventions in six cases. In this early series, only three patients had received behaviour therapy and six had received clomipramine pre-operatively. Adverse effects included seizures (3/33), decreased memory (1/33), suicide (4/33) and death from myocardial infarction 6 weeks post-operatively (1/33 with previous history of cardiac problems). Based on retrospective ratings of pre-operative symptoms of 14 patients who were interviewed, 8 (58%) showed moderate to marked improvement. However, in at least two cases, improvement was attributed to additional treatment. Overall, the authors conclude that 9/29 surviving patients showed significant improvement that could be attributed to psychosurgery.

Dougherty (2002) reported on a prospective study of 44 patients with treatment refractory OCD who received cingulotomy from 1989 onwards. Data from some of these patients had previously been reported elsewhere (Baer *et al.*, 1995; Spangler *et al.*, 1996). All patients had symptoms that had previously not responded to treatment regimes of both medication and behaviour therapy. Of the 44 patients operated on during the study period, 17 patients had received two cingulotomies and one had undergone a third. Clinical outcome measures, including the Y-BOCS, were administered pre- and post-intervention (mean of 6.7 months), at the first post-surgical procedure (mean of 7 months) after the second cingulotomy, and at a mean of 32 months' follow-up from the first cingulotomy. Response rate was defined as 35% improvement on the Y-BOCS and a CGI Global Improvement score of less than or equal to 2. In addition, to be considered as a responder, patients needed to attribute their improvement to the cingulotomy. At first follow-up, 5/44 patients met the response criteria and 14/44 at final follow-up. Nine patients (9/44) reported adverse effects post-operatively: memory problems (2/44), apathy (1/44), urinary disturbance (3/44), seizure (1/44), post-operative oedema with resulting hydrocephalus (1/44). One patient, who showed improvement in OCD symptoms, committed suicide 6 years after the operation. In all but two cases (one each of urinary incontinence and seizure), the adverse effects resolved.

Finally, 14 patients with refractory OCD underwent cingulotomy in a prospective study in Korea (Kim *et al.*, 2003). All patients referred for the trial had received behaviour therapy and medication. Patients were assessed using the Y-BOCS, CGI and the Hamilton Rating Scales for Depression and Anxiety. The response criteria were defined as at least 35% improvement on the Y-BOCS and a CGI score of 1 or 2 (improved or very much improved). Four patients met this criterion at 6-month follow-up and six at 12-month follow-up. Mean improvement rate on Y-BOCS was 36%. Two patients reported transient headache, three gained weight and one lost weight but normalised subsequently. There was no evidence of cognitive dysfunction on a range of measures.

In sum, these studies would suggest that cingulotomy has shown some effects with up to a third of patients meeting differing criteria for response. The two prospective studies are stronger designs than those found for some other interventions and

used conventional definitions of response (35% reduction in Y-BOCS score). Nevertheless, they remain limited by the lack of control groups. There were relatively few persistent adverse effects but the suicide rate in the retrospective study (Jenike *et al.*, 1991) would indicate caution.

8.4.3.4 Limbic leucotomy

Limbic leucotomy, a multi-target procedure, was developed in the UK by Desmond Kelly in the 1970s and consists of lesions corresponding to both cingulotomy and subcaudate tractotomy. An early study reported a high response rate with OCD (Mitchell-Heggs *et al.*, 1976) but was methodologically flawed and the results have been disputed (Chiocca & Matuza, 1990).

A number of reports describe an Australian series of 26 patients operated on from 1972 until 1989, most of whom (n = 17) received both cingulate and orbitomedial lesions similar to limbic leucotomy while six received cingulate lesions and four received orbitomedial lesions only (Cumming *et al.*, 1995; Hay *et al.*, 1993; Sachdev & Hay, 1995; Sachdev, 1996). The first six patients received the orbitomedial lesions through open neurosurgery; the remaining all received stereotactic interventions. Although this series is more difficult to interpret because of the mixed interventions, it is worth considering because of the extensive reports on cognitive and personality function. At follow-up of 10 years (mean), five (19%) were considered much improved or recovered, five were moderately improved, six were mildly improved, six showed no improvement, four were worse (Hay *et al.*, 1993). Of the four who were rated worse, three died by suicide and one had marked personality change. In addition to transient post-operative adverse effects in three patients, one suffered from post-operative haemorrhage, delirium, periods of psychosis and permanent personality change. An additional three were considered to have shown personality change and two suffered from recurrent seizures. In all, 6/26 suffered serious permanent adverse effects. In a report on self- and informant-rated personality change in a subgroup of 16, the majority reported little change in personality across 34 items (Sachdev & Hay, 1995).

Finally, 17 of this series were compared with a control group of patients with long-term OCD on a battery of neuropsychological tests. There was no evidence of impaired IQ or memory function compared with the OCD controls, but there was evidence of impaired performance in the operated group on executive function (Cumming *et al.*, 1995). Interestingly, a magnetic resonance imaging (MRI) study on 14 of these patients revealed accurately placed orbitomedial lesions (10/10), accurately placed cingulated lesions in only 10 of 13, and inadvertent lesions to the anterior capsule in three cases (Sachdev, 1996).

A more recent case series has been reported with 21 patients with major depressive disorder (MDD) or OCD who underwent stereotactic limbic leucotomy between 1993 and 1999 (Montoya *et al.*, 2002). All had symptoms that failed to respond to pharmacotherapy and over three quarters had received ECT.

In this study, there were 15 patients for whom the primary indication for surgery was OCD, although three of those whose primary indication was for MDD also had OCD. Eight of those in the OCD group also received a diagnosis of MDD.

For a significant proportion of the participants (76%), limbic leucotomy was the second (following unsuccessful bilateral anterior cingulotomy, n = 5) or third neurosurgical procedure (following enlargement of earlier cingulotomy lesions). It is unclear what percentage of patients with OCD had undergone a second or third operation as the authors have reported for the whole sample only.

Two patients, one with MDD and one with OCD, both with previous history of suicidal attempts, died by suicide during the follow-up period. Transient somnolence (6/21), apathy (5/21), and fever (2/21) were reported. Post-operative and persistent headache were reported by one patient. Four patients reported post-operative seizures that were transient in all but one. Five patients (5/21) reported bladder incontinence that was persistent in three (3/21). Finally, five patients (24%) reported short-term memory problems that were persistent for two people (10%).

Outcome measures used were the Y-BOCS and CGI Global Improvement scale. Response was determined in a similar way to other neurosurgical studies of OCD, namely, an improvement score of 1 or 2 on the CGI (very much or much improved) and a 35% decrease in Y-BOCS score. Of the 15 patients with OCD, only nine had pre- and post-operative ratings on the Y-BOCS and only eight had both Y-BOCS and CGI ratings. Of these eight patients, only two (25%) met the double criteria for response. When based on one or other of the measures the authors reported a 36% (Y-BOCS) to 62% (self-rated CGI) response. The authors also reported that for the entire cohort (N = 21), the response rate was better for those undergoing limbic leucotomy where it was the second or third procedure. In sum, this case series describes a group of chronically ill patients who had received multiple previous non-neurosurgical treatments and indeed 76% had also received one or more previous cingulotomies. The findings are limited by the partial data collected and the difficulty in separating out some of the findings for the OCD patients alone.

Given the shortcomings in the designs, the lack of robust evidence of efficacy and the high rate of persistent adverse events reported in both series, the evidence for this type of multi-target procedure is not promising.

8.4.3.5 Neurosurgery in BDD

Phillips (2002) noted one published case report and two personal communications describing benefit in three individuals with BDD (modified leucotomy in one, capsulotomy in one, and bilateral anterior cingulotomy and subcaudate tractotomy in one) and no benefit in two individuals (who received anterior capsulotomy).

8.4.3.6 Clinical summary

Although there are reports of improvement for three of the procedures (capsulotomy, cingulotomy and limbic leucotomy), there are also sufficient reports of both transient and persistent adverse effects to cause concern. The more recent prospective series all report on criteria for entry to these studies that include previous trials of pharmacological treatments and ERP, supporting the contention that at least in the last 15 years, these are generally treatments of last resort. Given the relative rarity of these interventions, studies are generally small and conducted over long periods. Importantly, none have control conditions, although it is unlikely that credible sham procedures could

be ethical for ablative procedures, nor are they likely to be possible as patients are unlikely to accept randomisation for treatments of last resort.

Some studies have used comparison groups to compare neuropsychological function and personality function in people with OCD who have received neurosurgery with those who have not. While these studies are attempting to answer important questions about effects of neurosurgery, the small sample size and the fact that they are essentially testing null hypotheses means that the most that can be concluded is the absence of large effects, but nothing can be said about whether smaller but potentially clinically significant differences may be present. Although there have been significant improvements in the measurement of obsessive-compulsive symptoms, there are still few prospective studies investigating neuropsychological changes and those measuring personality changes are using measures that are not widely known. In summary, the evidence on whether adverse changes occur in neuropsychological and personality function is inconclusive.

8.4.4 Non-ablative procedures

Concerns regarding the irreversibility and possible long-term adverse effects of ablative neurosurgical procedures have led to the investigation of a number of non-destructive neurophysiological interventions. Tissue damage could still occur during the intervention or through repeated stimulation, but the intervention does not seek to produce lesions.

8.4.4.1 Deep brain stimulation

Electrical deep brain stimulation is a relatively new technique and developed as a treatment for OCD through collaboration between Belgian and Swedish researchers. The effects of electrical stimulation of the brain have been previously investigated during stereotactic surgery for OCD before permanent lesions were made. For example, Laitinen and Singounas (1988) reported on the effects of stimulation in a series of 20 patients undergoing neurosurgery under local anaesthetic. However, the therapeutic use differs in that electrodes are implanted within brain structures, which are then stimulated via an external electrical source. Any lesions that result are not deliberate and are considered to be small relative to those made deliberately during ablative procedures. The efficacy and safety of DBS for the treatment of neurological conditions such as Parkinson's disease are well established. It has not yet been used for OCD in the UK although it is used for Parkinson's disease.

Essentially, DBS uses high frequency pulses that have complex effects including blocking of the targeted area and mimics the effect of tissue lesioning without destroying them (Tass *et al.*, 2003). In an initial report, Nuttin and colleagues (1999) described four patients who were treated with DBS; three were reported to have improved although little detail was provided. Since then, a series of six patients has been reported in several publications that address a range of variables as well as issues about placement of electrodes and stimulation parameters (Gabriels *et al.*, 2003; Nuttin *et al.*, 2003a; Nuttin *et al.*, 2003b). In this series, quadripolar electrodes stereo-

tactically implanted in both anterior limbs of the internal capsules in six patients with OCD, all of whom had been deemed to have severe OCD by a selection committee. Four of the patients were randomly crossed over from continuous stimulation to stimulation off. Two were not crossed over (one received a capsulotomy and one was still in the post-operative screening phase).

The authors reported improvement in the 'stimulator on' condition compared with 'stimulator off', but these data should be interpreted with caution due to the small size of the study. Of the four patients, one did not respond and 3 responded (improvement of >35% on the Y-BOCS). A number of side effects were reported including transient hypomanic states, swelling of the face, awareness of the leads attached to the electrodes, fatigue, and weight loss/gain (Nuttin *et al.*, 2003b). There were also technical difficulties due to broken electrical contacts and fracture of an electrode, but the main technical issue was short battery life necessitating replacement of the stimulators every 5 to 12 months. There is one additional independent report of DBS (Anderson & Ahmed, 2003) that reported a positive outcome in a case of severe OCD.

8.4.4.2 Clinical summary

DBS is a very recent procedure. The studies so far are too small to reach any conclusions about efficacy. The side effects reported so far suggest extreme vigilance especially as it is not yet established whether DBS is completely reversible. The technical issues around battery life would seem to present a significant limitation at the moment. The relatively non-destructive nature of the intervention means that well designed controlled trials would be possible.

8.4.5 Issues about neurosurgery for OCD

All of the recent series report selection criteria and despite agreement on general principles such as severity, disability and non-response to previous treatment, there is a degree of variability. There is also wide variability in terms of the range of assessments used although all recent studies have used the Y-BOCS. Consequently, combining or comparing data from different centres is almost impossible and centres with few operations do not contribute at all to these series. As several authors have pointed out, international agreement on these issues would allow the field to advance.

The main issues to be agreed, both in routine practice and research, are selection criteria that reflect severity and chronicity of OCD, the definition of adequate previous treatment, the need for independent oversight, a standardised assessment protocol pre- and post-operatively, and agreements and protocols about post-operative care.

Matthews and Eljamel (2003) have outlined a detailed series of criteria both for inclusion and for determining adequacy of previous treatment. These criteria are broadly in line with, and generally exceed, those reported by authors in Sweden, the US and Korea. These criteria, when interpreted in the spirit intended by the authors themselves, will result in decisions that properly balance the potential risks against the potential benefits and reserve consideration of such interventions for truly intractable cases of OCD. There is a potential problem with all guidelines, which results not from their inten-

tion, but from the possibility that they are applied in a superficial or unsophisticated way. In particular, the question of the adequacy of previous treatment is particularly difficult to determine. As Bejerot (2003) points out, 'yesterday's intractable OCD patient may well be treatable today and intractability may depend on the ambition of the prescriber'.

Although this statement apparently refers to pharmacological treatments, similar issues apply to psychological treatments. For psychological treatments, an adequate trial cannot be defined by number, duration and frequency of sessions alone. Engagement in therapy is believed to be essential to outcome and therapeutic strategies must be implemented. Unwillingness or inability to engage actively in therapy is a feature of some people with severe OCD. Sometimes several unsuccessful attempts to engage in therapy precede a successful attempt, perhaps with a therapist of equal skill who has a different style, or who is using the same basic techniques in a different manner. Consequently, guidelines such as those by Matthews and Eljamel (2003) must remain guidelines to inform sound clinical judgment as the authors themselves intend through thorough consideration of the complex issues involved in determining adequacy of previous treatment.

It is essential, as is commonly the practice in the UK, that multidisciplinary teams with specific expertise in the management and treatment of OCD be involved in treatment and assessment before concluding that a patient has not responded to adequate treatment. In addition, given that non-ablative methods are now being developed, it may be that such methods should be considered first when evidence, or more conclusive evidence, for their efficacy in OCD becomes available.

Assessment should include standardised measures of obsessive-compulsive symptoms, depression and anxiety, psychosocial functioning including quality of life, personality, cognitive function, and possible adverse effects. Ratings should be sought from independent assessors, the patient, and family members or friends. If it is decided to proceed with an intervention, protocols for post-operative care should be agreed that allow provision for pharmacological and psychological care.

8.4.6 Clinical summary

The evidence is generally inconclusive for each of the medical interventions considered. In addition to a variety of design issues that prevent strong inferences from being made, evidence for efficacy is limited. For those with the strongest supportive clinical evidence, the proportion of those who respond must be considered within the context that all neurosurgical techniques have the potential for serious adverse effects, including persistent ones as reported in many of the studies reviewed. However, it is important to note that, especially in the more recent studies, many of the participating patients (although not all) had severe OCD with high Y-BOCS scores, poor functioning, and significant levels of psychiatric comorbidity, including, in many cases, severe depression. In addition, most participants had previously received evidence-based standard treatments and independent selection committees had approved surgery. For such patients, considered refractory to other treatment

approaches, even the relatively modest rates and degrees of response reported in these studies may be clinically important.

At present, there is no compelling evidence comparing different neurosurgical procedures. Several studies have suggested multiple lesions may be needed for both capsulotomy and cingulotomy which may, as some data would suggest, increase the chance of response, but may also increase the risk of adverse effects. Small samples have prevented identification of predictors of response to any of the treatments. In conclusion, the quality of available evidence addressing the efficacy and safety of neurosurgical treatments is variable and does not support a clear recommendation. However, it is possible that a small number of patients with the most severe, chronic, disabling and treatment-refractory forms of OCD, for whom quality of life is very poor, will continue to be assessed as candidates for neurosurgery. This may, in the future, include non-ablative procedures. Such assessments should be conducted by established, expert, multidisciplinary teams who possess experience in the management of severe OCD, work from detailed pre- and post-operative protocols and structured long-term follow-up, are subject to appropriate independent oversight, and are committed to sharing and publication of audit information.

8.5 MEDICAL INTERVENTIONS IN CHILDREN WITH OCD DUE TO PANDAS

8.5.1 Introduction

There is accumulating research evidence that OCD may arise following infection with particular subtypes of streptococcal bacteria, Group A beta haemolytic streptococcus (GAS). This hypothesis and the subsequent studies, were stimulated by the longstanding observation that patients with Sydenham's chorea (SC) had high rates of obsessive-compulsive symptoms. SC is the neuropsychiatric manifestation of rheumatic fever, a disorder now known to be triggered by GAS. A sub-group of children with OCD were identified who had developed their condition following GAS infection, but did not meet criteria for SC. These children were given the acronym PANDAS (paediatric autoimmune disorders associated with streptococcal infection). The most striking feature of this sub-group of OCD is that the onset is very rapid, following streptococcal infection, and remits fully. Relapses occur with recurrent infection, giving these children an unusual fluctuating course to their OCD episodes.

The proposed mechanism of this disorder is one of 'molecular mimicry'. Antibodies generated by the body as part of the immune response against streptococcal infection, cross react with binding sites in the basal ganglia, a brain region thought to be important in OCD and related movement disorders. This autoimmune reaction only occurs in susceptible individuals, perhaps those with a genetic predisposition.

This proposed aetiology has suggested possibilities for novel prevention/treatment options in this subgroup. Could prevention of recurrent streptococcal infections prevent recurrences of OCD symptoms? Prophylactic antibiotics are given to

individuals with rheumatic heart disease; might a similar approach also be effective in a post-streptococcal neuropsychiatric condition? A second approach that has been explored is to modify the immune response which is thought to be the direct pathogenic mechanism. Antibody production could be inhibited by immunosupression, or antibodies removed by 'mopping-up' with another binding protein, or removing them with plasmaphoresis.

There have been limited trials of these approaches, and these are summarised below.

8.5.2 Current practice

PANDAS is difficult to diagnose, and a parental report of a sore throat preceding OCD, or a fluctuating history of OCD symptoms, is not adequate to make a diagnosis. The original definition of PANDAS described a homogenous group of children for the purpose of research into mechanisms, phenomenology and treatment. To diagnose PANDAS according to the criteria of the original investigators, streptococcal infection must be demonstrated in conjunction with at least two episodes of OCD symptoms, as well as demonstrating absence of infection and reduced antibodies during neuropsychiatric remission.

The clinical importance of a post-streptococcal subtype of OCD remains controversial. We do not know how common this sub-type of OCD might be, as there are no epidemiological studies in the general population. Children attending specialist psychiatric/OCD clinics may have higher than average evidence of previous streptococcal infections. However, even in the sub-group of children who are clearly thought to have PANDAS, there is no strong evidence to support immunomodulation or antibiotic prophylaxis currently, although these interventions remain the subject of active research. Similarly, there is no evidence to suggest that the post-streptococcal forms of OCD respond differently to medication or psychological treatments, as there have been no direct comparative studies of different OCD-subtypes.

Currently, there is no straightforward diagnostic system for determining whether an OCD patient meets PANDAS criteria, other than longitudinal follow-up, combined with repeat throat swabs and blood antibody tests. Even if there is a strong suggestion that they do fall into the post-streptococcal group, there is too little evidence currently to be recommending novel treatments, other than as part of a research program or clinical trial. In addition, children falling into this group should be given full trials of the conventional OCD treatments known to be effective (that is, CBT and SSRI medication).

8.5.3 Studies considered

Two double-blind randomised controlled trials were identified that tested whether a medical intervention targeting GAS infection would be effective in reducing OCD symptoms (Garvey *et al.*, 1999; Perlmutter *et al.*, 1999).

8.5.4 Descriptive review

Garvey and colleagues (1999) tested whether penicillin prophylaxis would reduce neuropsychiatric exacerbation in children with PANDAS by preventing streptococcal infections. The study was an 8-month double-blind cross-over trial, with 4 months in penicillin and 4 months in placebo. Children with a DSM-III or IV diagnosis for tic disorder and/or OCD, a history of a sudden onset of symptoms or an episodic course with abrupt symptom exacerbations interspersed with periods of partial or complete remission, and who showed evidence of an association between streptococcal infection and the onset or exacerbation of symptoms were included in the study. Participants received a standard prophylactic dose of 250 mg twice-daily penicillin V. Thirty-seven children entered the study, of whom 35% had both a primary diagnosis of OCD and tics and 35% had tics and subclinical OCD. Overall, there was no difference between the two groups on OCD and tic symptoms as measured by the Y-BOCS and the Yale Global Tics Severity Scale. There was also no difference between groups in the incidence of streptococcal infections, though fewer infections occurred in the penicillin than placebo phase. There was thus a failure to even achieve an adequate level of prophylaxis.

Perlmutter and colleagues (1999) tested whether plasma exchange and intravenous immunoglobulin (IVIG) would be better than placebo in decreasing neuropsychiatric symptoms in children with PANDAS. Children meeting similar inclusion and exclusion criteria to those in the Garvey and colleagues study were recruited to the study. Children were randomly assigned to plasma exchange, IVIG, or placebo. The plasma exchange procedure was done over 10–12 days, while the IVIG and placebo procedures were done over 2 days. Treatment outcome was assessed at 1 month and 1 year after start of therapy. Thirty children entered the study of which 63.33% had a primary diagnosis of OCD and 33.33% had a primary diagnosis of tic disorder. At 1-month follow-up, there was a significant improvement in symptom severity from baseline as measured by the Y-BOCS in the plasma exchange and IVIG groups. In turn, the plasma exchange group appeared to have greater improvement in OCD symptoms than the IVIG group, though this was not statistically significant. This improvement in OCD symptoms remained in the 17 children in the plasma exchange and IVIG groups who were followed-up at 1 year.

8.5.5 Clinical summary

Describing PANDAS as an autoimmune disorder rather than as a streptococcal infection has more benefit in terms of therapeutic gain. Treatments that target the autoimmune feature of PANDAS, such as plasma exchange and intravenous immunoglobulin, may improve OCD symptoms. Penicillin prophylaxis which aims to reduce streptococcal infection, however, does not seem to be effective in preventing OCD symptom exacerbations. Further research is needed to determine which children with OCD will benefit from immunomodulatory therapies.

8.6 CLINICAL PRACTICE RECOMMENDATIONS

The quality of available evidence addressing the efficacy and safety of neurosurgical treatments is variable and does not support a recommendation for neurosurgery in the treatment for OCD. However, it is recognised that some people may wish to consider this option when all other treatments have failed to produce an adequate response.

8.6.1.1 Neurosurgey is not recommended in the treatment of OCD. However, if a patient requests neurosurgery because they have severe OCD that is refractory to other forms of treatment, the following should be taken into consideration:

- Existing published criteria (for example, Matthews & Eljamel, 2003) should be used to guide decisions about suitability.
- Multidisciplinary teams with a high degree of expertise in the pharmacological and psychological treatment of OCD should have been recently involved in the patient's care. All pharmacological options should have been considered and every attempt should have been made to engage the individual in CBT (including ERP) and cognitive therapy, including very intensive and/or inpatient treatments.
- Standardised assessment protocols should be used pre- and post-operation and at medium- and long-term follow-ups in order to audit the interventions. These assessment protocols should include standardised measures of symptoms, quality of life, social and personality function, as well as comprehensive neuropsychological tests.
- Services offering assessment for neurosurgical treatments should have access to independent advice on issues such as adequacy of previous treatment and consent and should be subject to appropriate oversight.
- Post-operative care should be carefully considered, including pharmacological and psychological therapies.
- Services offering assessment for neurosurgical treatments should be committed to sharing and publishing audit information. **[GPP]**

9. USE OF HEALTH SERVICE RESOURCES

9.1 METHODS OF ECONOMIC EVALUATION

Methods of economic evaluation command a fairly high level of consensus and are reported in Drummond and colleagues (1997). However, where economic evaluations have been undertaken for anxiety disorders generally (see Issakidis *et al.*, 2004 for example), costing data for direct and indirect costs attributable to OCD are scarce[16]. Part of the issue is that OCD is a chronic, relapsing and remitting condition, where the result of treatment is modest. According to Bobes and colleagues (2001), OCD ranks tenth in the World Bank and World Health Organisation's leading causes of disability. The overall efficiency at which treatments are delivered is an important consideration when resources are limited. Beyond the healthcare perspective, the economic burden related to OCD comprises not only direct medical costs but also costs due to premature death, unemployment and reduced productivity when the condition is left untreated. Efficient service utilisation based upon rigorous health economic evaluations of OCD would reduce the social and economic burden of this condition, which would ensure optimal healthcare is delivered within the constraints of the national budget. Since OCD often starts in and continues beyond childhood and adolescence and in time increases in severity if left untreated, its economic burden accrues in the total direct and indirect costs that accumulate with time. The direct costs and cost effectiveness over a time course of 1-year are the focus of this section.

9.2 USE OF HEALTH SERVICE RESOURCES

Using the human capital approach, DuPont and colleagues (1995) estimated the direct and indirect costs of OCD to be $8,400 million in 1990 USD prices, 6% of their estimated $147,800 million cost of all mental illness in the United States. The same study estimated the indirect cost component of OCD, due to lost productivity, which amounted to $6,200 million, or 74% of the total estimated cost cited above (Dupont *et al.*, 1995).

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[16]The database searches for general health economic evidence for OCD resulted in a total of 41 references. Of these, nine were identified as potentially relevant. Secondary searches for relevant pharmacoeconomic papers resulted in a further eight references, of which, three were initially considered relevant to accepted criteria for health economic appraisal (as reported by Drummond *et al.*, 1997). A further four potentially eligible references were found by handsearching. Full texts of all potentially eligible studies (including those where relevance/eligibility was not clear from the abstract) were obtained, a total of 16 papers. At this stage inclusion was not limited to papers only from the UK.

9.3 PRIMARY CARE DRUG THERAPY VERSUS SECONDARY CARE CBT VERSUS COMBINED CBT PLUS SSRI THERAPY

There are a number of medications that are routinely administered in a primary care setting for the initial treatment of OCD. Traditionally, the most expensive of these has been the SSRIs. However, the differences in cost between alternative treatments need to be evaluated in light of their net effects beyond placebo. To accomplish this, one needs to know both the cost and net effectiveness of each treatment. For example, a 1-year course of the SSRI paroxetine (20 mg tablet in generic form) would cost an estimated £289, including three GP prescribing sessions[17] and follow-up costs,[18] which amounts to ±£10 in comparison with other generic drugs.

A meta-analysis was undertaken to obtain the weighted averages (according to number of patients in each study) of trial-based effectiveness data, based on 10 studies[19] reported in this guideline. The results suggest that approximately 43% of OCD patients respond following a 12-month course of SSRI therapy, in comparison with 27% in the placebo group. This translates into a 16% treatment-related response rate. Four sensitivity analyses are included in this section to show how cost effectiveness may change in light of changes to effectiveness assumptions, such as those estimated by experts in the GDG. For clinician-guided CBT, the results from Greist and colleagues (2002) and Cordioli and colleagues (2003) suggest that 56% and 70% of OCD patients respond following a 12 month course of CBT, in comparison with 14% (systematic relaxation) and 4% (waitlist control) of controls. Therefore, the average treatment-related response rate for CBT was 53%, meaning that 53/100 patients respond, due to the treatment alone, following clinician-guided CBT[20]. The treatment-related response rate of combined SSRI and clinician-guided CBT plus ERP is estimated to be slightly higher than these therapies alone, with 28 and 32% more respectively than SSRI and CBT as monotherapies. When the response rates of the individual treatments are averaged it is estimated that the overall response rates to combined treatment would be 63% within 3–12 months[21]. In comparison with SSRIs,

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[17]The number of sessions is based on the expert opinion of the GDG.

[18]£180 for the direct drug costs (West Midlands Medicines Information Service, 2003) plus £31 for first prescribing session (Netten & Curtis, 2003), plus 3 3 £26 for clinical consultations (GDG), including overheads and qualification costs = £289.

[19]Beasley *et al.* (1992), Burnham *et al.* (1993), Greist *et al.* (1995), Hollander *et al.* (2003b), Hollander *et al.* (2003d), Kamijima *et al.* (2004), Mallya *et al.* (1992), Montgomery (1993), Montgomery *et al.* (2001), Zohar & Judge (1996).

[20]Based on the midpoint of the weighted averages of 0.42 and 0.65 reported in Greist and colleagues (2002) and Cordioli and colleagues (2003), respectively.

[21]The combined ERP 1 clomipramine versus clomipramine alone study of Simpson (2004) yielded a net treatment-related response value of 0.32 (calculated as 0.64 for combined therapy 20.32 for the clomipramine control). When this value is added to the 0.12 net response rate for clomipramine alone (Burnham *et al.*, 1993; Stein *et al.*, 1992; Zohar & Judge, 1996), the combined response rate is 0.44 for CBT/ERP and clomipramine. We can call this value Alpha. The combined ERP 1 SRI versus ERP alone is adapted from the study by Hohagen and colleagues (1998). This study yielded a net treatment-related response value of 0.28 above the average of 0.53 that is the midpoint of the treatment-related responses calculated from Greist and colleagues(2002; 0.42) and Cordioli and colleagues(2003; 0.65). The combined response rate for ERP 1 SRI is therefore 0.81. We can call this value Beta. The midpoint of Alpha (0.44) and Beta (0.81) is 0.63. This estimate is included in Table 3 for combined therapy.

in the short term, treatment costs are known to be higher with clinician-guided CBT because such therapy is labour intensive and requires specialist knowledge for optimal delivery. The GDG estimated that approximately 16 1-hour sessions of CBT would be a realistic frequency and duration of therapy. Delivered by a clinical psychologist, the sum of these sessions will cost £1,056[22], or £767 more than the above SSRI monotherapy regimen.

Component costs and baseline effectiveness estimates are reported in Table 3. There are variations in both cost and effectiveness in comparing alternative monotherapies with their combination. To illustrate these differences, incremental cost-effectiveness ratios (ICERs) are compared in Tables 4 and 5, with GDG-suggested comparisons reported in parentheses.

9.4 INTERPRETATION

Comparing the treatments for OCD presented in Table 3, the average cost per treatment-related response was £1,806 for SSRI therapy, £1,992 for CBT, and £2,135 for combined SSRI plus CBT. In all cases the average cost per treatment-related response falls within generally accepted limits of cost effectiveness. Even if the response rates were .10 higher or lower, they would still fall within these limits. However, these average costs are unsuitable for decision making and it is important to consider the incremental cost effectiveness between the various treatments.

In the nominal scenario, CBT is dominated by (that is, has an inferior cost-effectiveness ratio) a linear mixture of SSRIs and combination therapy. In other words, this is a case of 'extended dominance' where treating the entire presenting population with CBT alone is more expensive and less effective than treating part of the population with SSRIs and the remainder with combination therapy. This means that the only relevant comparison is the incremental cost effectiveness of SSRI therapy versus combined CBT + SSRI therapy, which costs £2,247 to achieve an additional treatment-related response. Dividing this ICER value by the NICE cost effectiveness lower-threshold of £20,000 per QALY yields 0.11 of a QALY. This value is the minimum QALY gain per response that would be required for combined therapy to become cost effective in comparison with SSRI therapy alone, at the lower threshold. At a cost-effectiveness threshold of £30,000 per QALY (NICE upper-threshold) an additional responder would have to be worth 0.07 of a QALY (that is, a 7% gain in quality of life).

In terms of EuroQOL, a standardised and self-reporting mechanism of rating health outcome that is weighted to the preferences of the UK population, a move from level 2 (moderately anxious or depressed) to level 1 (not anxious or depressed) for 6 months represents a gain of at least 0.035 of a QALY, or a 3.5% gain in quality of life. In the case of OCD, where the response to treatment translates into a gain of at least a 7% gain in quality of life, a cost-effectiveness ratio of £2,247 to achieve an additional treatment-related response is likely to represent good value for money. Under the

[22]£66 (per hour of client contact, Netten & Curtis, 2003); 16 = £1,056.

Table 3: Cost and effectiveness estimates of alternative treatments for OCD

Per Patient	**Generic SSRI[23] 12-month course (GDG expert estimate for comparison)**	**Clinician-guided CBT (GDG expert estimate for comparison)**	**Combined SSRI and clinician-guided CBT (GDG expert estimate for comparison)**	**References**
Treatment-related response rate	16% (50%)	53% (50%)	63% (60%)	Drugs[24], CBT[25], Combined[26]
Cost (£) per case	289	1,056	1,345	Drugs[27], CBT[28], Combined[29]
Responders in cohort of 100 cases	16 (50)	53 (50)	63 (60)	As above
Cost in cohort of 100 cases	£28,900	£105,600	£134,500	As above
Average cost per patient per treatment-related response	£1,806	£1,992	£2,135	Calculated by dividing total cost per number of responders

[23]12-month course of the SSRI, paroxetine; calculations as cited previously.
[24]Crude response rate based on midpoint of the weighted average of 12 studies cited previously and compared in a sensitivity analysis to expert opinions of the GDG.
[25]Crude response rate based on midpoint of the weighted average of 2 studies cited previously and compared in a sensitivity analysis to expert opinions of the GDG.
[26]Crude response rate based on the midpoint of the response rates of CBT (including ERP) plus SSRI versus CBT (including ERP) alone (Hohagen *et al.*, 1998), and ERP with clomipramine versus clomipramine alone (Simpson, 2004); confirmed by expert opinions (GDG).
[27]West Midlands Medicines Information Service (2003).
[28]Netten and Curtis (2003).
[29]Sum of the cost of drug therapy plus a 12-month course of clinician-guided CBT.

Table 4: Incremental cost-effectiveness analysis: ICERs estimated for the nominal scenario (response rates = 16% for SSRI, 53% for CBT, and 63% for combined); assuming population of 100 patients treated

Comparison	£2003/4 cost per 100 treated	Number of additional treatment-related responders per 100 individuals in comparison to control group	£Cost per additional treatment-related response
Incremental cost SSRI versus CBT	76,700	–	–
Incremental effectiveness SSRI versus CBT	–	37	–
ICER SSRI versus CBT	–	–	2,073 (dominance)
Incremental cost SSRI versus combined	105,600	–	–
Incremental effectiveness SSRI versus combined	–	47	–
ICER SSRI versus combined	–	–	2,247
Incremental cost CBT versus combined	28,900	–	–
Incremental effectiveness CBT versus combined	–	10	–
ICER CBT versus combined	–	–	2,890 (dominance)

present assumptions, combined therapy is likely to be a cost-effective option. If at least an additional 0.07 of a QALY is gained per additional response, combination therapy will be more cost effective than SSRI therapy alone.

There will be sub-groups for whom SSRIs are not an option because they are contraindicated, ineffective for whatever reason, or because individuals refuse to take them. In such instances, CBT alone may be cost effective and further analyses will need to consider the 'do nothing' option compared with CBT alone. The results reported in these tables did not include 'do nothing' options for each of the three treatments because the control groups were not comparable across studies. Further,

Table 5: Sensitivity analysis 1: estimations of cost-effectiveness assuming estimates of the GDG (response rates = 50% for SSRI, 50% for CBT, and 60% for combined therapies); assuming population of 100 patients treated

Comparison	**£2003/4 cost per 100 treated**	**Number of additional treatment-related responders per 100 individuals in comparison with control group**	**£Cost per treatment-related response**
Incremental cost SSRI versus CBT	76,700	–	–
Incremental effectiveness SSRI versus CBT	–	0 (Cost-minimisation situation; no difference in effectiveness – only difference in cost)	–
ICER SSRI versus CBT	–	–	£76,700 to achieve no difference in outcome (dominance)
Incremental cost SSRI versus combined	105,600	–	–
Incremental effectiveness SSRI versus combined	–	10	–
ICER SSRI versus combined	–	–	10,560
Incremental cost CBT versus combined	28,900	–	–
Incremental effectiveness CBT versus combined	–	10	–
ICER CBT versus combined	–	–	2,890 (dominance)

the estimates of effectiveness for the CBT alone and combined SSRI and CBT treatments are based on very few studies and the control conditions are problematic. Consequently, the scenarios should be considered as illustrative only until more data from better controlled studies are available.

This model of 100 individuals did not consider the fact that patients often remain on drugs for much longer than a 12-month course of CBT, which will have an impact on the cost effectiveness. No value has been placed on patient choice: it has been assumed that all treatment options are equally acceptable to all patients. Another limitation is that response rates and response thresholds differed between the studies on which the weighted averages of effectiveness were based. Some utilised a 25% Y-BOCS threshold and others a 35% Y-BOCS score, for example. Others took the CGI as their scale of comparison. Future research should aim to utilise comparable scales of analysis and comparable samples of patients and presentations.

It is important to note that this model only considers up to a 12-month window of therapy. Quality of life gain due to response in the first and subsequent years must be weighed against the likelihood of relapse or a decline in response in the first and subsequent years. It may be that CBT, whether delivered as a monotherapy or as combined therapy, provides a more lasting response and/or less chance of relapse when medication is withdrawn. If there is a sufficient quality of life gain from response within 12 months to justify combined CBT plus SSRI therapy compared with SSRI therapy alone, any QALY gain beyond 12 months would only strengthen the cost effectiveness of this option.

9.4.1 Multi-way and extreme scenario sensitivity analyses based on GDG estimations and extreme effectiveness assumptions

Based on expert approximations of the GDG, the response rates for SSRI therapy, clinician-guided CBT and combined SSRI plus CBT therapy were estimated at 50%, 50%, and 60%, respectively. These estimates differ from approximations based on published data and are included in Sensitivity Analysis 1 (Table 5). If both SSRIs and CBT achieve a 50% treatment-related response rate, then simple cost-minimisation analysis will favour SSRIs as a first-line treatment because they are significantly cheaper. Thus, inclusion of CBT in this analysis is meaningless as it is dominated by SSRI therapy.

Using the GDG assumptions (reported in the parentheses of Table 3 that form the basis of Table 5), the incremental cost effectiveness of combined CBT plus SSRI therapy compared with SSRI monotherapy amounts to £2,890 per additional treatment-related response. Dividing this ICER value by the NICE cost effectiveness lower threshold of £20,000 per QALY yields 0.14 (14%) of a QALY, which is the minimum gain per response that would be required for combined therapy to become cost effective in comparison with SSRI monotherapy. At the upper-threshold of £30,000 per QALY, an additional responder would need to be worth 0.10 of a QALY, or a 10% gain in the quality of life, which is double the amount of gain required under the assumptions in Tables 3 and 4.

Tables 6–8 show extreme scenario analyses that demonstrate how sensitive cost effectiveness results are at present to changes in the effectiveness values.

Table 6: Sensitivity analysis 2: extreme scenario estimations of cost-effectiveness assuming equal treatment-related response rates of 25% between all treatments; assuming population of 100 patients treated

Comparison	**£2003/4 cost per 100 treated**	**Number of additional treatment-related responders per 100 individuals in comparison with control group**	**£Cost per treatment-related response**
Incremental cost SSRI versus CBT	76,700	–	–
Incremental effectiveness SSRI versus CBT	–	0 (Cost-minimisation situation; no difference in effectiveness – only difference in cost)	–
ICER SSRI versus CBT	–	–	£76,700 to achieve no difference in outcome (dominance)
Incremental cost SSRI versus combined	105,600	–	–
Incremental effectiveness SSRI versus combined	–	0 (Cost-minimisation situation; no difference in effectiveness – only difference in cost)	–
ICER SSRI versus combined	–	–	£105,600 to achieve no difference in outcome (dominance)
Incremental cost CBT versus combined	28,900	–	–

Continued

Table 6: (*Continued*)

Comparison	**£2003/4 cost per 100 treated**	**Number of additional treatment-related responders per 100 individuals in comparison to control group**	**£Cost per treatment-related response**
Incremental effectiveness CBT versus combined	–	0 (Cost-minimisation situation; no difference in effectiveness – only difference in cost)	–
ICER CBT versus combined	–	–	£28,900 to achieve no difference in outcome (dominance)

If all treatment-related response estimates were equal between the comparators at 25% each, Table 6 shows that a cost-minimisation situation would arise where it would not be cost effective to offer the comparator options since they would provide no benefit and accrue only a cost.

Table 7 assumes that the treatment-related response rates for SSRIs, CBT and combined treatments are 30%, 40% and 50%, respectively. In this case, as in the case of Table 4, CBT is dominated by a linear mixture of SSRIs and combination therapy. The cost effectiveness of SSRI compared with combined treatment is therefore the only meaningful comparison. At the upper-threshold of £30,000 per QALY, an additional responder would need to be worth a 35% gain in the quality of life, which is seven times the amount of gain required under the assumptions in Tables 3 and 4.

If the treatment-related response rates for SSRIs, CBT and combined treatments are inversed from the scenario in Table 7, then, as depicted in Table 8, SSRIs would become dominated by a mixture of CBT and combined therapies. Combined treatments compared with SSRIs would then be the only meaningful comparison and a response would need to be worth a 35% gain in the quality of life in order for SSRIs to be cost effective in comparison with combined treatments.

