Heredity and Evolutionary Theory

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"With an eye only seeing particles, and a speech only symbolizing them, there is no such thing as a study of process possible"

Riddle 1909

Introduction

Evolution by natural selection is often described as the outcome of three conditions; variation among individuals in their characteristics (phenotypic variation), that different variants leave different number of descendants (differential fitness), and that individuals resemble their parents more than they resemble unrelated individuals (heredity) (Lewontin 1970,1985; Godfrey-Smith 2009). Heredity is therefore fundamental to evolutionary theory. If the characters of offspring bear no relationship to the characters of their parents, fitness differences between individuals will not cause systematic shifts in the distribution of phenotypes in the following generation. Natural selection would be powerless and cumulative adaptive evolution would be impossible. This makes it desirable that we have a firm mechanistic and conceptual understanding of what heredity is, and what are the consequences of variation in the mechanisms of heredity for phenotypic evolution.

Heredity is readily observed in nature and it was accepted by the earliest writers on reproduction (e.g., Aristotle; Lennox 2000). The mechanisms of heredity remained obscure, however. Darwin observed patterns of shared features among individuals that told him that those features were inherited, but he could not provide a satisfactorily explanation for the underlying process by which those patterns were generated. Given that Darwin nevertheless could present a strong case for adaptive evolution by means of natural selection, it may at first seem as if the details of heredity are not that important. As long as traits *are* heritable perhaps it doesn't matter *how* they are inherited? But in fact disagreement about evolution often stems from disagreement about heredity. Perhaps the most important reason for this is that some mechanisms of heredity can render natural selection a relatively minor contributor to organismal design. In particular, adaptive evolution could be greatly facilitated if organisms were able to acquire new functional traits during their ontogeny via use and disuse and pass on those characters to their offspring. The mechanism of heredity was therefore of major interest to evolutionary biologists following the publication of the Origin of Species (e.g., Darwin 1875; Galton 1876; Weismann 1893; Romanes 1895).

Heredity as transmission genetics

Empirical research at the end of the 19th century and the beginning of the 20th century established that the inheritance of acquired characters through use and disuse was rare at best, and the modern evolutionary synthesis effectively removed it from being part of respectable evolutionary theorizing (Mayr 1982; Sapp 1987). In fact, the success of the modern synthesis can partly be explained by a changing concept of heredity. Mendel's work on the inheritance of discrete traits, Weismann's separation of soma and germ line, Johannsen's distinction between genotype and phenotype, and Morgan's breeding experiments with fruit flies all contributed to making heredity synonymous with the passing of trait determinants from parents to offspring (Amundson 2005; Müller-Wille & Rheinberger 2012). Under this scheme, parent-offspring similarity is ascribed to the (stable and regular) transmission of genes between generations ('transmission genetics'). Heredity-as-transmission-genetics thus reduces the complex biological process of gamete formation, fertilization, and parent-offspring interactions to a single parameter of importance; the passing of alleles from one generation to the next. As a

consequence, heredity can be studied as a pattern without paying attention to developmental process[1].

Heredity-as-transmission-genetics has been immensely useful and successful in evolutionary biology. There are good reasons for this. Even if transmission genetics is an abstraction that leaves out most of the complexities of reproduction, individuals with similar genotypes tend to have similar phenotypes. The transmission of genes from parents to offspring – leading to similarity of genotypes – is therefore causally important for the phenotypic similarity of parents and offspring[2]. Assuming Mendelian inheritance it is possible to use crosses between individuals with known phenotypes to establish the number of genes involved in production of a particular phenotype, the location of those genes relative to the others, sex-linkage and so on. Stable transmission of genes also enables differences in phenotypes between lineages to be maintained down many generations, which is crucial for building and maintaining complex adaptations[3]. Finally, the transgenerational dynamics of genes within populations can be formalized in mathematical terms (i.e., population genetics; Fisher 1930). It is difficult to overstate the importance of population genetics for the development of evolutionary theory (Provine 1971, 1986). It provided a tool that could be used to show that natural selection can drive different genetic variants (and hence phenotypes) to fixation in different environments, maintain polymorphism within populations, that chance events can result in fixation of deleterious traits, that phenotypes that never reproduce still can be favoured if they help their relatives to reproduce, that even related individuals can sometimes benefit from harming each other, and that genetic architectures can bias evolutionary outcomes. Predictions from population genetic models could be tested in natural or experimental systems, often with satisfactory results (e.g., summary of Dobzhansky's work in Lewontin et al. 2003).

The success of transmission and population genetics notwithstanding, there has been, and continues to be, dissatisfaction with describing the evolutionary process in

[1] Morgan and his lab members were particularly important for generating widespread acceptance that heredity could be equated with transmission genetics, for example through the publication of 'The mechanisms of Mendelian heredity' (Morgan et al. 1915); see Amundson 2005, pp. 148-152 and Allen 1978.

[2] Transmission genetics can also explain why parent-offspring similarity does not always hold, for example, because of recessive alleles that make some traits occur only under some combinations of paternal and maternal genotypes.

[3] Vertical genetic inheritance also ensures that relatedness becomes highly correlated across the genome, which facilitates the ability to build cumulative adaptations, in particular with respect to social traits (Grafen 1985; West & Gardner 2013)

purely genetic terms. Firstly, although the fact that allelic similarity predicts phenotypic similarity is strong evidence that DNA is causally important in heredity, this does not mean that a gene-centric view allows a complete description of the inheritance of biological features. Secondly, even though population genetic theory is often able to predict patterns observed in the wild or in the laboratory, this does not mean that population genetics provides a complete description of the evolutionary process. To show that heredity can satisfactorily be reduced to genetic inheritance in evolutionary theory, it must be shown that non-genetic interactions between parents and offspring – what we will refer to as non-genetic inheritance (Bonduriansky & Day 2009) – do not contribute to the rate or direction of phenotypic evolution.