In summary, the results depicted in this section are highly sensitive to changes in the effectiveness assumptions and future studies will need to establish confidence in these estimates of effectiveness before any estimates of cost effectiveness can be considered robust. In the meantime, it is anticipated that Tables 3–8 can serve as a

Table 7: Sensitivity analysis 3: extreme scenario estimations of cost-effectiveness assuming linear 10% *increase* in response rate with SSRI, CBT and combined treatments (response rates = 30% for SSRI, 40% for CBT, and 50% for combined therapies); assuming population of 100 patients treated

Comparison	£2003/4 cost per 100 treated	Number of additional treatment-related responders per 100 individuals in comparison with control group	£Cost per treatment-related response
Incremental cost SSRI versus CBT	76,700	–	–
Incremental effectiveness SSRI versus CBT	–	10	–
ICER SSRI versus CBT	–	–	7,670 (dominance)
Incremental cost SSRI versus combined	105,600	–	–
Incremental effectiveness SSRI versus combined	–	10	–
ICER SSRI versus combined	–	–	10,560
Incremental cost CBT versus combined	28,900	–	–
Incremental effectiveness CBT versus combined	–	10	–
ICER CBT versus combined	–	–	2,890 (dominance)

template which can be continuously updated as new data become available on both cost and effectiveness.

9.5 NON-HEALTHCARE BURDEN

Whereas 0.5–2% may have OCD in the background population, only 25% of sufferers actually present to a GP and fewer still continue through the full course of treatment when such is made available. OCD also presents a considerable economic

Table 8: Sensitivity analysis 4: extreme scenario estimations of cost-effectiveness assuming linear 10% *decline* in response rate with SSRI, CBT and combined treatments (response rates = 50% for SSRI, 40% for CBT, and 30% for combined therapies); assuming population of 100 patients treated

Comparison	£ 2003/4 cost per 100 treated	Number of additional treatment-related responders per 100 individuals in comparison with control group	£Cost per treatment-related response
Incremental cost CBT versus SSRI	76,700	–	–
Incremental effectiveness CBT versus SSRI	–	10	–
ICER CBT versus SSRI	–	–	7,670 (dominance)
Incremental cost combined versus SSRI	105,600	–	–
Incremental effectiveness combined versus SSRI	–	10	–
ICER combined vs. SSRI	–	–	10,560
Incremental cost combined versus CBT	28,900	–	–
Incremental effectiveness combined versus CBT	–	10	–
ICER combined versus CBT	–	–	2,890 (dominance)

Note: Comparisons are reversed compared with Table 7 to highlight that the direction of cost effectiveness has changed with these inverse, extreme-scenario assumptions.

burden to the individual, family, health services, and society as a whole. The total cost accrued as a result of OCD-related illness is difficult to measure, because it extends beyond the primary, secondary, and tertiary care settings. Often sufferers accrue costs attributed to work-related absences, and beyond the immediate family. These latter costs may arise from the affected individuals as well as from family and friends who care for them.

A survey of an OCD consumer advocacy group estimated that, on average, a person with OCD loses fully 3 years of wages over their lifetime (Hollander & Wong, 1995). If an OCD sufferer incurs losses of £483.04 for every week they are absent (Income data services, 2004), this would amount to a total of £75,354 due to unemployment over this 3-year period, not including lost opportunities for career advancement and the cost to families and carers over their respective working life-times. The long-term, societal costs are beyond the NICE scope of the healthcare perspective. Nonetheless, they highlight the importance of delivering interventions at the earliest signs of illness and strengthen the conclusion that combination therapy may be a cost-effective option.

9.6 CONCLUSIONS AND FUTURE RECOMMENDATIONS

The analyses presented in this section assume a presentation of OCD that is, on average, of moderate intensity. More severe presentations will obviously require more intensive treatments over a longer duration of time. Further analyses are needed to compare the cost effectiveness of the alternative therapies over realistic time courses. Also, more research is required to compare the stepped-care approaches with the monotherapies, when treatments are administered in their combination over different time courses.

The analyses presented in Tables 3–5 suggest that combined CBT plus SSRI therapy is a cost-effective option for individuals suffering from OCD who might otherwise receive only SSRI therapy. If the quality of life gains are not achieved at an acceptable incremental cost, however, SSRI would be the cost-effective option. CBT alone is unlikely to be cost effective with the assumptions reported in either Table 3 or Table 4. Delivery formats for CBT which require less therapist input per patient treated would result in lower treatment costs, and, depending on response rates, may prove to be cost effective. For individuals who are unable or unlikely to remit following SSRI therapy, CBT alone may be a cost-effective option depending on the quality of life gained per response and its cost effectiveness. From the perspective of the NHS, treating OCD as soon as it is identified, and through cost-effective means, is likely to be better value for money than treating a more severe presentation downstream.

10. SUMMARY OF RECOMMENDATIONS

Key priorities for implementation

All people with OCD or BDD

- Each PCT, mental healthcare trust and children's trust that provides mental health services should have access to a specialist obsessive-compulsive disorder (OCD)/body dysmorphic disorder (BDD) multidisciplinary team offering age-appropriate care. This team would perform the following functions: increase the skills of mental health professionals in the assessment and evidence-based treatment of people with OCD or BDD, provide high-quality advice, understand family and developmental needs, and, when appropriate, conduct expert assessment and specialist cognitive-behavioural and pharmacological treatment.
- OCD and BDD can have a fluctuating or episodic course, or relapse may occur after successful treatment. Therefore, people who have been successfully treated and discharged should be seen as soon as possible if re-referred with further occurrences of OCD or BDD, rather than placed on a routine waiting list. For those in whom there has been no response to treatment, care coordination (or other suitable processes) should be used at the end of any specific treatment programme to identify any need for continuing support and appropriate services to address it.

Adults with OCD or BDD

- In the initial treatment of adults with OCD, low intensity psychological treatments (including exposure and response prevention [ERP]) (up to 10 therapist hours per patient) should be offered if the patient's degree of functional impairment is mild and/or the patient expresses a preference for a low intensity approach. Low intensity treatments include:
  - brief individual cognitive behavioural therapy (CBT) (including ERP) using structured self-help materials
  - brief individual CBT (including ERP) by telephone
  - group CBT (including ERP) (note, the patient may be receiving more than 10 hours of therapy in this format).
- Adults with OCD with mild functional impairment who are unable to engage in low intensity CBT (including ERP), or for whom low intensity treatment has proved to be inadequate, should be offered the choice of either a course of a selective serotonin reuptake inhibitor (SSRI) or more intensive CBT (including ERP) (more than 10 therapist hours per patient), because these treatments appear to be comparably efficacious.

- Adults with OCD with moderate functional impairment should be offered the choice of either a course of an SSRI or more intensive CBT (including ERP) (more than 10 therapist hours per patient), because these treatments appear to be comparably efficacious.
- Adults with BDD with moderate functional impairment should be offered the choice of either a course of an SSRI or more intensive individual CBT (including ERP) that addresses key features of BDD.

Children and young people with OCD or BDD

- Children and young people with OCD with moderate to severe functional impairment, and those with OCD with mild functional impairment for whom guided self-help has been ineffective or refused, should be offered CBT (including ERP) that involves the family or carers and is adapted to suit the developmental age of the child as the treatment of choice. Group or individual formats should be offered depending upon the preference of the child or young person and their family or carers.
- Following multidisciplinary review, for a child (aged 8–11 years) with OCD or BDD with moderate to severe functional impairment, if there has not been an adequate response to CBT (including ERP) involving the family or carers, the addition of an SSRI to ongoing psychological treatment may be considered. Careful monitoring should be undertaken, particularly at the beginning of treatment.
- Following multidisciplinary review, for a young person (aged 12–18 years) with OCD or BDD with moderate to severe functional impairment if there has not been an adequate response to CBT (including ERP) involving the family or carers, the addition of an SSRI to ongoing psychological treatment should be offered. Careful monitoring should be undertaken, particularly at the beginning of treatment.
- All children and young people with BDD should be offered CBT (including ERP) that involves the family or carers and is adapted to suit the developmental age of the child or young person as first-line treatment.

10.1 GOOD PRACTICE POINTS RELEVANT TO THE CARE OF ALL PEOPLE WITH OCD OR BDD AND THEIR FAMILIES OR CARERS

10.1.1 Understanding

10.1.1.1 People with OCD or BDD are often ashamed and embarrassed by their condition and may find it very difficult to discuss their symptoms with healthcare professionals, friends, family or carers. Healthcare professionals should help patients, and their families or carers where appropriate, to understand the involuntary nature of the symptoms by providing accurate

information in an appropriate format on current understanding of the disorders from psychological and/or biological perspectives. **[GPP]**

10.1.1.2 When assessing people with OCD or BDD, healthcare professionals should sensitively explore the hidden distress and disability commonly associated with the disorders, providing explanation and information wherever necessary. In particular, people with OCD who are distressed by their obsessive thoughts should be informed that such thoughts are occasionally experienced by almost everybody, and when frequent and distressing are a typical feature of OCD. **[GPP]**

10.1.2 Continuity of care

10.1.2.1 OCD and BDD are frequently recurring or chronic conditions that often affect some of the most intimate aspects of a person's life. Healthcare professionals should therefore ensure continuity of care and minimise the need for multiple assessments by different healthcare professionals. **[GPP]**

10.1.2.2 Because OCD and BDD may occur across a person's lifespan, particular care should be given to the provision of appropriate care at all ages and a seamless transition between services aimed at specific ages, such as the transition from services for young people to services for adults. **[GPP]**

10.1.2.3 Careful consideration should be given to the effective integration and coordination of care of people with OCD and BDD across both primary and secondary care. There should be clear, written agreement among individual healthcare professionals about the responsibility for monitoring and treating people with OCD and BDD. A written copy of this agreement should be given to the patient. This should be in collaboration with the patient, and where appropriate

- the Care Programme Approach (CPA) should be used
- the patient's family or carers should be involved
- healthcare professionals should liaise with other professionals involved in providing care and support to the patient. **[GPP]**

10.1.3 Information and support

10.1.3.1 Treatment and care should take into account the individual needs and preferences of people with OCD or BDD. Patients should have the opportunity to make informed decisions about their care and treatment. Where patients do not have the capacity to make decisions, or children or young people are not old enough to do so, healthcare professionals should follow the Department of Health guidelines (*Reference guide to consent for examination or treatment* [2001]; available from www.dh.gov.uk). **[GPP]**

10.1.3.2 Good communication between healthcare professionals and people with OCD or BDD is essential. Provision of information, treatment and care

should be tailored to the needs of the individual, culturally appropriate, and provided in a form that is accessible to people who have additional needs, such as learning difficulties, physical or sensory disabilities, or limited competence in speaking or reading English. **[GPP]**

10.1.3.3 Healthcare professionals should consider informing people with OCD or BDD and their family or carers about local self-help and support groups, and encourage them to participate in such groups where appropriate. **[GPP]**

10.1.4 Religion and culture

10.1.4.1 Obsessive-compulsive symptoms may sometimes involve a person's religion, such as religious obsessions and scrupulosity, or cultural practices. When the boundary between religious or cultural practice and obsessive-compulsive symptoms is unclear, healthcare professionals should, with the patient's consent, consider seeking the advice and support of an appropriate religious or community leader to support the therapeutic process. **[GPP]**

10.1.5 Families and carers

10.1.5.1 Because OCD and BDD often have an impact on families and carers, healthcare professionals should promote a collaborative approach with people with OCD or BDD and their family or carers, wherever this is appropriate and possible. **[GPP]**

10.1.5.2 In the treatment and care of people with OCD or BDD, family members or carers should be provided with good information (both verbal and written) about the disorder, its likely causes, its course and its treatment. **[GPP]**

10.1.5.3 Assessment and treatment plans for people with OCD or BDD should, where appropriate, involve relevant family members or carers. In some cases, particularly with children and young people, when the symptoms of OCD or BDD interfere with academic or workplace performance, it may be appropriate to liaise with professionals from these organisations. Assessment should include the impact of rituals and compulsions on others (in particular on dependent children) and the degree to which carers are involved in supporting or carrying out behaviours related to the disorder. **[GPP]**

10.1.5.4 If dependent children are considered to be at risk of emotional, social or mental health problems as a result of the behaviour of a parent with OCD or BDD and/or the child's involvement in related activity, independent assessment of the child should be requested. If this is carried out, the parent should be kept informed at every stage of the assessment. **[GPP]**

10.1.5.5 In the treatment of people with OCD or BDD, especially when the disorder is moderate to severe or chronic, an assessment of their carer's social, occupational and mental health needs should be offered. **[GPP]**

10.2 STEPPED CARE FOR ADULTS, YOUNG PEOPLE AND CHILDREN WITH OCD OR BDD

The stepped-care model draws attention to the different needs of people with OCD and BDD, depending on the characteristics of their disorder, their personal and social circumstances, their age, and the responses that are required from services. It provides a framework in which to organise the provision of services in order to identify and access the most effective interventions (see Figure 4).

Stepped care attempts to provide the most effective but least intrusive treatments appropriate to a person's needs. It assumes that the course of the disorder is monitored and referral to the appropriate level of care is made depending on the person's difficulties. Each step introduces additional interventions; the higher steps normally assume interventions in the previous step have been offered and/or attempted, but there are situations where an individual may be referred to any appropriate level. The guidance follows the steps in the figure.

At all stages of assessment and treatment, families or carers should be involved as appropriate. This is particularly important in the treatment of children and young people with OCD or BDD where it may also be helpful to involve others in their network, for example teachers, school health advisors, educational psychologists, and educational social workers.

10.3 STEP 1: AWARENESS AND RECOGNITION

Although the more common forms of OCD are likely to be recognised when people report symptoms, less common forms of OCD and many cases of BDD may remain unrecognised, sometimes for many years. Relatively few mental health professionals or GPs have expertise in the recognition, assessment, diagnosis and treatment of the less common forms of OCD and BDD.

10.3.1.1 Each PCT, mental healthcare trust and children's trust that provides mental health services should have access to a specialist OCD/BDD multidisciplinary team offering age-appropriate care. This team would perform the following functions: increase the skills of mental health professionals in the assessment and evidence-based treatment of people with OCD or BDD, provide high-quality advice, understand family and developmental needs, and, when appropriate, conduct expert assessment and specialist cognitive-behavioural and pharmacological treatment. **[GPP]**

Figure 4: The stepped care model

Who is responsible for care?	What is the focus?	What do they do?
Step 6 Inpatient care or intensive treatment programmes CAMHS Tier 4	OCD or BDD with risk to life, severe self-neglect or severe distress or disability	Reassess, discuss options, care coordination, SSRI or clomipramine, CBT (including ERP), or combination of SSRI or clomipramine and CBT (including ERP), augmentation strategies, consider admission or special living arrangements
Step 5 Multidisciplinary care with expertise in OCD/BDD CAMHS Tier 3/4	OCD or BDD with significant comorbidity, or more severely impaired functioning and/or treatment resistance, partial response or relapse	Reassess, discuss options. **For adults:** SSRI or clomipramine, CBT (including ERP), or combination of SSRI or clomipramine and CBT (including ERP); consider care coordination, augmentation strategies, admission, social care. **For children and young people:** CBT (including ERP), then consider combined treatments of CBT (including ERP) with SSRI, alternative SSRI or clomipramine. For young people consider referral to specialist services outside CAMHS if appropriate
Step 4 Multidisciplinary care in primary or secondary care CAMHS Tier 2/3	OCD or BDD with comorbidity or poor response to initial treatment	Assess and review, discuss options. **For adults:** CBT (including ERP), SSRI, alternative SSRI or clomipramine, combined treatments. **For children and young people:** CBT (including ERP), then consider combined treatments of CBT (including ERP) with SSRI, alternative SSRI or clomipramine
Step 3 GP, primary care team, primary care mental health worker, family support team CAMHS Tier 1/2	Management and initial treatment of OCD or BDD	Assess and review, discuss options. **For adults according to impairment:** Brief individual CBT (including ERP) with self-help materials (for OCD), individual or group CBT (including ERP), SSRI, or consider combined treatments; consider involving the family/carers in ERP. **For children and young people:** Guided self-help (for OCD), CBT (including ERP), involve family or carers and consider involving school
Step 2 GP, practice nurses, school health advisers, health visitors, general health settings (including hospitals) CAMHS Tier 1	Recognition and assessment	Detect, educate, discuss treatment options, signpost voluntary support organisations, provide support to individuals/families/ work/schools, or refer to any of the appropriate levels
Step 1 Individuals, public organisations, NHS	Awareness and recognition	Provide, seek and share information about OCD or BDD and its impact on individualsand families/carers

10.3.1.2 Specialist mental health professionals in OCD or BDD should collaborate with local and national voluntary organisations to increase awareness and understanding of the disorders and improve access to high-quality information about them. Such information should also be made available to primary and secondary healthcare professionals, and to professionals from other public services who may come into contact with people of any age with OCD or BDD. **[GPP]**

10.3.1.3 Specialist OCD/BDD teams should collaborate with people with OCD or BDD and their families or carers to provide training for all mental health professionals, cosmetic surgeons and dermatology professionals. **[GPP]**

10.4 STEP 2: RECOGNITION AND ASSESSMENT

10.4.1 OCD

10.4.1.1 For people known to be at higher risk of OCD (such as individuals with symptoms of depression, anxiety, alcohol or substance misuse, BDD or an eating disorder), or for people attending dermatology clinics, healthcare professionals should routinely consider and explore the possibility of comorbid OCD by asking direct questions about possible symptoms such as the following:

- Do you wash or clean a lot?
- Do you check things a lot?
- Is there any thought that keeps bothering you that you would like to get rid of but can not?
- Do your daily activities take a long time to finish?
- Are you concerned about putting things in a special order or are you very upset by mess?
- Do these problems trouble you? **[C]**

10.4.1.2 In people who have been diagnosed with OCD, healthcare professionals should assess the risk of self-harm and suicide, especially if they have also been diagnosed with depression. Part of the risk assessment should include the impact of their compulsive behaviours on themselves or others. Other comorbid conditions and psychosocial factors that may contribute to risk should also be considered. **[GPP]**

10.4.1.3 If healthcare professionals are uncertain about the risks associated with intrusive sexual, aggressive or death-related thoughts reported by people with OCD, they should consult mental health professionals with specific expertise in the assessment and management of OCD. These themes are common in people with OCD at any age, and are often misinterpreted as indicating risk. **[GPP]**

10.4.2 BDD

10.4.2.1 For people known to be at higher risk of BDD (such as individuals with symptoms of depression, social phobia, alcohol or substance misuse, OCD or an eating disorder), or for people with mild disfigurements or blemishes who are seeking a cosmetic or dermatological procedure, healthcare professionals should routinely consider and explore the possibility of BDD. **[GPP]**

10.4.2.2 In the assessment of people at higher risk of BDD, the following five questions should be asked to help identify individuals with BDD:

- Do you worry a lot about the way you look and wish you could think about it less?
- What specific concerns do you have about your appearance?
- On a typical day, how many hours a day is your appearance on your mind? (More than 1 hour a day is considered excessive)
- What effect does it have on your life?
- Does it make it hard to do your work or be with friends? **[GPP]**

10.4.2.3 People with suspected or diagnosed BDD seeking cosmetic surgery or dermatological treatment should be assessed by a mental health professional with specific expertise in the management of BDD. **[GPP]**

10.4.2.4 In people who have been diagnosed with BDD, healthcare professionals should assess the risk of self-harm and suicide, especially if they have also been diagnosed with depression. Other comorbid conditions and psychosocial factors that may contribute to risk should also be considered. **[GPP]**

10.4.2.5 All children and young people who have been diagnosed with BDD should be assessed for suicidal ideation and a full risk assessment should be carried out before treatment is undertaken. If risks are identified, all professionals involved in primary and secondary care should be informed and appropriate risk management strategies put into place. **[GPP]**

10.4.2.6 Specialist mental health professionals in BDD should work in partnership with cosmetic surgeons and dermatologists to ensure that an agreed screening system is in place to accurately identify people with BDD and that agreed referral criteria have been established. They should help provide training opportunities for cosmetic surgeons and dermatologists to aid in the recognition of BDD. **[GPP]**

10.5 STEPS 3–5: TREATMENT OPTIONS FOR PEOPLE WITH OCD OR BDD

Effective treatments for OCD and BDD should be offered at all levels of the healthcare system. The difference in the treatments at the higher levels will reflect increasing experience and expertise in the implementation of a limited range of therapeutic options. For many people, initial treatment may be best provided in

primary care settings. However, people with more impaired functioning, higher levels of comorbidity, or poor response to initial treatment will require care from teams with greater levels of expertise and experience in the management of OCD/BDD.

Irrespective of the level of care, the following recommendations should be taken into account when selecting initial treatments for people with OCD or BDD. The specific recommendations as to how to provide these treatments follow in the subsequent sections.

Irrespective of the level of care, the following recommendations should be taken into account when selecting initial treatments for people with OCD or BDD. The specific recommendations on how to provide these treatments follow in the subsequent sections.

Regulatory authorities have identified that the use of SSRIs to treat depression in children and young people may be associated with the appearance of suicidal behaviour, self-harm or hostility, particularly at the beginning of treatment. There is no clear evidence of an increased risk of self-harm and suicidal thoughts in young adults aged 18 years or older. But individuals mature at different rates and young adults are at a higher background risk of suicidal behaviour than older adults. Hence, young adults treated with SSRIs should be closely monitored as a precautionary measure. The Committee on Safety of Medicine's Expert Working Group on SSRIs, at a meeting in February 2005, advised that it could not be ruled out that the risk of suicidal behaviour, hostility and other adverse reactions seen in the paediatric depression trials applies to use in children or young people in all indications. Consequently, the recommendations about the use of SSRIs for people of any age with OCD or BDD have taken account of the position of regulatory authorities.

10.5.1 Initial treatment options

Adults

The intensity of psychological treatment has been defined as the hours of therapist input per patient. By this definition, most group treatments are defined as low intensity treatment (less than 10 hours of therapist input per patient), although each patient may receive a much greater number of hours of therapy.

10.5.1.1 In the initial treatment of adults with OCD, low intensity psychological treatments (including ERP) (up to 10 therapist hours per patient) should be offered if the patient's degree of functional impairment is mild and/or the patient expresses a preference for a low intensity approach. Low intensity treatments include:

- brief individual CBT (including ERP) using structured self-help materials **[C]**
- brief individual CBT (including ERP) by telephone **[C]**

- group CBT (including ERP) (note, the patient may be receiving more than 10 hours of therapy in this format). **[C]**

10.5.1.2 Adults with OCD with mild functional impairment who are unable to engage in low intensity CBT (including ERP), or for whom low intensity treatment has proved to be inadequate, should be offered the choice of either a course of an SSRI or more intensive CBT (including ERP) (more than 10 therapist hours per patient), because these treatments appear to be comparably efficacious. **[C]**

10.5.1.3 Adults with OCD with moderate functional impairment should be offered the choice of either a course of an SSRI or more intensive CBT (including ERP) (more than 10 therapist hours per patient), because these treatments appear to be comparably efficacious. **[B]**

10.5.1.4 Adults with OCD with severe functional impairment should be offered combined treatment with an SSRI and CBT (including ERP). **[C]**

10.5.1.5 Adults with BDD with mild functional impairment should be offered a course of CBT (including ERP) that addresses key features of BDD in individual or group formats. The most appropriate format should be jointly decided by the patient and the healthcare professional. **[B]**

10.5.1.6 Adults with BDD with moderate functional impairment should be offered the choice of either a course of an SSRI or more intensive individual CBT (including ERP) that addresses key features of BDD. **[B]**

10.5.1.7 Adults with BDD with severe functional impairment should be offered combined treatment with an SSRI and CBT (including ERP) that addresses key features of BDD. **[C]**

Children and young people

10.5.1.8 For children and young people with OCD with mild functional impairment, guided self-help may be considered in conjunction with support and information for the family or carers. **[C]**

10.5.1.9 Children and young people with OCD with moderate to severe functional impairment, and those with OCD with mild functional impairment for whom guided self-help has been ineffective or refused, should be offered CBT (including ERP) involving the family or carers and adapted to suit the developmental age of the child as the treatment of choice. Group or individual formats should be offered depending upon the preference of the child or young person and their family or carers. **[B]**

10.5.1.10 All children and young people with BDD should be offered CBT (including ERP) that involves the family or carers and is adapted to suit the developmental age of the child or young person as first-line treatment. **[C]**

10.5.1.11 If psychological treatment is declined by children or young people with OCD or BDD and their families or carers, or they are unable to engage in

treatment, an SSRI may be considered with specific arrangements for careful monitoring for adverse events. **[B]**

10.5.1.12 The co-existence of comorbid conditions, learning disorders, persisting psychosocial risk factors such as family discord, or the presence of parental mental health problems, may be factors if the child or young person's OCD or BDD is not responding to any treatment. Additional or alternative interventions for these aspects should be considered. The child or young person will still require evidence-based treatments for his or her OCD or BDD. **[C]**

10.5.2 How to use psychological interventions

Training

10.5.2.1 All healthcare professionals offering psychological treatments to people of all ages with OCD or BDD should receive appropriate training in the interventions they are offering and receive ongoing clinical supervision in line with the recommendations in Organising and Delivering Psychological Therapies (Department of Health, 2004)[30]. **[GPP]**

Adults

10.5.2.2 For adults with obsessive thoughts who do not have overt compulsions, CBT (including exposure to obsessive thoughts and response prevention of mental rituals and neutralising strategies) should be considered. **[B]**

10.5.2.3 For adults with OCD, cognitive therapy adapted for OCD may be considered as an addition to ERP to enhance long-term symptom reduction. **[C]**

10.5.2.4 For adults with OCD living with their family or carers, involving a family member or carer as a co-therapist in ERP should be considered where this is appropriate and acceptable to those involved. **[B]**

10.5.2.5 For adults with OCD with more severe functional impairment who are housebound, unable or reluctant to attend a clinic, or have significant problems with hoarding, a period of home-based treatment may be considered. **[C]**

10.5.2.6 For adults with OCD with more severe functional impairment who are housebound and unable to undertake home-based treatment because of the nature of their symptoms (such as contamination concerns or hoarding that prevents therapists' access to the patient's home), a period of CBT by telephone may be considered. **[C]**

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[30]Department of Health (2004) *Organising and Delivering Psychological Therapies*. London: Department of Health. Available from www.dh.gov.uk.

10.5.2.7 For adults with OCD who refuse or cannot engage with treatments that include ERP, individual cognitive therapy specifically adapted for OCD may be considered. **[C]**

10.5.2.8 When adults with OCD request forms of psychological therapy other than cognitive and/or behavioural therapies as a specific treatment for OCD (such as psychoanalysis, transactional analysis, hypnosis, marital/couple therapy) they should be informed that there is as yet no convincing evidence for a clinically important effect of these treatments. **[C]**

10.5.2.9 When family members or carers of people with OCD or BDD have become involved in compulsive behaviours, avoidance or reassurance seeking, treatment plans should help them reduce their involvement in these behaviours in a sensitive and supportive manner. **[GPP]**

10.5.2.10 Adults with OCD or BDD with significant functional impairment may need access to appropriate support for travel and transport to allow them to attend for their treatment. **[GPP]**

10.5.2.11 Towards the end of treatment, healthcare professionals should inform adults with OCD or BDD about how the principles learned can be applied to the same or other symptoms if they occur in the future. **[GPP]**

Children and young people

Psychological treatments for children and young people should be collaborative and engage the family or carers. When using psychological treatments for children or young people, healthcare professionals should consider the wider context and other professionals involved with the individual. The recommendations on the use of psychological interventions for adults may also be considered, where appropriate.

10.5.2.12 In the cognitive-behavioural treatment of children and young people with OCD or BDD, particular attention should be given to:

- developing and maintaining a good therapeutic alliance with the child or young person, as well as their family or carers
- maintaining optimism in both the child or young person and their family or carers
- collaboratively identifying initial and subsequent treatment targets with the child or young person
- actively engaging the family or carers in planning treatment and in the treatment process, especially in ERP where, if appropriate and acceptable, they may be asked to assist the child or young person
- encouraging the use of ERP if new or different symptoms emerge after successful treatment
- liaising with other professionals involved in the child or young person's life, including teachers, social workers and other healthcare professionals, especially when compulsive activity interferes with the ordinary functioning of the child or young person
- offering one or more additional sessions if needed at review appointments after completion of CBT. **[GPP]**

10.5.2.13 In the psychological treatment of children and young people with OCD or BDD, healthcare professionals should consider including rewards in order to enhance their motivation and reinforce desired behaviour changes. **[C]**

10.5.3 How to use pharmacological interventions in adults

Current published evidence suggests that SSRIs are effective in treating adults with OCD or BDD, although evidence for the latter is limited and less certain. However, SSRIs may increase the risk of suicidal thoughts and self-harm in people with depression and in younger people. It is currently unclear whether there is an increased risk for people with OCD or BDD. Regulatory authorities recommend caution in the use of SSRIs until evidence for differential safety has been demonstrated.

Starting the treatment

10.5.3.1 Common concerns about taking medication for OCD or BDD should be addressed. Patients should be advised, both verbally and with written material, that:

- craving and tolerance do not occur **[C]**
- there is a risk of discontinuation/withdrawal symptoms on stopping the drug, missing doses, or reducing the dose **[C]**
- there is a range of potential side effects, including worsening anxiety, suicidal thoughts and self-harm, which need to be carefully monitored, especially in the first few weeks of treatment **[C]**
- there is commonly a delay in the onset of effect of up to 12 weeks, although depressive symptoms improve more quickly **[C]**
- taking medication should not be seen as a weakness. **[GPP]**

Monitoring risk

10.5.3.2 Adults with OCD or BDD started on SSRIs who are not considered to be at increased risk of suicide or self-harm should be monitored closely and seen on an appropriate and regular basis. The arrangements for monitoring should be agreed by the patient and the healthcare professional, and recorded in the notes. **[GPP]**

10.5.3.3 Because of the potential increased risk of suicidal thoughts and self-harm associated with the early stages of SSRI treatment, younger adults (younger than age 30 years) with OCD or BDD, or people with OCD or BDD with comorbid depression, or who are considered to be at an increased risk of suicide, should be carefully and frequently monitored by healthcare professionals. Where appropriate, other carers – as agreed by the patient and the healthcare professional – may also contribute to the monitoring until the risk is no longer considered significant. The arrangements for monitoring should be agreed by the patient and the healthcare professional, and recorded in the notes. **[C]**

10.5.3.4 For adults with OCD or BDD at a high risk of suicide, a limited quantity of medication should be prescribed. **[C]**

10.5.3.5 When adults with OCD or BDD, especially those with comorbid depression, are assessed to be at a high risk of suicide, the use of additional support such as more frequent direct contacts with primary care staff or telephone contacts should be considered, particularly during the first weeks of treatment. **[C]**

10.5.3.6 For adults with OCD or BDD, particularly in the initial stages of SSRI treatment, healthcare professionals should actively seek out signs of akathisia or restlessness, suicidal ideation and increased anxiety and agitation. They should also advise patients to seek help promptly if symptoms are at all distressing. **[C]**

10.5.3.7 Adults with OCD or BDD should be monitored around the time of dose changes for any new symptoms or worsening of their condition. **[C]**

Choice of drug treatment

Selective serotonin reuptake inhibitors (SSRIs)

10.5.3.8 For adults with OCD, the initial pharmacological treatment should be one of the following SSRIs: fluoxetine, fluvoxamine, paroxetine, sertraline or citalopram[31]. **[A]**

10.5.3.9 For adults with BDD (including those with beliefs of delusional intensity), the initial pharmacological treatment should be fluoxetine[32] because there is more evidence for its effectiveness in BDD than there is for other SSRIs. **[B]**

10.5.3.10 In the event that an adult with OCD or BDD develops marked and/or prolonged akathisia, restlessness or agitation while taking an SSRI, the use of the drug should be reviewed. If the patient prefers, the drug should be changed to a different SSRI. **[C]**

10.5.3.11 Healthcare professionals should be aware of the increased risk of drug interactions when prescribing an SSRI to adults with OCD or BDD who are taking other medications. **[GPP]**

10.5.3.12 For adults with OCD or BDD, if there has been no response to a full course of treatment with an SSRI, healthcare professionals should check that the patient has taken the drug regularly and in the prescribed dose and that there is no interference from alcohol or substance use. **[GPP]**

10.5.3.13 For adults with OCD or BDD, if there has not been an adequate response to a standard dose of an SSRI, and there are no significant side effects after 4–6 weeks, a gradual increase in dose should be considered in line with the schedule suggested by the Summary of Product Characteristics. **[C]**

10.5.3.14 For adults with OCD or BDD, the rate at which the dose of an SSRI should be increased should take into account therapeutic response, adverse effects

[31]Citalopram does not have a UK Marketing Authorisation for use in OCD in adults at the date of publication (November 2005).

[32]Fluoxetine does not have a UK Marketing Authorisation for use in BDD at the date of publication (November 2005).

and patient preference. Patients should be warned about, and monitored for, the emergence of side effects during dose increases. **[GPP]**

10.5.3.15 If treatment for OCD or BDD with an SSRI is effective, it should be continued for at least 12 months to prevent relapse and allow for further improvements. **[C]**

10.5.3.16 When an adult with OCD or BDD has taken an SSRI for 12 months after remission (symptoms are not clinically significant and the person is fully functioning for at least 12 weeks), healthcare professionals should review with the patient the need for continued treatment. This review should consider the severity and duration of the initial illness, the number of previous episodes, the presence of residual symptoms, and concurrent psychosocial difficulties. **[GPP]**

10.5.3.17 If treatment for OCD or BDD with an SSRI is continued for an extended period beyond 12 months after remission (symptoms are not clinically significant and the person is fully functioning for at least 12 weeks), the need for continuation should be reviewed at regular intervals, agreed between the patient and the prescriber, and written in the notes. **[GPP]**

10.5.3.18 For adults with OCD or BDD, to minimise discontinuation/withdrawal symptoms when reducing or stopping SSRIs, the dose should be tapered gradually over several weeks according to the person's need. The rate of reduction should take into account the starting dose, the drug half-life and particular profiles of adverse effects. **[C]**

10.5.3.19 Healthcare professionals should encourage adults with OCD or BDD who are discontinuing SSRI treatment to seek advice if they experience significant discontinuation/withdrawal symptoms. **[C]**

Other drugs

10.5.3.20 The following drugs should not normally be used to treat OCD or BDD without comorbidity:

- Tricyclic antidepressants other than clomipramine
- Tricyclic-related antidepressants
- Serotonin and noradrenaline reuptake inhibitors (SNRIs), including venlafaxine
- Monoamine-oxidase inhibitors (MAOIs)
- Anxiolytics (except cautiously for short periods to counter the early activation of SSRIs). **[C]**

10.5.3.21 Antipsychotics as a monotherapy should not normally be used for treating OCD. **[C]**

10.5.3.22 Antipsychotics as a monotherapy should not normally be used for treating BDD (including beliefs of delusional intensity). **[C]**

10.5.4 Poor response to initial treatment for adults

If initial treatment does not result in a clinically significant improvement in both symptoms and functioning, other treatment options should be considered. When additional treatment options also fail to produce an adequate response, multidisciplinary teams with specific expertise in OCD/BDD should become involved. Their role should include supporting and collaborating with those professionals already involved in an individual's care.

10.5.4.1 For adults with OCD or BDD, if there has not been an adequate response to treatment with an SSRI alone (within 12 weeks) or CBT (including ERP) alone (more than 10 therapist hours per patient), a multidisciplinary review should be carried out. **[GPP]**

10.5.4.2 Following multidisciplinary review, for adults with OCD or BDD, if there has not been an adequate response to treatment with an SSRI alone (within 12 weeks) or CBT (including ERP) alone (more than 10 therapist hours per patient), combined treatment with CBT (including ERP) and an SSRI should be offered. **[C]**

10.5.4.3 For adults with OCD or BDD, if there has not been an adequate response after 12 weeks of combined treatment with CBT (including ERP) and an SSRI, or there has been no response to an SSRI alone, or the patient has not engaged with CBT, a different SSRI or clomipramine should be offered. **[C]**

10.5.4.4 Clomipramine should be considered in the treatment of adults with OCD or BDD after an adequate trial of at least one SSRI has been ineffective or poorly tolerated, if the patient prefers clomipramine or has had a previous good response to it. **[C]**

10.5.4.5 For adults with OCD or BDD, if there has been no response to a full trial of at least one SSRI alone, a full trial of combined treatment with CBT (including ERP) and an SSRI, and a full trial of clomipramine alone, the patient should be referred to a multidisciplinary team with specific expertise in the treatment of OCD/BDD for assessment and further treatment planning. **[GPP]**

10.5.4.6 The assessment of adults with OCD or BDD referred to multidisciplinary teams with specific expertise in OCD/BDD should include a comprehensive assessment of their symptom profile, previous pharmacological and psychological treatment history, adherence to prescribed medication, history of side effects, comorbid conditions such as depression, suicide risk, psychosocial stressors, relationship with family and/or carers and personality factors. **[GPP]**

10.5.4.7 Following multidisciplinary review, for adults with OCD if there has been no response to a full trial of at least one SSRI alone, a full trial of combined treatment with CBT (including ERP) and an SSRI, and a full trial of clomipramine alone, the following treatment options should also be considered (note, there is no evidence of the optimal sequence of the options listed below):

- additional CBT (including ERP) or cognitive therapy **[C]**
- adding an antipsychotic to an SSRI or clomipramine **[C]**
- combining clomipramine and citalopram. **[C]**

10.5.4.8 Following multidisciplinary review, for adults with BDD, if there has been no response to a full trial of at least one SSRI alone, a full trial of combined treatment with CBT (including ERP) and an SSRI, and a full trial of clomipramine alone, the following treatment options should also be considered (note, there is no evidence of the optimal sequence of the options listed below):

- additional CBT or cognitive therapy by a different multidisciplinary team with expertise in BDD. **[GPP]**
- adding buspirone[33] to an SSRI. **[C]**

10.5.4.9 For adults with BDD, if there has been no response to treatment, or the patient is not receiving appropriate treatment, more intensive monitoring is needed because the risk of suicide is high in people with BDD. **[GPP]**

10.5.4.10 Treatments such as combined antidepressants and antipsychotic augmentation should not be routinely initiated in primary care. **[GPP]**

How to use clomipramine for adults

10.5.4.11 For adults with OCD or BDD who are at a significant risk of suicide, healthcare professionals should only prescribe small amounts of clomipramine at a time because of its toxicity in overdose[34]. The patient should be monitored regularly until the risk of suicide has subsided. **[GPP]**

10.5.4.12 An electrocardiogram (ECG) should be carried out and a blood pressure measurement taken before prescribing clomipramine for adults with OCD or BDD at significant risk of cardiovascular disease. **[C]**

10.5.4.13 For adults with OCD or BDD, if there has not been an adequate response to the standard dose of clomipramine, and there are no significant side effects, a gradual increase in dose should be considered in line with the schedule suggested by the Summary of Product Characteristics. **[C]**

10.5.4.14 For adults with OCD or BDD, treatment with clomipramine should be continued for at least 12 months if it appears to be effective and because there may be further improvement. **[B]**

10.5.4.15 For adults with OCD or BDD, when discontinuing clomipramine, doses should be reduced gradually in order to minimise potential discontinuation/withdrawal symptoms. **[C]**

10.5.5 Poor response to initial treatment in children and young people

Current published evidence suggests that SSRIs are effective in treating children and young people with OCD. The only SSRIs licensed for use in children and young

[33]Buspirone does not have a UK Marketing Authorisation for use in BDD at the date of publication (November 2005).

[34]Refer to the Summary of Product Characteristics for details about appropriate dosage.

people with OCD are fluvoxamine and sertraline. When used as a treatment for depression, SSRIs can cause significant adverse reactions, including increased suicidal thoughts and risk of self-harm, but it is not known whether this same risk occurs with their use in OCD. SSRIs may be safer in depression when combined with psychological treatments (see the NICE guideline *Depression in children and young people*, available from www.nice.org.uk/CG028). Given that the UK regulatory authority has advised that similar adverse reactions cannot be ruled out in OCD, appropriate caution should be observed, especially in the presence of comorbid depression.

10.5.5.1 For a child or young person with OCD or BDD, if there has not been an adequate response within 12 weeks to a full trial of CBT (including ERP) involving the family or carers, a multidisciplinary review should be carried out. **[GPP]**

10.5.5.2 Following multidisciplinary review, for a *child* (aged 8–11 years) with OCD or BDD with moderate to severe functional impairment, if there has not been an adequate response to CBT (including ERP) involving the family or carers, the addition of an SSRI to ongoing psychological treatment may be considered. Careful monitoring should be undertaken, particularly at the beginning of treatment. **[C]**

10.5.5.3 Following multidisciplinary review, for a *young person* (aged 12–18 years) with OCD or BDD with moderate to severe functional impairment, if there has not been an adequate response to CBT (including ERP) involving the family or carers, the addition of an SSRI to ongoing psychological treatment should be offered. Careful monitoring should be undertaken, particularly at the beginning of treatment. **[B]**

10.5.5.4 For a child or a young person with OCD or BDD, if treatment with an SSRI in combination with CBT (including ERP) involving the family or carers is unsuccessful or is not tolerated because of side effects, the use of another SSRI or clomipramine[35] with careful monitoring may be considered, especially if the child or young person has had a positive response to these alternatives in the past. This should also be in combination with CBT (including ERP). **[C]**

10.5.6 How to use pharmacological treatments in children and young people

In adults with OCD treated by medication, there is some clinical trial evidence regarding the onset of therapeutic response, the dose needed, the rate of increase of dose, the duration of treatment and the likelihood of relapse on discontinuation. Trials of these aspects have not been done in children and/or young people, but the following

[35]Clomipramine does not have a UK Marketing Authorisation for use in OCD and BDD in children and young people at the date of publication (November 2005).

good practice for prescribing SSRIs or clomipramine is based on adult trials and clinical experience.

How to use SSRIs in children and young people

10.5.6.1 An SSRI should only be prescribed to children and young people with OCD or BDD following assessment and diagnosis by a child and adolescent psychiatrist who should also be involved in decisions about dose changes and discontinuation. **[GPP]**

10.5.6.2 When an SSRI is prescribed to children and young people with OCD or BDD, it should be in combination with concurrent CBT (including ERP). If children and young people are unable to engage with concurrent CBT, specific arrangements should be made for careful monitoring of adverse events and these arrangements should be recorded in the notes. **[C]**

10.5.6.3 Children and young people with OCD or BDD starting treatment with SSRIs should be carefully and frequently monitored and seen on an appropriate and regular basis. This should be agreed by the patient, his or her family or carers and the healthcare professional, and recorded in the notes. **[GPP]**

10.5.6.4 A licensed medication (sertraline[36] or fluvoxamine[37]) should be used when an SSRI is prescribed to children and young people with OCD, except in patients with significant comorbid depression when fluoxetine[38] should be used, because of current regulatory requirements. **[A]**

10.5.6.5 Fluoxetine[39] should be used when an SSRI is prescribed to children and young people with BDD. **[C]**

10.5.6.6 For children and young people with OCD or BDD who also have significant depression, the NICE recommendations for the treatment of childhood depression[40] should be followed and there should be specific monitoring for suicidal thoughts or behaviours. **[GPP]**

10.5.6.7 Children and young people with OCD or BDD starting treatment with SSRIs should be informed about the rationale for the drug treatment, the delay in onset of therapeutic response (up to 12 weeks), the time course of treatment, the possible side effects and the need to take the medication as prescribed. Discussion of these issues should be supplemented by written information appropriate to the needs of the child or young person and their family or carers. **[GPP]**

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[36]Sertraline has a UK Marketing Authorisation for use in OCD in children over 6 years at the date of publication (November 2005).

[37]Fluvoxamine has a UK Marketing Authorisation for use in OCD in children over 8 years at the date of publication (November 2005).

[38]Fluoxetine does not have a UK Marketing Authorisation for use in OCD in children and young people at the date of publication (November 2005).

[39]Fluoxetine does not have a UK Marketing Authorisation for use in BDD at the date of publication (November 2005).

[40]Depression in children: identification and management of depression in children and young people in primary care and specialist services. *NICE Clinical Guideline* No. 28, available from www.nice.org.uk/CG028.

10.5.6.8 The starting dose of medication for children and young people with OCD or BDD should be low, especially in younger children. A half or quarter of the normal starting dose may be considered for the first week. **[C]**

10.5.6.9 If a lower dose of medication for children and young people with OCD or BDD is ineffective, the dose should be increased until a therapeutic response is obtained, with careful and close monitoring for adverse events. The rate of increase should be gradual and should take into account the delay in therapeutic response (up to 12 weeks) and the age of the patient. Maximum recommended doses for children and young people should not be exceeded. **[C]**

10.5.6.10 Children and young people prescribed an SSRI, and their families or carers, should be informed by the prescribing doctor about the possible appearance of suicidal behaviour, self-harm or hostility, particularly at the beginning of treatment. They should be advised that if there is any sign of new symptoms of these kinds, they should make urgent contact with their medical practitioner. **[GPP]**

10.5.6.11 Where children or young people with OCD or BDD respond to treatment with an SSRI, medication should be continued for at least 6 months post-remission (i.e. symptoms are not clinically significant and the child or young person is fully functioning for at least 12 weeks). **[C]**

How to use clomipramine in children and young people

10.5.6.12 Children and young people with OCD or BDD and their families or carers should be advised about the possible side effects of clomipramine, including toxicity in overdose. **[C]**

10.5.6.13 Before starting treatment with clomipramine in children and young people with OCD or BDD, an ECG should be carried out to exclude cardiac conduction abnormalities. **[C]**

10.5.6.14 For a child or young person with OCD or BDD, if there has not been an adequate response to the standard dose of clomipramine, and there are no significant side effects, a gradual increase in dose may be cautiously considered. **[C]**

10.5.6.15 Treatment of a child or young person with OCD or BDD with clomipramine should be continued for at least 6 months if the treatment appears to be effective, because there may be further improvement in symptoms. **[B]**

Stopping or reducing SSRIs and clomipramine in children and young people

10.5.6.16 In children and young people with OCD or BDD, an attempt should be made to withdraw medication if remission has been achieved (i.e. symptoms are no longer clinically significant and the child or young person is fully functioning) and maintained for at least 6 months, and if that is their wish. Patients and their family or carers should be warned that relapse and/or discontinuation/withdrawal symptoms may occur. They should be advised to contact their medical practitioner should symptoms of discontinuation/withdrawal arise. **[C]**

10.5.6.17 For children and young people with OCD or BDD, to minimise discontinuation/withdrawal symptoms on reducing or stopping antidepressants, particularly SSRIs, the dose should be tapered gradually over several weeks according to the individual's need. The rate of reduction should take into account the starting dose, the drug half-life and particular profiles of adverse effects. **[C]**

10.5.6.18 Children and young people with OCD or BDD should continue with psychological treatment throughout the period of drug discontinuation because this may reduce the risk of relapse. **[C]**

Other drugs

10.5.6.19 Tricyclic antidepressants other than clomipramine should not be used to treat OCD or BDD in children and young people. **[C]**

10.5.6.20 Other antidepressants (MAOIs, SNRIs) should not be used to treat OCD or BDD in children and young people. **[C]**

10.5.6.21 Antipsychotics should not be used alone in the routine treatment of OCD or BDD in children or young people, but may be considered as an augmentation strategy. **[C]**

10.6 STEP 6: INTENSIVE TREATMENT AND INPATIENT SERVICES FOR PEOPLE WITH OCD OR BDD

10.6.1.1 People with severe, chronic, treatment-refractory OCD or BDD should have continuing access to specialist treatment services staffed by multidisciplinary teams of healthcare professionals with expertise in the management of the disorders. **[C]**

10.6.1.2 Inpatient services, with specific expertise in OCD and BDD, are appropriate for a small proportion of people with these disorders, and may be considered when:

- there is risk to life
- there is severe self-neglect
- there is extreme distress or functional impairment
- there has been no response to adequate trials of pharmacological/psychological/combined treatments over long periods of time in other settings
- a person has additional diagnoses, such as severe depression, anorexia nervosa or schizophrenia, that make outpatient treatment more complex
- a person has a reversal of normal night/day patterns that make attendance at any daytime therapy impossible
- the compulsions and avoidance behaviour are so severe or habitual that they cannot undertake normal activities of daily living. **[GPP]**

10.6.1.3 A small minority of adults with long-standing and disabling obsessive-compulsive symptoms that interfere with daily living and have prevented them from developing a normal level of autonomy may, in addition to treatment, need suitable accommodation in a supportive environment that will enable them to develop life skills for independent living. **[GPP]**

10.6.1.4 Neurosurgery is not recommended in the treatment of OCD. However, if a patient requests neurosurgery because they have severe OCD that is refractory to other forms of treatment, the following should be taken into consideration:

- Existing published criteria (such as Matthews and Eljamel, 2003) should be used to guide decisions about suitability.
- Multidisciplinary teams with a high degree of expertise in the pharmacological and psychological treatment of OCD should have been recently involved in the patient's care. All pharmacological options should have been considered and every attempt should have been made to engage the individual in CBT (including ERP) and cognitive therapy, including very intensive and/or inpatient treatments.
- Standardised assessment protocols should be used at pre- and post-operation and at medium- and long-term follow-ups in order to audit the interventions. These assessment protocols should include standardised measures of symptoms, quality of life, social and personality function, as well as comprehensive neuropsychological tests.
- Services offering assessment for neurosurgical treatments should have access to independent advice on issues such as adequacy of previous treatment and consent and should be subject to appropriate oversight.
- Post-operative care should be carefully considered, including pharmacological and psychological therapies.
- Services offering assessment for neurosurgical treatments should be committed to sharing and publishing audit information. **[GPP]**

10.6.1.5 For children and young people with severe OCD or BDD with high levels of distress and/or functional impairment, if there has been no response to adequate treatment in outpatient settings, or there is significant self-neglect or risk of suicide, assessment for intensive inpatient treatment in units where specialist treatment for children or young people with OCD or BDD is available should be offered. **[GPP]**

10.7 DISCHARGE AFTER RECOVERY

10.7.1.1 When a person of any age with OCD or BDD is in remission (symptoms are not clinically significant and the person is fully functioning for 12 weeks), he or she should be reviewed regularly for 12 months by a mental health professional. The exact frequency of contact should be agreed between the professional and the person with OCD or BDD and/or the

family and/or carer and recorded in the notes. At the end of the 12-month period if recovery is maintained the person can be discharged to primary care. **[C]**

10.7.1.2 OCD and BDD can have a fluctuating or episodic course, or relapse may occur after successful treatment. Therefore, people who have been successfully treated and discharged should be seen as soon as possible if re-referred with further occurrences of OCD or BDD, rather than placed on a routine waiting list. For those in whom there has been no response to treatment, care coordination (or other suitable processes) should be used at the end of any specific treatment programme to identify any need for continuing support and appropriate services to address it. **[GPP]**

10.8 RESEARCH RECOMMENDATIONS

10.8.1 Treatment of OCD and BDD among young people and young adults

10.8.1.1 Appropriately blinded randomised controlled trials (RCTs) should be conducted to assess the acute and long-term efficacy (including measures of social function and quality of life), acceptability and the cost effectiveness of CBT and SSRIs, alone and in combination, compared with each other and with appropriate control treatments for both the psychological and pharmacological arms. These should be carried out in a broadly based sample of young people and young adults (e.g. aged 12–25 years) diagnosed with OCD and BDD across a range of functional impairment (using minimal exclusion criteria). The trials should be powered to examine the effect of treatment for combined versus single-strand treatments and involve a follow-up of 1, 2 and 5 years. Any treatment received in the follow-up period should also be recorded.

10.8.2 CBT treatment intensity formats among adults with OCD

10.8.2.1 Appropriately blinded RCTs should be conducted to assess the efficacy (including measures of social function and quality of life), acceptability and the cost effectiveness of different delivery formats of CBT that include ERP for adults with OCD, including brief individual CBT using structured self-help materials, brief individual CBT by telephone, group CBT and standard individual CBT compared with each other and with credible psychological treatment that is not specific to OCD and BDD (such as anxiety management training) in a broadly based sample of people diagnosed with OCD across a range of functional impairment (using minimal exclusion criteria). The trials should be powered to examine the effect of treatment in different bands of

severity or functional impairment and involve a follow-up of 1 and 2 years. Any treatment received in the follow-up period should also be recorded.

10.8.3 CBT for adults with OCD who have not responded to treatment

10.8.3.1 An appropriately blinded RCT should be conducted to assess the efficacy (including measures of social functioning and quality of life as well as OCD) of intensive versus spaced individual treatments (that include both ERP and cognitive therapy elements) compared with a treatment-as-usual control in a broadly based sample of adults diagnosed with OCD who have not responded to one or more adequate trials of an SSRI or clomipramine and one or more trials of CBT (that included ERP). The trial should be powered to examine the relative efficacy of intensive versus spaced treatment and involve a follow-up of 1 and 2 years. Any treatment received in the follow-up period should also be recorded.

10.8.4 Screening for OCD and BDD

10.8.4.1 Appropriately designed studies should be conducted to compare validated screening instruments for the detection of OCD and BDD in children, young people and adults. An emphasis should be placed on examining those that use computer technology and more age-appropriate methods of assessing both symptoms and functioning, taking into account cultural and ethnic variations in communication, and family values. For BDD, specific populations would include young people or adults who consult in dermatology or plastic surgery and those with other psychiatric disorders.

10.8.5 CBT for children and young people with OCD and BDD

10.8.5.1 An appropriately blinded RCT should be conducted to assess the efficacy (including measures of social functioning and quality of life) and the cost effectiveness of individual CBT and CBT involving the family or carers compared with each other and with a credible psychological treatment that is not specific to OCD and BDD (such as anxiety management training) in a broadly based sample of children and young people diagnosed with OCD and BDD (using minimal exclusion criteria). The trial should be powered to examine the effect of treatment in children and young people separately and involve a follow-up of at least 1 year.

10.9 Table 9: AUDIT TABLE

1. Possible objective for audit To improve access to specialist OCD/BDD multidisciplinary healthcare across the individual's lifespan		
Criterion	**Exception**	**Definition of terms**
Each PCT, mental healthcare trust, and children's trust that provides mental health services has access to a specialist multidisciplinary OCD/BDD team. a) Operational policies in each PCT, mental healthcare trust and children's trust that provides mental health services specify procedure for accessing specialist OCD/BDD team b) Specialist teams offer a liaison function to other mental health professionals	None	A specialist OCD/BDD team is able to conduct expert assessment, specialist cognitive-behavioural and pharmacological treatment and provide age-appropriate care A liaison function will aim to: increase skills in the assessment and evidence-based treatment of people with OCD or BDD; provide high-quality advice; aid understanding of the needs of family/carers and developmental needs
2. Possible objective for audit To decrease delays in the patient pathway for people who are re-referred for treatment of OCD/BDD		
Criterion	**Exception**	**Definition of terms**
People with OCD or BDD who have relapsed following successful treatment are seen by a healthcare professional as soon as possible if re-referred, and where there has been no response to treatment are appropriately supported. a) Operational policies indicate the re-referral pathway b) Operational policies indicate that care coordination or other suitable process is followed for people	Person with OCD or BDD refuses re-referral	None

where there has been no response to treatment		
3. Possible objective for audit To improve the initial treatment of adults who have mild OCD or BDD, or those who prefer a low intensity psychological treatment		
Criterion	**Exception**	**Definition of terms**
In their initial treatment, adults who have mild OCD or BDD, or those who express a preference, are offered a low intensity psychological treatment. a) Clinical notes indicate that people are informed of low intensity treatment options b) Clinical notes indicate the clinical outcome of low intensity interventions	● Adults with moderate to severe OCD or BDD ● Children and young people ● Adults who refuse this treatment	Low intensity treatments (less than 10 therapist hours) include: ● brief individual CBT (including ERP) using structured self-help materials ● brief individual CBT (including ERP) by telephone ● group CBT (includ ing ERP) – note the patient may be receiving more than 10 hours of therapy in this format
4. Possible objective for audit To improve the treatment of adults who have been unable to engage with, or where there has been no response to, low intensity treatment		
Criterion	**Exception**	**Definition of terms**
Where adults have been unable to engage with low intensity treatment, or there has been no response to low intensity treatment, adults with mild OCD are offered more intensive treatment interventions. a) Clinical notes indicate that people have been informed of the possibility of intensive CBT (including ERP) or an SSRI b) Clinical notes indicate the clinical outcome of the intervention offered	● Adults where there is improvement with low intensity interventions ● Children and young people ● Adults who refuse these treatments	More intensive treatment interven-tions include: a choice of either a course of an SSRI, or more intensive CBT (including ERP) (of more than 10 therapist hours per patient)

5. Possible objective for audit
To improve the treatment of adults who have OCD with moderate functional impairment

Criterion	Exception	Definition of terms
Adults who have OCD with moderate functional impairment are offered the choice of either a course of an SSRI or more intensive CBT (including ERP). a) Clinical notes indicate that people have been informed of the possibility of more intensive CBT (including ERP) or an SSRI b) Clinical notes indicate the clinical outcome of the intervention offered	Children and young people	More intensive CBT (including ERP) means: more than 10 therapist hours per patient

6. Possible objective for audit
To improve the treatment of adults who have BDD with moderate functional impairment

Criterion	Exception	Definition of terms
Adults who have moderate BDD are offered the choice of an SSRI or more intensive individual CBT (including ERP) or an SSRI. a) Clinical notes indicate that people have been informed of the possibility of intensive individual CBT (including ERP) or an SSRI b) Clinical notes indicate the clinical outcome of the intervention offered	Children and young people	CBT (including ERP) means: ERP that addresses key features of BDD.

7. Possible objective for audit
To improve the care of children and young people who have OCD with moderate to severe functional impairment and those who have OCD with mild functional impairment for whom guided self-help has been ineffective or refused

Criterion	Exception	Definition of terms
Children and young people who have OCD with moderate/severe	Children and young people	CBT (including ERP) means: treatment

impairment or those with mild impairment where there is no response to guided self-help, or where guided self-help has been refused, will be offered CBT (including ERP) as the treatment of choice. a) Clinical notes indicate that the child/young person and the family/carer were informed of possibility of CBT b) Clinical notes identify the clinical outcome of CBT	who refuse CBT (including ERP)	involving the family or carers and adapted to suit the developmental age of the child. Group or individual formats should be offered depending upon the preference of the child or young person and their family or carers
8. Possible objective for audit To improve the care of children (aged 8–11 years) who have OCD or BDD with moderate to severe functional impairment if there has not been an adequate response to CBT (including ERP) involving the family or carers		
Criterion	**Exception**	**Definition of terms**
Children who have OCD or BDD where there has not been an adequate response to CBT (including ERP) attend a multidisciplinary review (with family/carers) where the use of an SSRI is considered in addition to ongoing psychological treatment. a) Clinical notes indicate a multidisciplinary review occurred and identified that the use of an SSRI in addition to ongoing psychological treatment was explored in detail b) Clinical notes indicate that careful monitoring was carried out c) Clinical notes indicate the clinical outcome of the intervention offered	Children who respond to CBT (including ERP)	Children: aged 8–11 years Careful monitoring: being seen frequently on an appropriate and regular basis agreed by the patient, his or her family or carers and the healthcare professional, and recorded in the notes

9. Possible objective for audit
To improve the treatment of young people (aged 12–18 years) who have OCD or BDD with moderate to severe functional impairment if there has not been an adequate response to CBT (including ERP) involving the family or carers

Criterion	Exception	Definition of terms
Young people who have OCD or BDD where there has not been an adequate response to CBT (including ERP) attend a multidisciplinary review (with family/carers) where the use of an SSRI is considered in addition to ongoing psychological treatment. a) Clinical notes indicate a multidisciplinary review occurred and identified that the use of an SSRI in addition to ongoing psychological treatment was explored in detail b) Clinical notes indicate that careful monitoring was carried out c) Clinical notes indicate the clinical outcome of the intervention offered	Young people who respond to CBT (including ERP)	Young people: aged 12–18 years Careful monitoring: being seen frequently on an appropriate and regular basis agreed by the patient, his or her family or carers and the healthcare professional, and recorded in the notes

10. Possible objective for audit
To improve the treatment of children and young people who have BDD

Criterion	Exception	Definition of terms
Children and young people with BDD are considered for CBT (including ERP) as first-line treatment. a) Clinical notes indicate that the healthcare professional responsible has discussed the need for CBT (including ERP) and an arrangement has been made b) Clinical notes indicate the clinical outcome of the intervention offered	Children or young people who refuse treatment	Children: aged 8–11 years Young people: aged 12–18 years CBT (including ERP) means: involving the family or carers and adapted to the developmental age of the child or young person

11. APPENDICES

APPENDIX 1:
SCOPE FOR THE DEVELOPMENT OF A CLINICAL GUIDELINE ON THE MANAGEMENT OF OBSESSIVE-COMPULSIVE DISORDER

FINAL VERSION
31 July 2003

1. GUIDELINE TITLE

Obsessive-compulsive disorder: the management of obsessive-compulsive disorder in adults and children in primary and secondary care.[41]

1.1 Short title

Obsessive-compulsive disorder (OCD).

2. BACKGROUND

a) The National Institute for Health and Clinical Excellence ('NICE' or 'the Institute') has commissioned the National Collaborating Centre for Mental Health to develop a clinical guideline on the management of anxiety disorders for use in the NHS in England and Wales. This follows referral of the topic of anxiety disorders, by the Department of Health and Welsh Assembly Government (see Appendix). This document provides further detail on the specific issues relating to OCD and is a development of the original scope agreed for the anxiety disorders. The guideline will provide recommendations for good practice that are based on best available evidence of clinical and cost effectiveness.

[41]The title of the guideline changed in the development process to 'Obsessive-compulsive disorder: Core interventions in the treatment of obsessive-compulsive disorder and body dysmorphic disorder'.

b) The Institute's clinical guidelines will support the implementation of National Service Frameworks (NSFs) in those aspects of care where a Framework has been published. The statements in each NSF reflect the evidence that was used at the time the Framework was prepared. The clinical guidelines and technology appraisals published by the Institute after an NSF has been issued will have the effect of updating the Framework.

3. CLINICAL NEED FOR THE GUIDELINE

a) Obsessive-compulsive disorder (OCD) is a potentially life-long disabling disorder. Diagnostic features include recurrent obsessions or compulsions that are distressing, time-consuming, that interfere with occupational or educational functioning and social activities or relationships.
b) In the UK, the prevalence of OCD is 1.2% of the adult population between 16–64 years of age, with it affecting a slightly higher proportion of women (1.5%) than men (1.0%). DSM-IV estimates a lifetime prevalence of 2.5% and 1-year prevalence of 1.5%–2.1%. The disorder can occur at any age. Because OCD is often a 'hidden' disorder, it is neither identified nor reported accurately. Thus, these figures should be viewed as underestimates.
c) Individuals with OCD and related disorders are currently treated in a range of NHS settings including primary care services, general mental health services and specialist secondary care mental health services. The provision and uptake of such services varies across England and Wales and in part reflects presence or absence of specialist services.
d) A number of guidelines, consensus statements and local protocols exist. This guideline will review evidence of clinical and cost-effective practice, together with current guidelines, and will offer guidance on best practice.

4. THE GUIDELINE

a) The guideline development process is described in detail in three booklets that are available from the NICE website (see 'Further information'). The *Guideline Development Process: Information for Stakeholders* describes how organisations can become involved in the development of a guideline.
b) This document is the scope. It defines exactly what this guideline will (and will not) examine, and what the guideline developers will consider. The scope is based on the referral from the Department of Health and Welsh Assembly Government (see Appendix to the scope).
c) The areas that will be addressed by the guideline are described in the following sections.

4.1. Population

4.1.1. Groups that will be covered

The recommendations made in the guideline will cover management of the following groups.

a) Children and adults who meet the standard diagnostic criteria of obsessive-compulsive disorder and body dysmorphic disorders.

4.1.2. Groups that will not be covered

a) Although the guidelines will be of relevance to all people with OCD whether or not it is accompanied by other illnesses, it will not address separately or specifically the management of individuals with other physical or psychiatric conditions.

4.2. Healthcare setting

a) The guideline will cover the care provided in primary and secondary care and that provided by healthcare professionals who have direct contact with and make decisions concerning the care of patients with OCD.
b) The guideline will also be relevant to the work of, but will not provide specific recommendations to the following non NHS services. However it will consider the interface between health care services and these services:
   - Social services
   - Voluntary sector
   - Education.

4.3. Clinical management – areas that will be covered

The guideline will cover the following areas of clinical practice:

a) The full range of care routinely made available by the NHS with regard to OCD.
b) Clarification and confirmation of diagnostic criteria currently in use and therefore the diagnostic factors that trigger the use of this guideline and assessment and instruments that might be used in this process. The definition of the condition in relation to other anxiety disorders will be precise.
c) Pathways to treatment.
d) Psychological interventions including type, format, frequency, duration and intensity. This will include computerised cognitive behaviour therapy (CCBT).[42]

[42]CCBT was subsequently covered by a NICE Technology Appraisal, 'Depression and anxiety: Computerised cognitive behaviour therapy' (CCBT) (No. 51) (June 2005).

e) Pharmacological treatments including type, dose and duration. When referring to pharmacological treatments, normally guidelines will recommend within the licence indications. However, where the evidence clearly supports it, recommendations for use outside the licence indications may be made in exceptional circumstances. It is the responsibility of prescribers to be aware of circumstances where medication is contraindicated. The guideline will assume that prescribers are familiar with the side-effect profile and contraindications of medication they prescribe for patients with depression. The guideline will consider the side effects, toxicity and other disadvantages of treatments.
f) Appropriate use of combined pharmacological and psychological interventions.
g) Psychosurgery and deep brain stimulation.
h) Self-care.
i) Sensitivity to cross-cultural and religious factors.
j) The role of the family in the treatment and support of patients.

a. Clinical management – areas that will not be covered

The guideline will not cover treatments that are not normally available on the NHS.

b. Audit support within the guideline

The guideline will include review criteria for audit, for key recommendation, which will enable objective measurements to be made of the extent and nature of local implementation of this guidance, particularly its impact upon practice and outcomes for people with OCD.

c. Status

i. Scope

This is the final version of the scope. It has been derived from the scope on Generalised Anxiety which formerly included OCD and which was subject to a 4-week period of consultation with stakeholders and review by the Guidelines Advisory Committee. As a result of that consultation, a decision was taken to prepare a separate guideline for OCD and this separate scope was drafted and submitted to the Institute's Guideline Programme Director and Executive Lead for approval.

ii. Guideline

The development of the guideline will begin in June 2003.

d. Further information

Information on the guideline development process is provided in:

- *The Guideline Development Process – Information for the Public and the NHS*
- *The Guideline Development Process – Information for Stakeholders*
- *The Guideline Development Process – Information for National Collaborating Centres and Guideline Development Groups.*

These booklets are available as PDF files from the NICE website (www.nice.org.uk). Information on the progress of the guideline will also be available from the website.

Appendix to the scope – Referral from the Department of Health and Welsh Assembly Government

The Department of Health and Welsh Assembly Government asked the Institute:

'To prepare a clinical guideline and audit tool for the NHS in England and Wales for "talking" therapies, drug treatments and prescribing for anxiety and related common mental disorders, including generalised anxiety disorder, panic disorder (with or without agoraphobia), post-traumatic stress disorder, and obsessive-compulsive disorder (OCD). The audit tool should include a dataset, database and audit methodology'.

APPENDIX 2: STAKEHOLDERS WHO RESPONDED TO EARLY REQUESTS FOR EVIDENCE

Amicus
British Association of Behavioural and Cognitive Psychotherapy
CIS'ters
College of Occupational Therapy
Cyberonics
Eating Disorders Association
Inner Cities Mental Health Group
National Phobics Society
Pfizer
Royal College of Nursing
Solvay Healthcare Ltd
Wyeth

APPENDIX 3: STAKEHOLDERS AND EXPERTS WHO RESPONDED TO THE FIRST CONSULTATION DRAFT OF THE GUIDELINE

STAKEHOLDERS

Association for Family Therapy
AstraZeneca UK Ltd
British Association for Counselling and Psychotherapy
British Association for Psychopharmacology
Cambridgeshire and Peterborough Mental Health Trust
Camden and Islington Mental Health and Social Care Trust
College of Occupational Therapists
Department of Health
GlaxoSmithKline UK
Hampshire Partnership NHS Trust
Lundbeck Limited
North Staffordshire Combined Healthcare NHS Trust
OCD-UK
Oxfordshire Mental Healthcare NHS Trust
Pfizer UK Limited
Rethink
Royal College of Nursing
Royal College of Paediatrics
Royal College of Psychiatrists
Royal Pharmaceutical Society of Great Britain
ST Solutions Limited
Tavistock and Portman NHS Trust
UK Council for Psychotherapy
Welsh Assembly Government

EXPERTS

Daniel A. Geller
Professor Peter Hill
Prof Emeritus Isaac Marks
Professor Keith Matthews
Professor Eric Taylor

APPENDIX 4: RESEARCHERS CONTACTED TO REQUEST INFORMATION ABOUT UNPUBLISHED OR SOON-TO-BE PUBLISHED STUDIES

Jonathan Abramowitz
Margaret Altemus
Jambur Ananth
Martin M. Antony
Lee Baer
Donald Black
Pierre Blier
Martine Bouvard
Maria Lynn Buttolph
Alexander Bystritsky
Cheryl Carmin
Diane L. Chambless
David A. Clark
Edwin H. Cook Jr.
Jean Cottraux
Jonathan Robert Davidson
Pedro Delgado
Paul M.G. Emmelkamp
Brian A. Fallon
Martine Flament
Edna Foa
Martin Franklin
Randy Frost
Tim M. Gale
Daniel A. Geller
Wayne K. Goodman
Tana A. Grady-Weliky
Benjamin D. Greenberg
John H. Greist
Gregory L. Hanna
Jeffrey E. Hecker
William Hewlett
Eric Hollander
Jonathan D. Huppert
Bruce M. Hyman
James W. Jefferson
Michael A. Jenike
David J. Katzelnick
Suck Won Kim
Lorrin M Koran
Michael J. Kozak
James F. Leckman
Henrietta Leonard
Christopher McDougle
Richard J. McNally
Charles Mansueto
Isaac Marks
Arturo Marrero
Fugen A. Neziroglu
Michele Pato
Maggie Pekar
Frederick Penzel
Katharine A. Phillips
Teresa A. Pigott
Alec Pollard
Lawrence Price
S. Rachman
Adam S. Radomsky
Judith L. Rapoport
Scott Rauch
Mark Riddle
Jerilyn Ross
Barbara Rothbaum
Paul Salkovskis
Jeffrey M. Schwartz
H. Blair Simpson
David A. Spiegel
Dan J. Stein
Gail Steketee
Susan E. Swedo

Richard Swinson
Dana S. Thordarson
Barbara Van Noppen
Dr Patricia Van Oppen
Lorne Warneke
Maureen Whittal
Tim Williams
Jose Yaryura-Tobias

APPENDIX 5:
CLINICAL QUESTIONS

A.	**Psychological intervention**
1.	For people with OCD, does BT, when compared with waitlist control/ relaxation/anxiety management, behavioural stress management/another active psychological intervention produce benefits/harms on the specified outcomes?
2.	For people with OCD, does CT, when compared with waitlist control/ relaxation/anxiety management, behavioural stress management/another active psychological intervention produce benefits/harms on the specified outcomes?
3.	For people with OCD, does CBT, when compared with waitlist control/ relaxation/anxiety management, behavioural stress management/another active psychological intervention produce benefits/harms on the specified outcomes?
4.	For people with OCD, does rational-emotive therapy (RET), when compared with waitlist control/relaxation/anxiety management, behavioural stress management/another active psychological intervention produce benefits/harms on the specified outcomes?
5.	For people with OCD, does psychoanalysis, psychoanalytic psychotherapy, psychodynamic psychotherapy or supportive psychotherapy, when compared with waitlist control/relaxation/anxiety management – behavioural stress management/another active psychological intervention produce benefits/harms on the specified outcomes?
6.	For people with OCD, does mind body therapy (MBT), when compared with waitlist control/relaxation/anxiety management, behavioural stress management/another active psychological intervention produce benefits/harms on the specified outcomes?
7.	For people with OCD, does family therapy, when compared with waitlist control/relaxation/anxiety management, behavioural stress management/ another active psychological intervention produce benefits/harms on the specified outcomes?
8.	For people with OCD, does any other psychological intervention (yoga), when compared with waitlist control/relaxation/anxiety management, behavioural stress management/another active psychological intervention produce benefits/harms on the specified outcomes?

B.	**Pharmacological interventions**
1.	For people with OCD (BDD), do tricyclic antidepressants (amitriptyline, desipramine, imipramine, nortriptyline, excluding clomipramine), when compared with placebo/comparator drug, produce benefits/harms on the specified outcomes?
2.	For people with OCD (BDD), does clomipramine, when compared with placebo/comparator drug, produce benefits/harms on the specified outcomes?
3.	For people with OCD (BDD), do SSRIs (citalopram, fluoxetine, fluvoxamine, paroxetine, sertraline), when compared with placebo/comparator drug, produce benefits/harms on the specified outcomes?
4.	For people with OCD (BDD), do atypical SRIs, when compared with placebo/comparator drug, produce benefits/harms on the specified outcomes?
5.	For people with OCD (BDD) do SNRIs (venlafaxine), when compared with placebo/comparator drug, produce benefits/harms on the specified outcomes?
6.	For people with OCD, do MAOIs (phenelzine), when compared with placebo/comparator drug, produce benefits/harms on the specified outcomes?
7.	For people with OCD (BDD), do anxiolytics (buspirone, clonazepam), when compared with placebo/comparator drug, produce benefits/harms on the specified outcomes?
8.	For people with OCD (BDD), do antipsychotics, when compared with placebo/comparator drug, produce benefits/harms on the specified outcomes?
9.	For people with OCD (BDD), does any other pharmacological intervention (inositol, oxytocin), when compared with placebo/comparator drug, produce benefits/harms on the specified outcomes?
10.	For people with OCD, do augmentation strategies (buspirone, clomipramine, desipramine, haloperidol, inositol, lithium, nortriptyline, olanzapine, pindolol, quetiapine, risperidone, triiodothyronine), when compared with placebo/comparator drug, produce benefits/harms on the specified outcomes?
11.	For people with OCD, does any drug treatment, when compared with any psychological intervention, produce benefits/harms on the specified outcomes?

12. For people with OCD, does the combination of a drug treatment and a psychological intervention, when compared with a drug treatment alone/ psychological intervention alone, produce benefits/harms on the specified outcomes?
C. Other biological interventions
1. For people with OCD, does neurosurgery (stereotactic anterior capsulotomy/ cingulotomy), when compared with placebo/waitlist control/drug treatment/ any psychological intervention, produce benefits/harms on the specified outcomes?
2. For people with OCD, does deep brain stimulation (electrical capsular stimulation), when compared with placebo/waitlist control/drug treatment/any psychological intervention, produce benefits/harms on the specified outcomes?
3. For people with OCD, does transcranial magnetic stimulation, when compared with placebo/waitlist control/drug treatment/any psychological intervention, produce benefits/harms on the specified outcomes?
4. For people with OCD, does ECT, when compared with placebo/waitlist control/drug treatment/any psychological intervention, produce benefits/harms on the specified outcomes?
5. For people with OCD, do other interventions, when compared with placebo/waitlist control/drug treatment/any psychological intervention, produce benefits/harms on the specified outcomes?

APPENDIX 6: SEARCH STRATEGIES FOR THE IDENTIFICATION OF CLINICAL STUDIES

OCD SEARCH FILTER

MEDLINE, CINAHL, EMBASE, PSYCINFO

1. compulsive behavior.sh.
2. obsessive-compulsive disorder.sh.
3. obsessive behavior.sh.
4. compulsions.sh.
5. obsession.sh.
6. body dysmorphic disorder.sh.
7. obsessive compulsive neuros$.tw.
8. obsessive compulsive disorder$.tw.
9. (recurr$ adj obsession$).tw.
10. (recurr$ adj thought$).tw.
11. (obsession or obsessions or obsessional).tw.
12. (clean$ adj response$).tw.
13. OCD.tw.
14. Osteochondr$.tw.
15. [(obsess$ adj ruminat$) or scrupulosity or body dysmorphi$ or dysmorphophobi$ or imagine$ ugl$].mp.
16. (compulsion or compulsions or compulsional).tw.
17. [(symmetr$ or count$ or arrang$ or order$ or wash$ or repeat$ or hoard$ or clean$ or check$) adj compulsi$].mp.
18. or/1–11
19. 13 not 14
20. or/15–19

BDD SEARCH FILTER

1. body dysmorphic disorder.sh.
2. (body dysmorphi$ or dysmorphophobi$ or imagine$ ugl$).mp.
3. 1 or 2
4. remove duplicates from 3

SYSTEMATIC REVIEW SEARCH FILTER

MEDLINE, CINAHL, EMBASE, PSYCINFO

1. meta analysis/

2. meta analysis.fc.
3. meta-analysis.pt.
4. (review, academic or review, multicase).pt.
5. exp literature searching/
6. systematic review.pt.
7. (metaanaly$ or meta analy$ or meta?analy$).tw.
8. [(systematic or quantitative or methodologic$) adj (overview$ or review$)].tw.
9. (research review$ or research integration).tw.
10. (handsearch$ or [(hand or manual) adj search$)].tw.
11. (mantel haenszel or peto or dersimonian or der simonian).tw.
12. [fixed effect$ or random effect$ or (pooled adj data)].tw.
13. (medline or embase or scisearch or science citation or isi citation or "web of science").tw.
14. or/1–13

RANDOMISED CONTROLLED TRIALS SEARCH FILTERS

MEDLINE, CINAHL, EMBASE, PsycINFO

1. exp clinical trials/or cross-over studies/or random allocation/or double-blind method/or single-blind method/
2. random$.pt.
3. exp clinical trial/or crossover procedure/or double blind procedure/or single blind procedure/or randomisation/
4. exp clinical trials/or crossover design/or random assignment/
5. exp clinical trials/or double blind method/or random allocation/
6. random$.mp.
7. [cross-over or cross?over or (clinical adj2 trial$) or single-blind$ or single? blind$ or double-blind or double?blind$ or triple-blind or triple?blind].tw.
8. or/1–7
9. animals/not (animals/and human$.mp.)
10. animal$/not (animal$/and human$/)
11. meta-analysis/
12. meta-analysis.pt.
13. systematic review/
14. or/9–13
15. 8 not 14

SEARCH STRINGS SUPPORTING SPECIFIC REVIEWS

Other psychological

Date of search	30.10.2003	
Databases searched	MEDLINE, CINAHL, EMBASE, PsycINFO	
No. of hits	406	Dedup'ed: 369

[1–20 OCD search filter above]
21. psychoanalysis.sh.
22. (gestalt therapy or counseling or hypnosis or transactional analysis).sh.
23. exp psychoanalysis/
24. exp hypnotherapy/or exp counseling/or (supportive psychotherapy or eye movement desensitization therapy).sh.
25. (psychoanaly$ or psychodynamic$ or support$ psychotherap$).tw.
26. (EMDR or eye movement desensiti$ or gestalt or counseling or hypnotherap$ or transactional analys$ or cognitive analytic).tw.
27. or/23,25
28. 20 and 27
29. remove duplicates from 28
30. or/22,24,26
31. 20 and 30
32. remove duplicates from 31

Augmentation

Date of search	13.11.2003
Databases searched	MEDLINE, CINAHL, EMBASE, PsycINFO
No. of hits after Dedup'ed	369

[1–20 OCD search filter above]
21. (adjunct$ or augment$ or "add on" or addition$ or supplement$ or resist$ or refract$ or nonrespon$ or intractable).ti,ab.
22. 20 and 21
23. remove duplicates from 22
24. exp inositol/or exp pindolol/or exp antipsychotic agents/or exp tryptophan/or (valproic acid or lithium).sh.
25. exp antipsychotic agents/or (inositol or lithium or valproic acid or tryptophan).sh.
26. exp lithium/or exp tryptophan/or exp neuroleptic drugs/or valrpoic acid.sh.
27. exp neuroleptic agent/or (gabapentin or inositol or lithium or pindolol or valproic acid or tryptophan).sh.
28. (anti-testosterone or gabapentin or inositol or lithium or pindolol or valproate or valproic acid or triptans or tryptophan).ti,ab.
29. (benperidol or chlorpromazine or flupentixol or fluphenazine or haloperidol or levomepromazine or methotrimeprazine or perioyazine or perphenazine or pimozide or prochlorperazine or promazine or sulpiride or thioridazine or trifluoperazine or zuclopenthixol or amisulpride or clozapine or olanzapine or quetiapine or risperidone or sertindole or zotepine).mp.
30. (loxapine or pericyazine or buspirone or fenfluramine or trazodone).mp.
31. or/24–30
32. 20 and 31

33. remove duplicates from 32
34. 23 and 33

Other pharmacological

Date of search	29.04.2004
Databases searched	CINAHL: 48 hits EMBASE: 549 hits PsychINFO: 147 hits MEDLINE: 230 hits
No. of hits	48

1. inositol or pindolol or tryptophan or gabapentin or triptans or anti-testosterone or john* wort or kava kava or gingko biloba or ginkgo biloba or amphetamine or oxytocin or clonidine or practolol or beta-blocker* or ondansetron or ritanserin or anti-androgen or cyproterone
2. OCD not osteochondr*
3. (obsess* near ruminat*) or scrupulosity or body dysmorphi* or dysmorpho-phobi* or (imagin* ugl*)
4. obsessive compulsive neuros* or obsessive compulsive disorder* or (recurr* near obsess*) or (recurr* near thought*) or obsession or obsessions or obsessional or compulsion or compulsions or compulsiona
5. (compulsive behavior or obsessive-compulsive disorder or obsessive behavior or compulsions or obsession or body dysmorphic disorder)

Other medical

Date of search	20.10.2003	
Databases searched	MEDLINE, CINAHL, EMBASE, PsycINFO	
No. of hits	843	Dedup'ed: 602

[1–20 OCD search filter above]
21. neurosurgery.sh.
22. psychosurgery.sh.
23. exp brain stimulation/
24. electroconvulsive therapy.sh.
25. electroconvulsive shock therapy.sh.
26. brain depth stimulation.sh.
27. transcranial magnetic stimulation.sh.
28. tractotomy.sh.