In this chapter we will address both of these issues. We will argue that we need a concept of heredity in biology that is not gene-centric. Firstly, we review why transmission genetics is causally and explanatory insufficient for both the inheritance of features and the inheritance of differences in features. Having established the need for a non-genetic contribution to heredity, we briefly outline three concepts of heredity in evolutionary biology that allow non-genetic inheritance (i.e., mechanisms in addition to the transmission of DNA): heredity-as-phenotypic-covariance, heredity-as-intergenerational-communication, and heredity-as-developmental-process. These perspectives each emphasize different aspects, and implications, of non-genetic inheritance for phenotypic evolution and we explain what these differences are. The last two sections expand on the role of non-genetic inheritance in evolution, firstly, using a general description of change within populations (the Price Equation) and, secondly, by discussing the role of non-genetic inheritance in the proposed 'extended evolutionary synthesis', which emphasizes evolutionary processes that were marginalized in the Modern Synthesis.

The explanatory insufficiency of transmission genetics

Heredity fundamentally refers to the like-begets-like phenomenon. A theory of heredity therefore needs to be able to explain the reliable recurrence of parental features in offspring. This includes characters that are shared among all members of a lineage, such as human-specific features, but also characters that differ between lineages within populations, such as hair colour. Mameli (2005) made this distinction by introducing the concepts of F-inheritance and D-inheritance, where the F stands for *F*eatures and

the D for *D*ifferences in features (see also Mameli 2004,2007). F-inheritance requires reference to the full set of mechanisms that result in recurrence of phenotypes down generations. Although genes obviously contribute to species-typical features, the fact that, for example, a lizard egg differs from the egg of a bird should make it obvious that transfer of DNA from parents to offspring is not causally sufficient to explain why offspring of lizards look like lizards rather than like birds[4]. Experimental transfer of DNA between eggs of closely related species has indeed demonstrated that some species-typical features are determined by egg content, not the zygotic DNA (e.g., Sun et al. 2005). Developmental biologists are increasingly revealing such maternal regulation of early development (e.g., Pelegri et al. 2003; Gilbert 2010; Li et al. 2010; see East 1934a,b and Sapp 1987 for reviews of the early literature). Similarly, development of species-typical behaviours require parent-offspring interactions that go well beyond the transmission of DNA (e.g., Moore 1995; Gottlieb 1997; Hood et al. 2010; Slagsvold & Wiebe 2011).

However, adaptive evolution requires transgenerational stability of phenotypic differences, not similarities. Consequently, evolutionary theory has focused on D-inheritance. This only requires reference to those mechanisms that contribute to the recurrence of phenotypic differences down generations. Some causal factors in development that contribute to F-inheritance (such as species-typical environments) can therefore be considered 'background conditions' under D-inheritance. It is possible to acknowledge the importance of non-genetic inheritance for heredity of features, while arguing that non-genetic inheritance is of no relevance for adaptive evolution (e.g., Dawkins 1982). Transgenerationally stable differences between lineages within populations are typically assumed to be due to genetic differences. This is not always the case, however. In a famous example, cross-fostering of rat pups between mothers of different parenting style (assessed by their licking and grooming behaviours) found that these differences are induced and maintained via behavioural interactions between the mother and her pups and not because of genetic differences (Francis et al 1999; Weaver et al. 2004). Other examples of non-genetic inheritance of behavioural phenotypes that can be maintained over several generations include differences in migration to breeding or overwintering sites (Harrison et al. 2008; Brown & Shine 2010), foraging and

[4] In fact, development typically fails if DNA is transferred from one species to the other unless the species are closely related.

exploration behaviours (Slagsvold & Wiebe 2011; Schuett 2013), and preferences for food (Robinson & Méndez-Gallardo 2010) and sexual partners (Freeberg 2000). In addition, there is increasing evidence that some epigenetic variants may be transmitted through the germ line and that this contribute to stable differences between lineages within populations (e.g., Roux et al. 2010; reviewed in Jablonka & Raz 2009; Jablonka 2012; Lim & Brunet 2013).

A common response to many of these examples is to argue that non-genetic inheritance can be reduced to genetic inheritance of parental traits and hence are best viewed as being genetically determined (e.g., Toyama 1913; Dobzhansky 1935; Dawkins 2004; Dickins et al. 2009; Dickins & Rahman 2012). It is of course true that genes are important for development of the parental phenotypes that 'transmits' non-genetic factors to offspring, and hence that a full causal explanation for the differences between lineages may also need to refer to the genetics of parents. However, this does not show that genes are causally prior to, or more important than, non-genetic components with respect to the reconstruction of life cycles. Arguing that it is would imply ascribing genetic causes a more important or fundamental role than non-genetic causes not only in evolution, but also in development. As has been pointed out by many authors, this position is not defensible (e.g., Oyama 1985; Nijhout 1990; Griffiths & Gray 1994; Godfrey-Smith 2000; Maynard-Smith 2000; Shea 2007; see Griffiths & Stotz 2013 for a recent summary).