29. (neurosurg$ or brain stimulat$ or transcranial or TMS or magnetic stimulat$ or ECT or electroconvulsive).tw.
30. (cingulotom$ or cingulectom$ or leucotom$ or leukotom$ or capsulotom$ or tractotom$ or electric$ capsular$).tw.
31. or/21–30
32. 20 and 31
33. remove duplicates from 32

Child psychotherapy

Date of search	05.11.2003
Databases searched	MEDLINE, CINAHL, EMBASE, PsycINFO
No. of hits after Dedup'ed	791

[1–20 OCD search filter above]
21. exp child/or exp adolescent/
22. exp pediatrics/
23. (child$ or adolescen$).tw.
24. or/21–23
25. 20 and 24
26. limit 25 to (adult ,19 to 44 years. or aged ,65 to 79 years. or "aged ,80 and over." or middle age ,45 to 64 years.)
27. limit 26 to (all adult ,19 plus years. or "all aged ,65 and over.")
28. limit 27 to adulthood ,181 years.
29. limit 28 to (adult ,18 to 64 years. or aged ,651 years.)
30. 25 not 29
31. exp psychotherapy/
32. (cognitive therapy or behavior therapy or family therapy).sh.
33. psychotherapy, rational-emotive.sh.
34. rational emotive therapy.sh.
35. systematic desensitisation therapy.sh.
36. [(cognitive or behavior$ or behaviour$ or family or systemic or strategic or structural) adj1 (therap$ or treatment$)].tw.
37. {rational emotive or RET or CBT or [multimodal adj1 (behavior or behaviour)] or MBT}.tw.
38. or/31–37
39. 30 and 38
40. remove duplicates from 39

Psychoanalysis

[1–20 OCD search filter above]
21. psychoanalysis.sh.

22. (gestalt therapy or counseling or hypnosis or transactional analysis).sh.
23. exp psychoanalysis/
24. exp hypnotherapy/or exp counseling/or (supportive psychotherapy or eye movement desensitisation therapy).sh.
25. (psychoanaly$ or psychodynamic$ or support$ psychotherap$).tw.
26. (EMDR or eye movement desensiti$ or gestalt or counseling or hypnotherap$ or transactional analys$ or cognitive analytic).tw.
27. or/23,25
28. 20 and 27
29. remove duplicates from 28
30. or/22,24,26
31. 20 and 30
32. remove duplicates from 31
33. 29 not 32

Screening

Date of search	05.11.2003
Databases searched	PsycINFO
No. of hits after Dedup'ed	130

1. (RELIABILITY OR SENSITIVITY OR SPECIFICITY OR SCREENING).AB.
2. OBSESSIVE ADJ COMPULSIVE ADJ DISORDER
3. OBSESSIVE-COMPULSIVE-DISORDER.DE.
4. 3 AND 1
5. 3 AND 1
6. (TEST OR QUESTIONNAIRE OR SCALE OR INVENTORY).AB.
7. 6 AND 5
8. primary ADJ care
9. PRIMARY-HEALTH-CARE.DE. OR PHYSICIANS.W.DE. OR FAMILY-PHYSICIANS.DE. OR GENERAL-PRACTITIONERS.DE.
10. 7 AND 9
11. primary ADJ care
12. general ADJ practitioner
13. physician
14. 7 AND (11 OR 12 OR 13)
15. 10 OR 14

APPENDIX 7:
SYSTEMATIC REVIEW QUALITY CHECKLIST

Depression Guideline **Quality checklist for a systematic review (notes for reviewer are in italics)**			
Checklist completed by:		**Report reference ID:**	
SECTION 1: VALIDITY			
Evaluation criteria		**Comments**	
1.1	Does the review address an appropriate and clearly focused question?		
Unless a clear and well-defined question is specified, it will be difficult to assess how well the study has met its objectives or how relevant it is to the question you are trying to answer on the basis of its conclusions.			
1.2	Does the review include a description of the methodology used?		
A systematic review should include a detailed description of the methods used to identify and evaluate individual studies. If this description is not present, it is not possible to make a thorough evaluation of the quality of the review, and it should be rejected as a source of Level 1 evidence. (Though it may be useable as Level 4 evidence, if no better evidence can be found.) Unless a clear and well-defined question is specified, it will be difficult to assess how well the study has met its objectives or how relevant it is to the question you are trying to answer on the basis of its conclusions.			
1.3	Was the literature search sufficiently rigorous to identify all relevant studies?		
Consider whether the review used an electronic search of at least one bibliographic database (searching for studies dating at least 10 years before publication of the review). Any indication that hand-searching of key journals, or follow-up of reference lists of included studies, were carried out in addition to electronic database searches can normally be taken as evidence of a well-conducted review.			
1.4	Was study quality assessed and taken into account?		
A well-conducted systematic review should have used clear criteria to assess whether individual studies had been well conducted before deciding whether to include or exclude them. At a minimum, the authors should have checked that there was adequate concealment of allocation, that the rate of drop out was minimised, and that the results were analysed on an 'intention to treat' basis. If there is no indication of such an assessment, the review should be rejected as a source of Level 1 evidence. If details of the assessment are poor, or the methods considered to be inadequate, the quality of the review should be downgraded.			
SECTION 2: OVERALL ASSESSMENT		**Comments**	**Code**
2.1	Low risk of bias Moderate risk of bias High risk of bias	*All or most criteria met* *Most criteria partly met* *Few or no criteria met*	**A** **B** **C**

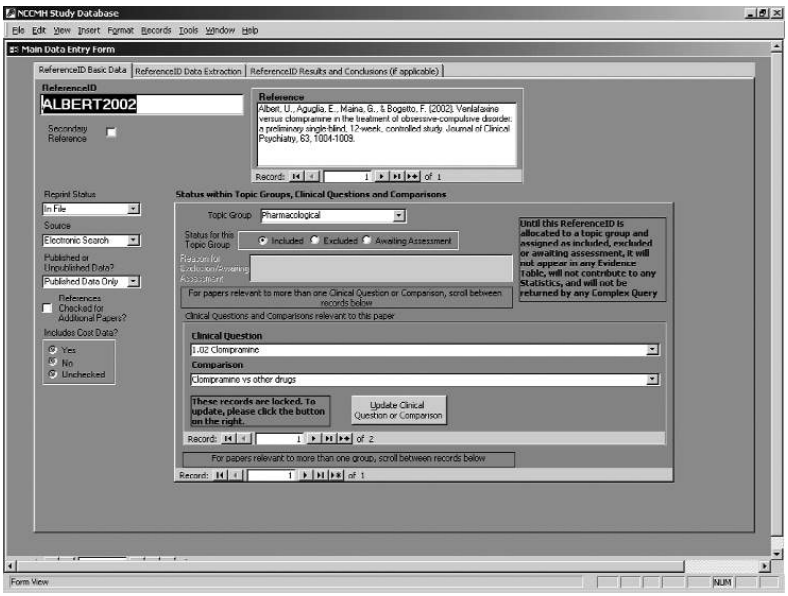
APPENDIX 8:

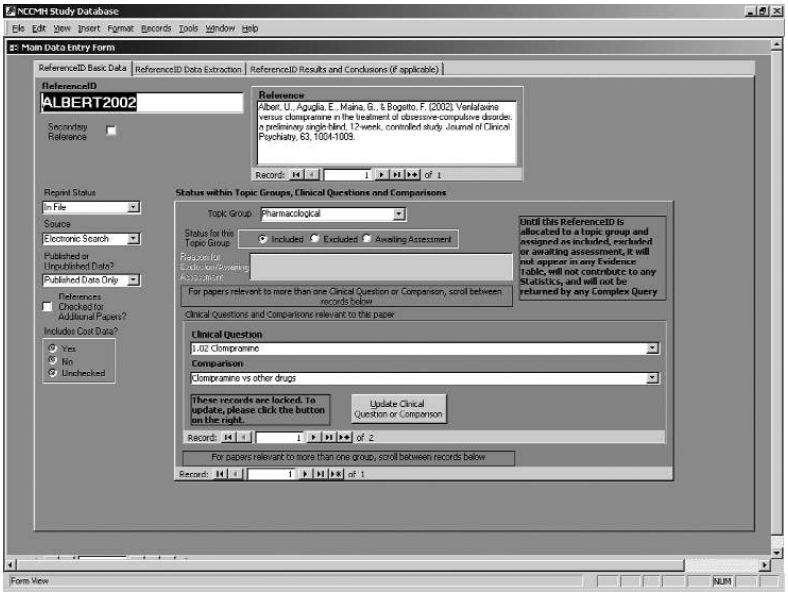
RCT METHODOLOGICAL QUALITY CHECKLIST

Depression Guideline **Quality checklist for an RCT**			
Report reference ID:			
Checklist completed by:		**Date completed:**	
SECTION 1: INTERNAL VALIDITY			
Evaluation criteria		**How well is this criterion addressed?**	
1.1	Was the assignment of subjects to treatment groups randomised?		
If there is no indication of randomisation, the study should be rejected*. If the description of randomisation is poor, or the process used is not truly random (e.g., allocation by date, alternating between one group and another) or can otherwise be seen as flawed, the study should be given a lower quality rating.*			
1.2	Was an adequate concealment method used?		
Centralised allocation, computerised allocation systems, or the use of coded identical containers would all be regarded as adequate methods of concealment, and may be taken as indicators of a well-conducted study. If the method of concealment used is regarded as poor, or relatively easy to subvert, the study must be given a lower quality rating, and can be rejected if the concealment method is seen as inadequate.			
ASSESSMENT		**Comments**	**Code**
2.1	Low risk of bias Moderate risk of bias High risk of bias	*Both criteria met* *One or more criteria partly met* *One or more criteria not met*	**A** **B** **C**

APPENDIX 9:
CLINICAL STUDY DATA EXTRACTION FORMS

STUDY CHARACTERISTICS EXTRACTION FORM





RCT DATA EXTRACTION FORM

Data extraction form for a randomised controlled trial											
Completed by:						**Report reference ID:**					
1 TREATMENT GROUP:											
Dropouts			**Treatment Responders**			**Side Effects (total)**					
n	*N*		*n*	*N*		*n*	*N*		*n*	*N*	
Definition of responders											
Post-treatment means											
	n	*Mean*	*SD*	*n*	*Mean*	*SD*	*n*	*Mean*	*SD*	*n*	*Mean*	*SD*
Other data												
	n	*N*		*n*	*N*		*n*	*Mean*	*SD*	*n*	*Mean*	*SD*

2 TREATMENT GROUP:											
Dropouts			**Treatment Responders**			**Side Effects (total)**					
n	*N*		*n*	*N*		*n*	*N*		*n*	*N*	
Definition of responders											
Post-treatment means											
	n	*Mean*	*SD*	*n*	*Mean*	*SD*	*n*	*Mean*	*SD*	*n*	*Mean*	*SD*
Other data												
	n	*N*		*n*	*N*		*n*	*Mean*	*SD*	*n*	*Mean*	*SD*

Comparisons entered:

APPENDIX 10: FORMULAE FOR CALCULATING STANDARD DEVIATIONS

The following formulae were used to calculate standard deviations (SD) where these were not available in study reports:

(n = sample size of group)

$$SD = Standard\ Error \times \sqrt{n}$$

$$SD = \frac{(upper\ 95\%\ Confidence\ Interval - mean)}{1.96} \times \sqrt{n}$$

$$SD = \frac{(mean_1 - mean_2)}{\sqrt{F}\left(\frac{\sqrt{1}}{n_1}\right) + \left(\frac{\sqrt{1}}{n_2}\right)}$$

(If F ratio is not given, then $F = t_2$)

APPENDIX 11:
HEALTH ECONOMICS SEARCH STRATEGY

Date of search	08.04.2004
Databases searched	PsycINFO

#1 (obsessive compulsive disorder or compulsions or obsessions or body dysmorphic disorder) in DE,SU (6196 records)
#2 obsessive compulsive neuros* or obsessive compulsive disorder* or obsession or obsessions or obsessional (8579 records)
#3 OCD not osteochondr* (2409 records)
#4 scrupulosity or body dysmorphi* or dysmorphophobi* or imagine* ugl* (446 records)
#5 #1 or #2 or #3 or #4 (9446 records)
#6 (burden near illness) or (burden near disease) or (cost* near evaluat*) or (cost* near benefit*) or (cost* near utilit*) or (cost* near minimi*) or (cost* near illness) or (cost* near disease) or (cost* near analys*) or (cost* near assess*) or (cost* near study) or (cost* near studies) or (cost* near allocation) or (cost* near outcome*) or (cost* near consequence*) or (cost* near offset*) or (cost* near off-set*) or (cost* near effect*) or (cost* near treatment*) (20441 records)
#7 (economic near evaluat*) or (economic near analys*) or (economic near burden) or (economic near study) or (economic near studies) or (economic near assess*) or (economic near consequence*) or (economic near outcome*) or (health service* near (us* or utili*)) or (health care near (us* or utili*)) or (healthcare near (us* or utili*)) or health utility or health utilities or quality adjusted life year* or quality-adjusted-life-year* or qaly* or (resource near (us* or utili* or allocation*)) or expenditure* (38791 records)
#8 explode 'economics' or explode 'costs and cost analysis' (9300 records)
#9 #6 or #7 or #8 (57686 records)
#10 #5 and #9 (141 records)

Date of search	08.04.2004
Databases searched	Medline

#1 cost* (226082 records)
#2 economic (75343 records)
#3 health service or health care or healthcare (378473 records)

#4 quality adjusted life year* or qaly or resource utili* or resource allocation* or expenditure* (34265 records)
#5 (obsessive compulsive disorder or compulsive behavior or obsessive behavior) in KW,MESH,PS (7109 records)
#6 obessive compulsive neuros* or obsessive compulsive disorder* or obsession or obsessions or obsessional (7133 records)
#7 OCD not osteochondr* (1867 records)
#8 scrupulosity or body dysmorphi* or dysmorphophobi* or imagine* ugl* (334 records)
#9 #5 or #6 or #7 or #8 (8688 records)
#10 #1 and #9 (105 records)
#11 #2 and #9 (30 records)
#12 #3 and #9 (216 records)
#13 #4 and #9 (9 records)
#14 #10 or #11 or #12 or #13 (318 records)

Date of search	08.04.2004
Databases searched	EMBASE

#1 (obsessive compulsive disorder or compulsion or obsession or body dysmorphic disorder) in SU (8256 records)
#2 obessive compulsive neuros* or obsessive compulsive disorder* or obsession or obsessions or obsessional (7554 records)
#3 OCD not osteochondr* (2159 records)
#4 scrupulosity or body dysmorphi* or dysmorphophobi* or imagine* ugl* (469 records)
#5 #1 or #2 or #3 or #4 (9129 records)
#6 (burden near illness) or (burden near disease) or (cost* near evaluat*) or (cost* near benefit*) or (cost* near utilit*) or (cost* near minimi*) or (cost* near illness) or (cost* near disease) or (cost* near analys*) or (cost* near assess*) or (cost* near study) or (cost* near studies) or (cost* near allocation) or (cost* near outcome*) or (cost* near consequence*) or (cost* near offset*) or (cost* near off-set*) or (cost* near effect*) or (cost* near treatment*) (91709 records)
#7 (economic near evaluat*) or (economic near analys*) or (economic near burden) or (economic near study) or (economic near studies) or (economic near assess*) or (economic near consequence*) or (economic near outcome*) or (health service* near (us* or utili*)) or (health care near (us* or utili*)) or (healthcare near (us* or utili*)) or health utility or health utilities or quality adjusted life year* or quality-adjusted-life-year* or qaly* or (resource near (us* or utili* or allocation*)) or expenditure* (60659 records)
#8 (explode 'cost'/all subheadings or explode 'economics'/all subheadings or explode 'health economics'/all subheadings) in SU (154319 records)
#9 #6 or #7 or #8 (218918 records)
#10 #5 and #9 (242 records)

Date of search	08.04.2004
Databases searched	EconLit

#1 obsessive or compulsive or obsession or obsessions or obsessional or compulsion or compulsions or compusional or body dysmorphi* or dysmorphophobi* or OCD (116 records)

Date of search	08.04.2004
Databases searched	NHS EED

"obsess*" = 2

APPENDIX 12: SELECTION CRITERIA FOR ECONOMIC STUDIES

COST-OF-ILLNESS/ECONOMIC BURDEN STUDIES

1. There was no restriction placed on language or publication status of the papers.
2. Studies published between 1980 and 2004 were included. This date restriction was imposed in order to obtain data relevant to current healthcare settings and costs.
3. Only studies from the UK/OECD were included, as the aim of the review was to identify economic burden information relevant to the national context.
4. Selection criteria based on types of clinical conditions and patients were identical to the clinical literature review.
5. Studies were included provided that sufficient details regarding methods and results were available to enable the methodological quality of the study to be assessed, and provided that the study's data and results were extractable.

ECONOMIC EVALUATIONS

1. Studies were included provided they had used cost-minimisation analysis, cost-effectiveness analysis, cost-utility analysis or cost-benefit analysis.
2. Clinical evidence from a meta-analysis, a randomised controlled trial, a quasi-experimental trial or a cohort study was used.
3. There was no restriction placed on language or publication status of the papers.
4. Studies published between 1980 and 2004 were included. This date restriction was imposed in order to obtain data relevant to current healthcare settings and costs.
5. Only studies from the UK/OECD were considered, as the aim of the review was to identify economic evaluation information relevant to the national context.
6. Selection criteria were based on types of clinical conditions, patients, treatments and settings to which agreed by the GDG (2004).
7. Studies were included provided that sufficient details regarding methods and results were available to enable the methodological quality of the study to be assessed, and provided that the study's data and results were extractable.
8. In cases where no published data were available, estimations were made by the GDG (2004) based upon expert opinions.

HEALTH STATE AND UTILITY STUDIES

1. Studies reporting health state and utilities for OCD were considered for inclusion.
2. There was no restriction placed on language or publication status of the papers.
3. Studies published between 1980 and 2004 were included.
4. Only studies from OECD countries were considered to assure the generalisability of the results to the UK context.
5. Selection criteria based on types of clinical conditions, patients, treatments and settings were identical to the clinical literature review.

APPENDIX 13: DATA EXTRACTION FORM FOR ECONOMIC STUDIES

Reviewer: **Date of review:**
Authors: **Publication date:**
Title:
Country: **Language:**

Interventions compared:
Treatment:
Comparator:
Patient population:
Setting:
Economic study design:

CEA	CMA
CBA	CCA
CUA	CA

Perspective of the analysis:

Health care system	Societal
Health care provider	Patient and family
Third party payer	Other:

Time frame of the analysis:
Modelling:

NO	YES

Source of data for effect size measures:

	Meta-analysis
	Non-systematic review
RCT	RCT
Quasi-experimental study	Quasi-experimental study
Cohort study	Cohort study
Mirror-image (before after) study	Mirror-image (before after) study
	Expert opinion

Comments:

Primary outcome measures:
Costs included:

Direct medical	Direct non-medical	Lost productivity
direct treatment	social care	income forgone due to illness
inpatient	social benefits	income forgone due to death

outpatient	travel costs	income forgone by caregiver
day care	caregiver out-of-pocket	
community health care	criminal justice	
medication	training of staff	

or

Staff
Medication
Consumables
Overhead
Capital equipment
Real estate
Others:

Source of resource use and unit costs:
Currency: **Price year:**
Discounting (costs/benefits):
Sensitivity analysis:
Effectiveness results:
Cost results:
Cost-effectiveness results:
Authors' conclusions:
Comments – limitations:

APPENDIX 14: QUALITY CHECKLIST – FULL ECONOMIC EVALUATIONS

Author: Date of publication:

Title:

Study design

1.	The research question is stated		Yes	No
2.	The economic importance of the research question is stated		Yes	No
3.	The viewpoint(s) of the analysis are clearly stated and justified		Yes	No
4.	The rationale for choosing the alternative programmes or interventions compared is stated		Yes	No
5.	The alternatives being compared are clearly described		Yes	No
6.	The form of economic evaluation used is stated		Yes	No
7.	The choice of form of economic evaluation used is justified in relation to the questions addressed		Yes	No

Data collection

1.	The source of effectiveness estimates used is stated	Yes	No	
2.	Details of the design and results of effectiveness study are given (if based on a single study)	Yes	No	N/A
3.	Details of the method of synthesis or meta-analysis of estimates are given (if based on an overview of a number of effectiveness studies)	Yes	No	N/A
4.	The primary outcome measure(s) for the economic evaluation are clearly stated	Yes	No	
5.	Methods to value health states and other benefits are stated	Yes	No	N/A
6.	Details of the subjects from whom valuations were obtained are given	Yes	No	N/A
7.	Indirect costs (if included) are reported separately	Yes	No	N/A
8.	The relevance of indirect costs to the study question is discussed	Yes	No	N/A
9.	Quantities of resources are reported separately from their unit costs	Yes	No	
10.	Methods for the estimation of quantities and unit costs are described	Yes	No	
11.	Currency and price data are recorded	Yes	No	
12.	Details of currency, price adjustments for inflation or currency conversion are given	Yes	No	

13. Details of any model used are given	Yes	No	N/A
14. The choice of model used and the key parameters on which it is based are justified	Yes	No	N/A

Analysis and interpretation of results

1. Time horizon of costs and benefits is stated	Yes	No	
2. The discount rate(s) is stated	Yes	No	N/A
3. The choice of rate(s) is justified	Yes	No	N/A
4. An explanation is given if costs or benefits are not discounted	Yes	No	N/A
5. Details of statistical tests and confidence intervals are given for stochastic data	Yes	No	N/A
6. The approach to sensitivity analysis is given	Yes	No	N/A
7. The choice of variables for sensitivity analysis is given	Yes	No	N/A
8. The ranges over which the variables are varied are stated	Yes	No	N/A
9. Relevant alternatives are compared	Yes	No	
10. Incremental analysis is reported	Yes	No	N/A
11. Major outcomes are presented in a disaggregated as well as aggregated form	Yes	No	
12. The answer to the study question is given	Yes	No	
13. Conclusions follow from the data reported	Yes	No	
14. Conclusions are accompanied by the appropriate caveats	Yes	No	

Validity score: Yes/No/NA:

APPENDIX 15:
DIAGNOSTIC CRITERIA

Table 10: Diagnostic criteria for OCD in ICD-10 and DSM-IV

ICD-10 clinical descriptions and diagnostic guidelines	ICD-10 research diagnostic criteria	DSM-IV criteria
Definitions		
Obsessional thoughts: distressing ideas, images, or impulses that enter a person's mind repeatedly. Often violent, obscene, or perceived to be senseless, the person finds these ideas difficult to resist.		**Obsessions:** persistent ideas, thoughts, impulses, or images that are experienced as inappropriate or intrusive and that cause anxiety and distress. The content of the obsession is often perceived as alien and not under the person's control.
Compulsive acts or rituals: stereotyped behaviours that are not enjoyable that are repeated over and over and are perceived to prevent an unlikely event that is in reality unlikely to occur. The person often recognises that the behaviour is ineffectual and makes attempts to resist it, but is unable to.		**Compulsions:** repetitive behaviours or mental acts that are carried out to reduce or prevent anxiety or distress and are perceived to prevent a dreaded event or situation.
Diagnostic criteria		

Continued

Table 1: (*Continued*)

1. Obsessional symptoms or compulsive acts or both must be present on most days for at least 2 successive weeks and be a source of distress or interference with activities.	1. Either obsessions or compulsions (or both) are present on most days for a period of at least 2 weeks.	1. The obsessions or compulsions cause marked distress, are time consuming (take more than 1 hour a day) or significantly interfere with the person's normal routine, occupational (or academic) functioning, or usual social activities or relationships.
2. Obsessional symptoms should have the following characteristics: (a) they must be recognised as the individual's own thoughts or impulses. (b) there must be at least one thought or act that is still resisted unsuccessfully, even though others may be present which the sufferer no longer resists. (c) the thought of carrying out the act must not in itself be pleasurable (simple relief of tension or anxiety is not regarded as pleasure in this sense).	2. Obsessions (thoughts, ideas, or images) and compulsions (acts) share the following features, all of which must be present: (a) they are acknowledged as originating in the mind of the patient, and are not imposed by outside persons or influences. (b) they are repetitive and unpleasant, and at least one obsession or compulsion that is acknowledged as excessive or unreasonable must be present. (c) the patient tries to resist them (but resistance to very long-standing obsessions or compulsions may be minimal). At least one obsession or compulsion that is unsuccessfully resisted must be present.	2. If another Axis I disorder is present, the content of the obsessions or compulsions is not restricted to it (e.g., preoccupation with food in the presence of an Eating Disorder; hair pulling in the presence of Trichotillomania; concern with appearance in the presence of Body Dysmorphic Disorder; preoccupation with drugs in the presence of a Substance Use Disorder; preoccupation with having a serious illness in the presence of Hypochondriasis; preoccupation with sexual urges or fantasies in the presence of a Paraphilia; or guilty ruminations in the presence of a

ICD-10 clinical descriptions and diagnostic guidelines	ICD-10 research diagnostic criteria	DSM-IV criteria
(d) the thoughts, images, or impulses must be unpleasantly repetitive.	(d) experiencing the obsessive thought or carrying out the compulsive act is not in itself pleasurable. (This should be distinguished from the temporary relief of tensions or anxiety.)	Major Depressive Disorder.
	3. The obsessions or compulsions cause distress or interfere with the patient's social or individual functioning, usually by wasting time.	3. The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition.
Differential diagnosis		
Differentiating between obsessive-compulsive disorder and a depressive disorder may be difficult because the two types of symptoms frequently occur together. In an acute episode, presence should be given to the symptoms that developed first; when both types are present but neither predominates, it is usually best to regard the depression as primary.	n/a	Obsessive-compulsive disorder must be distinguished from: ● Anxiety disorder due to a general medical condition. ● Substance-induced anxiety disorder Recurrent or intrusive thoughts, impulses, images or behaviours may occur in the context of many other mental disorders. OCD is not diagnosed if the thoughts or activities is exclusively related to another disorder, such as

Continued

Table 1: (*Continued*)

In chronic disorders, the symptoms that most frequently persist in the absence of the other should be given priority. Occasional panic attacks or mild phobic symptoms are no bar to the diagnosis. However, obsessional symptoms developing in the presence of schizophrenia, Tourette's syndrome, or organic mental disorder should be regarded as part of these conditions. Although obsessional thoughts and compulsive acts commonly coexist, it is useful to be able to specify one set of symptoms as predominant in some individuals, since they may respond to different treatments.		● Body dysmorphic disorder ● Specific or social phobia ● Hair pulling in trichotillomania Worries or ruminations are mood-congruent and aspects of the condition and are not ego-dystonic in ● Major depressive episode Worries are related to real-life circumstances in ● Generalised anxiety disorder Distressing thoughts are exclusively related to fears based on misinterpretation of bodily symptoms in: ● Hypochondriasis Ruminative delusional thoughts and stereotyped behaviours differ from obsessions and compulsions because they are not ego-dystonic and not subject to reality testing in: ● Schizophrenia Movements are typically less complex and are not aimed at neutralising an obsession in: ● Tic disorder ● Stereotypic movement disorder

CD-10 clinical descriptions and diagnostic guidelines	ICD-10 research diagnostic criteria	DSM-IV criteria
		Activities are not considered to be compulsions because pleasure is usually derived in: ● Eating disorder ● Paraphilia ● Pathological gambling ● Alcohol dependence or abuse Condition is not characterised by the presence of obsessions and compulsions and instead involves a pervasive pattern of preoccupation with orderliness and cleanliness and must begin by early adulthood in: ● Obsessive compulsive personality disorder An additional diagnosis of OCD may be warranted if there are obsessions or compulsions not related to the other mental disorder.

Adapted with permission from *The ICD-10 Classification of Mental and Behavioural Disorders: Clinical Descriptions and Diagnostic Guidelines (10th edn), Vol.1. Geneva: World Health Organization (1993)* and *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (Copyright 2000). American Psychiatric Association.*

Table 11: Diagnostic criteria for BDD in DSM-IV

DSM-IV Criteria
Diagnostic criteria
1. Preoccupation with an imagined defect in appearance. If a slight physical anomaly is present, the person's concern is markedly excessive. 2. The preoccupation causes clinically significant distress or impairment in social, occupational, or other important areas of functioning. 3. The preoccupation is not better accounted for by another mental disorder (e.g. dissatisfaction with body shape and size in anorexia nervosa).
Differential diagnosis
Unlike normal concerns about appearance, the preoccupation with appearance is excessively time consuming and associated with significant distress or impairment in social, occupational, or other areas of functioning. Condition is not characterised by an excessive preoccupation restricted to fatness as in: • Eating disorders Condition is not characterised by a sense of inappropriateness about primary and secondary sex characteristics as in • Gender identity disorder The preoccupation is not limited to mood-congruent ruminations involving appearance that occur exclusively during a: • Major depressive episode Concerns with appearance are not prominent, persistent, distressing, time consuming and impairing in: • Avoidant personality disorder • Social phobia A separate diagnosis of BDD is only given when obsessions and compulsions are not restricted to concerns about appearance in • Obsessive-compulsive disorder Removing body hair or picking skin does not occur in response to appearance concerns in • Trichotillomania People with BDD may receive an additional diagnosis of delusional disorder, somatic type, if their preoccupation with an imagined defect in appearance is held with a delusional intensity.
Adapted with permission from: *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (Copyright 2000). American Psychiatric Association.*

12. REFERENCES

AACP (1998) Practice parameters for the assessment and treatment of children and adolescents with obsessive-compulsive disorder. AACAP. *Journal of the American Academy of Child and Adolescent Psychiatry*, *37* (Suppl 10), 27S–45S.

Abram, K.M., Teplin, L.A., McClelland, G.M., *et al.* (2003) Comorbid psychiatric disorders in youth in juvenile detention. *American Journal of Psychiatry*, *60*, 1097–1108.

Abramowitz, J.S. (1997) Effectiveness of psychological and pharmacological treatments for obsessive-compulsive disorder: a quantitative review. *Journal of Consulting and Clinical Psychology*, *65*, 44–52.

Abramowitz, J.S. (2004) Treatment of obsessive-compulsive disorder in patients who have comorbid major depression. *Journal of Clinical Psychology, 60,* 1133–1141.

Abramowitz, J.S., Schwartz, S.A., Moore, K.M. *et al.* (2003) Obsessive-compulsive symptoms in pregnancy and the puerperium: a review of the literature. *Journal of Anxiety Disorders, 17,* 461–478.

Ackerman, D.L. & Greenland, S. (2002) Multivariate meta-analysis of controlled drug studies for obsessive-compulsive disorder. *Journal of Clinical Psychopharmacology*, *22*, 309–317.

Albano, A.M. & DiBartolo, P.M. (1997) Cognitive-behavioral treatment of obsessive-compulsive disorder and social phobia in children and adolescents. In *Innovations in Clinical Practice* (Ed. L. Vande Creek), Vol. 15, pp.41–58. Sarasota, Florida: Professional Resource Exchange Inc.

Albert, U., Aguglia, E., Maina, G., *et al.* (2002) Venlafaxine versus clomipramine in the treatment of obsessive-compulsive disorder: a preliminary single-blind, 12-week, controlled study. *Journal of Clinical Psychiatry*, *63*, 1004–1009.

Albert, U., Maina, G., Forner, F., *et al.* (2004) DSM-IV obsessive-compulsive personality disorder: Prevalence in patients with anxiety disorders and in healthy comparison subjects. *Comprehensive Psychiatry*, *45*, 325–332.

Albertini, R.S., & Phillips, K.A. (1999) Thirty-three cases of body dysmorphic disorder in children and adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*, *38*, 453–459.

Alonso, P., Pujol, J., Cardoner, N., *et al.* (2001) Right prefrontal repetitive transcranial magnetic stimulation in obsessive-compulsive disorder: a double-blind, placebo-controlled study. *American Journal of Psychiatry*, *158*, 1143–1145.

American Psychiatric Association (1980) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn) (DSM-III). Washington, DC: APA.

American Psychiatric Association (1994) *Diagnostic and Statistical Manual of Mental Disorders* (4th edn) (DSM-IV). Washington, DC: APA.

Amin, M.M., Ban, T.A., Pecknold, J.C., *et al.* (1977) Clomipramine (Anafranil) and behaviour therapy in obsessive-compulsive and phobic disorders. *Journal of International Medical Research*, *5*, 33–37.

Amir, N., Freshman, M., & Foa, E.B. (2000) Family distress and involvement in relatives of obsessive-compulsive disorder patients. *Journal of Anxiety Disorders*, *14*, 209–217.

Ananth, J., Pecknold, J.C., van den Steen, N., *et al.* (1981) Double-blind comparative study of clomipramine and amitriptyline in obsessive neurosis. *Progress in Neuro-Psychopharmacology*, *5*, 257–262.

Anderson, D., & Ahmed, A. (2003) Treatment of patients with intractable obsessive-compulsive disorder with anterior capsular stimulation: case report. *Journal of Neurosurgery*, *98*, 1104–1108.

Ansseau, M., Legros, J.J., Mormont, C., *et al.* (1987) Intranasal oxytocin in obsessive-compulsive disorder. *Psychoneuroendocrinology*, *12*, 231–236.

Ansseau, M., Bejerot, S., Blauwblomme, J.F., *et al.* A double-blind study to compare the maintenance of efficacy and relapse rates in patients with obsessive-compulsive disorder who responded to paroxetine, clomipramine or placebo in the short-term study 136. *http://ctr.gsk.co.uk/Summary/paroxetine/studylist.asp* [Study No.: MY-1056/BRL-029060/1/CPMS-241 (PAR 241)] (Accessed April 2004).

Antonelli, F., de Gregorio, M. & Dionisio, A. (1973) Trazodone in the treatment of psychoneuroses: a double-blind study. *Current Therapeutic Research, Clinical and Experimental*, *15*, 799–804.

Antony, M.M., Roth, D., Swinson, R.P., *et al.* (1998) Illness intrusiveness in individuals with panic disorder, obsessive-compulsive disorder, or social phobia. *Journal of Nervous and Mental Disease*, *186*, 311–315.

Aouizerate, B., Pujol, H., Grabot, D., *et al.* (2003) Body dysmorphic disorder in a sample of cosmetic surgery applicants. *European Psychiatry*, *18*, 365–368.

Apter, A., Bernhout, E., & Tyano, S. (1984) Severe obsessive compulsive disorder in adolescence: a report of eight cases. *Journal of Adolescence*, *7*, 349–358.

Apter, A., Horesh, N., Gothelf, D., *et al.* (2003) Depression and suicidal behavior in adolescent inpatients with obsessive compulsive disorder. *Journal of Affective Disorders*, *75*, 181–189.

Askin, R., Turan, M., Cilli, A.S., *et al*. (1999) Clomipramine versus sertraline in the treatment of obsessive compulsive disorder. *Klinik Psikofarmakoloji Buelteni*, *9*, 133–138.

Atmaca, M., Kuloglu, M., Tezcan, E., *et al.* (2002) Quetiapine augmentation in patients with treatment resistant obsessive-compulsive disorder: a single-blind, placebo-controlled study. *International Clinical Psychopharmacology*, *17*, 115–119.

Baer, L., & Greist, J.H. (1997) An interactive computer-administered self-assessment and self-help program for behavior therapy. *Journal of Clinical Psychiatry*, *58* (Suppl 12), 23–28.

Baer, L., Rauch, S.L., Ballantine, H.T., Jr., *et al.* (1995) Cingulotomy for intractable obsessive-compulsive disorder: Prospective long-term follow-up of 18 patients. *Archives of General Psychiatry*, *52*, 384–392.

Bailer, D.C., Burnham, D. & Oakes, R. Long-term treatment with paroxetine of outpatients with obsessive-compulsive disorder: an extension of the companion study. *http://ctr.gsk.co.uk/Summary/paroxetine/studylist.asp* [Study No.: MY-1053/BRL-029060/CPMS-127] (Accessed April 2004).

Bakken, K., Landheim, A.S., & Vaglum, P. (2003) Primary and secondary substance misusers: do they differ in substance-induced and substance-independent mental disorders? *Alcohol and Alcoholism*, *38*, 54–59.

Barker, A.T., Jalinous, R., & Freeston, I.L. (1985) Non-invasive magnetic stimulation of human motor cortex. *Lancet*, 1 (8437), 1106–1107.

Barr, L.C., Goodman, W.K., Anand, A., *et al.* (1997) Addition of desipramine to serotonin reuptake inhibitors in treatment-resistant obsessive-compulsive disorder. *American Journal of Psychiatry*, *154*, 1293–1295.

Barrett, P., Shortt, A., & Healy, L. (2002) Do parent and child behaviours differentiate families whose children have obsessive-compulsive disorder from other clinic and non-clinic families? *Journal of Child Psychology and Psychiatry*, *43*, 597–607.

Barrett, P., Healy, L., & March, J.S. (2003) Behavioral avoidance test for childhood obsessive-compulsive disorder: a home-based observation. *American Journal of Psychotherapy*, *57*, 80–100.

Barrett, P., Healy-Farrell, L., & March, J.S. (2004) Cognitive-behavioral family treatment of childhood obsessive-compulsive disorder: a controlled trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, *43*, 46–62.

Barth, F.D. (1990) Obsessional thinking as 'paradoxical action'. *Bulletin of the Menninger Clinic*, *54*, 499–511.

Bartlett, J.R., & Bridges, P.K. (1977) The extended subcaudate tractotomy lesion. In *Neurosurgical Treatment in Psychiatry, Pain and Epilepsy* (eds. W.H. Sweet, S. Obrador & J.G. Martin-Rodriguez). Baltimore: University Park Press.

Beale, M.D., Kellner, C.H., Pritchett, J.T., *et al.* (1995) ECT for OCD. *Journal of Clinical Psychiatry*, *56*, 81–82.

Beasley, C.M., Jr., Potvin, J.H., Masica, D.N., *et al.* (1992) Fluoxetine: no association with suicidality in obsessive-compulsive disorder. *Journal of Affective Disorders*, *24*, 1–10.

Becker, D., & North, M.M. (1998) The virtual reality therapy system: VRT-2002. *Cyberpsychology and Behavior*, *1*, 401–403.

Bejerot, S. (2003) Psychosurgery for obsessive-compulsive disorder: concerns remain. *Acta Psychiatrica Scandinavica*, *107*, 241–243.

Bejerot, S., & Bodlund, O. (1998) Response to high doses of citalopram in treatment-resistant obsessive-compulsive disorder. *Acta Psychiatrica Scandinavica*, *98*, 423–424.

Benazon, N.R., Ager, J., & Rosenberg, D.R. (2002) Cognitive behavior therapy in treatment-naive children and adolescents with obsessive-compulsive disorder: an open trial. *Behaviour Research and Therapy*, *40*, 529–539.

Bergeron, R., Ravindran, A.V., Chaput, Y., *et al.* (2002) Sertraline and fluoxetine treatment of obsessive-compulsive disorder: results of a double-blind, 6-month treatment study. *Journal of Clinical Psychopharmacology*, *22*, 148–154.

Berle, D., & Starcevic, V. (2005) Thought-action fusion: review of the literature and future directions. *Clinical Psychology Review, 25*, 263–284.

Berlin, J.A. (2001) Does blinding of readers affect the results of meta-analysis? University of Pennsylvania Meta-Analysis Blinding Study Group. *Lancet*, *350* (9072), 185–186.

Biederman, J., Petty, C., Faraone, S.V., *et al.* (2004) Moderating effects of major depression on patterns of comorbidity in patients with panic disorder. *Psychiatry Research*, *126*, 143–149.

Bisserbe, J.C., Lane, R.M., & Flament, M.F. (1997) A double-blind comparison of sertraline and clomipramine in outpatients with obsessive-compulsive disorder. *European Psychiatry*, *12*, 82–93.

Black, A. (1974) The natural history of obsessional neurosis. In *Obsessional States* (eds. H.R. Beech), pp. 19–54. London: Methuen.

Black, D., & Noyes, R. (1997) Obsessive-compulsive disorder and axis II. *International Review of Psychiatry*, *9*, 111–118.

Black, D.W., Gaffney, G., Schlosser, S., *et al.* (1998) The impact of obsessive-compulsive disorder on the family: preliminary findings. *Journal of Nervous and Mental Disease*, *186*, 440–442.

Black, D.W., Gaffney, G.R., Schlosser, S., *et al.* (2003) Children of parents with obsessive-compulsive disorder: a 2-year follow-up study. *Acta Psychiatrica Scandinavica*, *107*, 305–313.

Blier, P. & Bergeron, R. (1996) Sequential administration of augmentation strategies in treatment-resistant obsessive-compulsive disorder: preliminary findings. *International Clinical Psychopharmacology*, *11*, 37–44.

Bloch, S. & Glue, P. (1988) Psychotherapy and dysmorphophobia: a case report. *British Journal of Psychiatry*, *152*, 271–274.