A second counter-argument in favour of gene-centrism is that even if non-genetic inheritance in principle could contribute to the recurrence of phenotypic differences between lineages, those mechanisms do not allow cumulative adaptive change because they lack certain features that DNA exhibits. Important features of DNA that make it particularly useful as an inheritance system include stable replication during reproduction, potential for transmission of large ('unlimited') number of messages and modularity (Maynard Smith & Szathmáry 1995; Bergstrom & Rosvall 2011). These are good reasons for why genetic inheritance plays a fundamental role in evolutionary models. However, as the examples above show, other mechanisms of inheritance also enable stable inheritance of phenotypes. Thus, the difference between DNA and at least some non-genetic inheritance mechanisms is one of degree, not of kind (see e.g., Jablonka & Raz 2009 for discussion of tentative examples of epigenetically inherited phenotypes).

A gene-centric view of heredity therefore fails to hold up to scrutiny. This does not mean that transmission of genes is not important for the recurrence of phenotypic differences between lineages. Given that most populations probably harbour substantial amounts of genetic variation it likely is. Indeed, it remains an open question to what extent non-genetic inheritance contributes to stable inheritance of differences in phenotypes. Nevertheless, a complete explanation of both inheritance of features and inheritance of differences in features from one generation to the next will have to include all those mechanisms that contribute to parent-offspring similarity, and not just to the transmission of DNA.

Alternative Perspectives on Heredity in Evolutionary Theory

That many modern definitions of heredity refer specifically to the transmission of genes (Table 1) reflects that for many biologists transmission genetics is not just a heuristic that makes modelling the evolutionary process feasible, it is an accurate and satisfactory description of the mechanism of heredity for the vast majority of organisms (humans often excluded because of our extensive capacity for culture). But as we have seen transmission genetics cannot be taken to be causally or explanatory sufficient for hereditary phenomena. This suggests that we need an alternative concept of heredity in evolutionary biology. Here we will briefly discuss three ways to conceptualize heredity that does not force heredity to be (exclusively) a genetic phenomenon. In the following sections we discuss to what extent these perspectives are able to capture how non-genetic inheritance contribute to the evolutionary process.

Heredity as Phenotypic Covariance

Lewontin's (1970,1985) three necessary conditions for evolution by natural selection that opened up this chapter imply that adaptive evolution does not rely on particulate inheritance, but that offspring resemble their parents more than they resemble unrelated individuals. Rather than treating the relationship between parents and offspring as transmission of discrete particles, we can treat it statistically in terms of the

covariance between the phenotypes of parents and offspring[5]. The covariance between two random variables, X and Y, is defined as

$$\mathrm{cov}(X,Y) = E\left[\left(X - E[X]\right)\left(Y - E[Y]\right)\right]$$

$$= E[XY] - E[X]E[Y]$$

Where $E[X]$ represents the expected value for variable X.

Using this statistical measure of covariance between the average phenotype of the parents (known as the mid-parent phenotype) and the phenotype in the offspring gives us the breeder's equation for change in a phenotype, z, from one generation to the next,

$$\Delta z = \frac{\mathrm{cov}(z_o, z_p)}{\mathrm{var}(z_P)} \mathrm{cov}(w, z_p) ,$$

where subscripts denote phenotypic values in parents, *p*, and offspring, *o*, respectively (see e.g., Falconer & Mackay 1996; Rice 2004 for mathematical details). The covariance between phenotype values in parents and offspring divided by the total phenotypic variance in parents is equal to the slope of the regression of offspring phenotype on midparent phenotype (i.e., β_{z_o, z_p}), which is also equal to the heritability, usually denoted h^2 (Rice 2004). This is multiplied by the covariance between phenotype and fitness, which is known as the selection differential. The selection differential measures the change in phenotype due to differential survival or reproduction. The breeder's equation, typically written as $R = h^2 S$, shows that natural selection can be effective, i.e., can cause a change in the average phenotype from one generation to the next, as long as the covariance between parents and offspring is non-zero. This equation occurs in virtually all textbooks on evolution.

The concept of heredity as a phenotypic covariance is representative of the field of quantitative genetics (Falconer & Mackay 1996; Rice 2004). However, in quantitative genetics, heritability is not always defined as a regression slope, but instead as the ratio of additive (roughly equal to 'transmitted') genetic variance over total phenotypic variance, i.e., $h^2 = \frac{V_A}{V_P}$. The additive genetic variance cannot be observed directly, but it can be estimated by comparing traits in relatives (e.g., parents and offspring, half- and

[5] The use of a statistical approach to heredity has a long history that goes back to Galton (e.g., Galton 1876) and the biometricians that clashed with the Mendelians about the nature of heredity following rediscovery of Mendel's work; see Provine 1971 for a historical account.

full-sibs). The focus on additive genetic variance is a consequence of a gene-centric definition of heredity, and it does not imply that the additive genetic variance divided by the total phenotypic variance accurately captures how differences in fitness will translate into phenotypic change from one generation to the next. In fact, the covariance between the phenotype of parents and offspring is only equal to the additive genetic variance when the phenotype is determined additively by genes and environment and there are no correlations between, for example, the genotype of the parent and the environment of the offspring or the environment of parents and offspring (Lewontin 1974; Rice 2004). Thus, the additive genetic variance of a phenotype is only an estimate of the covariance between the phenotype of parents and offspring.

This insight has some important consequences for understanding the evolutionary implications of non-genetic inheritance. Firstly, any mechanism that allows covariance between phenotypes of parents and offspring can contribute to heritability. This means that it should also be possible to empirically estimate additive epigenetic variance, additive behavioural variance, and so on (Tal et al. 2010; Furrow et al. 2011; Danchin et al. 2011). Together these sum up as the total additive, transmitted, variance (which when divided by the total phenotypic variance represents a quantity termed inclusive heritability by Danchin & Wagner 2010). Secondly, offspring phenotype is not only determined by the additive components of inheritance and its own environment, but also by aspects of the phenotype of its parent that are not 'transmitted' additively. This means that some of the variation in offspring phenotype can be statistically attributed to non-additive genetic and non-genetic variation in parental phenotypes, which can cause the covariance between parents and offspring to be negative despite that a negative heritability is not possible under the additive genetic variance definition of heritability. Empirical studies suggest that a substantial proportion of variance in traits in natural populations can be ascribed to variation in parental, in particular maternal, phenotypes ('parental effects'; e.g., Mousseau & Fox 1998; Maestripieri & Mateo 2009).

Quantitative genetic models that incorporate parental effects were first developed in the field of animal breeding (e.g., Dickerson 1948; Willham 1963). Over the last decades those models have been put to use for addressing how phenotypes evolve in the presence of non-genetic mechanisms that contribute to the covariance between parental and offspring phenotypes (e.g., Cheverud 1984; Kirkpatrick & Lande

1989; Moore et al. 1997; recent reviews in Cheverud & Wolf 2009; Hadfield 2012). These models show that parental effects can affect the rate and direction of evolution, which has been confirmed empirically in natural populations of animals (e.g., McAdam & Boutin 2004; Wilson et al. 2005). The expansion of quantitative genetic models to include epigenetic inheritance, which focuses on the resetting and environmental-dependence of epigenetic marks, is ongoing (e.g., Johannes et al. 2008; Tal et al. 2010).

In summary, the heredity-as-phenotypic-covariance perspective treats (at least in principle) all mechanisms that contribute to the covariance between parental and offspring phenotypes as mechanisms of heredity. It is thus conceptually different from heredity-as-transmission-genetics both in that it does not assume particulate heredity and that it does not assume that DNA is causally or explanatory sufficient for the inheritance of phenotypic differences. Nevertheless, quantitative genetic models often assume genetic inheritance only and reduce the relationship between parents and offspring to a single parameter of evolutionary relevance; heritability, h^2, estimated as the ratio of additive genetic variance over phenotypic variance. More recent models that relax this assumption by allowing parental effects show that non-genetic inheritance can have evolutionary consequences, both for the rate and direction of phenotypic change.

Heredity as Intergenerational Information Transfer

Heredity is often described as the passing of information between generations. This is true for population genetic models (e.g., Frank 2009), quantitative genetic models that allow non-genetic inheritance (e.g., Danchin et al. 2011), cultural evolution models (Feldman & Cavalli-Sforza, 1981; Boyd & Richerson, 1985) and for general discussions about non-genetic inheritance (e.g., Jablonka & Lamb 2005)[6]. The use of information language suggests that heredity could be seen as a form of communication between parents and offspring (Bergstrom & Rosvall 2011; Shea 2012). Consequently, inheritance mechanisms could be seen as adaptive features that contribute to the fit between organism and environment by allowing parents to transmit information about the state of the world to their offspring, thereby enabling offspring to match their phenotype accordingly. Effects of the parental phenotype on offspring phenotype are

[6] For example, two of the strongest proponents of non-genetic inheritance, Eva Jablonka and Marion Lamb, tend to define inheritance in terms of information transmission, see e.g, the Prologue in *Evolution in four dimensions* (Jablonka & Lamb 2005).

306 often interpreted in this way in behavioural ecology, where they are referred to as
307 maternal (or parental) effects (adopting the terminology from quantitative genetics[7])
308 (Uller et al. 2013). However, the number of theoretical models that explicitly take an
309 information perspective on the evolution of non-genetic inheritance is still limited.
310 The rationale for treating inheritance as parent-offspring communication is that
311 mechanisms of inheritance can carry correlational information about the state of the
312 world (Jablonka 2002; Shea 2011; Shea et al. 2011; Bergstrom & Rosvall 2011).
313 Correlational information is found whenever some entity's being in a particular state
314 changes the probability that some other entity is in another particular state. For
315 example, the presence of smoke increases the probability that there is a fire nearby. As
316 Figure 1 shows, different mechanisms of inheritance can also carry information about
317 the state of the world. For DNA, correlational information can arise because DNA is
318 transmitted down generations unchanged (with the exception of mutations) which
319 enables natural selection to build up statistical correlations between genotypes and
320 environments (Fig 1). Thus, the passing of DNA from parents to offspring also passes
321 information about the historical state of the environment, which makes selection an
322 information-generating process (Kimura 1961; Frank 2009). DNA is not the only
323 information-carrying entity in heredity, however. Parental phenotypes can also carry
324 information about the state of the environment that the offspring are likely to
325 experience (Fig. 1). For example, maternal hormone levels during breeding can carry
326 information about the quality of the local habitat (e.g., Tschirren et al. 2007). Offspring
327 could capitalize on this information if there are mechanisms that enable development of
328 alternative phenotypes, e.g., dispersive versus non-dispersive behaviour, in response to
329 hormone exposure in utero or in the egg yolk. Parents (the signaller) may also evolve to
330 increase the information content of the hormonal signal, for example, by modification of
331 the timing, strength or duration of their endocrine response to appropriate cues
332 (Badyaev & Oh 2008) [8]. Other mechanisms of non-genetic inheritance, including

[7] It is a potential source of confusion that maternal effects in quantitative genetics refer to a proportion of variance in offspring phenotype attributed to variation in maternal phenotype (which can further be divided into variation due to genetic and environmental differences between mothers), whereas in behavioural ecology it tends to refer to a causal, potentially adaptive, effect of the maternal phenotype on offspring phenotype. Recently, quantitative geneticists have suggested that a causal rather than statistical definition should be adopted (Wolf & Wade 2009), which largely avoids this problem. For more on the relationship between parental effects and 'non-genetic inheritance' see Bonduriansky & Day 2009; Uller 2012.