Bobes, J., Gonzalez, M.P., Bascaran, M.T., *et al.* (2001) Quality of life and disability in patients with obsessive-compulsive disorder. *European Psychiatry, 16,* 239–245.

Boehm, T. (2002) Sara in her fourth analytic year. *International Journal of Psycho-Analysis*, *83*, 1003–1008.

Boersma, K., Den Hengst, S., Dekker, J., *et al.* (1976) Exposure and response prevention in the natural environment: a comparison with obsessive-compulsive patients. *Behaviour Research and Therapy, 14*, 19–24.

Bogetto, F., Bellino, S., Vaschetto, P., *et al.* (2000) Olanzapine augmentation of fluvoxamine-refractory obsessive-compulsive disorder (OCD): a 12-week open trial. *Psychiatry Research*, *96*, 91–98.

Bogetto, F., Albert, U., & Maina, G. (2002) Sertraline treatment of obsessive-compulsive disorder: efficacy and tolerability of a rapid titration regimen. *European Neuropsychopharmacology*, *12*, 181–186.

Bohne, A., Wilhelm, S., & Keuthen, N.J. (2002) Prevalence of body dysmorphic disorder in a German college student sample. *Psychiatry Research*, *109*, 101–104.

Bolton, D. & Turner, T. (1984) Obsessive-compulsive neurosis with conduct disorder in adolescence: a report of two cases. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *25*, 133–139.

Bolton, D., Collins, S., & Steinberg, D. (1983) The treatment of obsessive-compulsive disorder in adolescence: a report of fifteen cases. *British Journal of Psychiatry*, *142*, 456–464.

Bolton, D., Luckie, M., & Steinberg, D. (1996) Obsessive-compulsive disorder treated in adolescence: 14 long-term case histories. *Clinical Child Psychology and Psychiatry*, *1*, 409–430.

Bouvard, M. (2002) A standardized cognitive-behavioural group treatment program for obsessive compulsive disorder: preliminary outcomes. *Encephale, 28*, 439–446.

Braddock, L.E. (1982) Dysmorphophobia in adolescence: a case report. *British Journal of Psychiatry*, *140*, 199–201.

Brawman-Mintzer, O., Lydiard, R.B., Phillips, K.A., *et al.* (1995) Body dysmorphic disorder in patients with anxiety disorders and major depression: a comorbidity study. *American Journal of Psychiatry*, *152*, 1665–1667.

Brown, K.W., McGoldrick, T., & Buchanan, R. (1997) Body dysmorphic disorder: seven cases treated with eye movement desensitization and reprocessing. *Behavioural and Cognitive Psychotherapy*, *25*, 203–207.

Burgy, M. (2001) The narcissistic function in obsessive-compulsive neurosis. *American Journal of Psychotherapy*, *55*, 65–73.

Burnham, D.B., Apter, J., Ballenger, J.C., *et al.* Paroxetine versus clomipramine and placebo in the treatment of obsessive-compulsive disorder. *http://ctr.gsk.co.uk/Summary/paroxetine/studylist.asp* [Study No.: MY-1028/BRL-029060/1/CPMS-118] (Accessed April 2004).

Butters, J.W. & Cash, T.F. (1987) Cognitive-behavioral treatment of women's body-image dissatisfaction. *Journal of Consulting and Clinical Psychology*, *55*, 889–897.

Byerly, M.J., Goodman, W.K., & Christensen, R. (1996) High doses of sertraline for treatment-resistant obsessive-compulsive disorder. *American Journal of Psychiatry*, *153*, 1232–1233.

Calvocoressi, L., Lewis, B., Harris, M., *et al.* (1995) Family accommodation in obsessive-compulsive disorder. *American Journal of Psychiatry*, *152*, 441–443.

Calvocoressi, L., Mazure, C.M., Kasl, S.V., *et al.* (1999) Family accommodation of obsessive-compulsive symptoms: instrument development and assessment of family behavior. *Journal of Nervous and Mental Disease*, *187*, 636–642.

Campisi, T.A. (1995) Exposure plus response prevention in the treatment of body dysmorphic disorder. PhD thesis, Hofstra University, Hempstead, New York.

Carpenter, D.J., Schaefer, D.M., Lawnnson, S., *et al.* A 38-week, two phase, multicentre study to investigate the safety and effectiveness of paroxetine (10–60 mg/day) in the treatment of children and adolescent outpatients with obsessive-compulsive disorder. *http://ctr.gsk.co.uk/Summary/paroxetine/studylist.asp* [Study No.: BRL-029060/RSD-100W81/1/CPMS-453] (Accessed April 2004).

Carroll, B.J. (1994) Response of major depression with psychosis and body dysmorphic disorder to ECT. *American Journal of Psychiatry*, *158*, 288–289.

Carter, L. (2001) Body dysmorphia. In Electronic Response to Katharine A. Phillips & David J. Castle, Body dysmorphic disorder in men. *BMJ, 323,* 1015–1016. http://bmj.bmjjournals.com/cgi/eletters/323/7320/1015#17324.

Casas, M., Alvarez, E., Duro, P., *et al.* (1986) Antiandrogenic treatment of obsessive-compulsive neurosis. *Acta Psychiatrica Scandinavica*, *73*, 221–222.

Casey, D.A., & Davis, M.H. (1994) Obsessive-compulsive disorder responsive to electroconvulsive therapy in an elderly woman. *South African Medical Journal*, *87*, 862–864.

Cassano, G.B., Castrogiovanni, P., & Mauri, M. (1981) A multicenter controlled trial in phobic-obsessive psychoneurosis: the effect of chlorimipramine and of its combinations with haloperidol and diazepam. *Progress in Neuro-Psychopharmacology, 5*, 129–138.

Castle, D.J., Molton, M., Hoffman, K., *et al.* (2004) Correlates of dysmorphic concern in people seeking cosmetic enhancement. *Australian and New Zealand Journal of Psychiatry*, *38*, 439–444.

Cela, J.A. (1995) A classical case of a severe obsessive compulsive defense. *Modern Psychoanalysis*, *20*, 271–277.

Chakrabarti, S., Kulhara, P., & Verma, S.K. (1993) The pattern of burden in families of neurotic patients. *Social Psychiatry and Psychiatric Epidemiology*, *28*, 172–177.

Chambless, D.L. & Steketee, G. (1999) Expressed emotion and behavior therapy outcome: a prospective study with obsessive-compulsive and agoraphobic outpatients. *Journal of Consulting and Clinical Psychology, 67*, 658–665.

Chambless, D.L., Bryan, A.D., Aiken, L.S., *et al.* (2001) Predicting expressed emotion: a study with families of obsessive-compulsive and agoraphobic outpatients. *Journal of Family Psychology*, *15*, 225–240.

Chatterji, N.N. (1963) Psychoanalysis of an artist with obsessional symptoms. *Samiksa*, *17*, 173–197.

Chiocca, E.A., & Matuza, R.L. (1990) Neurosurgical therapy of obsessive compulsive disorder. In *Obsessive-Compulsive Disorders: Theory and Management* (eds. M.A. Jenike, L. Baer, & W.E. Minichiello), pp. 283–294. Chicago: Year Book Medical.

Chouinard, G. (1992) Sertraline in the treatment of obsessive compulsive disorder: two double-blind, placebo-controlled studies. *International Clinical Psychopharmacology*, *7*, 37–41.

Chouinard, G., Goodman, W., Greist, J., *et al.* (1990) Results of a double-blind placebo controlled trial of a new serotonin uptake inhibitor, sertraline, in the treatment of obsessive-compulsive disorder. *Psychopharmacology Bulletin*, *26*, 279–284.

Chowdhury, U., Frampton, I., & Heyman, I. (2004) Clinical characteristics of young people referred to an obsessive compulsive disorder clinic in the United Kingdom. *Clinical Child Psychology and Psychiatry*, *9*, 395–401.

Christensen, H., Hadzi-Pavlovic, D., Andrews, G., *et al.* (1987) Behavior therapy and tricyclic medication in the treatment of obsessive-compulsive disorder: a quantitative review. *Journal of Consulting and Clinical Psychology*, *55*, 701–711.

Christensen, D.D., Laitinen, L.V., Schmidt, L.J., *et al.* (2002) Anterior capsulotomy for treatment of refractory obsessive-compulsive disorder: results in a young and an old patient. *Stereotactic and Functional Neurosurgery*, *79*, 234–244.

Chung, M.S., Huang, S.C., Tsai, S.J., *et al.* (2001) Mania associated with electroconvulsive therapy in a patient with obsessive compulsive disorder. *International Medical Journal*, *8*, 185–186.

Churchill, J.E. (1986) Hypnotherapy and conjoint family therapy: a viable treatment combination. *American Journal of Clinical Hypnosis*, *28*, 170–176.

Clark, D.A. (2004) *Cognitive-Behavioral Therapy for OCD*. New York, NY: Guilford Press.

Clark, D.A. & Beck, A.T. (2002) *Clark-Beck Obsessive Compulsive Inventory*. San Antonio, TX: The Psychological Corporation.

Clark, D.A., Sugrim, I., & Bolton, D. (1982) Primary obsessional slowness: a nursing treatment programme with a 13-year-old male adolescent. *Behaviour Research and Therapy*, *20*, 289–292.

Clarke, D.J., Boer, H., Whittington, J., *et al.* (2002) Prader-Willi syndrome, compulsive and ritualistic behaviours: the first population-based survey. *British Journal of Psychiatry*, *180*, 358–362.

Clomipramine Collaborative Study Group (CCSG) (1991) Clomipramine in the treatment of patients with obsessive-compulsive disorder. *Archives of General Psychiatry, 48,* 730–738.

Cochrane Collaboration (2004) Review Manager (Rev Man). [4.2.7 for Windows].Oxford, England: The Cochrane Collaboration.

Cordioli, A.V., Heldt, E., Bochi, D.B., *et al.* (2003) Cognitive-behavioral group therapy in obsessive-compulsive disorder: a randomized clinical trial. *Psychotherapy and Psychosomatics*, *72*, 211–216.

Coryell, W.H., Black, D.W., Kelly, M.W., *et al.* (1989) HPA axis disturbance in obsessive-compulsive disorder. *Psychiatry Research, 30*, 243–251.

Cosyns, P., Caemaert, J., Haaijman, W., *et al.* (1994) Functional stereotactic neurosurgery for psychiatric disorders: an experience in Belgium and The Netherlands. *Advances and Technical Standards in Neurosurgery*, *21*, 239–279.

Cottraux, J., Mollard, E., Bouvard, M., *et al.* (1990) A controlled study of fluvoxamine and exposure in obsessive-compulsive disorder. *International Clinical Psychopharmacology*, *5*, 17–30.

Cottraux, J., Messy, P., Marks, I.M., *et al*. (1993) Predictive factors in the treatment of obsessive-compulsive disorders with fluvoxamine and/or behaviour therapy. *Behavioural Psychotherapy*, *21*, 45–50.

Cottraux, J., Note, I., Yao, S.N., *et al.* (2001) A randomized controlled trial of cognitive therapy versus intensive behavior therapy in obsessive compulsive disorder. *Psychotherapy and Psychosomatics*, *70*, 288–297.

Cox, B.J., Swinson, R.P., Morrison, B., *et al.* (1993) Clomipramine, fluoxetine, and behavior therapy in the treatment of obsessive-compulsive disorder: a meta-analysis. *Journal of Behavior Therapy and Experimental Psychiatry*, *24*, 149–153.

Crago, H. (1995) The anxious boys' newsletter. *Australian and New Zealand Journal of Family Therapy*, *16*, 29–37.

Crocq, M.A. (2002) Open-label olanzapine in obsessive-compulsive disorder refractory to antidepressant treatment. *European Psychiatry*, *17*, 296–297.

Cromarty, P., & Marks, I. (1995) Does rational role-play enhance the outcome of exposure therapy in dysmorphophobia? A case study. *British Journal of Psychiatry*, *167*, 399–402.

Cui, Y.H. (1986) A double-blind trial of chlorimipramine and doxepin in obsessive-compulsive neurosis. [Chinese]. *Chung-Hua Shen Ching Ching Shen Ko Tsa Chih [Chinese Journal of Neurology and Psychiatry]*, *19*, 279–281.

Cumming, S., Hay, P., Lee, T., *et al.* (1995) Neuropsychological outcome from psychosurgery for obsessive-compulsive disorder. *Australian and New Zealand Journal of Psychiatry*, *29*, 293–298.

Dale, R.C., & Heyman, I. (2002) Post-streptococcal autoimmune psychiatric and movement disorders in children. *British Journal of Psychiatry*, *181*, 188–190.

Dalton, P. (1983) Family treatment of an obsessive-compulsive child: a case report. *Family Process*, *22*, 99–108.

D'Amico, G., Cedro, C., Muscatello, M.R., *et al.* (2003) Olanzapine augmentation of paroxetine-refractory obsessive-compulsive disorder. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *27*, 619–623.

Dannon, P.N., Sasson, Y., Hirschmann, S., *et al.* (2000) Pindolol augmentation in treatment-resistant obsessive compulsive disorder: a double-blind placebo controlled trial. *European Neuropsychopharmacology*, *10*, 165–169.

De Araujo, L.A., Ito, L.M., Marks, I.M., *et al.* (1995) Does imagined exposure to the consequences of not ritualising enhance live exposure for OCD? A controlled study. I. Main outcome. *British Journal of Psychiatry*, *167*, 65–70.

De Araujo, L.A., Ito, L.M., & Marks, I.M. (1996) Early compliance and other factors predicting outcome of exposure for obsessive-compulsive disorder. *British Journal of Psychiatry*, *169*, 747–752.

De Haan, E., Hoogduin, K.A., Buitelaar, J.K., *et al.* (1998) Behavior therapy versus clomipramine for the treatment of obsessive-compulsive disorder in children and adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*, *37*, 1022–1029.

Den Boer, J.A., & Westenberg, H.G. (1992) Oxytocin in obsessive compulsive disorder. *Peptides*, *13*, 1083–1085.

Den Boer, J.A., Westenberg, H.G., Kamerbeek, W.D., *et al.* (1987) Effect of serotonin uptake inhibitors in anxiety disorders: a double-blind comparison of clomipramine and fluvoxamine. *International Clinical Psychopharmacology, 2*, 21–32.

Denys, D., van der Wee, N., van Megen, H.J., *et al.* (2002) Double blind comparison of venlafaxine and paroxetine in obsessive-compulsive disorder. *International Journal of Neuropsychopharmacology*, *5*, S131–S132.

Denys, D., van Megen, H., & Westenberg, H. (2002a) Quetiapine addition to serotonin reuptake inhibitor treatment in patients with treatment-refractory obsessive-compulsive disorder: an open-label study. *Journal of Clinical Psychiatry*, *63* (8), 700–703.

Denys, D., van Megen H. & Westenberg, H. (2002b) The adequacy of pharmacotherapy in outpatients with obsessive-compulsive disorder. *International Clinical Psychopharmacology*, *17*, 109–114.

Denys, D., van der Wee, N., van Megen, H., *et al.* (2003) A double blind comparison of venlafaxine and paroxetine in obsessive-compulsive disorder. *Journal of Clinical Psychopharmacology*, *23*, 568–575.

Denys, D., de Geus, F., van Megen, H., *et al.* (2004) A double-blind, randomized, placebo-controlled trial of quetiapine addition in patients with obsessive-compulsive disorder refractory to serotonin reuptake inhibitors. *Journal of Clinical Psychiatry*, *65*, 1040–1048.

Department of Health (2001) *Mental Health NSF (and the NHS Plan) Workforce Planning, Education and Training-Underpinning Programme: Adult Mental Health Services – Final Report by the Workforce Action Team*. London: Department of Health.

Department of Health (2004) *Organising and Delivering Psychological Therapies.* London: Department of Health.

Deri, S. (1990) Case presentation. *Psychoanalytic Review*, *77*, 535–554.

DerSimonian, R., & Laird, N. (1986) Meta-analysis in Clinical Trials. *Controlled Clinical Trials*, *7*, 177–188.

Desmarais, P.A., & Lavallee, Y.J. (1988) Severe obsessive-compulsive syndrome in a 10 year old: a 3 year follow-up. *Canadian Journal of Psychiatry – Revue Canadienne de Psychiatrie*, *33*, 405–408.

Detweiler, M.F., & Albano, A.M. (2001) Covert symptoms of obsessive-compulsive disorder in children: a case study. *Journal of Cognitive Psychotherapy*, *15*, 75–86.

DeVeaugh-Geiss, J., Landau, P. & Katz, R. (1989) Treatment of obsessive compulsive disorder with clomipramine. *Psychiatric Annals, 19*, 97–101.

DeVeaugh-Geiss, J., Katz, R., Landau, P., *et al.* (1990) Clinical predictors of treatment response in obsessive compulsive disorder: exploratory analyses from multicenter trials of clomipramine. *Psychopharmacology Bulletin, 26*, 54–59.

DeVeaugh-Geiss, J., Katz, R., Landau, P., *et al.* (1991) Clomipramine hydrochloride (Anafranil) in the treatment of obsessive-compulsive disorder: results from three multicentre trials. In *Understanding Obsessive-Compulsive Disorder* (OCD) (eds. M.A. Jenike & M. Asberg), pp. 46–51. Toronto: Hogrefe and Huber Publishers; Bern: Hans Huber.

DeVeaugh-Geiss, J., Moroz, G., Biederman, J., *et al.* (1992) Clomipramine hydrochloride in childhood and adolescent obsessive-compulsive disorder: a multicenter trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, *31*, 45–49.

De Waal, M.W.M., Arnold, I.A., Eekhof, J.A.H., *et al.* (2004) Prevalence, functional impairment and comorbidity with anxiety and depressive disorders. *British Journal of Psychiatry*, *184*, 479–476.

Diamond, B.I., Borison, R.L., Katz, R., *et al.* (1989) Rebound withdrawal reactions due to clomipramine. *Psychopharmacology Bulletin, 25*, 209–212.

Di Blasi, Z., Harkness, E., Ernst, E., *et al.* (2001) Influence of context effects on health outcomes: a systematic review. *Lancet*, *357* (9258), 757–762.

Doherr, L., Reynolds, S., Wetherly, J., *et al.* (2005) Young children's ability to engage in cognitive therapy tasks: associations with age and educational experience. *Behavioural and Cognitive Psychotherapy*, *33*, 201–215.

Dormaar, J.M.M. (1987) February man revisited. *American Journal of Clinical Hypnosis*, *30*, 28–35.

Do Rosario-Campos, M.C., Leckman, J.F., Curi, M., *et al.* (2005) A family study of early-onset obsessive-compulsive disorder. *American Journal of Medical Genetics Part B, Neuropsychiatric Genetics: The Official Publication of the International Society of Psychiatric Genetics*, *136*, 92–97.

Dougherty, D.D., Baer, L., Cosgrove, G.R., *et al.* (2002) Prospective long-term follow-up of 44 patients who received cingulotomy for treatment-refractory obsessive-compulsive disorder. *American Journal of Psychiatry*, *159*, 269–275.

Dreessen, L., Hoekstra, R., & Arntz, A. (1997) Personality disorders do not influence the results of cognitive and behavior therapy for obsessive compulsive disorder. *Journal of Anxiety Disorders, 11*, 503–521.

Drummond, M.F., O'Brien, B., Stoddart, G.I., *et al.* (1997) *Methods for the Economic Evaluation of Health Care Programmes*. Oxford: Oxford University Press.

Dubois, A.M. (1983) A double-blind study of two types of psychiatric treatments. [French]. *Genitif*, *5*, 91–101.

Dufresne, R.G., Phillips, K.A., Vittorio, C.C., *et al.* (2001) A screening questionnaire for body dysmorphic disorder in a cosmetic dermatologic surgery practice. *Dermatologic Surgery*, *27*, 457–462.

DuPont, R.L., Rice, D.P., Shiraki, S., *et al.* (1995) Economic costs of obsessive compulsive disorder. *Medical Interface*, *8*, 102–109.

Eccles, M., & Mason, J. (2001) How to develop cost-conscious guidelines. *Health Technology Assessment*, *5*, 1–69.

Eccles, M., Freemantle, N., & Mason, J. (1998) North of England evidence-based guideline development project: methods of developing guidelines for efficient drug use in primary care. *BMJ*, *316*, 1232–1235.

Eddy, M.F., & Walbroehl, G.S. (1998) Recognition and treatment of obsessive-compulsive disorder. *American Family Physician*, *57*, 1623–1624.

Einat, H., Shaldubina, A., & Belmaker, R.H. (2000) Epi-inositol: a potential antidepressant. *Drug Development Research, 50*, 309–315.

El-Khatib, H.E., & Dickey, T.O. (1995) Sertraline for body dysmorphic disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *34*, 1404–1405.

Emmelkamp, P.M. & Beens, H. (1991) Cognitive therapy with obsessive-compulsive disorder: a comparative evaluation. *Behaviour Research and Therapy*, *29*, 293–300.

Emmelkamp, P.M.G. & de Lange, I. (1983) Spouse involvement in the treatment of obsessive-compulsive patients. *Behaviour Research and Therapy*, *21*, 341–346.

Emmelkamp, P.M.G. & Foa, E.B. (1983) Failures are a challenge. In *Failures in Behaviour Therapy* (eds. E.B. Foa & P.M.G. Emmelkamp), pp. 1–9. New York: Wiley.

Emmelkamp, P.M.G. & Giesselbach, P. (1981) Treatment of obsessions: relevant vs. irrelevant exposure. *Behavioural Psychotherapy, 9*, 322–329.

Emmelkamp, P.M.G. & Kraanen, J. (1977) Therapist-controlled exposure in vivo versus self-controlled exposure in vivo: a comparison with obsessive-compulsive patients. *Behaviour Research and Therapy*, *15*, 491–495.

Emmelkamp, P.M.G. & van der Heyden, H. (1980) Treatment of harming obsessions. *Behavioural Analysis and Modification, 4*, 28–35.

Emmelkamp, P.M., van der Heyden, H., van Zanten, B.L., *et al.* (1980) Treatment of obsessive-compulsive patients: the contribution of self-instructional training to the effectiveness of exposure. *Behaviour Research and Therapy, 18*, 61–66.

Emmelkamp, P.M., Visser, S. & Hoekstra, R.J. (1988) Cognitive therapy vs exposure in vivo in the treatment of obsessive-compulsives. *Cognitive Therapy and Research*, *12*, 103–114.

Emmelkamp, P.M., van Linden van den Heuvell, C., Ruphan, M., *et al.* (1989) Home-based treatment of obsessive-compulsive patients: intersession interval and therapist involvement. *Behaviour Research and Therapy, 27*, 89–93.

Emmelkamp, P.M.G., de Haan, E., & Hoogduin, C.A.L. (1990) Marital adjustment and obsessive-compulsive disorder. *British Journal of Psychiatry, 156*, 55–60.

Enright S.J. (1991) Group treatment for obsessive-compulsive disorder: an evaluation. *Behavioral Psychotherapy*, *19*, 183–92.

Erickson, M.H. (1973) Psychotherapy achieved by a reversal of the neurotic processes in a case of ejaculatio precox. *American Journal of Clinical Hypnosis*, *15*, 217–222.

Eriksson, T. (2000) Antiandrogenic treatment for obsessive-compulsive disorder. *American Journal of Psychiatry*, *157*, 483.

Erzegovesi, S., Ronchi, P., & Smeraldi, E. (1992) 5HT-2 receptor and fluvoxamine effect in obsessive-compulsive disorder. *Human Psychopharmacology, 7*, 287–289.

Esman, A.H. (2001) Obsessive-compulsive disorder: current views. *Psychoanalytic Inquiry*, *21*, 145–156.

Fallon, B.A., Liebowitz, M.R., Campeas, R., *et al.* (1998) Intravenous clomipramine for obsessive-compulsive disorder refractory to oral clomipramine: a placebo-controlled study. *Archives of General Psychiatry*, *55*, 918–924.

Fals-Stewart, W., & Angarano. K. (1994) Obsessive-compulsive disorder among patients entering substance abuse treatment: prevalence and accuracy of diagnosis. *Journal of Nervous and Mental Disease*, *182*, 715–719.

Fals-Stewart, W., Marks, A.P. & Schafer, J. (1993) A comparison of behavioral group therapy and individual behavior therapy in treating obsessive-compulsive disorder. *Journal of Nervous and Mental Disease, 181*, 189–193.

Faravelli, C., Salvatori, S., Galassi, F., *et al.* (1997) Epidemiology of somatoform disorders: a community survey in Florence. *Social Psychiatry and Psychiatric Epidemiology*, *32*, 24–29.

Feldman, J.D., Noshirvani, H., & Chu, C. (1988) Improvement in female patients with severe obsessions and/or compulsions treated with cyproterone acetate. *Acta Psychiatrica Scandinavica*, *78*, 254.

Fields, L. (1998) An integrative brief treatment approach for obsessive-compulsive disorder. *Journal of Psychotherapy Integration*, *8*, 161–172.

Fine, S. (1973) Family therapy and a behavioral approach to childhood obsessive-compulsive neurosis. *Archives of General Psychiatry*, *28*, 695–697.

Fineberg, N.A., & Gale, T.M. (2004) Evidence-based pharmacotherapy of obsessive-compulsive disorder. *International Journal of Neuropsychopharmacology*, 7, 1–23.

Fineberg, N.A., & Roberts, A. (2001) Obsessive compulsive disorder: a twenty-first century perspective. In *Obsessive Compulsive Disorder: A Practical Guide* (eds. N.A. Fineberg, D. Marazziti, & D. Stein), pp. 1–13. London: Martin Dunitz.

Fineberg, N.A., O'Doherty, C., Rajagopal, S., *et al.* (2003) How common is obsessive compulsive disorder in a dermatology outpatient clinic? *Journal of Clinical Psychiatry*, *64*, 152–155.

Finell, J.S., & McDougall, J. (1985) A case presentation: the treatment of a man with a severe obsessional disorder. *Current Issues in Psychoanalytic Practice*, *2*, 3–24.

Fingert Chused, J. (1999) Obsessional manifestations in children. *Psychoanalytic Study of the Child*, *54*, 219–232.

First, M.B., Spitzer, R.L., Gibbon, M., *et al.* (1997) *Structured Clinical Interview for DSM-IV Axis I Disorders – Clinician Version (SCID-CV)*. Washington DC: American Psychiatric Press.

Fischer, D.J., Himle, J.A., & Hanna, G.L. (1998) Group behavioral therapy for adolescents with obsessive-compulsive disorder: preliminary outcomes. *Research on Social Work Practice*, *8*, 629–636.

Flament, M.F., Rapoport, J.L., Berg, C.J., *et al.* (1985) Clomipramine treatment of childhood obsessive-compulsive disorder: a double-blind controlled study. *Archives of General Psychiatry*, *42*, 977–983.

Flament, M.F., Whitaker, A., Rapoport, J.L., *et al.* (1988) Obsessive compulsive disorder in adolescence: an epidemiological study. *Journal of the American Academy of Child and Adolescent Psychiatry*, *27*, 764–771.

Foa, E.B., & Franklin, M.E. (2000) Psychotherapies for obsessive-compulsive disorder: a review. In *Obsessive-Compulsive Disorder* (eds. M. Maj, N. Sartorius, A. Okasha *et al.*), WPA series evidence and experience in psychiatry, pp. 93–146. New York: John Wiley & Sons Ltd.

Foa, E.B., & Goldstein, A.J. (1978) Continuous exposure and complete response prevention in the treatment of obsessive-compulsive neurosis. *Behavior Therapy*, *9*, 821–829.

Foa, E.B., & Kozak, M.J. (1996) Psychological treatment for obsessive-compulsive disorder. In *Long-Term Treatments of Anxiety Disorders* (eds. M.R. Mavissakalian & R.F. Prien), pp. 285–309. Washington, DC: American Psychiatric Association.

Foa, E.B., Steketee, G., & Milby, J.B. (1980) Differential effects of exposure and response prevention in obsessive-compulsive washers. *Journal of Consulting and Clinical Psychology*, *48*, 71–79.

Foa, E.B., Steketee, G.S., Grayson, J.B., *et al.* (1984) Deliberate exposure and blocking of obsessive-compulsive rituals: immediate and long-term effects. *Behavior Therapy, 15*, 450–472.

Foa, E.B., Steketee, G.S., & Oazrow, B.J. (1985) Behavior therapy with obsessive-compulsives: from theory to treatment. In *Obsessive-Compulsive Disorder: Psychological and Pharmacological Treatments* (eds. M. Mavissakeliam, S.M. Turner & L. Michelson), pp. 49–120. New York: Plenum.

Foa, E.B., Kozak, M.J., Goodman, W.K., *et al.* (1995) DSM-IV field trial: obsessive compulsive disorder. *American Journal of Psychiatry*, *152*, 990–996.

Foa, E.B., Kozak, M.J., Michael, J. *et al.* (1998) The validation of a new obsessive-compulsive disorder scale: the obsessive-compulsive inventory. *Psychological Assessment*, *10*, 206–214.

Foa, E.B., Franklin, M.E., & Moser, J. (2002a) Context in the clinic: how well do cognitive-behavioral therapies and medications work in combination? *Biological Psychiatry*, *52*, 987–997.

Foa, E.B., Huppert, J.D., Leiberg, S., *et al.* (2002b) The Obsessive-Compulsive Inventory: development and validation of a short version. *Psychological Assessment*, *14*, 485–496.

Foa, E.B., Liebowitz, M.R., Kozak, M.J., *et al.* (2005) Randomized, placebo-controlled trial of exposure and ritual prevention, clomipramine, and their combination in the treatment of obsessive-compulsive disorder. *American Journal of Psychiatry*, *162*, 151–161.

Fodstad, H., Strandman, E., Karlsson, B., *et al.* (1982) Treatment of chronic obsessive compulsive states with stereotactic anterior capsulotomy or cingulotomy. *Acta Neurochirurgica*, *62*, 1–23.

Fontenelle, L.F., Mendlowicz, M.V., Marques, C., *et al.* (2004) Trans-cultural aspects of obsessive-compulsive disorder: a description of a Brazilian sample and a systematic review of international clinical studies. *Journal of Psychiatric Research*, *38*, 403–411.

Francis, G. (1988) Childhood obsessive-compulsive disorder: extinction of compulsive reassurance-seeking. *Journal of Anxiety Disorders*, *2*, 361–366.

Francobandiera, G. (2001) Olanzapine augmentation of serotonin uptake inhibitors in obsessive-compulsive disorder: an open study. *Canadian Journal of Psychiatry*, *46*, 356–358.

Franklin, M.E., Kozak, M.J., Cashman, L.A., *et al.* (1998) Cognitive-behavioral treatment of pediatric obsessive-compulsive disorder: an open clinical trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, *37*, 412–419.

Franklin, M.E., Tolin, D.F., March, J.S., *et al.* (2001) Treatment of pediatric obsessive-compulsive disorder: a case example of intensive cognitive-behavioral therapy involving exposure and ritual prevention. *Cognitive and Behavioral Practice*, *8*, 297–304.

Franklin, M.E., Rynn, M., Foa, E.B., *et al.* (2003) Treatment of obsessive compulsive disorder. In *Cognitive Therapy With Children and Adolescents: A Casebook for Clinical Practice* (eds. M.A. Reinecke, F.M. Datilio, & A. Freeman), pp. 162–188. New York & London: Guilford Press.

Frare, P. & Lebel, A. (1996) The use of 'thought-stopping' in the treatment of obsessive-compulsive disorder in a 9-year-old girl. *Canadian Journal of Psychiatry – Revue Canadienne de Psychiatrie*, *41*, 367–370.

Frare, F., Perugi, G., Ruffolo, G., *et al.* (2004) Obsessive-compulsive disorder and body dysmorphic disorder: a comparison of clinical features. *European Psychiatry*, *19*, 292–298.

Freeling, P., Rao, B.M., Paykel, E.S., *et al.* (1985) Unrecognised depression in general practice. *BMJ (Clinical Research Edition)*, *290*, 1880–1883.

Freeman, C.P., Trimble, M.R., Deakin, J.F., *et al.* (1994) Fluvoxamine versus clomipramine in the treatment of obsessive compulsive disorder: a multicenter, randomized, double-blind, parallel group comparison. *Journal of Clinical Psychiatry*, *55*, 301–305.

Freeman, C., Crossley, D., & Eccleston, D. (2000) *Neurosurgery for Mental Disorder: Report from the Neurosurgery Working Group of the Royal College of Psychiatrists*. London: Royal College of Psychiatrists, CR89.

Freeston, M.H. (2001) Cognitive-behavioural treatment of a 14-year-old teenager with obsessive-compulsive disorder. *Behavioural and Cognitive Psychotherapy*, *29*, 71–84.

Freeston, M., Rheaume, J., & Ladouceur, R. (1996) Correcting faulty appraisals of obsessive thoughts. *Behaviour Research and Therapy*, *34*, 446.

Freeston, M.H., Ladouceur, R., Gagnon, F., *et al.* (1997) Cognitive-behavioral treatment of obsessive thoughts: a controlled study. *Journal of Consulting and Clinical Psychology*, *65*, 405–413.

Freud, S. (1909) *Notes Upon a Case of Obsessional Neurosis* (Reprinted 1955) in *The Standard Edition of the Complete Psychological Works of Sigmund Freud* (translated and edited by J. Strachey), Vol. 10. London: Hogarth Press.

Friedmann, C.T., & Silvers, F.M. (1977) A multimodality approach to inpatient treatment of obsessive-compulsive disorder. *American Journal of Psychotherapy*, *31*, 456–465.

Fritzler, B.K., Hecker, J.E., & Losee, M.C. (1997) Self-directed treatment with minimal therapist contact: preliminary findings for obsessive-compulsive disorder. *Behaviour Research and Therapy*, *35*, 627–631.

Frost, R., & Steketee, G. (1999) Issues in the treatment of compulsive hoarding. *Cognitive and Behavioral Practice*, *6*, 397–407.

Frost, R., & Steketee, G. (2002) *Cognitive Approaches to Obsessions and Compulsions: Theory, Assessment, and Treatment*. Amsterdam, Netherlands: Pergamon/Elsevier Science Inc.

Furth, G.M., Smith, R., & Kubler-Ross, E. (2000) *Amputee Identity Disorder: Information, Questions, Answers and Recommendations About Self-Demand Amputation*. Bloomington: 1st Books Library.

Fux, M., Levine, J., Aviv, A., *et al.* (1996) Inositol treatment of obsessive-compulsive disorder. *American Journal of Psychiatry*, *153*, 1219–1221.

Fux, M., Benjamin, J., & Belmaker, R.H. (1999) Inositol versus placebo augmentation of serotonin reuptake inhibitors in the treatment of obsessive-compulsive disorder: a double-blind cross-over study. *International Journal of Neuropsychopharmacology*, *2*, 193–195.

Gabbard, G.O. (2001) Psychoanalytically informed approaches to the treatment of obsessive-compulsive disorder. *Psychoanalytic Inquiry*, *21*, 208–221.

Gabriels, L., Cosyns, P., Nuttin, B., *et al.* (2003) Deep brain stimulation for treatment-refractory obsessive-compulsive disorder: psychopathological and neuropsychological outcome in three cases. *Acta Psychiatrica Scandinavica*, *107*, 275–282.

Garvey, M.A., Perlmutter, S.J., Allen, A.J., *et al.* (1999) A pilot study of penicillin prophylaxis for neuropsychiatric exacerbations triggered by streptococcal infections. *Biological Psychiatry*, *45*, 1564–1571.

Geffken, G.R., Pincus, D.B., & Zelikovsky, N. (1999) Obsessive compulsive disorder in children and adolescents: review of background, assessment, and treatment. *Journal of Psychological Practice*, *5*, 15–31.

Geisler, A., & Schou, M. (1969) [Lithium for obsessive-compulsive neuroses: a double-blind therapeutic trial]. [Danish]. *Nordisk Psykiatrisk Tidsskrift*, *23*, 493–495.

Geller, D., Biederman, J., Jones, J., *et al.* (1998) Is juvenile obsessive-compulsive disorder a developmental subtype of the disorder? A review of the pediatric literature. *Journal of the American Academy of Child and Adolescent Psychiatry*, *37*, 420–427.

Geller, D.A., Hoog, S.L., Heiligenstein, J.H., *et al.* (2001) Fluoxetine treatment for obsessive-compulsive disorder in children and adolescents: a placebo-controlled clinical trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, *40*, 773–779.

Geller, D.A., Biederman, J., Stewart, S.E., *et al.* (2003) Impact of comorbidity on treatment response to paroxetine in pediatric obsessive-compulsive disorder: is the use of exclusion criteria empirically supported in randomized clinical trials? *Journal of Child and Adolescent Psychopharmacology*, *13* (Suppl 1), S19–S29.

Geller, D.A., Wagner, K.D., Emslie, G., *et al.* (2004) Paroxetine treatment in children and adolescents with obsessive-compulsive disorder: a randomized, multicenter, double-blind, placebo-controlled trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, *43*, 1387–1396.

George, M.S. (2000) New methods of brain stimulation are improving research and therapy in obsessive-compulsive disorder. *CNS Spectrum*, *5* (6 (Suppl 4)), 12–17.

Geremia, G.M., & Neziroglu, F. (2001) Cognitive therapy in the treatment of body dysmorphic disorder. *Clinical Psychology and Psychotherapy*, *8*, 243–241.

Gilbody, S.M., Whitty, P.M., Grimshaw, J.M., *et al.* (2003) Improving the detection and management of depression in primary care. *Quality and Safety in Health Care*, *12*, 149–155.

Goktepe, E.O., Young, L.B., & Bridges, P.K. (1975) A further review of the results of stereotactic subcaudate tractotomy. *British Journal of Psychiatry*, *126*, 270–280.

Gold-Steinberg, S., & Logan, D. (1999) Integrating play therapy in the treatment of children with obsessive-compulsive disorder. *American Journal of Orthopsychiatry*, *69*, 495–503.

Gomez de Setien, D.I. (1982) Transactional analysis and combined system of therapy for the modification of conduct. *Transactional Analysis Journal*, *12*, 267–269.

Gomez-Perez, J.C., Marks, I.M., & Gutierrez-Fisac, J.L. (1994) Dysmorphophobia: clinical features and outcome with behavior therapy. *European Psychiatry*, *9*, 229–235.

Gomibuchi, T., Gomibuchi, K., Akiyama, T., *et al.* (2000) Obsession of hearing music: from the viewpoint of morita theory. *Psychiatry and Clinical Neurosciences*, *54*, 203–206.

Goodman, R. (2001) Psychometric properties of the strengths and difficulties questionnaire. *Journal of the American Academy of Child and Adolescent Psychiatry*, *40*, 1337–1345.

Goodman, R.W. (1988) Applications of strategic therapy with children. *Journal of Child and Adolescent Psychotherapy*, *5* (1), 11–14.

Goodman, W.K., Price, L.H., Rasmussen, S.A., *et al.* (1989) Efficacy of fluvoxamine in obsessive-compulsive disorder. A double-blind comparison with placebo. *Archives of General Psychiatry*, *46*, 36–44.

Goodman, W.K., Price, L.H., Delgado, P.L., *et al.* (1990) Specificity of serotonin reuptake inhibitors in the treatment of obsessive-compulsive disorder. Comparison of fluvoxamine and desipramine. *Archives of General Psychiatry*, *47*, 577–585.

Goodman, W.K., Kozak, M.J., Liebowitz, M., *et al.* (1996) Treatment of obsessive-compulsive disorder with fluvoxamine: a multicentre, double-blind, placebo-controlled trial. *International Clinical Psychopharmacology*, *11*, 21–29.

Gothelf, D., Aharonovsky, O., Horesh, N., *et al.* (2004) Life events and personality factors in children and adolescents with obsessive-compulsive disorder and other anxiety disorders. *Comprehensive Psychiatry*, *45*, 192–198.

Gournay, K., Veale, D., & Walburn, J. (1997) Body dysmorphic disorder: pilot randomized controlled trial of treatment; implications for nurse therapy research and practice. Including commentary by Newell R and Latham M with author response. *Clinical Effectiveness in Nursing, 1*, 38–46.

Gournay, K., Denford, L., Parr, A.M., *et al.* (2000) British nurses in behavioural psychotherapy: a 25-year follow-up. *Journal of Advanced Nursing*, *32*, 343–351.

Grady, T.A., Pigott, T.A., L'Heureux, F., *et al.* (1993) Double-blind study of adjuvant buspirone for fluoxetine-treated patients with obsessive-compulsive disorder. *American Journal of Psychiatry*, *150*, 819–821.

Grant, J.E. (2001) Successful treatment of nondelusional body dysmorphic disorder with olanzapine: a case report. *Journal of Clinical Psychiatry*, *62*, 297–298.

Grant, J.E., Won Kim, S., & Eckert, E.D. (2002) Body dysmorphic disorder in patients with anorexia nervosa: prevalence, clinical features, and delusionality of body image. *International Journal of Eating Disorders*, *32*, 291–300.

Green, D. (1980) A behavioural approach to the treatment of obsessional rituals: an adolescent case study. *Journal of Adolescence*, *3*, 297–306.

Greenberg, B.D., & Rezai, A.R. (2003) Mechanisms and the current state of deep brain stimulation in neuropsychiatry. *CNS Spectrum*, *8*, 522–526.