[8] Parents could also change the state of the world to fit the offspring phenotype (Odling-Smee et al. 2003; see below).

epigenetic and behavioural mechanisms, can carry information in similar ways (Shea et al. 2011).

[INSERT FIG 1 ABOUT HERE]

The information perspective on heredity thus establishes a difference between mechanisms of inheritance that is based on the underlying cause(s) for mechanism *X* to carry correlational information about future environmental state *Y*. Shea et al. (2011) named these two ways by which inheritance mechanisms carry information selection-based – when the information is generated through selection on stably transmitted variants – and detection-based – when the parent responds to an adaptively relevant feature of the environment in ways that communicate the state of the world to the offspring (Fig. 1). The distinction between selection-based and detection-based information transmission helps to evaluate some claims regarding the evolutionary function of non-genetic inheritance. Specifically, it shows that even if different mechanisms of heredity can be on a par in terms of their causal effects on development, they need not be on a par with respect to their role in evolution (Shea 2011). Several authors have pointed out that DNA is very good at storing and transmitting an arbitrary sequence and hence that it may have been under selection for its ability to generate long-run heredity of selected phenotypes (Maynard-Smith & Szathmary 1995; Bergstrom & Rosvall 2011). This would make DNA an inheritance system in a more strict sense than, say, maternal hormones that are less able to sustain consistent lineage differences in phenotypes down generations (partly because they are so sensitive to context). However, not only DNA is an inheritance system in this strict sense. Any mechanism that enables variants to be faithfully passed on can result in selected-based information (Shea 2011). For example, some epigenetic variants are replicated with sufficiently high fidelity to suggest that they acquire information through a selective process (Jablonka & Raz 2009). More complex interactions between parents and offspring also enable information to be generated in the same way, as in instances of behavioural imitation where complex parental behaviours are faithfully replicated in offspring (Weaver et al. 2004; Jablonka & Lamb 2005; Shea 2009). On this account, DNA plays a special, but not unique, informational role in heredity and evolution (Shea 2011).

In summary, considering heredity as transmission of information between generations emphasizes an important role for both genetic and non-genetic inheritance. In contrast to heredity-as-phenotypic-covariance, which focuses on the evolution of phenotypes subject to different forms of inheritance, heredity-as-intergenerational-communication emphasizes the adaptive evolution of inheritance mechanisms themselves[9]. However, both perspectives single out statistical properties of parent-offspring relations as the key to understanding evolutionary phenomena. In contrast, our last perspective on heredity attempts to explain heredity in a causal-mechanistic way.

Heredity as Developmental Process

Until the 18th century, heredity and development were not seen as the two distinct processes that are so entrenched in biological thinking today (Amundsen 2005; Müller-Wille & Rheinberger 2007, 2012). In fact, their separation has been hailed as a crucial step in advancing evolutionary theory (e.g., Mayr 1982). However, some biologists and philosophers of biology view the distinction with suspicion. Advocates of a developmental perspective, like those ascribing to 'developmental systems theory' (DST; Oyama 2000; Oyama et al. 2001), tend to view all causal mechanisms that contribute to parent-offspring similarity as inheritance in a broad sense. They thus view heredity not as transmission of adaptively tuned information through discrete channels, but as the entire process of reconstruction of life cycles to which the parents contribute (Griffiths & Gray 2001; Oyama 2000; 2001 Ch 4; Badyaev 2011). This of course involves the replication and transmission of DNA to the gametes, but it also includes all non-genetic interactions that contribute to the reliable recurrence of phenotypic development down generations.

This developmental perspective on heredity is associated with several major conceptual differences compared to the standard narratives of development and evolution. Perhaps the most conspicuous is a rejection of the notion that some aspects of the organism can be considered to be due to nature and some due to nurture (Oyama 2000; Moore 2013). If life cycles are reconstructed, one cannot point to any single causal mechanism and say that it is prior to, or more fundamental than, the rest of the

[9] Note that selection is not always concordant across generations and hence that there is potential for 'parent-offspring conflict' (Trivers 1974), which can influence the evolution of non-genetic inheritance (e.g., Uller & Pen 2011).

developmental system (Oyama 1985; Gray & Griffiths 1994; Laland et al. 2013). Developmental system theorists thus reject that genes have a privileged role as causes in development, which implies they are also not privileged as causes of heredity. Heredity cannot be reduced to the transmission of traits; what is transmitted are the developmental means that enable the reconstruction of life cycle,s and this includes more than DNA (Oyama 2000). Selection is suggested to be redefined to 'changes in the distribution of developmental systems that occur when traits are differentially associated with different lineages and the variants interact with their environment in ways that confer on them different probabilities of being perpetuated' (Oyama 2001, p. 81). Natural selection can generate adaptations because the organism itself contributes to the reconstruction of life cycles, which enables cumulative evolution of organism-environment complexes. Evolution is not defined genetically but instead as any changes in the composition of the developmental systems that enable perpetuation of life cycles (Oyama 2001, Ch. 4).