Greenberg, B.D., George, M.S., Martin, J.D., *et al.* (1997) Effect of prefrontal repetitive transcranial magnetic stimulation in obsessive-compulsive disorder: a preliminary study. *American Journal of Psychiatry*, *154*, 867–869.

Greist, J.H., Jefferson, J.W., Rosenfeld, R., *et al.* (1990) Clomipramine and obsessive compulsive disorder: a placebo-controlled double-blind study of 32 patients. *Journal of Clinical Psychiatry, 51*, 292–297.

Greist, J.H., Jefferson, J.W., Kobak, K.A., *et al.* (1995) A 1 year double-blind placebo-controlled fixed dose study of sertraline in the treatment of obsessive-compulsive disorder. *International Clinical Psychopharmacology*, *10*, 57–65.

Greist, J.H., Marks, I.M., Baer, L., *et al.* (2002) Behavior therapy for obsessive-compulsive disorder guided by a computer or by a clinician compared with relaxation as a control. *Journal of Clinical Psychiatry*, *63*, 138–145.

Greist, J.H., Bandelow, B., Hollander, E., *et al.* (2003) WCA recommendations for the long-term treatment of obsessive-compulsive disorder in adults. *CNS Spectrum*, *8* (8 (Suppl 1)), 7–16.

Haaga, D.A. (2000) Introduction to the special section on stepped care models in psychotherapy. *Journal of Consulting and Clinical Psychology*, *68*, 547–548.

Hackmann, A., & McLean, C. (1975) A comparison of flooding and thought stopping in the treatment of obsessional neurosis. *Behaviour Research and Therapy, 13*, 263–269.

Hafner, J.R., Gilchrist, P., Bowling, J., *et al.* (1981) The treatment of obsessional neurosis in a family setting. *Australian and New Zealand Journal of Psychiatry*, *15*, 145–151.

Hafner, R.J. (1982) Marital interaction in persisting obsessive-compulsive disorders. *Australian and New Zealand Journal of Psychiatry*, *16*, 171–178.

Hallam, R.S. (1974) Extinction of ruminations: a case study. *Behavior Therapy*, *5*, 565–568.

Hammad, T.A. (2004) Relationship between psychotropic drugs and pediatric suicidality. *http://www.fda.gov/ohrms/dockets/ac/04/briefing/2004–4065b1–10-TAB08-Hammads-Review.pdf*. Center for Drug Evaluation and Research, FDA.

Hand, I. (1988) Obsessive-compulsive patients and their families. In *Handbook of Behavioral Family Therapy* (eds. M. Halloon), pp. 231–256. New York, NY: Guilford Press.

Hanna, G.L., Himle, J.A., Curtis, G.C., *et al.* (2005) A family study of obsessive-compulsive disorder with pediatric probands. *American Journal of Medical Genetics Part B, Neuropsychiatric Genetics: The Official Publication of the International Society of Psychiatric Genetics, 134,* 13–19.

Harbin, H.T. (1979) Cure by ordeal: treatment of an obsessive-compulsive neurotic. *International Journal of Family Therapy, 1*, 324–332.

Harris, C.V. & Wiebe, D.J. (1992) An analysis of response prevention and flooding procedures in the treatment of adolescent obsessive compulsive disorder. *Journal of Behavior Therapy and Experimental Psychiatry*, *23*, 107–115.

Harris, D.L., & Carr, A.T. (2001) Prevalence of concern about physical appearance in the general population. *British Journal of Plastic Surgery*, *54*, 223–226.

Harvey, M.A., & Green, C.L. (1990) Looking into a deaf child's future: a brief treatment approach. *American Annals for the Deaf*, *135*, 364–370.

Hay, P., Sachdev, P., Cumming, S., *et al.* (1993) Treatment of obsessive-compulsive disorder by psychosurgery. *Acta Psychiatrica Scandinavica*, *87*, 197–207.

Hembree, E.A., Riggs, D.S., Kozak, M.J., *et al.* (2003) Long-term efficacy of exposure and ritual prevention therapy and serotonergic medications for obsessive-compulsive disorder. *CNS Spectrum*, *8*, 363–371.

Hesso, R., & Thorell, L.H. (1969) [Lithium treatment for obsessive-compulsive neuroses. A little clinical-therapeutic pilot study of the double-blind cross-over type]. [Swedish]. *Nordisk Psykiatrisk Tidsskrift, 23*, 496–499.

Hettema, J.M., Neale, M.C., & Kendler, K.S. (2001) A review and meta-analysis of the genetic epidemiology of anxiety disorders. *American Journal of Psychiatry*, *158*, 1568–1578.

Hewlett, W.A., Vinogradov, S., & Agras, W.S. (1992) Clomipramine, clonazepam, and clonidine treatment of obsessive-compulsive disorder. *Journal of Clinical Psychopharmacology*, *12*, 420–430.

Hewlett, W.A., Schmid, S.P., & Salomon, R.M. (2003) Pilot trial of ondansetron in the treatment of 8 patients with obsessive-compulsive disorder. *Journal of Clinical Psychiatry*, *64*, 1025–1030.

Heyman, I. (1997) Children with obsessive compulsive disorder. *BMJ*, *315* (7106), 444.

Heyman, I., Fombonne, E., Simmons, H., *et al.* (2001) Prevalence of obsessive compulsive disorder in the British nationwide survey of child mental health. *British Journal of Psychiatry*, *179*, 324–329.

Higgins, J.P.T., & Thompson, S.G. (2002) Qualifying heterogeneity in a meta-analysis. *Statistics in Medicine*, *21*, 1539–1558.

Himle, J.A., van Etten, M., & Fischer, D.J. (2003) Group cognitive behavioral therapy for obsessive-compulsive disorder. *Brief Treatment and Crisis Intervention*, *3*, 217–229.

Hiss, H., Foa, E.B., & Kozak, M.J. (1994) Relapse prevention program for treatment of obsessive-compulsive disorder. *Journal of Consulting and Clinical Psychology*, *62*, 801–808.

Hodgkiss, A.D., Malizia, A.L., Bartlett, J.R., *et al.* (1995) Outcome after the psychosurgical operation of stereotactic subcaudate tractotomy, 1979–1991. *Journal of Neuropsychiatry and Clinical Neurosciences*, *7*, 230–234.

Hoehn-Saric, R., Ninan, P., Black, D.W., *et al.* (2000) Multicenter double-blind comparison of sertraline and desipramine for concurrent obsessive-compulsive and major depressive disorders. *Archives of General Psychiatry*, *57*, 76–82.

Hohagen, F., Winkelmann, G., Rasche-Ruchle, H., *et al.* (1998) Combination of behaviour therapy with fluvoxamine in comparison with behaviour therapy and placebo. Results of a multicentre study. *British Journal of Psychiatry – Supplementum*, *173* (Suppl 35), 71–78.

Hollander, E. (1998) Treatment of obsessive-compulsive spectrum disorders with SSRIs. *British Journal of Psychiatry, 173* (Suppl 35), 7–12.

Hollander, E., & Wong, C.M. (1995) Obsessive-compulsive spectrum disorders. *Journal of Clinical Psychology*, *56* (Suppl 4), 53–55.

Hollander, E., & Wong, C. (1998) Psychosocial functions and economic costs of obsessive compulsive disorder. *CNS Spectrum*, *3* (5), 48–58.

Hollander, E., Liebowitz, M.R., Winchel, R., *et al.* (1989) Treatment of body-dysmorphic disorder with serotonin reuptake blockers. *American Journal of Psychiatry*, *146*, 768–770.

Hollander, E., Stein, D.J., DeCaria, C.M., *et al* (1993) A pilot study of biological predictors of treatment outcome in obsessive-compulsive disorder. *Biological Psychiatry, 33*, 747–749.

Hollander, E., Allen, A., Kwon, J., *et al.* (1999) Clomipramine vs desipramine crossover trial in body dysmorphic disorder: selective efficacy of a serotonin reuptake inhibitor in imagined ugliness. *Archives of General Psychiatry*, *56*, 1033–1039.

Hollander, E., Koran, L.M., Goodman, W.K., *et al.* (2003) A double-blind, placebo-controlled study of the efficacy and safety of controlled-release fluvoxamine in patients with obsessive-compulsive disorder. *Journal of Clinical Psychiatry*, *64*, 640–647.

Hollander, E., Baldini, R.N., Sood, E., *et al.* (2003a) Risperidone augmentation in treatment-resistant obsessive-compulsive disorder: a double-blind, placebo-controlled study. *International Journal of Neuropsychopharmacology*, *6*, 397–401.

Hollander, E., Friedberg, J., Wasserman, S., *et al.* (2003b) Venlafaxine in treatment-resistant obsessive-compulsive disorder. *Journal of Clinical Psychiatry*, *64*, 546–550.

Hollander, E., Kaplan, A., & Stahl, S.M. (2003c) A double-blind, placebo-controlled trial of clonazepam in obsessive-compulsive disorder. *World Journal of Biological Psychiatry*, *4*, 30–34.

Hollander, E., Allen, A., Steiner, M., *et al.* (2003d) Acute and long-term treatment and prevention of relapse of obsessive-compulsive disorder with paroxetine. *Journal of Clinical Psychiatry*, *64*, 1113–1121.

Horowitz, K., Gorfinkle, K., Lewis, O., *et al.* (2002) Body dysmorphic disorder in an adolescent girl. *Journal of the American Academy of Child and Adolescent Psychiatry*, *41*, 1503–1509.

Horwath, E., & Weissman, M.M. (2000) The epidemiology and cross-national presentation of obsessive-compulsive disorder. *Psychiatric Clinics of North America*, *23*, 493–507.

Hughes, A., Fineberg, N., Roberts, A., *et al.* (2004) Group cognitive behaviour therapy in obsessive compulsive disorder (OCD): a controlled study. Unpublished trial.

Husain, M.M., Lewis, S.F., & Thornton, W.L. (1993) Maintenance ECT for refractory obsessive-compulsive disorder. *American Journal of Psychiatry*, *150*, 1899–1900.

Income data services (2004) *http://www.incomesdata.co.uk/index/html.*

Insel, T.R., Alterman, I., & Murphy, D.L. (1982) Antiobsessional and antidepressant effects of clomipramine in the treatment of obsessive-compulsive disorder. *Psychopharmacology Bulletin, 18*, 115–117.

Insel, T.R., Murphy, D.L., Cohen, R.M., *et al.* (1983) Obsessive-compulsive disorder: a double-blind trial of clomipramine and clorgyline. *Archives of General Psychiatry*, *40*, 605–612.

Insel, T.R., Mueller, E.A., Alterman, I., *et al.* (1985) Obsessive-compulsive disorder and serotonin: is there a connection? *Biological Psychiatry, 20*, 1174–1188.

Ishigooka, J., Mitsuhiro, I., Makihiko, S., *et al.* (1998) Demographic features of patients seeking cosmetic surgery. *Psychiatry and Clinical Neuroscience*, *52*, 283–287.

Issakidis, C., Sanderson, K., Corry, J., *et al.* (2004) Modelling the population cost-effectiveness of current and evidence-based optimal treatment for anxiety disorders. *Psychological Medicine*, *34*, 19–35.

Iyasu, S. (2004) Report of the Audit of the Columbia Suicidality Classification Methodology. *http://www.fda.gov/ohrms/dockets/ac/04/briefing/2004-4065b1-09-TAB07-Iyasu-Audit_report.htm*.

Jadad, A.R., Moore, R.A., Carroll, D., *et al.* (1996) Assessing the quality of reports of randomised clinical trials: is blinding necessary? *Controlled Clinical Trials*, *17*, 1–12.

Jenike, M.A. (1984) A case report of successful treatment of dysmorphophobia with tranylcypromine. *American Journal of Psychiatry*, *141*, 1463–1464.

Jenike, M.A., Hyman, S., Baer, L., *et al.* (1990a) A controlled trial of fluvoxamine in obsessive-compulsive disorder: implications for a serotonergic theory. *American Journal of Psychiatry*, *147*, 1209–1215.

Jenike, M.A., Baer, L., Summergrad, P., *et al.* (1990b) Sertraline in obsessive-compulsive disorder: a double-blind comparison with placebo. *American Journal of Psychiatry*, *147*, 923–928.

Jenike, M.A., Baer, L., Ballantine, T., *et al.* (1991) Cingulotomy for refractory obsessive-compulsive disorder. A long-term follow-up of 33 patients. *Archives of General Psychiatry*, *48*, 548–555.

Jenike, M.A., Baer, L., Minichiello, W.E., *et al.* (1997) Placebo-controlled trial of fluoxetine and phenelzine for obsessive-compulsive disorder. *American Journal of Psychiatry*, *154*, 1261–1264.

Jianxun, L., Hu, X., & Haiying, D. (1998) Clinical controlled study of paroxetine and clomipramine in treatment of obsessive-compulsive disorder. [Chinese]. *Chinese Journal of Psychiatry, 31*, 215–217.

Johnson, G.M., & Hallenbeck, C.E. (1985) A case of obsessional fears treated by brief hypno-imagery intervention. *American Journal of Clinical Hypnosis*, *27*, 232–236.

Jones, M.K., & Menzies, R.G. (1998) Danger ideation reduction therapy (DIRT) for obsessive-compulsive washers. A controlled trial. *Behaviour Research and Therapy*, *36*, 959–970.

Jordan, J., Joyce, P.R., Carter, F.A., *et al.* (2003) Anxiety and psychoactive substance use disorder comorbidity in anorexia nervosa or depression. *International Journal of Eating Disorders*, *34*, 211–219.

Juni, S. (1987) From the analysis of an obsessive hummer: theoretical and clinical implications. *Psychoanalytic Review*, *74*, 63–81.

Kamijima, K., Murasaki, M., Asai, M., *et al.* (2004) Paroxetine in the treatment of obsessive-compulsive disorder: randomized, double-blind, placebo-controlled study in Japanese patients. *Psychiatry and Clinical Neuroscience*, *58*, 427–433.

Karabanow, O. (1977) Double-blind controlled study in phobias and obsessions. *Journal of International Medical Research, 5*, 42–48.

Karush, R.K. (1998) The use of dream analysis in the treatment of a nine-year-old obsessional boy. *Psychoanalytic Study of the Child*, *53*, 199–211.

Kasvikis, Y., & Marks, I.M. (1988) Clomipramine in obsessive-compulsive ritualisers treated with exposure therapy: relations between dose, plasma levels, outcome and side effects. *Psychopharmacology*, 95, 113–118.

Kasvikis, Y., & Marks, I.M. (1988a) Clomipramine, self-exposure, and therapist-accompanied exposure in obsessive-compulsive ritualizers: two-year follow-up. *Journal of Anxiety Disorders, 2*, 291–298.

Katz, R.J., DeVeaugh-Geiss, J., & Landau, P. (1990) Clomipramine in obsessive-compulsive disorder. *Biological Psychiatry*, *28*, 401–414.

Kay, J. (1996) Is psychoanalytic psychotherapy relevant to the treatment of OCD? *Journal of Psychotherapy Practice and Research*, *5*, 341–354.

Kazarian, S.S., & Evans, D.R. (1977) Modification of obsessional ruminations: a comparative study. *Canadian Journal of Behavioural Science, 9*, 91–100.

Kearney, C.A., & Silverman, W.K. (1990) Treatment of an adolescent with obsessive-compulsive disorder by alternating response prevention and cognitive therapy: an empirical analysis. *Journal of Behavior Therapy and Experimental Psychiatry*, *21*, 39–47.

Keijsers, G.P., Hoogduin, C.A., & Schaap, C.P. (1994) Predictors of treatment outcome in the behavioural treatment of obsessive-compulsive disorder. *British Journal of Psychiatry*, *165*, 781–786.

Keiley, M.K. (2002) Affect regulation and attachment focused treatment of a husband with obsessive-compulsive disorder and his wife. *Journal of Couple and Relationship Therapy*, *1*, 25–44.

Kellerman, J. (1981) Hypnosis as an adjunct to thought-stopping and covert reinforcement in the treatment of homicidal obsessions in a twelve-year-old boy. *International Journal of Clinical and Experimental Hypnosis*, *29*, 128–135.

Kenwright, M., Marks, I., Graham, C., *et al.* (2005) Brief scheduled phone support from a clinician to enhance computer-aided self-help for obsessive-compulsive disorder: randomised controlled-trial. *Journal of Clinical Psychology, 61,* 1499–1508.

Keuler, D.J., Altemus, M., Michelson, D., *et al.* (1996) Behavioural effects of naloxone infusion in obsessive-compulsive disorder. *Biological Psychiatry, 40*, 154–156.

Khan, A., Leventhal, R.M., Khan, S., *et al.* (2002) Suicide risk in patients with anxiety disorders: a meta-analysis of the FDA database. *Journal of Affective Disorders*, *68*, 183–190.

Khanna, S., Rajendra, P.N., & Channabasavanna, S.M. (1988) Clomipramine in resistant obsessive compulsive disorder. *Indian Journal of Psychiatry*, *30*, 375–379.

Khanna, S., Gangadhar, B.N., Sinha, V., *et al.* (1988a) Electroconvulsive therapy in obsessive-compulsive disorder. *Convulsive Therapy*, *4*, 314–320.

Khanna, S., Rajendra, P.N., & Channabasavanna, S.M. (1988b) Life events and onset of obsessive compulsive disorder. *International Journal of Social Psychiatry*, *34*, 305–309.

Kiloh, L.G., Smith, J.S., & Johnston, G.F. (1988) Psychosurgery (limbic surgery). In *Physical Treatments in Psychiatry* (eds. L.G. Kiloh, J.S. Smith, & G.F. Johnston), pp. 277–333. Melbourne: Blackwell.

Kim, C.H., Chang, J.W., Koo, M.S., *et al.* (2003) Anterior cingulotomy for refractory obsessive-compulsive disorder. *Acta Psychiatrica Scandinavica*, *107*, 283–290.

Kim, S.W., Dysken, M.W., Kushner, M.G., *et al.* (1997) Phenomenological and pharmacological study of provoked obsessive/anxiety symptoms in obsessive-compulsive disorder: a preliminary study. *Biological Psychiatry, 42*, 969–975.

King, R.A., & Scahill, L. (1999) The assessment and coordination of treatment of children and adolescents with OCD. *Child and Adolescent Psychiatric Clinics of North America*, *8*, 577–597.

King, R.A., Leonard, H., March, J., *et al.* (1998) Summary of the practice parameters for the assessment and treatment of children and adolescents with obsessive-compulsive disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *37*, 1110–1116.

Klassen, A., Jenkinson, C., Fitzpatrick, R., *et al.* (1996) Patients' health related quality of life before and after aesthetic surgery. *British Journal of Plastic Surgery*, *49*, 433–438.

Klerman, G.L., Weissman, M.M., Rounsaville, B.J., *et al.* (1984) *Interpersonal Psychotherapy of Depression*. New York: Basic Books.

Knesevich, J.W. (1982) Successful treatment of obsessive-compulsive disorder with clonidine hydrochloride. *American Journal of Psychiatry*, *139*, 364–365.

Knight, G. (1969) Stereotactic surgery for the relief of suicidal and severe depression and intractable psychoneurosis. *Postgraduate Medical Journal*, *45*, 1–13.

Knox, L.S., Albano, A.M., & Barlow, D.H. (1996) Parental involvement in the treatment of childhood obsessive compulsive disorder: a multiple-baseline examination incorporating parents. *Behavior Therapy*, *27*, 93–115.

Kobak, K.A., Taylor, L.H., Dottl, S.L., *et al.* (1997) A computer-administered telephone interview to identify mental disorders. *Journal of the American Medical Association*, *278*, 905–910.

Kobak, K.A., Greist, J.H., Jefferson, J.W., *et al.* (1998) Behavioral versus pharmacological treatments of obsessive compulsive disorder: a meta-analysis. *Psychopharmacology*, *136*, 205–216.

Koran, L.M. (2000) Quality of life in obsessive-compulsive disorder. *Psychiatric Clinics of North America*, *23*, 509–517.

Koran, L.M., Cain, J.W., Dominguez, R.A., *et al.* (1996) Are fluoxetine plasma levels related to outcome in obsessive-compulsive disorder? *American Journal of Psychiatry, 153*, 1450–1454.

Koran, L.M., McElroy, S.L., Davidson, J.R., *et al.* (1996a) Fluvoxamine versus clomipramine for obsessive-compulsive disorder: a double-blind comparison. *Journal of Clinical Psychopharmacology*, *16*, 121–129.

Koran, L.M., Sallee, F.R., & Pallanti, S. (1997) Rapid benefit of intravenous pulse loading of clomipramine in obsessive-compulsive disorder. *American Journal of Psychiatry*, *154*, 396–401.

Koran, L.M., Ringold, A.L., & Elliott, M.A. (2000) Olanzapine augmentation for treatment-resistant obsessive-compulsive disorder. *Journal of Clinical Psychiatry*, *61*, 514–517.

Koran, L.M., Pallanti, S., & Quercioli, L. (2001) Sumatriptan, 5-HT(1D) receptors and obsessive-compulsive disorder. *European Neuropsychopharmacology*, *11*, 169–172.

Koran, L.M., Hackett, E., Rubin, A., *et al.* (2002) Efficacy of sertraline in the long-term treatment of obsessive-compulsive disorder. *American Journal of Psychiatry*, *159*, 88–95.

Kozak, M.J., Leibowitz, M.R., & Foa, E.B. (2000) Cognitive behaviour therapy and pharmacotherapy for obsessive-compulsive disorder: the NIMH-sponsored collaborative study. In *Obsessive-Compulsive Disorder: Contemporary Issues in Treatment* (eds. W.K. Goodman, M.V. Rudorfer, & J.D. Maser), pp. 501–530. Mahwah, New Jersey: Lawrence Erlbaum Associates.

Krochmalik, A., Jones, M.K., & Menzies, R.G. (2001) Danger ideation reduction therapy (DIRT) for treatment-resistant compulsive washing. *Behaviour Research and Therapy*, *39*, 897–912

Krone, K.P., Himle, J.A., & Nesse, R.M. (1991) A standardized behavioral group treatment program for obsessive-compulsive disorder: preliminary outcomes. *Behaviour Research and Therapy*, *29*, 627–631.

Kronig, M.H., Apter, J., Asnis, G., *et al.* (1999) Placebo-controlled, multicenter study of sertraline treatment for obsessive-compulsive disorder. *Journal of Clinical Psychopharmacology*, *19*, 172–176.

Laidlaw, T.M., Falloon, I.R., Barnfather, D., *et al.* (1999) The stress of caring for people with obsessive compulsive disorders. *Community Mental Health Journal*, *35*, 443–450.

Laitinen, L.V., & Singounas, E. (1988) Intraoperative electrical stimulation of the brain in patients with obsessive-compulsive neurosis. *Applied Neurophysiology*, *51*, 317–323.

Lang, H. (1997) Obsessive-compulsive disorders in neurosis and psychosis. *Journal of the American Academy of Psychoanalysis*, *25*, 143–150.

LaSalle, V.H., Cromer, K.R., Nelson, K.N., *et al.* (2004) Diagnostic interview assessed neuropsychiatric disorder comorbidity in 334 individuals with obsessive-compulsive disorder. *Depression and Anxiety*, *19*, 163–173.

Last, C.G. & Strauss, C.C. (1989) Obsessive-compulsive disorder in childhood. *Journal of Anxiety Disorders, 3,* 295–302.

Lax, T., Basoglu, M., & Marks, I.M. (1992) Expectancy and compliance as predictors of outcome in obsessive-compulsive disorder. *Behavioural Psychotherapy*, *20*, 257–266.

Lecrubier, Y., Sheehan, D., Weiller, E., *et al.* (1997) The MINI International Neuropsychiatric Interview (M.I.N.I.). A Short Diagnostic Structured Interview: Reliability and Validity According to the CIDI. *European Psychiatry*, *12*, 224–231.

Leib, P.T. (2001) Integrating behavior modification and pharmacotherapy with the psychoanalytic treatment of obsessive-compulsive disorder: a case study. *Psychoanalytic Inquiry*, *21*, 222–241.

Leon, A.C., Portera, L., & Weissman, M.M. (1995) The social costs of anxiety disorders. *British Journal of Psychiatry*, *166* (Suppl 27), 19–22.

Leonard, H.L., Swedo, S.E., Rapoport, J.L., *et al.* (1989) Treatment of obsessive-compulsive disorder with clomipramine and desipramine in children and adolescents. A double-blind crossover comparison. *Archives of General Psychiatry*, *46*, 1088–1092.

Leonard, H.L., Swedo, S.E., Lenane, M.C., *et al.* (1991) A double-blind desipramine substitution during long-term clomipramine treatment in children and adolescents with obsessive-compulsive disorder. *Archives of General Psychiatry*, *48*, 922–927.

Leonard, H.L., Lenane, M.C., Swedo, S.E., *et al.* (1992) Tics and Tourette's disorder: A 2- to 7-year follow-up of 54 obsessive-compulsive children. *American Journal of Psychiatry, 149,* 1244–1251.

Leonard, H.L., Swedo, S.E., Lenane, M.C., *et al.* (1993) A 2- to 7-year follow-up study of 54 obsessive-compulsive children and adolescents. *Archives of General Psychiatry*, *50*, 429–439.

Leonard, H.L., Meyer, M.C., Swedo, S.E., *et al.* (1995) Electrocardiographic changes during desipramine and clomipramine treatment in children and adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry, 34*, 1460–1468.

Liebowitz, M.R., Turner, S.M., Piacentini, J., *et al.* (2002) Fluoxetine in children and adolescents with OCD: a placebo-controlled trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, *41*, 1431–1438.

Lin, H.N., & Chen, C.C. (1979) A double-blind test on the effect of bromazepam in obsessive-compulsive neurosis. *Taiwan i Hsueh Hui Tsa Chih – Journal of the Formosan Medical Association, 78*, 267–275.

Lindsay, M., Crino, R., & Andrews, G. (1997) Controlled trial of exposure and response prevention in obsessive-compulsive disorder. *British Journal of Psychiatry*, *171*, 135–139.

Lippitz, B., Mindus, P., Meyerson, B.A., *et al.* (1997) Obsessive compulsive disorder and the right hemisphere: topographic analysis of lesions after anterior capsulotomy performed with thermocoagulation. *Acta Neurochirurgica – Supplementum*, *68*, 61–63.

Lippitz, B.E., Mindus, P., Meyerson, B.A., *et al.* (1999) Lesion topography and outcome after thermocapsulotomy or gamma knife capsulotomy for obsessive-compulsive disorder: relevance of the right hemisphere. *Neurosurgery*, *44*, 452–458.

Lochner, C., & Stein, D.J. (2003) Heterogeneity of obsessive-compulsive disorder: a literature review. *Harvard Review of Psychiatry*, *11*, 113–132.

Lochner, C., Mogotsi, M., du Toit, P.L., *et al.* (2003) Quality of life in anxiety disorders: a comparison of obsessive-compulsive disorder, social anxiety disorder, and panic disorder. *Psychopathology*, *36*, 255–262.

Lopez-Ibor, J.J., Jr., Saiz, J., Cottraux, J., *et al.* (1996) Double-blind comparison of fluoxetine versus clomipramine in the treatment of obsessive compulsive disorder. *European Neuropsychopharmacology*, *6*, 111–118.

Lovell, K., & Richards, D.A. (2000) Multiple access points and levels of entry (MAPLE): ensuring choice, accessibility and equity for CBT services. *Behavioural and Cognitive Psychotherapy*, *28*, 379–392.

Lovell, K., Marks, I.M., Noshirvani, H., *et al.* (1994) Should treatment distinguish anxiogenic from anxiolytic obsessive-compulsive ruminations? Results of a pilot controlled study and of a clinical audit. *Psychotherapy and Psychosomatics*, *61*, 150–155.

Lovell, K., Fullalove, L., Garvey, R., *et al.* (2000) Telephone treatment of obssessive-compulsive disorder. *Behavioural and Cognitive Psychotherapy*, *28*, 87–91.

Lovell, K., Cox, D., Garvey, R., *et al.* (2003) Agoraphobia: nurse therapist-facilitated self-help manual. *Journal of Advanced Nursing*, *43*, 623–630.

Lovell, K., Ekers, D., Fullford, J., *et al.* (2005) A pilot study of a self-help manual with minimal therapist contact in the treatment of obsessive-compulsive disorder. *Clinical Effectiveness in Nursing, 8,* 122–127.

Luo, F., Leckman, J.F., Katsovich, L., *et al.* (2004) Prospective longitudinal study of children with tic disorders and/or obsessive-compulsive disorder: relationship of symptom exacerbations to newly acquired streptococcal infections. *Pediatrics*, *113*, e578–e585.

McCarter, R.H. (1997) Directive activity and repair of the self in the cognitive behavioral treatment of obsessive compulsive disorder: a case example. *Journal of Psychotherapy Integration*, *7*, 75–88.

McDougle, C.J., & Walsh, K.H. (2001) Treatment of refractory OCD. In *Obsessive Compulsive Disorder: A Practical Guide* (eds. N.A. Fineberg, D. Marazziti, & D. Stein), pp. 135–152. London: Martin Dunitz.

McDougle, C.J., Goodman, W.K., Price, L.H., *et al.* (1990) Neuroleptic addition in fluvoxamine-refractory obsessive-compulsive disorder. *American Journal of Psychiatry*, *147*, 652–654.

McDougle, C.J., Price, L.H., Goodman, W.K., *et al.* (1991) A controlled trial of lithium augmentation in fluvoxamine-refractory obsessive-compulsive disorder: Lack of efficacy. *Journal of Clinical Psychopharmacology*, *11*, 175–184.

McDougle, C.J., Goodman, W.K., Leckman, J.F., *et al.* (1993) Limited therapeutic effect of addition of buspirone in fluvoxamine-refractory obsessive-compulsive disorder. *American Journal of Psychiatry*, *150*, 647–649.

McDougle, C.J., Goodman, W.K., Leckman, J.F., *et al.* (1994) Haloperidol addition in fluvoxamine-refractory obsessive-compulsive disorder: a double-blind, placebo-controlled study in patients with and without tics. *Archives of General Psychiatry*, *51*, 302–308.

McDougle, C.J., Barr, L.C., Goodman, W.K., *et al.* (1995a) Lack of efficacy of clozapine monotherapy in refractory obsessive-compulsive disorder. *American Journal of Psychiatry*, *152*, 1812–1814.

McDougle, C.J., Fleischmann, R.L., Epperson, C.N., *et al.* (1995b) Risperidone addition in fluvoxamine-refractory obsessive-compulsive disorder: three cases. *Journal of Clinical Psychiatry*, *56*, 526–528.

McDougle, C.J., Kresch, L.E., Goodman, W.K., *et al.* (1995c) A case-controlled study of repetitive thoughts and behavior in adults with autistic disorder and obsessive-compulsive disorder. *American Journal of Psychiatry*, *152*, 772–777.

McDougle, C.J., Epperson, C.N., Pelton, G.H., *et al.* (2000a) A double-blind, placebo-controlled study of risperidone addition in serotonin reuptake inhibitor-

refractory obsessive-compulsive disorder. *Archives of General Psychiatry*, *57*, 794–801.

McDougle, C.J., Kresch, L.E., & Posey, D.J. (2000b) Repetitive thoughts and behavior in pervasive developmental disorders: treatment with serotonin reuptake inhibitors. *Journal of Autism and Developmental Disorders*, *30*, 427–435.

McKay, D. (1999) Two-year follow-up of behavioral treatment and maintenance for body dysmorphic disorder. *Behavior Modification*, *23*, 620–629.

McKay, D., Neziroglu, F., Todaro, J.F., *et al.* (1996) Changes in personality disorders following behaviour therapy for obsessive-compulsive disorder. *Journal of Anxiety Disorders*, *10*, 47–57.

McKay, D., Todaro, J., Neziroglu, F., *et al.* (1997) Body dysmorphic disorder: a preliminary evaluation of treatment and maintenance using exposure and response prevention. *Behaviour Research and Therapy*, *35*, 67–70.

McLean, P.D., Whittal, M.L., Thordarson, D.S., *et al.* (2001) Cognitive versus behavior therapy in the group treatment of obsessive-compulsive disorder. *Journal of Consulting and Clinical Psychology*, *69*, 205–214.

Magliano, L., Tosini, P., Guarneri, M., *et al.* (1996) Burden on the families of patients with obsessive-compulsive disorder: a pilot study. *European Psychiatry*, *11*, 192–197.

Maina, G., Albert, U., Ziero, S., *et al.* (2003) Antipsychotic augmentation for treatment resistant obsessive-compulsive disorder: what if antipsychotic is discontinued? *International Clinical Psychopharmacology*, *18*, 23–28.

Maletzky, B., McFarland, B., & Burt, A. (1994) Refractory obsessive compulsive disorder and ECT. *Convulsive Therapy*, *10*, 34–42.

Mallery, E. (1996) *The Subjective Experience of OCD. A Questionnaire-Based Study from Members of the Self-Help Organisation Obsessive Action.* London: Conway Hall.

Mallya, G.K., White, K., Waternaux, C., *et al.* (1992) Short- and long-term treatment of obsessive-compulsive disorder with fluvoxamine. *Annals of Clinical Psychiatry*, *4*, 77–80.

Mann, T. (1996) *Clinical Guidelines: Using Clinical Guidelines to Improve Patient Care Within the NHS*. London: Department of Health.

Marazziti, D., Pfanner, C., Palego, L., *et al.* (1997) Changes in platelet markers of obsessive-compulsive patients during a double-blind trial of fluvoxamine versus clomipramine. *Pharmacopsychiatry, 30*, 245–249.

March, J.S. (1995) Cognitive-behavioral psychotherapy for children and adolescents with OCD: a review and recommendations for treatment. *Journal of the American Academy of Child and Adolescent Psychiatry*, *34*, 7–18.

March, J.S. & Mulle, K. (1995) Manualized cognitive-behavioral psychotherapy for obsessive-compulsive disorder in childhood: a preliminary single case study. *Journal of Anxiety Disorders*, *9*, 175–184.

March, J.S. & Mulle, K. (1996) Banishing OCD: cognitive-behavioral psychotherapy for obsessive-compulsive disorders. In *Psychosocial Treatments for Child and Adolescent Disorders. Empirically Based Strategies for Clinical Practice* (eds.

E.D. Hibbs & P.S. Jensen), pp. 83–102. Washington, DC: American Psychological Association.

March, J.S. & Mulle, K. (1998) *OCD in Children and Adolescents: A Cognitive-Behavioural Treatment Manual*. New York: Guilford.

March, J.S., Johnston, H., Jefferson, J.W., *et al.* (1990) Do subtle neurological impairments predict treatment resistance to clomipramine in children and adolescents with obsessive-compulsive disorder? *Journal of Child and Adolescent Psychopharmacology*, *1*, 133–140.

March, J.S., Mulle, K., & Herbel, B. (1994) Behavioral psychotherapy for children and adolescents with obsessive-compulsive disorder: an open trial of a new protocol-driven treatment package. *Journal of the American Academy of Child and Adolescent Psychiatry*, *33*, 333–341.

March, J.S., Frances, A., Carpenter, D., *et al.* (1997) The expert consensus guideline series: treatment of obsessive-compulsive disorder. *Journal of Clinical Psychiatry*, *58* (Suppl 4), 13–72.

March, J.S., Biederman, J., Wolkow, R., *et al.* (1998) Sertraline in children and adolescents with obsessive-compulsive disorder: a multicenter randomized controlled trial. *Journal of the American Medical Association*, *280*, 1752–1756.

March, J.S., Franklin, M., Nelson, A., *et al.* (2001) Cognitive-behavioral psychotherapy for pediatric obsessive-compulsive disorder. *Journal of Clinical Child Psychology*, *30*, 8–18.

Marks, I. (1997) Behaviour therapy for obsessive-compulsive disorder: a decade of progress. *Canadian Journal of Psychiatry*, *42*, 1021–1027.

Marks, I., & Mishan, J. (1988) Dysmorphophobic avoidance with disturbed bodily perception: a pilot study of exposure therapy. *British Journal of Psychiatry*, *152*, 674–678.

Marks, I.M., Hodgson, R., & Rachman, S. (1975) Treatment of chronic obsessive-compulsive neurosis by in-vivo exposure: a two-year follow-up and issues in treatment. *British Journal of Psychiatry*, *127*, 349–364.

Marks, I.M., Stern, R.S., Mawson, D., *et al.* (1980) Clomipramine and exposure for obsessive-compulsive rituals: i. *British Journal of Psychiatry*, *136*, 1–25.

Marks, I.M., Lelliott, P., Basoglu, M., *et al.* (1988) Clomipramine, self-exposure and therapist-aided exposure for obsessive-compulsive rituals. *British Journal of Psychiatry, 152*, 522–534.

Martin, J.L., Barbanoj, M.J., Perez, V., *et al.* (2003) Transcranial magnetic stimulation for the treatment of obsessive-compulsive disorder. *Cochrane Database of Systematic Reviews*, *3*, CD003387.

Mataix-Cols, D., Rauch, S.L., Manzo, P.A., *et al.* (1999) Use of factor-analyzed symptom dimensions to predict outcome with serotonin reuptake inhibitors and placebo in the treatment of obsessive-compulsive disorder. *American Journal of Psychiatry*, *156*, 1409–1416.

Mataix-Cols, D., Marks, I.M., Greist, J.H., *et al.* (2002a) Obsessive-compulsive symptom dimensions as predictors of compliance with and response to behaviour therapy: results from a controlled trial. *Psychotherapy and Psychosomatics*, *71*, 255–262.

Mataix-Cols, D., Rauch, S.L., Baer, L., *et al.* (2002b) Symptom stability in adult obsessive-compulsive disorder: data from a naturalistic two-year follow-up study. *American Journal of Psychiatry*, *159*, 263–268.

Matthews, K., & Eljamel, M.S. (2003) Status of neurosurgery for mental disorder in Scotland. Selective literature review and overview of current clinical activity. *British Journal of Psychiatry*, *182*, 404–411.

Mavissakalian, M., & Michelson, L. (1983) Tricyclic antidepressants in obsessive-compulsive disorder: antiobsessional or antidepressant agents? *Journal of Nervous and Mental Disease, 171*, 301–306.

Mavissakalian, M., Turner, S.M., Michelson, L., *et al.* (1986) The relationship of antiobsessional and antidepressant effects of tricyclic antidepressants. *Psychopharmacology Bulletin, 22*, 161–166.

Mawson, D., Marks, I.M., & Ramm, L. (1982) Clomipramine and exposure for chronic obsessive-compulsive rituals: III. Two year follow-up and further findings. *British Journal of Psychiatry, 140*, 11–18.

Mehta, M. (1990) A comparative study of family-based and patient-based behavioural management in obsessive-compulsive disorder. *British Journal of Psychiatry*, *157*, 133–135.

Mellman, L.A., & Gorman, J.M. (1984) Successful treatment of obsessive-compulsive disorder with ECT. *American Journal of Psychiatry*, *141*, 596–597.

Mendez, M.F., Perryman, K.M., Miller, B.L., *et al.* (1997) Compulsive behaviors as presenting symptoms of frontotemporal dementia. *Journal of Geriatric Psychiatry and Neurology*, *10*, 154–157.

Metin, O., Yazici, K., Tot, S., *et al.* (2003) Amisulpiride augmentation in treatment resistant obsessive-compulsive disorder: an open trial. *Human Psychopharmacology*, *18*, 463–467.

Meyer, V. (1966) Modification of expectations in cases with obsessional rituals. *Behaviour Research and Therapy*, *4*, 273–280.

Milanfranchi, A., Ravagli, S., Lensi, P., *et al.* (1997) A double-blind study of fluvoxamine and clomipramine in the treatment of obsessive-compulsive disorder. *International Clinical Psychopharmacology*, *12*, 131–136.

Mindus, P., Rasmussen, S.A., & Lindquist, C. (1994) Neurosurgical treatment for refractory obsessive-compulsive disorder: implications for understanding frontal lobe function. *Journal of Neuropsychiatry and Clinical Neurosciences*, *6*, 467–477.

Mindus, P., Edman, G., & Andreewitch, S. (1999) A prospective, long-term study of personality traits in patients with intractable obsessional illness treated by capsulotomy. *Acta Psychiatrica Scandinavica*, *99*, 40–50.

Mitchell-Heggs, N., Kelly, D., & Richardson, A. (1976) Stereotactic limbic leucotomy: a follow-up at 16 months. *British Journal of Psychiatry*, *128*, 226–240.

Mitzman, S., & Duigan, I. (1993) One man's group: brief cognitive-analytic group therapy and the use of sequential diagrammatic reformulation. *Counselling Psychology Quarterly*, *6*, 183–192.

Mohr, N., Vythilingum, B., Emsley, R.A., *et al.* (2002) Quetiapine augmentation of serotonin reuptake inhibitors in obsessive-compulsive disorder. *International Clinical Psychopharmacology*, *17*, 37–40.

Monteiro, W.O., Noshirvani, H.F., Marks, I.M., *et al.* (1987) Anorgasmia from clomipramine in obsessive-compulsive disorder. A controlled trial. *British Journal of Psychiatry, 151*, 107–112.