Considering heredity a developmental process extends the role of non-genetic inheritance beyond heritability and information transmission. Specifically, it raises the possibility that non-genetic mechanisms of heredity also can contribute to the origin of novelties (Badyaev 2008,2009; Rice 2012). This can be due to changes in genes that contribute to parental transfer of developmental resources ('maternal effect genes'; Gilbert 2009, Ch. 6) but, as West-Eberhard (2003) and others have argued, environmentally induced phenotypes may have even greater evolutionary potential. Responses to novel environmental input are often accommodated in functional ways ('phenotypic accommodation') and, in contrast to DNA mutations, can affect many individuals within a single population. If the ability to respond is heritable selection can result in the spread and further modification of environmentally induced phenotypic accommodations. Such heritable variation will often be due to genetic differences between individuals[10], but the retention and spread of new variation can also be due to non-genetic inheritance, including epigenetic and behavioural mechanisms (e.g., by offspring imitation of parental behaviour). This raises the possibility that heredity itself evolves as a result of increasing stabilization of life cycles under natural selection (Badyaev 2009), but to our knowledge this process has not yet been formally modelled.

[10] This is often described as variation among individuals in the slope of reaction norms (Schichtling & Pigliucci 1998). Lande (2009) presents a quantitative genetic model of evolution following environmental change that explicitly considers genetic variation in norms of reaction.

In summary, it is possible to reject a fundamental distinction between heredity and development and consider heredity as the developmental process by which offspring come to resemble their parents. This may have some potentially radical consequences for evolutionary theory. In contrast to heredity-as-phenotypic-covariance and heredity-as-information-transmission, heredity-as-developmental-process emphasizes the origin of adaptations in addition to their spread within populations, and downplays the adaptive function of inheritance in terms of information transmission. Indeed, developmental systems theorists are deeply suspicious of the notion that any mechanism of inheritance can usefully be considered to transmit information (e.g., Oyama 1985).

Heredity in Evolutionary Theory Revisited

The four perspectives on heredity that we have discussed look quite different (Table 2), and it is not immediately obvious how they are related. A comprehensive analysis is beyond the scope of this chapter. Our aim is instead to clarify the evolutionary implications of non-genetic inheritance and the explanatory sufficiency of different perspectives on heredity in two ways. Firstly, we will show that even mechanisms that generate transient or partial inheritance are of evolutionary significance and that those mechanisms can contribute to phenotypic evolution even if they do not affect parent-offspring covariance or adaptively transmit information. Importantly, this analysis also helps to clarify differences and similarities between different mechanisms of inheritance, which we exemplify by discussing epigenetic and ecological inheritance. Secondly, we explain what is needed of a concept of heredity to encompass changes in the structure of evolutionary theory that are associated with calls for an extended evolutionary synthesis. Both of these exercises points towards considering heredity a developmental process and we end with a brief discussion of the consequences of this for evolutionary theory.

Heredity and the Price Equation

We have seen that the breeder's equation shows that an evolutionary response to selection requires a covariance between parental and offspring phenotypes. Non-genetic inheritance can contribute to this covariance in both the short (e.g., parental effects) and long-term (e.g., 'epialleles'), and could therefore be important for predicting

how populations evolve. This formulation of evolutionary change is based on some simplifying assumptions, however. Here we will therefore start with a general description of change in average phenotype between two populations (e.g., ancestors and descendants) (see also Helanterä & Uller 2010; Day & Bonduriansky 2011; Uller & Helanterä 2013). This is known as the Price Equation, which can be written as

$$\Delta\bar{z} = \frac{1}{\bar{w}}\left[\operatorname{cov}(w, z) + E(w\Delta z)\right] \qquad (1)$$

where $\Delta\bar{z}$ is the change in the average phenotype in the population, $\bar{w}$ is the mean number of descendants per individual, $\operatorname{cov}(w, z)$ is the covariance between fitness and trait value, and $E(w\Delta z)$ is the expected value of the product of fitness and the average phenotypic difference between parent and offspring in the absence of selection (Price 1970,1972). The last two terms can be interpreted as the change due to differential reproduction and survival and the change that occurs as a result of reproduction and the mechanisms of inheritance, respectively (Rice 2004; it is important to note that the expected value is also affected by external changes between generations that cause shifts in phenotypes, such as environmental change). Division by the mean number of descendants means that fitness is relative and not absolute.

Because the Price Equation does not make any assumptions of the underlying mechanisms of parent-offspring similarity it can be used to derive the standard single-locus and quantitative genetic formulations of evolutionary change (Rice 2004). However, none of the terms in Eq. (1) specifically refer to the covariance between parental and offspring phenotypes, which makes this difficult to see. We will therefore rewrite the equation as

$$\Delta z = \frac{1}{\bar{w}}\left(\beta_{z_o,z} Cov(w, z) + E(\Delta z) + Cov(w, z_o \,|\, z)\right) \qquad (2)$$

This decomposes change in population mean phenotype into three components (Fig. 3; see Heywood 2005; Lynch & Walsh 2013 for mathematical details on how to get from Eq. 1 to Eq. 2). The first term in the parenthesis on the right hand side consists of $\beta_{z_o,z}$, the best linear slope of the parent-offspring regression or the heritability of the phenotype (using selected parents; Heywood 2005), times the covariance between fitness and trait value, i.e., the selection differential. Thus, if the remaining terms are zero, this equation corresponds to the breeder's equation discussed above (Falconer &