Monteleone, P., Catapano, F., Di Martino, S., *et al.* (1997) Prolactin response to d-fenfluramine in obsessive-compulsive patients, and outcome of fluvoxamine treatment. *British Journal of Psychiatry, 170*, 554–557.

Montgomery, S.A. (1980) Clomipramine in obsessional neurosis: a placebo-controlled trial. *Pharmacological Medicine*, *1*, 189–192.

Montgomery, S.A. (1993) Obsessive compulsive disorder is not an anxiety disorder. *International Clinical Psychopharmacology, 8* (Suppl 1), 57–62.

Montgomery, S.A., Montgomery, D.B., & Fineberg, N. (1990) Early response with clomipramine in obsessive compulsive disorder: a placebo controlled study. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 14*, 719–727.

Montgomery, S.A., McIntyre, A., Osterheider, M., *et al.* (1993) A double-blind, placebo-controlled study of fluoxetine in patients with DSM-III-R obsessive-compulsive disorder. *European Neuropsychopharmacology, 3*, 143–152.

Montgomery, S.A., Kasper, S., Stein, D.J., *et al.* (2001) Citalopram 20 mg, 40 mg and 60 mg are all effective and well tolerated compared with placebo in obsessive-compulsive disorder. *International Clinical Psychopharmacology*, *16*, 75–86.

Montoya, A., Weiss, A.P., Price, B.H., *et al.* (2002) Magnetic resonance imaging-guided stereotactic limbic leukotomy for treatment of intractable psychiatric disease. *Neurosurgery*, *50*, 1043–1052.

Moore, K.A., & Burrows, G.D. (1991) Hypnosis in the treatment of obsessive-compulsive disorder. *Australian Journal of Clinical and Experimental Hypnosis*, *19*, 63–75.

Morelli, G. (1983) Adolescent compulsion: a case study involving cognitive-behavioral treatment. *Psychological Reports*, *53*, 519–522.

Moritz, E.K. (1998) *Behavior Therapy in Game Format for the Treatment of Childhood Obsessive Compulsive Disorder.* PhD Thesis, Hofstra University, Hempstead, New York.

Morphy, R. (1980) An inner view of obsessional neurosis. *Gestalt Journal*, *3*, 120–136.

Muller, N., Putz, A., Kathmann, N., *et al.* (1997) Characteristics of obsessive-compulsive symptoms in Tourette's syndrome, obsessive-compulsive disorder, and Parkinson's disease. *Psychiatry Research*, *70*, 105–114.

Mundo, E., Bellodi, L., & Smeraldi, E. (1995a) Effects of acute intravenous clomipramine on obsessive-compulsive symptoms and response to chronic treatment. *Biological Psychiatry, 38*, 525–531.

Mundo, E., Bareggi, S.R., Pirola, R., *et al.* (1997) Long-term pharmacotherapy of obsessive-compulsive disorder: a double-blind controlled study. *Journal of Clinical Psychopharmacology, 17*, 4–10.

Mundo, E., Bianchi, L., & Bellodi, L. (1997a) Efficacy of fluvoxamine, paroxetine, and citalopram in the treatment of obsessive-compulsive disorder: a single-blind study. *Journal of Clinical Psychopharmacology*, *17*, 267–271.

Mundo, E., Guglielmo, E., & Bellodi, L. (1998) Effect of adjuvant pindolol on the antiobsessional response to fluvoxamine: a double-blind, placebo-controlled study. *International Clinical Psychopharmacology*, *13*, 219–224.

Mundo, E., Bareggi, S.R., Pirola, R., *et al.* (1999) Effect of acute intravenous clomipramine and antiobsessional response to proserotonergic drugs: is gender a predictive variable? *Biological Psychiatry*, *45*, 290–294.

Mundo, E., Rouillon, F., Figuera, M.L., *et al.* (2001) Fluvoxamine in obsessive-compulsive disorder: similar efficacy but superior tolerability in comparison with clomipramine. *Human Psychopharmacology*, *16*, 461–468.

Munjack, D.J. (1978) The behavioral treatment of dysmorphophobia. *Journal of Behavior Therapy and Experimental Psychiatry*, *9*, 53–56.

National Collaborating Centre for Mental Health (2003) *Schizophrenia: Core Interventions in the Treatment and Management of Schizophrenia in Primary and Secondary Care*. London: Gaskell & the British Psychological Society.

National Collaborating Centre for Mental Health (2004) *Eating Disorders: Core Interventions in the Treatment and Management of Anorexia Nervosa, Bulimia Nervosa, and Related Eating Disorders*. London & Leicester: Gaskell & the British Psychological Society.

National Collaborating Centre for Mental Health (2005a) *Depression: Management of Depression in Primary and Secondary Care*. London & Leicester: Gaskell & the British Psychological Society.

National Collaborating Centre for Mental Health (2005b) *Depression in Children and Young People: Identification and Management in Primary, Community and Secondary Care*. Leicester: British Psychological Society & the Royal College of Psychiatrists.

Nestadt, G., Addington, A., Samuels, J., *et al.* (2003) The identification of OCD: related subgroups based on comorbidity. *Biological Psychiatry*, *53*, 914–920.

Nestadt, G., Addington, A., Samuels, J., *et al.* (2004) The identification of OCD-related subgroups based on comorbidity: reply. *Biological Psychiatry*, *55*, 960–961.

Netten, A., & Curtis, L. (2003) *Unit Costs of Health and Social Care.* Canterbury: University of Kent Personal Social Services Research Unit.

Newell, R., & Shrubb, S. (1994) Attitude change and behaviour therapy in body dysmorphic disorder: two case reports. *Behavioural and Cognitive Psychotherapy*, *22*, 163–169.

Newth, S., & Rachman, S. (2001) The concealment of obsessions. *Behaviour Research and Therapy*, *39*, 457–464.

Neziroglu, F., & Khemlani-Patel, S. (2002) A review of cognitive and behavioural treatment for body dysmorphic disorder. *CNS Spectrum*, *7*, 465–471.

Neziroglu, F., & Yaryura-Tobias, J.A. (1993a) Body dysmorphic disorder: phenomenology and case descriptions. *Behavioural Psychotherapy*, *21*, 27–36.

Neziroglu, F., & Yaryura-Tobias, J.A. (1993b) Exposure, response prevention, and cognitive therapy in the treatment of body dysmorphic disorder. *Behavior Therapy*, *24*, 431–438.

Neziroglu, F., McKay, D., Todaro, J., *et al.* (1996) Effect of cognitive behavior therapy on persons with body dysmorphic disorder and comorbid axis II diagnoses. *Behavior Therapy*, *27*, 67–77.

Neziroglu, F., Yaryura-Tobias, J.A., Walz, J., *et al.* (2000) The effect of fluvoxamine and behavior therapy on children and adolescents with obsessive-compulsive disorder. *Journal of Child and Adolescent Psychopharmacology*, *10*, 295–306.

Neziroglu, F., Stevens, K.P., McKay, D., *et al.* (2001) Predictive validity of the overvalued ideas scale: outcome in obsessive-compulsive and body dysmorphic disorders. *Behaviour Research and Therapy, 39*, 745–756.

NICE (2002) *The Guideline Development Process: Information for National Collaborating Centres and Guideline Development Groups*. London: National Institute of Clinical Excellence.

NICE (2003) *Guidance on the Use of Electroconvulsive Therapy*. London: NICE, Technology Appraisal 59.

Nierenberg, A.A., Phillips, K.A., Petersen, T.J., *et al.* (2002) Body dysmorphic disorder in outpatients with major depression. *Journal of Affective Disorders*, *69*, 141–148.

Noorbala, A.A., Hosseini, S.H., Mohammadi, M.R., *et al.* (1998) Combination of clomipramine and nortriptyline in the treatment of obsessive-compulsive disorder: a double-blind, placebo-controlled trial. *Journal of Clinical Pharmacy and Therapeutics*, *23*, 155–159.

Norland, M. (1988) Peculiar and characteristic symptoms: a case. *Homeopathy*, *7*, 190–197.

Nuttin, B., Cosyns, P., Demeulemeester, H., *et al.* (1999) Electrical stimulation in anterior limbs of internal capsules in patients with obsessive-compulsive disorder. *Lancet*, *354* (9189), 1526.

Nuttin, B.J., Gabriels, L., van Kuyck, K., *et al.* (2003a) Electrical stimulation of the anterior limbs of the internal capsules in patients with severe obsessive-compulsive disorder: anecdotal reports. *Neurosurgery Clinics of North America*, *14*, 267–274.

Nuttin, B.J., Gabriels, L.A., Cosyns, P.R., *et al.* (2003b) Long-term electrical capsular stimulation in patients with obsessive-compulsive disorder. *Neurosurgery*, *52*, 1263–1272.

Nyman, H., Andreewitch, S., Lundback, E., *et al.* (2001) Executive and cognitive functions in patients with extreme obsessive-compulsive disorder treated by capsulotomy. *Applied Neuropsychology*, *8*, 91–98.

O'Connor, J.J. (1983) Why can't I get hives: brief strategic therapy with an obsessional child. *Family Process*, *22*, 201–209.

O'Connor, K., Todorov, C., Robillard, S., *et al.* (1999) Cognitive-behaviour therapy and medication in the treatment of obsessive-compulsive disorder: a controlled study. *Canadian Journal of Psychiatry – Revue Canadienne de Psychiatrie, 44*, 64–71.

Oliver, B., Gascon, J., Aparicio, A., *et al.* (2003) Bilateral anterior capsulotomy for refractory obsessive-compulsive disorders. *Stereotactic and Functional Neurosurgery*, *81*, 90–95.

Ong, S.B., & Leng, Y.K. (1979) The treatment of an obsessive-compulsive girl in the context of Malaysian Chinese culture. *Australian and New Zealand Journal of Psychiatry*, *13*, 255–259.

Ongur, D. & Goff, D.C. (2005) Obsessive-compulsive symptoms in schizophrenia: associated clinical features, cognitive function and medication status. *Schizophrenia Research, 75,* 349–362.

Orvin, G.H. (1967) Treatment of the phobic obsessive-compulsive patient with oxazepam, an improved benzodiazepine compound. *Psychosomatics*, *8*, 278–280.

O'Sullivan, G., Noshirvani, H., Marks, I., *et al.* (1991) Six-year follow-up after exposure and clomipramine therapy for obsessive compulsive disorder. *Journal of Clinical Psychiatry, 52*, 150–155.

Otto, M., Wilhelm, S., Cohen, L.S., *et al.* (2001) Prevalence of body dysmorphic disorder in a community sample of women. *American Journal of Psychiatry*, *158*, 2061–2063.

Owens, E.B., & Piacentini, J. (1998) Case study: behavioral treatment of obsessive-compulsive disorder in a boy with comorbid disruptive behavior problems. *Journal of the American Academy of Child and Adolescent Psychiatry*, *37*, 443–446.

Ownby, R.L. (1983) A cognitive behavioral intervention for compulsive handwashing with a thirteen-year-old boy. *Psychology in the Schools*, *20*, 219–222.

Pallanti, S., & Koran, L.M. (1996) Intravenous, pulse loaded clomipramine in body dysmorphic disorder: two case reports. *CNS Spectrum*, *1*, 54–57.

Pallanti, S., Quercioli, L., Paiva, R.S., *et al.* (1999) Citalopram for treatment-resistant obsessive-compulsive disorder. *European Psychiatry: The Journal of the Association of European Psychiatrists*, *14*, 101–106.

Pallanti, S., Hollander, E., Bienstock, C., *et al.* (2002a) Treatment non-response in OCD: methodological issues and operational definitions. *International Journal of Neuropsychopharmacology*, *5*, 181–191.

Pallanti, S., Quercioli, L., & Koran, L.M. (2002b) Citalopram intravenous infusion in resistant obsessive-compulsive disorder: an open trial. *Journal of Clinical Psychiatry*, *63*, 796–801.

Parfitt, A. (1999) Notes on some technical problems in a case of obsessional neurosis. *Journal of Child Psychotherapy*, *25*, 115–139.

Pato, M.T., Zohar-Kadouch, R., Zohar, J., *et al*. (1988) Return of symptoms after discontinuation of clomipramine in patients with obsessive-compulsive disorder. *American Journal of Psychiatry*, *145*, 1521–1525.

Pato, M.T., Pigott, T.A., Hill, J.L., *et al.* (1991) Controlled comparison of buspirone and clomipramine in obsessive-compulsive disorder. *American Journal of Psychiatry*, *148*, 127–129.

Pauls, D.L., Alsobrook, J.P., Goodman, W., *et al.* (1995) A family study of obsessive-compulsive disorder. *American Journal of Psychiatry*, *152*, 76–84.

Pelton, C.L. (1987) Born obsessing. *Psychotherapy Patient, 3*, 131–142.

Perlmutter, S.J., Leitman, S.F., Garvey, M.A., *et al.* (1999) Therapeutic plasma exchange and intravenous immunoglobulin for obsessive-compulsive disorder and tic disorders in childhood. *Lancet*, *354* (9185), 1153–1158.

Perse, T.L., Greist, J.H., Jefferson, J.W., *et al.* (1987) Fluvoxamine treatment of obsessive-compulsive disorder. *American Journal of Psychiatry*, *144*, 1543–1548.

Perugi, G., Giannotti, D., Di Vaio, S., *et al.* (1996) Fluvoxamine in the treatment of body dysmorphic disorder (dysmorphophobia) *International Clinical Psychopharmacology*, *11*, 247–254.

Peter, H., Tabrizian, S., & Hand, I. (2000) Serum cholesterol in patients with obsessive compulsive disorder during treatment with behavior therapy and SSRI or placebo. *International Journal of Psychiatry in Medicine*, *30*, 27–39.

Philippopulis, G.S. (1979) The analysis of a case of dysmorphophobia. *Canadian Journal of Psychiatry*, *24*, 397–401.

Phillips, K.A. (1991) Body dysmorphic disorder: the distress of imagined ugliness. *American Journal of Psychiatry*, *148*, 1138–1149.

Phillips, K.A. (1996a) An open study of buspirone augmentation of serotonin-reuptake inhibitors in body dysmorphic disorder. *Psychopharmacology Bulletin*, *32*, 175–180.

Phillips, K.A. (1996b) Pharmacologic treatment of body dysmorphic disorder. *Psychopharmacology Bulletin*, *32*, 597–605.

Phillips, K.A. (2000) Quality of life for patients with body dysmorphic disorder. *Journal of Nervous and Mental Disease*, *188*, 170–175.

Phillips, K.A. (2002) Pharmacologic treatment of body dysmorphic disorder: review of the evidence and a recommended treatment approach. *CNS Spectrum*, *7*, 453–460.

Phillips, K.A. (2005) Placebo-controlled study of pimozide augmentation of fluoxetine in body dysmorphic disorder. *American Journal of Psychiatry*, *162*, 377–379.

Phillips, K.A., & Diaz, S.F. (1997) Gender differences in body dysmorphic disorder. *Journal of Nervous and Mental Disease*, *185*, 570–577.

Phillips, K.A., & Najjar, F. (2003) An open-label study of citalopram in body dysmorphic disorder. *Journal of Clinical Psychiatry*, *64*, 715–720.

Phillips, K.A., McElroy, S.L., Keck, P.E., Jr., *et al.* (1993) Body dysmorphic disorder: 30 cases of imagined ugliness. *American Journal of Psychiatry*, *150*, 302–308.

Phillips, K.A., Kim, J.M., & Hudson, J.I. (1995) Body image disturbance in body dysmorphic disorder and eating disorders. Obsessions or delusions? *Psychiatric Clinics of North America*, *18*, 317–334.

Phillips, K.A., Dwight, M.M., & McElroy, S.L. (1998) Efficacy and safety of fluvoxamine in body dysmorphic disorder. *Journal of Clinical Psychiatry*, *59*, 165–171.

Phillips, K.A., Dufresne, R.G.Jr, Wilkel, C.S. *et al.* (2000) Rate of body dysmorphic disorder in dermatology patients. *Journal of the Americal Academy of Dermatology, 42,* 436–441.

Phillips, K.A., Albertini, R.S., Siniscalchi, J.M., *et al.* (2001) Effectiveness of pharmacotherapy for body dysmorphic disorder: a chart-review study. *Journal of Clinical Psychiatry*, *62*, 721–727.

Phillips, K.A., Albertini, R.S., & Rasmussen, S.A. (2002) A randomized placebo-controlled trial of fluoxetine in body dysmorphic disorder. *Archives of General Psychiatry*, *59*, 381–388.

Piacentini, J. (1999) Cognitive behavioral therapy of childhood OCD. *Child and Adolescent Psychiatric Clinics of North America*, *8*, 599–616.

Piacentini, J., Gitow, A., Jaffer, M., *et al.* (1994) Outpatient behavioral treatment of child and adolescent obsessive compulsive disorder. *Journal of Anxiety Disorders*, *8*, 277–289.

Piacentini, J., Bergman, R.L., Jacobs, C., *et al.* (2002) Open trial of cognitive behavior therapy for childhood obsessive-compulsive disorder. *Journal of Anxiety Disorders*, *16*, 207–219.

Pidrman, V., & Tuma, I. (1997) Fenfluramine challenge test in obsessive-compulsive disorder: first results. *Acta Medica (Hradec Kralove), 40*, 99–102.

Pigott, T.A., Pato, M.T., Bernstein, S.E., *et al.* (1990) Controlled comparisons of clomipramine and fluoxetine in the treatment of obsessive-compulsive disorder: behavioral and biological results. *Archives of General Psychiatry*, *47*, 926–932.

Pigott, T.A., Pato, M.T., L'Heureux, F., *et al.* (1991) A controlled comparison of adjuvant lithium carbonate or thyroid hormone in clomipramine-treated patients with obsessive-compulsive disorder. *Journal of Clinical Psychopharmacology*, *11*, 242–248.

Pigott, T.A., L'Heureux, F., Rubenstein, C.S., *et al.* (1992) A double-blind, placebo controlled study of trazodone in patients with obsessive-compulsive disorder. *Journal of Clinical Psychopharmacology*, *12*, 156–162.

Pigott, T.A., L'Heureux, F., Hill, J.L., *et al.* (1992a) A double-blind study of adjuvant buspirone hydrochloride in clomipramine-treated patients with obsessive-compulsive disorder. *Journal of Clinical Psychopharmacology*, *12*, 11–18.

Pigott, T.A., L'Heureux, F., Rubenstein, C.F., *et al.* (1992b) A controlled trial of clonazepam augmentation in OCD patients treated with clomipramine or fluoxetine. New Research Abstracts NR144 presented at the 145th Annual Meeting of the American Psychiatric Association, Washington, DC: USA.

Pitman, R.K., Green, R.C., Jenike, M.A., *et al*. (1987) Clinical comparison of Tourette's disorder and obsessive-compulsive disorder. *American Journal of Psychiatry, 144*, 1166–1171.

Pollard, J.W. (2001) Don't go there: impulse control in stage-specific short term counselling. *Journal of College Student Psychotherapy*, *16*, 65–84.

POTS (2004) Cognitive-behavior therapy, sertraline, and their combination for children and adolescents with obsessive-compulsive disorder: the Pediatric OCD Treatment Study (POTS) randomized controlled trial. *Journal of the American Medical Association*, *292*, 1969–1976.

Prasad, A. (1984) A double blind study of imipramine versus zimelidine in treatment of obsessive compulsive neurosis. *Pharmacopsychiatry, 17*, 61–62.

Price, L.H., Goodman, W.K., Charney, D.S., *et al.* (1987) Treatment of severe obsessive-compulsive disorder with fluvoxamine. *American Journal of Psychiatry, 144*, 1059–1061.

Querioz, L.O.S., Motta, M.A., Madi, M.B.B.P., *et al.* (1981) A functional analysis of obsessive-compulsive problems with related therapeutic procedures. *Behaviour Research and Therapy*, *19*, 377–388.

Rabavilas, A.D., Boulougouris, J.C., & Perissaki, C. (1979) Therapist qualities related to outcome with exposure in vivo in neurotic patients. *Journal of Behavior Therapy and Experimental Psychiatry*, *10*, 293–294.

Rachman, S. (1994) Pollution of the mind. *Behaviour Research and Therapy*, *32*, 311–314.

Rachman, S. (1997) A cognitive theory of obsessions. *Behaviour Research and Therapy*, *35*, 793–802.

Rachman, S. (1998) A cognitive theory of obsessions: elaborations. *Behaviour Research and Therapy*, *36*, 385–401.

Rachman, S. (2002) A cognitive theory of compulsive checking. *Behaviour Research and Therapy*, *40*, 624–639.

Rachman, S.J. (2003) *The Treatment of Obsessions*. Oxford: Oxford University Press.

Rachman, S. (2004) Fear of contamination. *Behaviour Research and Therapy*, *42*, 1227–1255.

Rachman, S.J., & de Silva, P. (1978) Abnormal and normal obsessions. *Behaviour Research and Therapy*, *16*, 233–238.

Rachman, S., Hodgson, R., & Marks, I.M. (1971) The treatment of chronic obsessive-compulsive neurosis. *Behaviour Research and Therapy*, *9*, 237–247.

Rachman, S., Marks, I.M., & Hodgson, R. (1973) The treatment of obsessive-compulsive neurotics by modelling and flooding in vivo. *Behaviour Research and Therapy*, *11*, 463–471.

Rao, S. (2002) Fluoxetine was safe and effective for body dysmorphic disorder. *Evidence-Based Mental Health, 5*, 119.

Raphael, F.J., Rani, S., Bale, R., *et al.* (1996) Religion, ethnicity and obsessive-compulsive disorder. *International Journal of Social Psychiatry*, *42*, 38–44.

Rapoport, J.L., & Inoff-Germain, G. (2000) Treatment of obsessive-compulsive disorder in children and adolescents. *Journal of Child Psychology and Psychiatry*, *41*, 419–431.

Rapoport, J., Elkins, R., & Mikkelsen, E. (1980) Clinical controlled trial of chlorimipramine in adolescents with obsessive-compulsive disorder. *Psychopharmacology Bulletin*, *16*, 61–63.

Rapoport, J.L., Leonard, H.L., Swedo, S.E., *et al.* (1993) Obsessive compulsive disorder in children and adolescents: issues in management. *Journal of Clinical Psychiatry*, *54* (Suppl 6), 27–29.

Rasmussen, S.A., & Eisen, J.L. (1990a) Epidemiology and clinical features of obsessive compulsive disorder. In *Obsessive Compulsive Disorders: Theory and Management* (eds. M.A. Jenike, L. Bae, & W.E. Minichiello), pp. 10–27. Chicago: Year Book Medical.

Rasmussen, S.A., & Eisen, J.L. (1990b) Epidemiology of obsessive compulsive disorder. *Journal of Clinical Psychiatry*, *51* (Suppl), 10–13.

Rasmussen, S.A., & Eisen, J.L. (1994) The epidemiology and differential diagnosis of obsessive compulsive disorder. *Journal of Clinical Psychiatry*, *55*, 5–14.

Rasmussen, S., Greenberg, B., Mindus, P., *et al.* (2000) Neurosurgical approaches to intractable obsessive-compulsive disorder. *CNS Spectrum*, *5*, 23–34.

Ravizza, L., Barzega, G., Bellino, S., *et al.* (1995) Predictors of drug treatment response in obsessive-compulsive disorder. *Journal of Clinical Psychiatry*, *56*, 368–373.

Ravizza, L., Barzega, G., Bellino, S., *et al.* (1996) Drug treatment of obsessive-compulsive disorder (OCD): long-term trial with clomipramine and selective serotonin reuptake inhibitors (SSRIs) *Psychopharmacology Bulletin*, *32*, 167–173.

Reichenberg, U.J., & Ullman, R. (1998a) A violent child with an attitude. *Townsend Letter*, *178*, 52.

Reichenberg, U.J. & Ullman, R. (1998b) A grieving mother with obsessive-compulsive tendencies. *Townsend Letter*, *177*, 38–39.

Reichenberg, U.J., & Ullman, R. (1999) Everything in its place: the torment of obsessive-compulsive disorder. *Townsend Letter*, *195*, 132–135.

Reichenberg, U.J., & Ullman, R. (2000) The fascinating and debilitating world of OCD. *Journal of the American Institute of Homeopathy*, *93*, 132–139.

Rettew, D.C., Swedo, S.E., Leonard, H.L., *et al.* (1992) Obsessions and compulsions across time in 79 children and adolescents with obsessive-compulsive disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *31*, 1050–1056.

Rheaume, J., Freeston, M., Leger, E., *et al.* (1998) Bad luck: an underestimated factor in the development of obsessive-compulsive disorder. *Clinical Psychology and Psychotherapy*, *5*, 1–12.

Riddle, M.A., Scahill, L., King, R.A., *et al.* (1992) Double-blind, crossover trial of fluoxetine and placebo in children and adolescents with obsessive-compulsive disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *31*, 1062–1069.

Riddle, M.A., Reeve, E.A., Yaryura-Tobias, J.A., *et al.* (2001) Fluvoxamine for children and adolescents with obsessive-compulsive disorder: a randomized, controlled, multicenter trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, *40*, 222–229.

Riding, J., & Munro, A. (1975) Pimozide in the treatment of monosymptomatic hypochondriacal psychosis. *Acta Psychiatrica Scandinavica*, *52*, 23–30.

Robins, L.N., Holzer, J.E., & Weissman, M.M. (1984) Lifetime prevalence of psychiatric disorders in three sites. *Archives of General Psychiatry*, *41*, 949–958.

Romano, S., Goodman, W., Tamura, R., *et al.* (2001) Long-term treatment of obsessive-compulsive disorder after an acute response: a comparison of fluoxetine versus placebo. *Journal of Clinical Psychopharmacology*, *21*, 46–52.

Rosen, J.C., Saltzberg, E., & Srebnik, D. (1989) Cognitive behavior therapy for negative body image. *Behavior Therapy*, *20*, 393–404.

Rosen, J.C., Reiter, J., & Orosan, P. (1995) Cognitive-behavioral body image therapy for body dysmorphic disorder. *Journal of Consulting and Clinical Psychology*, *63*, 263–269.

Rosenfeld, R., Dar, R., Anderson, D., *et al.* (1992) A computer-administered version of the Yale-Brown Obsessive Compulsive Scale. *Psychological Assessment*, *4*, 329–332.

Rosqvist, J., Egan, D., Manzo, P., *et al.* (2001) Home-based behavior therapy for obsessive-compulsive disorder: a case series with data. *Journal of Anxiety Disorders, 15*, 395–400.

Rosso, S.M., Roks, G., Stevens, M., *et al.* (2001) Complex compulsive behaviour in the temporal variant of frontotemporal dementia. *Journal of Neurology*, *248*, 965–970.

Roth, A., & Fonagy, P. (2004) *What Works for Whom? A Critical Review of Psychotherapy Research.* (2nd edn). London: Guilford.

Rothbaum, B.O. & Fran, S. (2000) Behavioural treatment of obsessive-compulsive disorder in a naturalistic setting. *Cognitive and Behavioural Practice, 7,* 262–270.

Sachdev, P. (1996) Site and size of lesion and psychosurgical outcome in obsessive-compulsive disorder: a magnetic resonance imaging study. *Biological Psychiatry, 39*, 739–742.

Sachdev, P., & Hay, P. (1995) Does neurosurgery for obsessive-compulsive disorder produce personality change? *Journal of Nervous and Mental Disease*, *183*, 408–413.

Sachdev, P.S., McBride, R., Loo, C.K., *et al.* (2001) Right versus left prefrontal transcranial magnetic stimulation for obsessive-compulsive disorder: a preliminary investigation. *Journal of Clinical Psychiatry*, *62*, 981–984.

Salkovskis, P.M. (1985) Obsessive-compulsive problems: a cognitive behavioural analysis. *Behaviour Research and Therapy*, *23*, 571–583.

Salkovskis, P.M. (1999) Understanding and treating obsessive-compulsive disorder. *Behaviour Research and Therapy, 37* (Suppl 1), S29–S52.

Salkovskis, P.M., & Warwick, H.M. (1986) Cognitive therapy of obsessive-compulsive disorder: treating treatment failures. *Behavioural Psychotherapy*, *13*, 243–255.

Salkovskis, P.M., Richards, C.H., & Forrester, E. (1995) The relationship between obsessional problems and intrusive thoughts. *Behavioural and Cognitive Psychotherapy*, *23*, 281–299.

Salkovskis, P.M., Shafran, R., Rachman, S., *et al.* (1999) Multiple pathways to inflated responsibility beliefs in obsessional problems: possible origins and implications for therapy and research. *Behaviour Research and Therapy*, *37*, 1055–1072.

Salkovskis, P.M., Thorpe, S.J., Wahl, K., *et al.* (2003) Neutralizing increases discomfort associated with obsessional thoughts: an experimental study with obsessional patients. *Journal of Abnormal Psychology, 112*, 709–715.

Sallee, F.R., Koran, L.M., Pallanti, S., *et al.* (1998) Intravenous clomipramine challenge in obsessive-compulsive disorder: predicting response to oral therapy at eight weeks. *Biological Psychiatry, 44*, 220–227.

Salzberg, A.D., & Swedo, S.E. (1992) Oxytocin and vasopressin in obsessive-compulsive disorder. *American Journal of Psychiatry*, *149*, 713–714.

Salzman, L. (1983) Psychoanalytic therapy of the obsessional patient. *Current Psychiatric Therapies*, *22*, 53–59.

Salzman, L. (1997) Therapy of the obsessive personality. In *Essential Papers in Psychoanalysis, Essential Papers on Obsessive-Compulsive Disorder* (eds. D.J. Stein & M.H. Stone), pp. 124–160. New York: New York University Press.

Sarwer, D.B., Wadden, T.A., Pertschuk, M.J., *et al.* (1998) Body image dissatisfaction and body dysmorphic disorder in 100 cosmetic surgery patients. *Plastic and Reconstructive Surgery*, *101*, 1644–1649.

Saxena, S., Brody, A.L., Schwartz, J.M., *et al.* (1998) Neuroimaging and frontal-subcortical circuitry in obsessive-compulsive disorder. *British Journal of Psychiatry, 173* (Suppl 35), 26–37.

Scahill, L., Vitulano, L.A., Brenner, E.M., *et al.* (1996) Behavioral therapy in children and adolescents with obsessive-compulsive disorder: a pilot study. *Journal of Child and Adolescent Psychopharmacology*, *6*, 191–202.

Scahill, L., Riddle, M.A., McSwiggin, H.M., *et al.* (1997) Children's Yale-Brown Obsessive Compulsive Scale: reliability and validity. *Journal of the American Academy of Child and Adolescent Psychiatry*, *36*, 844–852.

Schmidt, N.B., & Harrington, P. (1995) Cognitive-behavioral treatment of body dysmorphic disorder: a case report. *Journal of Behavior Therapy and Experimental Psychiatry*, *26*, 161–167.

Schwartz, E.K. (1972) The treatment of the obsessive patient in the group therapy setting. *American Journal of Psychotherapy*, *26*, 352–361.

Schwartz, J.M., Stoessel, P.W., Baxter, L. Jr., *et al.* (1996) Systematic changes in cerebral glucose metabolic rate after successful behaviour modification treatment of obsessive-compulsive disorder. *Archives of General Psychiatry*, *53*, 109–113.

Scrignar, C.B. (1981) Rapid treatment of contamination phobia with hand-washing compulsion by flooding with hypnosis. *American Journal of Clinical Hypnosis*, *23*, 252–257.

Sevincok, L., & Topuz, A. (2003) Lack of efficacy of low doses of quetiapine addition in refractory obsessive-compulsive disorder. *Journal of Clinical Psychopharmacology*, *23*, 448–450.

Shafran, R. (1997) The manipulation of responsibility in obsessive-compulsive disorder. *British Journal of Clinical Psychology*, *36* (Pt 3), 397–407.

Shafran, R., Ralph, J., & Tallis, F. (1995) Obsessive-compulsive symptoms and the family. *Bulletin of the Menninger Clinic*, *59*, 472–479.

Shafran, R., Frampton, I., Heyman, I., *et al.* (2003) The preliminary development of a new self-report measure for OCD in young people. *Journal of Adolescence*, *26*, 137–142.

Shannahoff-Khalsa, D.S., Ray, L.E., Levine, S., *et al.* (1999) Randomized controlled trial of yogic meditation techniques for patients with obsessive-compulsive disorder. *CNS Spectrum*, *4*, 34–47.

Shaomei, L., & Numerous, C. (2000) Combination of clomipramine with exposure therapy in treatment of obsessive-compulsive disorder. [Chinese]. *Chinese Mental Health Journal, 15*, 239–240.

Shapira, N.A., Ward, H.E., Mandoki, M., *et al.* (2004) A double-blind, placebo-controlled trial of olanzapine addition in fluoxetine-refractory obsessive-compulsive disorder. *Biological Psychiatry*, *55*, 553–555.

Shapiro, D.A., Cavanagh, K., & Lomas, H. (2003) Geographic inequity in the availability of cognitive behavioural therapy in England and Wales. *Behavioural and Cognitive Psychotherapy*, *31*, 185–192.

Sheinberg, M. (1988) Obsessions/counter-obsessions: a construction/reconstruction of meaning. *Family Process*, *27*, 305–316.

Simpson, H.B., Liebowitz, M.R., Foa, E.B., *et al.* (2004) Post-treatment effects of exposure therapy and clomipramine in obsessive-compulsive disorder. *Depression and Anxiety*, *19*, 225–233.

Skoog, G., & Skoog, I. (1999) A 40-year follow-up of patients with obsessive-compulsive disorder. *Archives of General Psychiatry*, *56*, 121–127.

Slattery, M.J., Dubbert, B.K., Allen, A.J., *et al.* (2004) Prevalence of obsessive-compulsive disorder in patients with systemic lupus erythematosus. *Journal of Clinical Psychiatry*, *65*, 301–306.

Smeraldi, E., Erzegovesi, S., Bianchi, I., *et al.* (1992) Fluvoxamine vs clomipramine treatment in obsessive-compulsive disorder: a preliminary study. *New Trends in Experimental and Clinical Psychiatry*, *8*, 63–65.

Smith, R., & Fisher, K. (2003) Healthy limb amputation: ethical and legal aspects. *Clinical Medicine*, *3*, 188.

Sobanski, E., & Schmidt, M.H. (2000) 'Everybody looks at my pubic bone': a case report of an adolescent patient with body dysmorphic disorder. *Acta Psychiatrica Scandinavica*, *101*, 80–82.

Solyom, L., DiNicola, V.F., Phil, M., *et al.* (1985) Is there an obsessive psychosis? Aetiological and prognostic factors of an atypical form of obsessive-compulsive neurosis. *Canadian Journal of Psychiatry*, *30*, 372–379.

Sondheimer, A. (1988) Clomipramine treatment of delusional disorder-somatic type. *Journal of the American Academy of Child and Adolescent Psychiatry*, *27*, 188–192.

Soomro, G.M. (2002) Sertraline reduced patient dropout rates and acute exacerbation of symptoms in obsessive compulsive disorder. *Evidence-Based Mental Health, 5*, 115.

Spangler, W.J., Cosgrove, G.R., Ballantine, H.T., Jr., *et al.* (1996) Magnetic resonance image-guided stereotactic cingulotomy for intractable psychiatric disease. *Neurosurgery*, *38*, 1071–1076.

Stanley, L. (1980) Treatment of ritualistic behaviour in an eight-year-old girl by response prevention: a case report. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *21*, 85–90.

Stanley, M.A., & Turner, S.M. (1995) Current status of pharmacological and behaviour treatment of obsessive-compulsive disorder. *Behaviour Therapy*, *26*, 163–168.

Stein, D.J. (2000) Neurobiology of the obsessive-compulsive spectrum disorders. *Biological Psychiatry*, *47*, 296–304.

Stein, D.J. (2002) Obsessive-compulsive disorder. *Lancet*, *360*, 397–405.

Stein, D.J., Hollander, E., Mullen, L.S., *et al.* (1992) Comparison of clomipramine, alprazolam and placebo in the treatment of obsessive-compulsive disorder. *Human Psychopharmacology*, *7*, 389–395.

Stein, D.J., Laszlo, B., Marais, E., *et al.* (1997a) Hoarding symptoms in patients on a geriatric psychiatry inpatient unit. *South African Medical Journal*, *87*, 1138–1140.

Stein, D.J., Van Heerden, B., Wessels, C.J., *et al.* (1999) Single photon emission computed tomography of the brain with Tc-99 m HMPAO during sumatriptan challenge in obsessive-compulsive disorder: Investigating the functional role of the serotonin auto-receptor. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 23*, 1079–1099.

Stein, D.J., Montgomery, S.A., Kasper, S., *et al.* (2001) Predictors of response to pharmacotherapy with citalopram in obsessive-compulsive disorder. *International Clinical Psychopharmacology, 16*, 357–361.

Stein, M.B., Forde, D.R., Anderson, G., *et al.* (1997b) Obsessive-compulsive disorder in the community: an epidemiologic survey with clinical reappraisal. *American Journal of Psychiatry*, *154*, 1120–1126.

Stein, M.B., Sherbourne, C.D., Craske, M.G., *et al.* (2004) Quality of care for primary care patients with anxiety disorders. *American Journal of Psychiatry*, *161*, 2230–2237.

Steketee, G. (1993) *Treatment of Obsessive Compulsive Disorder*. New York: Guilford Press.

Steketee, G. (1994) Behavioural assessment and treatment planning with obsessive compulsive disorder: a review emphasizing clinical application. *Behavior Therapy, 25,* 613–633.

Steketee, G., & Frost, R. (2003) Compulsive hoarding: current status of the research. *Clinical Psychology Reviews, 23*, 905–927.

Steketee, G., Foa, E.B., & Grayson, J.B. (1982) Recent advances in the behavioral treatment of obsessive-compulsives. *Archives of General Psychiatry*, *39*, 1365–1371.

Steketee, G., Frost, R., & Bogart, K. (1996) The Yale-Brown Obsessive Compulsive Scale: interview versus self-report. *Behaviour Research and Therapy*, *34*, 675–684.

Stengler-Wenzke, K., Trosbach, J., Dietrich, S., *et al.* (2004) Experience of stigmatization by relatives of patients with obsessive compulsive disorder. *Archives of Psychiatric Nursing*, *18*, 88–96.

Stern, R.S. (1973) Contract therapy in obsessive-compulsive neurosis with marital discord. *British Journal of Psychiatry*, *123*, 681–684.

Stern, R.S., Lipsedge, M.S., & Marks, I.M. (1973) Obsessive ruminations: a controlled trial of thought-stopping technique. *Behaviour Research and Therapy, 11*, 659–662.

Stern, R.S., Cobb, J.P., Marks, I.M., *et al.* (1977) A preliminary report on clinical response and plasma levels of clomipramine and desmethylclomipramine in obsessive-compulsive neurosis. *Postgraduate Medical Journal, 53*, 97–103.

Stern, R.S., Marks, I.M., Wright, J., *et al.* (1980) Clomipramine: plasma levels, side effects and outcome in obsessive-compulsive neurosis. *Postgraduate Medical Journal, 56*, 134–139.

Stewart, M.A. (1995) Effective physician-patient communication and health outcomes: a review. *Canadian Association Medical Journal*, *152*, 1423–1433.

Stewart, S.E., Geller, D.A., Jenike, M., *et al.* (2004) Long-term outcome of pediatric obsessive-compulsive disorder: a meta-analysis and qualitative review of the literature. *Acta Psychiatrica Scandinavica, 110*, 4–13.

Strom-Olsen, R., & Carlisle, S. (1971) Bi-frontal stereotactic tractotomy: a follow-up study of its effects on 210 patients. *British Journal of Psychiatry*, *118*, 141–154.

Swedo, S.E., Rapoport, J.L., Leonard, H.L., *et al.* (1989) Obsessive-compulsive disorder in children and adolescents: clinical phenomenology of 70 consecutive cases. *Archives of General Psychiatry*, *46*, 335–341.

Swedo, S.E., Leonard, H.L., Garvey, M., *et al.* (1998) Pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections: clinical description of the first 50 cases. *American Journal of Psychiatry*, *155*, 264–271.

Szegedi, A., Wetzel, H., Leal, M., *et al.* (1996) Combination treatment with clomipramine and fluvoxamine: drug monitoring, safety, and tolerability data. *Journal of Clinical Psychiatry*, *57*, 257–264.

Tass, P.A., Klosterkotter, J., Schneider, F., *et al.* (2003) Obsessive-compulsive disorder: development of demand-controlled deep brain stimulation with methods from stochastic phase resetting. *Neuropsychopharmacology*, *28* (Suppl 1), S27–S34.

Taylor, L.H., & Kobak, K.A. (2000) An open-label trial of St. John's Wort (Hypericum perforatum) in obsessive-compulsive disorder. *Journal of Clinical Psychiatry*, *61*, 575–578.

Taylor, R.E. (1985) Imagery for the treatment of obsessional behavior: a case study. *American Journal of Clinical Hypnosis*, *27*, 175–179.

Taylor, S. (1995) Assessment of obsessions and compulsions: reliability, validity, and sensitivity to treatment effects. *Clinical Psychology Reviews*, *15*, 261–296.

Taylor, S., Thordarson, D.S., & Sachting, I. (2002) Obsessive-compulsive disorder. In *Handbook of Assessment and Treatment Planning for Psychological Disorders* (eds. M.M. Anthony & D.H. Barlow), pp. 182–214. New York: Guilford Press.

Taylor, S., Thordarson, D.S., Spring, T., *et al.* (2003) Telephone-administered cognitive behavior therapy for obsessive-compulsive disorder. *Cognitive Behaviour Therapy*, *32*, 13–25.

Tek, C., & Ulug, B. (2001) Religiosity and religious obsessions in obsessive-compulsive disorder. *Psychiatry Research*, *104*, 99–108.

Tenney, N.H., Denys, D.A., van Megen, H.J., *et al.* (2003) Effect of a pharmacological intervention on quality of life in patients with obsessive-compulsive disorder. *International Clinical Psychopharmacology*, *18*, 29–33.

Thienemann, M., Martin, J., Cregger, B., *et al.* (2001) Manual-driven group cognitive-behavioral therapy for adolescents with obsessive-compulsive disorder: a pilot study. *Journal of the American Academy of Child and Adolescent Psychiatry*, *40*, 1254–1260.

Thomas, S.G., & Kellner, C.H. (2003) Remission of major depression and obsessive-compulsive disorder after a single unilateral ECT. *Journal of ECT*, *19*, 50–51.

Thoren, P., Asberg, M., Cronholm, B., *et al.* (1980) Clomipramine treatment of obsessive-compulsive disorder. I. A controlled clinical trial. *Archives of General Psychiatry*, *37*, 1281–1285.

Tippin, J., & Henn, F.A. (1982) Modified leukotomy in the treatment of intractable obsessional neurosis. *American Journal of Psychiatry*, *139*, 1601–1603.

Tolin, D.F. (2001) Case study: bibliotherapy and extinction treatment of obsessive-compulsive disorder in a 5-year-old boy. *Journal of the American Academy of Child and Adolescent Psychiatry*, *40*, 1111–1114.

Tolin, D.F., & Franklin, M.E. (2002) Prospects for the use of cognitive-behavioral therapy in childhood obsessive-compulsive disorder. *Expert Review of Neurotherapeutics*, *2*, 539–548.

Tollefson, G.D., Birkett, M., Koran, L., *et al.* (1994) Continuation treatment of OCD: double-blind and open-label experience with fluoxetine. *Journal of Clinical Psychiatry*, *55*, 69–76.

Turgeon, L., O'Connor, K.P., Marchand, A., *et al.* (2002) Recollections of parent-child relationships in patients with obsessive-compulsive disorder and panic disorder with agoraphobia. *Acta Psychiatrica Scandinavica*, *105*, 310–316.

Turner, S.M., Jacob, R.G., Beidel, D.C., *et al.* (1985) Fluoxetine treatment of obsessive-compulsive disorder. *Journal of Clinical Psychopharmacology, 5*, 207–212.

Ushijima, S., Kamijima, K., Asai, M., *et al.* (1997) Clinical evaluation of sertraline hydrochloride, a selective serotonin reuptake inhibitor in the treatment of obsessive-compulsive disorder: a double blind placebo controlled trial. [Japanese]. *Japanese Journal of Neuropsychopharmacology, 19*, 603–623.

Uzun, O., Basoglu, C., Akar, A., *et al.* (2003) Body dysmorphic disorder in patients with acne. *Comprehensive Psychiatry*, *44*, 415–419.

Vallejo, J., Olivares, J., Marcos, T., *et al.* (1992) Clomipramine versus phenelzine in obsessive-compulsive disorder. A controlled clinical trial. *British Journal of Psychiatry*, *161*, 665–670.

Valleni-Basile, L.A., Garrison, C.Z., Jackson, K.L., *et al.* (1994) Frequency of obsessive-compulsive disorder in a community sample of young adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*, *33*, 782–791.

Van Balkom, A.J.L.M., van Oppen, P., Vermeulen, A.W.A., *et al.* (1994) A meta-analysis on the treatment of obsessive compulsive disorder: a comparison of antidepressants, behavior, and cognitive therapy. *Clinical Psychology Review*, *14*, 359–381.

Van Balkom, A.J., de Haan, E., van Oppen, P., *et al.* (1998) Cognitive and behavioral therapies alone versus in combination with fluvoxamine in the treatment of obsessive compulsive disorder. *Journal of Nervous and Mental Disease*, *186*, 492–499.

Van Noppen, B., Steketee, G., McCorkle, B.H., *et al.* (1997) Group and multifamily behavioral treatment for obsessive compulsive disorder: a pilot study. *Journal of Anxiety Disorders, 11*, 431–446.

Van Noppen, B.L., Pato, M.T., Marsland, R., *et al.* (1998) A time-limited behavioral group for treatment of obsessive-compulsive disorder. *Journal of Psychotherapy Practice and Research*, 7, 272–280.

Van Oppen, P., & Arntz, A. (1994) Cognitive therapy for obsessive-compulsive disorder. *Behaviour Research and Therapy*, *32*, 79–87.

Van Oppen, P., de Haan, E., van Balkom, A.J., *et al.* (1995) Cognitive therapy and exposure in vivo in the treatment of obsessive compulsive disorder. *Behaviour Research and Therapy*, *33*, 379–390.

Veale, D. (2000) Outcome of cosmetic surgery and 'DIY' surgery in patients with body dysmorphic disorder. *Psychiatric Bulletin*, *24*, 218–221.

Veale, D. (2002) Overvalued ideas: a conceptual analysis. *Behaviour Research and Therapy*, *40*, 383–400.

Veale, D. (2004) Advances in a cognitive behavioural understanding of body dysmorphic disorder. *Body Image*, *1*, 113–125.

Veale, D., Boocock, A., Gournay, K., *et al.* (1996a) Body dysmorphic disorder: a survey of fifty cases. *British Journal of Psychiatry*, *169*, 196–201.

Veale, D., Gournay, K., Dryden, W., *et al.* (1996b) Body dysmorphic disorder: a cognitive behavioural model and pilot randomised controlled trial. *Behaviour Research and Therapy*, *34*, 717–729.

Vindigni, V., Pavan, C., Semenzin, S., *et al.* (2002) The importance of recognizing body dysmorphic disorder in cosmetic surgery patients: do our patients need a pre-operative psychiatric evaluation? *European Journal of Plastic Surgery*, *25*, 305–308.

Vitiello, B., & DeLeon, J. (1990) Dysmorphophobia misdiagnosed as obsessive-compulsive disorder. *Psychosomatics*, *31*, 220–222.

Vogel, P.A., Stiles, T.C., & Nordahl, H.M. (1997) Recollections of parent-child relationships in OCD out-patients compared to depressed out-patients and healthy controls. *Acta Psychiatrica Scandinavica*, *96*, 469–474.

Vogel, P.A., Stiles, T.C., & Gotestam, G. (2004) Adding cognitive therapy elements to exposure therapy for obsessive compulsive disorder: a controlled study. *Behavioural and Cognitive Psychotherapy*, *32*, 275–290.

Volavka, J., Neziroglu, F., & Yaryura-Tobias, J.A. (1985) Clomipramine and imipramine in obsessive-compulsive disorder. *Psychiatry Research*, *14*, 85–93.

Walker, W.L. (1981) The secret room for problem solving. *Australian Journal of Clinical and Experimental Hypnosis*, *9*, 102–103.

Warneke, L.B. (1985) Intravenous chlorimipramine in the treatment of obsessional disorder in adolescence: case report. *Journal of Clinical Psychiatry*, *46*, 100–103.

Waters, T.L., Barrett, P.M., & March, J.S. (2001) Cognitive-behavioral family treatment of childhood obsessive-compulsive disorder: preliminary findings. *American Journal of Psychotherapy*, *55*, 372–387.

Watts, F.N. (1990) Aversion to personal body hair: a case study in the integration of behavioural and interpretative methods. *British Journal of Medical Psychology*, *63*, 335–340.

Waxman, D. (1977) A clinical trial of clomipramine and diazepam in the treatment of phobic and obsessional illness. *Journal of International Medical Research, 5*, 99–110.

Waziri, R. (1990) Psychosurgery for anxiety and obsessive-compulsive disorders. In *The Treatment of Anxiety: Handbook of Anxiety* (eds. R. Noyes, M. Roth, & G.D. Burrows). Amsterdam: Elsevier.

Weiner, I.B. (1967) Behaviour therapy in obsessive-compulsive neurosis treatment of an adolescent boy. *Psychotherapy: Theory, Research and Practice*, *4*, 27–29.

Weir, E. (2000) Treating obsessive-compulsive and tic disorders. *CMAJ: Canadian Medical Association Journal, 163*, 82.

Weiss, E.L., Potenza, M.N., McDougle, C.J., *et al.* (1999) Olanzapine addition in obsessive-compulsive disorder refractory to selective serotonin reuptake inhibitors: an open-label case series. *Journal of Clinical Psychiatry*, *60*, 524–527.

Weissman, M.M., Bland, R.C., Canino, G.J., *et al* (1994) The cross national epidemiology of obsessive compulsive disorder. The Cross National Collaborative Group. *Journal of Clinical Psychiatry*, *55* (Suppl), 5–10.

Weissman, M.M., Broadhead, W.E., Olfson, M., *et al.* (1998) A diagnostic aid for detecting (DSM-IV) mental disorders in primary care. *General Hospital Psychiatry*, *20*, 1–11.

Welkowitz, L.A., Struening, E.L., Pittman, J., *et al.* (2000) Obsessive-compulsive disorder and comorbid anxiety problems in a national anxiety screening sample. *Journal of Anxiety Disorders, 14,* 471–482.

Wells, A. (2000) *Emotional Disorders and Metacognition: Innovative Cognitive Therapy*. New York, NY: John Wiley & Sons.

Wells, M.C. (1990) Treating obsessive-compulsive personalities in psychodynamic/ interpersonal group therapy. *Psychotherapy: Theory, Research, Practice, Training*, *27*, 366–379.

West Midlands Medicines Information Service (2003) UKMi Midlands cost comparison chart: a comparison of the NHS cost of drugs at standard doses. www.ukmicentral.nhs.uk/pressupp/costs/4_3_antidepress.PDF

Wever, C., & Rey, J.M. (1997) Juvenile obsessive-compulsive disorder. *Australian and New Zealand Journal of Family Therapy*, *31*, 105–113.

Whiteside, S.P., Port, J.D., & Abramowitz, J.S. (2004) A meta-analysis of functional neuroimaging in obsessive-compulsive disorder. *Psychiatry Research*, *132*, 69–79.

Whittal, M.L., Thordarson, D.S., McLean, P.D. (2005) Treatment of obsessive-compulsive disorder: cognitive behavior therapy vs. exposure and response prevention. *Behaviour Research and Therapy, 43,* 1559–1576.

Wilhelm, S. (2000) Cognitive therapy for obsessive-compulsive disorder. *Journal of Cognitive Psychotherapy*, *14*, 245–259.

Wilhelm, S., Otto, M.W., Zucker, B.G., *et al.* (1997) Prevalence of body dysmorphic disorder in patients with anxiety disorders. *Journal of Anxiety Disorders*, *11*, 499–502.

Wilhelm, S., Otto, M.W., Lohr, B., *et al.* (1999) Cognitive behavior group therapy for body dysmorphic disorder: a case series. *Behaviour Research and Therapy, 37*, 71–75.

Williams, T.I., & Allsopp, M. (1999) Obsessional compulsive disorder in adolescent populations: experiences of offering a combined pharmacological-psychological approach. *Child Psychology and Psychiatry Review*, *4*, 162–169.

Williams, T.I., Salkovskis, P.M., Forrester, E.A., *et al.* (2002) Changes in symptoms of OCD and appraisal of responsibility during cognitive behavioural treatment: a pilot study. *Behavioural and Cognitive Psychotherapy*, *30*, 69–78.

Willick, M.S. (1995) Obsessive-compulsive disorder? Or, a narcissistic solution in an obsessional man? A nineteen-year treatment in progress. *Journal of Clinical Psychoanalysis, 4*, 149–168.

Willmuth, M.E. (1988) Cognitive-behavioral and insight-oriented psychotherapy of an eleven-year-old boy with obsessive-compulsive disorder. *American Journal of Psychotherapy*, *42*, 472–478.

Wolff, R., & Rapoport, J. (1998) Behavioral treatment of childhood obsessive-compulsive disorder. *Behavior Modification*, *12*, 252–266.

Wolff, R.P., & Wolff, L.S. (1991) Assessment and treatment of obsessive-compulsive disorder in children. *Behavior Modification*, *15*, 372–393.

World Health Organization (1992) *The ICD-10 Classification of Mental and Behavioural Disorders: Clinical Descriptions and Diagnostic Guidelines*. Geneva: World Health Organization.

Yamagami, T. (1978) Changes of behavior, fear and thought in the treatment by response prevention: a case study of obsessive compulsive disorder. *Folia Psychiatrica et Neurologica Japonica, 32*, 77–83.

Yargic, L.I., Enderer, M., Imre, H., *et al.* (1995) A randomized single blind comparison of clomipramine and fluvoxamine in OCD patients. [Turkish]. *Noropsikiyatri Arsivi, 32*, 70–75.

Yaryura-Tobias, J.A., & Bhagavan, H.N. (1977) L-tryptophan in obsessive-compulsive disorders. *American Journal of Psychiatry*, *134*, 1298–1299.

Yaryura-Tobias, J.A., & Neziroglu, F.A. (1996) Venlafaxine in obsessive-compulsive disorder. *Archives of General Psychiatry, 53*, 653–654.

Yaryura-Tobias, J.A., Neziroglu, F., & Bergman, L. (1976) Chlorimipramine, for obsessive-compulsive neurosis: an organic approach. *Current Therapeutic Research, 20*, 541–548.

Yoder, J. (1994) Paradoxical intention: the case of Ron. *International Forum for Logotherapy*, *17*, 108–113.

Zahn, T.P., Insel, T.R., & Murphy, D.L. (1984) Psychophysiological changes during pharmacological treatment of patients with obsessive compulsive disorder. *British Journal of Psychiatry, 145*, 39–44.

Zhang, P.G., Zhong, Q., & Yu, Y.E. (2002) Controlled observation on electro-acupuncture combined with clomipramine in treating obsession. [Chinese]. *Zhongguo Zhong Xi Yi Jie He Za Zhi Zhongguo Zhongxiyi Jiehe Zazhi, 22*, 215–216.

Zikis, P. (1983) Treatment of an 11 year old obsessive compulsive ritualizer and tiquear girl with in vivo exposure and response prevention. *Behavioural Psychotherapy*, *11*, 75–81.

Zimmerman, M., & Mattia, J.I. (1998) Body dysmorphic disorder in psychiatric outpatients: recognition, prevalence, comorbidity, demographic, and clinical correlates. *Comprehensive Psychiatry, 39*, 265–270.

Zitterl, W., Meszaros, K., Hornik, K., *et al.* (1999) Efficacy of fluoxetine in Austrian patients with obsessive-compulsive disorder. *Wiener Klinische Wochenschrift, 111*, 439–442.

Zohar, J., & Insel, T.R. (1987) Obsessive-compulsive disorder: psychobiological approaches to diagnosis, treatment, and pathophysiology. *Biological Psychiatry*, *22*, 667–687.

Zohar, J., & Judge, R. (1996) Paroxetine versus clomipramine in the treatment of obsessive-compulsive disorder. OCD Paroxetine Study Investigators. *British Journal of Psychiatry*, *169*, 468–474.

Zohar, J., Mueller, E.A., Insel, T.R., *et al.* (1987) Serotonergic responsivity in obsessive-compulsive disorder: comparison of patients and healthy controls. *Archives of General Psychiatry, 44*, 946–951.

13. ABBREVIATIONS

A, B, C, GPP	Grades of evidence forming the basis for guideline recommendations (for full definition see Text Box 1, Section 4.4.5.1)
AMED	Allied and Complementary Medicine Database
AMT	Anxiety management training
BDD	Body dysmorphic disorder
BT	Behaviour therapy
CAMHS	Child and adolescent mental health services
CBT	Cognitive behavioural therapy
CBT (including ERP)	Cognitive behavioural therapy (including exposure and response prevention)
CBFT	Cognitive behavioural family therapy
CGI	Clinical Global Impressions
CI	Confidence interval
CINAHL	Cumulative Index to Nursing and Allied Health Literature
CPN	Community psychiatric nurse
CPRS – OC	Comprehensive Psychopathological Rating Scale Obsessive-Compulsive subscale
CT	Cognitive Therapy
CY-BOCS	Children's Yale-Brown Obsessive-Compulsive Scale
DBS	Deep brain stimulation
Df	Degrees of freedom
DSM	Diagnostic and Statistical Manual of Mental Disorders (versions III, III-R and IV)
ECT	Electroconvulsive therapy
EMBASE	Excerpta Medica Database
ERP	Exposure and response prevention
F	The statistic calculated by analysis of variance (F Ratio)
FAD	McMaster Family Assessment Device
FDA	(US) Food and Drugs Administration
GAS	Group A beta haemolytic streptococcus

GDG	Guideline development group
GPP	Good practice point
ICD-10	International Classification of Diseases, 10th Edition
ICER	Incremental cost-effectiveness ratio
IVIG	Intravenous immunoglobulin
K	Number of studies
LOI-CV	Leyton Obsessional Inventory – child version
MAOI	Monoamine-oxidase inhibitor
MDD	Major depressive disorder
MEDLINE	Compiled by the US National Library of Medicine (NLM) and published on the web by Community of Science, MEDLINE is a source of life sciences and biomedical bibliographic information
n	Number of participants in a group
N	Total number of participants
NCCMH	National Collaborating Centre for Mental Health
NHS	National Health Service
NICE	National Institute for Health and Clinical Excellence
NIMH – OC	National Institute of Mental Health Obsessive-Compulsive Global Scale
OCD	Obsessive-Compulsive Disorder
OECD	Organisation for Economic Co-operation and Development
OR	Odds ratio
p	Probability
PANDAS	Paediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcus
PCT	Primary Care Trust
PILOTS	Published International Literature on Traumatic Stress
PsycINFO	An abstract (not full text) database of psychological literature from the 1800s to the present
RCT	Randomised controlled trials
RET	Rational emotive therapy
RR	Relative risk, risk ratio
rTMS	Repetitive transcranial magnetic stimulation

SC	Sydenham's chorea
SCID – IV	Structured Clinical Interview for DSM-IV
SD	Standard deviation
SIGLE	System for Information on Grey Literature in Europe
SMD	Standardised mean difference
SNRI	Serotonin and noradrenaline reuptake inhibitors
SRI	Serotonin reuptake inhibitor
SSRI	Selective serotonin reuptake inhibitor
t	t-statistic
TMS	Transcranial magnetic stimulation
WHOQOL – BREF	World Health Organization Quality of Life
WMD	Weighted mean difference
Y-BOCS	Yale-Brown Obsessive-Compulsive Scale

14. GLOSSARY

Ablative neurosurgery: Surgery in which parts of the brain are disconnected from one another.

Adherence: The behaviour of taking medicine according to treatment dosage and schedule as intended by the prescriber. In this guideline, the term 'adherence' is used in preference to 'compliance', but is not synonymous with 'concordance', which has a number of meanings.

Adverse event: Any untoward medical occurrence in a patient who was given a pharmaceutical product, and which does not necessarily have a causal relationship with this treatment.

Anxiety management training (AMT): A psychological approach aimed at teaching people a set of skills to help them manage their own anxiety and stress. These include:

- Relaxation training: teaching techniques for relaxing major muscle groups in a way that decreases anxiety.
- Breathing retraining: teaching techniques of slow, abdominal breathing to avoid hyperventilation and the unpleasant physical sensations that accompany it.
- Positive thinking and self-talk: positive statements (for example, 'I did it before and I can do it again') are written on cards and rehearsed so that they can be used to replace the negative thoughts that often occur during stressful experiences.
- Assertiveness training: teaching the person how to express wishes, opinions, and emotions appropriately and without alienating others.

Anxiolytics: Drugs used to alleviate anxiety states.

Antipsychotics: This group of medicines all act on a brain chemical, dopamine. Their main use is in psychotic illness, but their dopamine blocking properties can help, when used together with other medicines, in some people with OCD, especially those who do not respond to standard treatments.

Augmentation: The addition of more than one potentially effective treatment together with the aim of enhancing the benefits.

Avoidance: People with OCD and BDD may be unable to engage in particular behaviours, go to specific places or interact with certain people related to their main fears and preoccupations. Even if they wished to do these things, they may find this impossible to do so because of the distress that would arise or through fear of unacceptably dangerous consequences. In OCD, people tend to avoid situations and objects as

they can potentially trigger obsessional thoughts and compulsions. The individual sees such situations as risky and knows that they lead to a high level of anxiety and tension. For example, people with obsessions about germs and cleaning compulsions usually strive to avoid objects and situations that they believe to be contaminating. Therapeutic approaches such as BT and CBT seek to help people to overcome avoidance.

Behaviour therapy (BT): A psychological therapy and an umbrella term for a range of interventions including exposure and response prevention and behavioural activation (see below). Behaviour therapy, also called behaviour modification or behavioural psychotherapy refers to the use of learning theory in the treatment of psychological disorders. It is based on the belief that psychological problems are caused by faulty learning rather than a medical disease. BT aims primarily to help people to manage/change unhelpful behaviours. For example, in OCD, behaviour therapy often involves confronting feared situations (exposure) and refraining from performing rituals (response prevention). For OCD and BDD, BT is often synonymous with exposure and response prevention (see below).

Body dysmorphic disorder (BDD): A preoccupation with an 'imagined' defect in one's appearance or where there is a slight physical anomaly, the person's concern is markedly excessive. To fulfil diagnostic criteria in DSM-IV, the person must be either significantly distressed or impaired in their occupational or social functioning. The older term, 'dysmorphophobia' was first introduced by an Italian psychiatrist Morselli in 1886 although it is now falling into disuse probably because ICD-10 has discarded it and subsumed it under that of hypochondriacal disorder.

Case series: A study of the treatment of a number of people that is normally evaluated with standardised instruments at different times such as before treatment, after treatment and at follow-up some time after treatment. Unlike controlled trials or cohort studies, there is usually no control or comparison group. Although useful in early studies of new treatments, they are not considered to be a rigorous test of a treatment,

Case study: A detailed description of the treatment of a single individual. Such studies may have an important role in the development of new treatments, but do not generally allow strong conclusions to be made about effectiveness.

Child: A person younger than 12 years of age.

Cognitive analytic therapy: A form of psychotherapy that is based on a combination of ideas from psychodynamic theory, with techniques from cognitive therapy. It involves looking at the individual's patterns of relating to others, as well as their behaviours and problems, which is based on a model called the Procedural Sequence Model.

Cognitive behavioural therapy (CBT): In the treatment of OCD and BDD, CBT generally combines elements of BT such as ERP and elements of CT such as

techniques to change beliefs about the things that they find distressing. For people with OCD this often involves reducing catastrophic thinking and the exaggerated sense of responsibility often seen in people with OCD. All CBT programs for OCD include an element of psychoeducation about obsessions and compulsions and a rationale for the interventions.

Cognitive therapy (CT): A psychological therapy and an umbrella term that covers a range of interventions that focus on altering/modifying unhelpful thoughts and behaviour. Typically it helps people to modify unhelpful negative cognitions (that is, interpretations, thoughts and beliefs) that lead to disturbing emotions, unhelpful behaviours and impaired functioning. For OCD, this may be about taking excessive responsibility or giving too much importance to obsessive thoughts. For BDD, this may be about altering extreme self-focused attention on a distorted body image and the meaning that is attached to the body image. People may be encouraged to test out the new ways of thinking through behavioural experiments, but CT does not usually rely on repeated exposure as in ERP.

Cohort study (also known as follow-up, incidence, longitudinal, or prospective study): An observational study in which a defined group of people (the cohort) is followed over time and outcomes are compared in subsets of the cohort who were exposed or not exposed, or exposed at different levels, to an intervention, or other factor of interest. Cohorts can be assembled in the present and followed into the future (a 'concurrent cohort study'), or identified from past records and followed forward from that time up to the present (a 'historical cohort study'). Because random allocation is not used, matching or statistical adjustment must be used to ensure that the comparison groups are as similar as possible.

Comorbidity: More than one diagnosis/disorder occurring in the same person.

Compulsions: Compulsions, sometimes known as rituals, are behaviours that people feel pressured to do to reduce anxiety, guilt and distress, or to prevent harm from occurring. Compulsions are often repeated, conducted according to strict rules, and time consuming. Although the goal may be to reduce anxiety, performing them can also lead to distress and frustration. The pressure to engage in these behaviours can prevent people from doing other things that they wish to do and cause significant impact on their lives and the lives of those around them. Compulsions can take almost any form but common forms include washing and cleaning, checking, hoarding, ordering and arranging, and repeated questions. Many compulsions are overt, that is, they could be observed by others, for example, hand washing rituals. However, other compulsions are covert, that is, they could not be seen by others because they are of a mental nature, for example, mentally repeating sentences. Although many people may try to resist these behaviours, they may find themselves unable to do so either because of the distress caused by resisting them or because they believe that the consequences of not performing the compulsion are unacceptably dangerous.

Confidence interval (CI): The range within which the 'true' values (for example, size of effect of an intervention) are expected to lie with a given degree of certainty (for example, 95% or 99%). Confidence intervals represent the probability of random errors, but not systematic errors or bias.

Controlled trial: An experiment in which investigators allocate eligible people into groups to receive or not to receive one or more interventions that are being compared.

Cost-effectiveness analysis: A type of full economic evaluation that compares competing alternatives of which the costs and consequences vary. The outcomes are measured in the same non-monetary (natural) unit. It expresses the result in the form of an incremental (or average or marginal) cost-effectiveness ratio.

Costs (direct): The costs of all the goods, services and other resources that are consumed in the provision of a health intervention. They can be medical or non-medical.

Costs (indirect): The lost productivity suffered by the national economy as a result of an employee's absence from the workplace through illness, decreased efficiency or premature death.

Counselling and supportive psychotherapy: A range of counselling methods are used in practice, including supportive, psychodynamic, and cognitive behavioural counselling. The most widely practiced form of counselling is supportive counselling/psychotherapy. This is defined as a way of relating and responding to another person, so that the person is helped to explore their thoughts, feelings and behaviour; to reach clearer self-understanding; and then is helped to find and use their strengths so that they cope more effectively with their lives by making appropriate decisions, or by taking relevant action. Counselling is essentially a purposeful relationship in which one person helps another to help him- or herself. In most cases it does not attempt to directly change the key features of OCD or BDD but can address a range of issues that may affect the individual.

Crossover study design: The administration of two or more experimental therapies one after the other in a specified or random order to the same group of patients.

Double-blind: A trial in which neither the participants nor the investigators (outcomes assessors) are aware of which intervention the participants are given. The purpose of blinding the participants (recipients and providers of care) is to prevent performance bias. The purpose of blinding the investigators (outcome assessors) is to protect against detection bias.

Drop out: A term no longer used to indicate leaving a study before its completion (the term 'leaving the study early' is now preferred).

Economic evaluation: Technique developed to assess both costs and consequences of alternative health strategies and to provide a decision-making framework.

Effectiveness: The extent to which a specific intervention, when used under ordinary circumstances, does what it is intended to do. Clinical trials that assess effectiveness are sometimes called management trials.

Efficacy: The extent to which an intervention produces a beneficial result under ideal conditions. Clinical trials that assess efficacy are sometimes called explanatory trials and are restricted to participants who fully co-operate. The randomised controlled trial is the accepted 'gold standard' for evaluating the efficacy of an intervention.

Exposure and response prevention (ERP): The person is encouraged to confront the feared object, situation, or thought that provokes anxiety (exposure) and resist engaging in the compulsive or other behaviour that would rapidly reduce anxiety (response prevention) until the anxiety gradually reduces of its own accord. For example, people with obsessions about contamination are encouraged to stay in contact with the 'germy' object (for example, handling money) until their anxiety decreases (habituates). Thus, through repeated exposure, the person is said to habituate so that the object, situation or thought no longer provokes anxiety and the urge to engage in the compulsive behaviour is no longer present. The ERP programme is usually conducted in a progressive way, starting with objects, situations, or thoughts that produce relatively low levels of distress. Exposure can be conducted in a variety of ways including *in vivo* (that is direct confrontation with the feared object or situation) and *in imagination* (where the person repeatedly imagines the feared object, thought, or situation). Taped scenarios can be used for exposure, especially when the cause of distress is particular obsessive thoughts. The technique can be used within structured therapy with the therapist present or with instructions from the therapist who is not present. It can also be used with support from a family member or other members of self-help groups, or alone as part of a pure self-help programme.

Eye movement desensitisation and reprocessing (EMDR): Originally developed for the treatment of trauma, patients are instructed to focus on a trauma-related image and its accompanying feelings, sensations and thoughts, while visually tracking the therapist's fingers as they move back and forth in front of the patient's eyes. After each set of approximately 20 eye movements, patients are instructed let go of the memory and to discuss their reactions. This process is repeated, and includes focusing on different memories that come up in connection with the trauma. Once distress is reduced, patients are instructed to focus on the target image while rehearsing a positive thought connected to the image.

Family therapy: A form of psychotherapy that is based on the idea that the behaviours of individuals and families is influenced and maintained by the way other individuals and systems interact with them both within and outside of the family. When

a member of the family has a problem that is persistent it can often dominate family life and impact significant on family function, interaction and communication. The aim of family therapy is to help family members recognise and understand how they function as a family and in particular how their patterns of interaction may have become organised around the symptoms or problems of one of their members. Where these patterns of interaction have become unhelpful and perhaps contribute to the maintenances of the problem, the family is helped to develop more functional patterns of organising and interacting within the family. Families are generally seen together but may at times be seen by the family therapists individually or with only some members of the family. Family therapy draws on a number of theoretical principles/approaches that may be used singularly or together in therapy. In the treatment of OCD various forms of family therapy are used, these include systemic, strategic, cognitive behavioural, and narrative family therapy although in practice these approaches tend to overlap:

- Systemic family therapy tends to focus on the meaning of OCD symptoms within the family unit. The therapy tends to see OCD symptoms as a sign that the family unit is stressed, leading to difficult, unspoken emotions between family members. A systemic treatment might involve the therapists exploring 'OCD stories' within a family, thus changing the way that the family members have co-created the meaning of OCD. This is aimed at improving relationships within the family and in turn, changing the meaning of the individual's symptoms to allow for changes in their behaviour.
- Strategic family therapy tends to focus on power issues within the family, which in turn may well impact on both family members' understanding and response to OCD, and the OCD symptoms themselves. The rationale is that a more flexible, creative family structure may reduce stress, and challenge the 'power' of the OCD symptoms.
- Cognitive behavioural family therapy (CBFT) as a treatment for OCD is based on the recognition that families of people with OCD become involved in trying to help manage the distress and the interference caused by the compulsions. The OCD thus disrupts family relationships. The focus of the treatment is to help the family understand how their well-intentioned involvement can inadvertently maintain the disorder and then help them withdraw from the compulsions. In some cases, especially with children, family members may act as co-therapists to help with ERP. Although the aim is to improve family relationships, the focus is more on reducing a particular individual's obsessive-compulsive symptoms in which other family members have become involved.
- Narrative family therapy proposes that individuals and families acquire certain stories about themselves which have the effect of filtering their experiences and thereby selecting what information gets focused on how it is understood and how this determines how individual problems within the family are perceived and addressed. Narrative family therapy is concerned with finding ways for families to develop new stories or expressions through which to enable them to change their lives.

Forest plot: A graphical display of results from individual studies on a common scale, allowing visual comparison of trial results and examination of the degree of heterogeneity between studies.

Good practice point (GPP): Recommended good practice based on the clinical experience of the Guideline Development Group.

Group therapy: A way of providing psychotherapy that usually involves one or more healthcare professionals and a group of patients. The patients are encouraged to understand their own and one another's difficulties with the goal of making changes. Most individual psychological therapies have also been provided in group format; for example, group cognitive behavioural therapy or group behaviour therapy

Guided self-help (GSH): A self-help programme for OCD in which a healthcare professional provides support, guidance and encouragement but does not take on the role of a formal therapist. Such programmes may use a self-help manual and the healthcare professional may work face-to-face, by telephone or by computer.

Heterogeneity: This occurs when there is more variation between the study results (in a systematic review) than would be expected to occur by chance alone.

Hypnosis: This involves giving the patient instructions (for example, 'focus on your right arm and on the sensation that it is getting lighter and lighter') to induce a state of highly focused attention, a reduced awareness of peripheral stimuli and a heightened responsiveness to social cues (suggestibility).

Marketing authorisation: A process in which the doses, indications, cautions, contraindications, and side effects of a drug are authorised for use by regulatory authorities. The decision to apply for a license for a patient condition depends on many factors. Drugs may be used outside their licensed indications.

Meta-analysis: The use of statistical techniques in a systematic review to integrate the results of the included studies. Also used to refer to systematic reviews that use meta-analysis.

Mindfulness: A meditation based approach to treatment that has been developed in particular for relapse prevention. Mindfulness has been defined as 'paying attention in a particular way: on purpose, in the present moment and non-judgmentally'. Mindfulness-based cognitive therapy aims to help patients make a shift in their relationship to thoughts, feelings and sensations, learning to perceive them as 'events in the mind' rather than as 'self' or necessarily true.

Monoamine-oxidase inhibitors (MAOIs): A group of drugs that act by inhibiting the enzyme monoamine-oxidase.

Odds ratio (OR): A measure of the relative benefit of the experimental treatment that can be obtained by dividing the experimental odds by the control odds.

Obsessions: Sometimes known as obsessive thoughts or ruminations, obsessions are unwanted and recurrent thoughts, doubts, or images that intrude into one's mind despite attempts to resist or control them. They may be fleeting thoughts, or they may stick in one's mind for long periods of time despite attempts to dislodge them. They are upsetting and may cause anxiety, guilt and shame. They usually lead to compulsions and/or avoidance (see above) as people try to remove or control the thoughts, deal with the situations that the thoughts refer to, or reduce the distress. The thoughts may be about almost any content, but common themes include dirt and contamination and their effects on self or others, harm that one may cause to others or failure to prevent harm from occurring, personally unacceptable blasphemous, immoral, or sexual thoughts, or excessive preoccupation with moral, religious or existential questions.

PANDAS (Paediatric Autoimmune Neuropsychiatric Disorder Associated with Streptococcus): Refers to research studies which show that in some individuals OCD appears to be triggered by streptococcal infection.

Patient: The terms 'patient' or 'person with OCD/BDD' are used in this guideline to identify the person presently or formerly with the condition and/or receiving services in the present or past. The term 'sufferer' is sometimes used by self-help groups.

Psychoanalysis: A school of psychology and a method of treating mental health problems that is based upon the teachings of Sigmund Freud (1856–1939). There are many different psychoanalytic theories of OCD. Obsessions and compulsions are seen as symptoms of some deeper problem in the person's unconscious mind. The compulsive acts and obsessional thoughts are seen as defensive reactions that suppress the real hidden anxieties.

Psychodynamic psychotherapy: Focuses on understanding the meaning of the target symptoms such as obsessions in the context of the individual's personality, attitudes and early experiences. The emphasis lies on resolving the unconscious conflicts that are thought to underlie the symptoms. Treatment strategies include exploratory insight-oriented, supportive or directive activity, working with transference, but with the therapist using a less strict technique than that used in psychoanalysis.

Psychoeducation: Educating people with OCD/BDD and their families about the symptoms of the condition, possible origins of the condition, its evolution over time, and the various treatments available. It also includes education about the symptoms and treatment of any comorbid disorders such as depression.

Quality of life (QOL): Used in some treatment studies to show improvement in a person's condition beyond reduction in symptoms, measures of QOL can be

defined broadly and include satisfaction, especially within important areas of one's life, the level of functioning in different areas, and the objective circumstances in which one lives. In many studies, however, QOL is defined narrowly as the level of functioning or degree of handicap which is one important aspect but limited as a marker of quality.

Randomisation: Method used to generate a random allocation sequence, such as using tables of random numbers or computer-generated random sequences. The method of randomisation should be distinguished from concealment of allocation, because if the latter is inadequate selection bias may occur despite the use of randomisation. For instance, a list of random numbers may be used to randomise participants, but if the list were open to the individuals responsible for recruiting and allocating participants, those individuals could influence the allocation process, either knowingly or unknowingly.

Randomised controlled trial (RCT): Also termed 'randomised clinical trial', this is an experiment in which investigators randomly allocate eligible people into groups to receive or not to receive one or more interventions that are being compared. The results are assessed by comparing outcomes from the different groups. Through randomisation, the groups should be similar in all aspects apart from the treatment they receive during the study.

Recommendation: A systematically developed statement that is derived from the best available research evidence, using predetermined and systematic methods to identify and evaluate evidence relating to the specific condition in question.

Relative risk (RR): Also known as risk ratio; the ratio of risk in the intervention group to the risk in the control group. The risk (proportion, probability or rate) is the ratio of people with an event in a group to the total in the group. A relative risk of 1 indicates no difference between comparison groups. For undesirable outcomes, an RR that is less than 1 indicates that the intervention was effective in reducing the risk of that outcome.

Relaxation: Relaxation therapy is aimed at teaching a patient to reduce the uncomfortable physical sensations that anxiety produces. Although there is a range of relaxation techniques, the most common one aims to help people relax by systematically tensing and relaxing various muscle groups. This can be used by itself to help people cope with stressful situations or as a part of desensitisation to specific fears. It is also a part of anxiety management training. In some trials of psychological treatments, relaxation has been used as a comparator condition.

Rituals: See *Compulsions*

Ruminations: *See Obsessions*

Selective serotonin reuptake inhibitors (SSRIs): Medicines that more selectively inhibit the reuptake of the neurotransmitter serotonin into the presynaptic neurone (see Serotonin reuptake inhibitors).

Self-help: This involves following a self-help programme for OCD, either on one's own (pure self-help), with the support of a group (self-help groups), or with support, guidance and encouragement from a mental health professional (guided self-help). Self-help programmes can use books, computerised materials, audio and videotapes. Effective programmes aim to improve self-management skills in addition to providing knowledge and information.

Self-help groups: Many people with health problems find it helpful to meet others with similar difficulties, for support, advice and social contact. These are usually run by people with the disorder, for people with the disorder (and sometimes their carers/families). Some groups also have professional support for some of their activities such as providing information.

Sensitivity analysis: Sensitivity analysis is a technique used in economic analysis or decision-making to allow for uncertainty by testing whether plausible changes in the values of the main variables affect the results of the analysis.

Serotonin reuptake inhibitors (SRIs): A group of drugs that act by inhibiting the neuronal reuptake of the neurotransmitter serotonin. These include SSRIs and other medicines such as clomipramine that act on other neurotransmitters as well as serotonin.

Stepped-care model: A sequence of treatment options aiming to provide the most appropriate and cost-effective interventions according to both patient need and locally available resources. When appropriate, simpler and less expensive interventions will be offered first, moving to more complex and intensive interventions if the patient has not benefited.

Sufferer: See patient

Tics: A tic is an involuntary, rapid, recurrent movement or sound. Examples of simple motor tics include eye-blinking and neck jerking. Simple vocal tics include throat clearing, barking noises, sniffing. Complex tics include jumping, or saying whole words. There is some suggestion that tics seem to be genetically related to OCD in that they can run in families; some people with OCD also have tics.

Tourette's syndrome (or Gilles de la Tourette syndrome): A chronic form of tic disorder where both vocal and motor tics have been present for a year or more. Some studies suggest that about half of people with Tourette's syndrome also have OCD although the opposite has not been shown.

Transcranial magnetic stimulation (TMS): An intervention involving the use of a pulsed magnetic field to induce changes in function in cortical structures. It was developed in 1985 (Barker *et al.*, 1985) to investigate the cerebral cortical activity and more recently has been used therapeutically for some mental disorders, namely, depression and OCD. In OCD this treatment aims to modify prefrontal cortical activity in order to influence obsessive–compulsive symptoms.

Treatment resistance: A relative failure to respond adequately to a treatment.

Uncontrolled trial: A treatment trial where no attempt is made to compare the investigated treatment with a matched comparator, either active or neutral placebo.

Vagus nerve stimulation (VNS): An intervention developed for the control of epilepsy that cannot be managed by normal medical treatment. It involves the electrical stimulation of the vagus nerve within the neck by an electrode connected to a programmable stimulator.

Virtual reality therapy: An intervention consisting of the use of computer technology to support ERP by exposing the patient to a virtual representation of the environment that contains the feared situation rather than taking the patient into the actual environment or having the patient imagine the stimulus.

Weighted mean difference (WMD): A method of meta-analysis used to combine measures on continuous scales (such as the Y-BOCS), where the mean, standard deviation and sample size in each group are known. The weight given to each study (for example, how much influence each study has on the overall results of the meta-analysis) is determined by the precision of its estimate of effect and, in the statistical software used by the NCCMH, is equal to the inverse of the variance. This method assumes that all of the trials have measured the outcome on the same scale.

Young person: A person between the ages of 12 and 18.