Mackay 1996). The second term represents the expected phenotypic change in the absence of fitness differences, which is often referred to as transmission bias (e.g., Heywood 2005). Mathematically this is represented by the intercept of the parent-offspring regression and could be, for example, changes that occur because of intergenerational environmental change that affects phenotypic development (Rice 2004). The third term is the covariance between the residuals for the regression of fitness on parental phenotype and for the residuals for parent-offspring regression (named 'spurious response to selection' by Heywood 2005) (Fig. 2). Why would the residuals covary? As shown in Figure 3, this can happen because when we describe evolutionary change in this form of the Price Equation we are forcing the slope of the regression to be linear (Lynch & Walsh 2013, Ch. 13). But this is not always true (e.g., Gimmelfarb 1968; see Rice 2012). If the relationship is non-linear, the residuals are biased across parental phenotypic values (Fig. 2). Because fitness may also show non-linear relationships with phenotype, the residuals may be correlated and hence $Cov(w, z_o \mid z)$ may be non-zero. An alternative reason for why $Cov(w, z_o \mid z)$ could be non-zero is that, even if one or both regressions are linear, the residuals are correlated via a third variable (Heywood 2005). Heywood (2005) discusses a case with breeding date in birds, where there is a spurious response to selection even when both regressions are linear. This is because a third variable, nutritional status, covaries with both the residuals of breeding date on fitness and the residuals of parental breeding date on offspring breeding date.

[INSERT FIG 2 ABOUT HERE]

In summary, Equation 2 describes the change in the population mean phenotype from one generation to the next in terms of the product of the parent-offspring regression ('heritability') and the covariance between phenotype and fitness ('selection differential'), and two terms that can be affected by mechanisms of inheritance and environmental effects ('transmission bias' and 'spurious response to selection'). Quantitative genetics typically assume that the last two terms are zero (hence the breeder's equation), but they may be non-zero even under pure genetic inheritance (Heywood 2005; Lynch & Walsh 2013). Explicitly addressing how the mechanisms of heredity and development influences each of the components of the Price Equation

helps to establish the consequences of different forms of inheritance for phenotypic evolution. For example, by separating evolutionary change into that produced by genetic and non-genetic inheritance, Day and Bonduriansky (2011) developed a series of models that exemplify how different non-genetic inheritance mechanisms can affect evolution via their effects on phenotypic covariance and transmission bias. Furthermore, Rice (2008, 2012) has shown how a stochastic version of the Price Equation makes explicit the importance of non-genetic inheritance for evolution because of its effect on the shape of the parent-offspring phenotype distribution. Here we will exemplify these points by discussing two 'inheritance systems', epigenetic inheritance and ecological inheritance.

Epigenetic inheritance

Epigenetic inheritance, such as DNA methylation, differs from genetic inheritance in several ways (Jablonka & Lamb 1995,2005). Although epigenetic variants can be reliably inherited through meiosis in some multicellular organisms, their stability appears to be relatively short lived compared to transmission of DNA sequence variation. Epigenetic variation can be environmentally induced but, unlike DNA mutation (which can also vary across environments), a broader range of environments are apparently able to modify epigenetic states, perhaps in non-random directions. (Rando & Verstepen 2007; Jablonka 2012a). The degree to which offspring pass on the same 'epiallele' as they received from their parents can therefore depend on the similarity of environments across generations, the parental phenotype, and perhaps the epigenetic state itself (Jablonka & Lamb 2005; Richards 2006; Jablonka 2012b; Uller 2012).

From Equation 2 we can see that epigenetic mechanisms can contribute to phenotypic change in several ways. Firstly, direct transmission of epigenetic variants means that epigenetic mechanisms can cause offspring to resemble their parents, i.e., that the (linear) slope of regression of offspring phenotype on parental phenotype is non-zero. Thus, as mentioned above, epigenetic mechanisms contribute to the overall heritability of a character (e.g., Tal et al. 2010; Furrow et al. 2011; Danchin et al. 2011). The long-term consequences of epigenetic inheritance will depend on the stability of these variants and their pattern of inheritance (e.g., non-Mendelian), which itself can be a function of phenotypic and environmental change. For example, Day and

Bonduriansky (2011) have shown that differences in the stability of epialleles can affect both evolutionary trajectories and equilibria of genotype and phenotype values within populations (see also Geoghegan & Spencer 2012).

Secondly, the environment-dependence of epigenetic inheritance is likely to cause significant transmission bias, which makes $E(\Delta z)$ non-zero as well (Helanterä & Uller 2010; Day & Bonduriansky 2011). This affects the predicted evolutionary change from one generation to the next because epigenetic inheritance, or environmental epigenetic effects, means that phenotypes can change more or less predictably from one generation to the next even in the absence of parent-offspring covariance (e.g., due to a common plastic response in the population). This shows that mechanisms that are shared among all lineages of the population, but do not contribute to inheritance of differences in phenotypes, nevertheless have consequences for how populations evolve.

Finally, epigenetic inheritance may be more likely than genetic inheritance to generate a spurious response to selection. The stochastic nature of epigenetic inheritance and its dependence on the phenotypic character state of the parent and the environment may make it more likely that there will be non-linear relationships between parent and offspring phenotype or biased distribution of residuals of the regressions in Figure 2. Non-linearity could actually be common whenever there are parental effects as they tend to skew the distribution of phenotypes from that expected under additive genetic variance (Rice 2012). For example, biased transmission stability of DNA methylation may result from passive loss of methylation with age. This can result in a spurious response for both reasons mentioned above (Fig. 2). Firstly, it could lead to non-linear relationships between phenotypes in parents and offspring and hence residuals may become correlated even in the absence of a causal link. Secondly, age may covary both with the residuals of fitness for a focal trait (e.g., older individuals may be more experienced and thus have higher breeding performance for a given trait value) and the residuals of the parent-offspring regression (e.g., older parents may be less likely to transmit the same epigenetic mark as they themselves inherited because of stochastic loss of DNA methylation with age). This line of reasoning suggests that establishing the pattern of parent-offspring similarity (e.g., if it is linear), and its underlying mechanism (e.g., if there is environment-specific transmission of epigenetic states), is an important task if we are to understand and predict the extent to which

589 epigenetic mechanisms contribute to short- and long-term evolution (Day &
590 Bonduriansky 2011; Rice 2012).
591
592 *Ecological inheritance*
593 Parents (or more generally ancestors) do not only 'transmit' resources to their offspring
594 (descendants), they also modify the environment that the offspring encounter by
595 choosing nest sites, constructing burrows, and so on ('niche construction'; Odling-Smee
596 et al. 2003). As a result, the environmental context of the offspring is partly determined
597 by the phenotypes of the parents, a phenomenon that has been termed 'ecological
598 inheritance' (Odling-Smee et al. 2003). Just like epigenetic inheritance, the evolutionary
599 consequences of ecological inheritance for a particular phenotypic trait (not necessarily
600 the niche constructing trait itself) can appear through its effect on the linear slope of the
601 parent-offspring regression ('heritability'), its intercept ('transmission bias'), or the
602 covariance between the residuals of the two regressions in Figure 2 ('spurious
603 response'). Firstly, when ecological inheritance causes environmental similarity within
604 lineages, but maintains environmental differences between lineages, it increases the
605 parent-offspring covariance of phenotypes that show environmental-dependence in
606 their expression, and hence heritability (e.g., Furrow et al. 2011). Such effects are
607 probably common in organisms where resources are unevenly distributed and passed
608 on to offspring, as occurs in species where offspring take over the territory of their
609 parents. Secondly, even without differences among lineages within populations, the
610 collective actions of organisms can make the environment change in ways that influence
611 offspring development. For example, as population densities increase, more frequent
612 encounter rates with other individuals can stimulate development of more aggressive
613 behaviours, which could result in directional shifts in aggression even in the absence of
614 parent-offspring covariance. Human niche construction is also a good example of such
615 transmission bias, since collective cultural inheritance may underlie directional changes
616 in many traits independently of a direct response to selection (e.g., changes in body size,
617 sexual behaviours, language use; Boyd & Richerson, 1985; Laland & O'Brien 2010).
618 Thirdly, ecological inheritance could cause $Cov(w, z_o \mid z)$ to be non-zero. For example, the
619 deviation from the expected value of offspring size from parental size may be a function
620 of the available resources as determined by territory quality. If larger individuals
621 benefit disproportionally from having good territories, the residuals from a size-fitness

622 relationship could also be biased across territory qualities. Ecological inheritance of
623 territories would thus cause a spurious response to selection. Of course, ecological
624 inheritance may also cause non-linear relationships between parents and offspring that,
625 as outlined above, can cause residuals to correlate even in the absence of a causal link.
626 An added complexity with ecological inheritance is that its definition[11]
627 emphasizes another mechanism by which it can have evolutionary consequences.
628 Specifically, the niche constructing activities of organisms do not only change the
629 'transmission terms' of the Price Equation, but also potentially cause $Cov(w,z)$ to be a
630 function of z, or other phenotypic traits, in immediate or more distant ancestors. For
631 example, assume that we follow the evolutionary change in a specific phenotype, P, that
632 originally has a mean value of z. Further assume that the trait is heritable and that
633 phenotypic covariance is only due to genetic variation and that there is no transmission
634 bias. The trait would predictably evolve in response to natural selection according to
635 equation 2 with $E(\Delta z) = 0$ and $Cov(w, z^0 | z) = 0$. Now assume that the average trait
636 value in the population affects the external environment in ways that does not affect
637 how P is inherited, but that change what value of P that has the highest fitness. For
638 example, if z is large the environment in the next generation could deteriorate, which
639 favours a lower z in the next generation. But when z is low the environment may
640 improve, which favour a large z in the next generation. That the average trait value in a
641 population would have strong effects on selection across two generations is probably
642 unlikely, but it illustrates the importance of ecological inheritance for the dynamics of
643 covariance between phenotype and fitness (Laland et al, 1996, 1999; Odling-Smee et al.
644 2003). Importantly, traits do not evolve in isolation. This means that evolution of one
645 particular character can contribute to the dynamics of $Cov(w,z)$ for other characters,
646 which implies that predicting evolutionary dynamics will often be difficult and require
647 explicit consideration of trait interactions and the spatial structure of populations
648 (Silver & Di Paolo 2005).

[11] Odling-Smee et al. 2003 (p.42) define ecological inheritance as 'any case in which organisms encounter a modified feature-factor relationship between themselves and their environment where the change in selective pressures is a consequence of the prior niche construction by parents or other ancestral organisms'. Odling-Smee 2010 (p.180-181) defines ecological inheritance as occurring 'whenever the environmental consequences of prior niche constructing activities of organisms (e.g., the presence of burrows, mounds, and dams or, on a larger scale, changed atmospheric states, soil states, substrate states, or sea states) persist or accumulate in environments as modified natural selection pressures, relative to successive generations of organisms'.

This example suggests that 'ecological inheritance' does not only refer to parent-offspring resemblance, i.e., how most biologists would understand the terms inheritance or heredity, but also to patterns of fitness, i.e. selection. Thus, the term 'ecological inheritance' can be somewhat misleading since it contributes to phenotypic evolution in so many ways; (i) it could affect heritability (regression slope) of a particular phenotype (D-inheritance), (ii) it could affect the expected change in the trait in the population in addition to the response to selection via transmission bias or spurious responses caused by niche constructing activities (which can include F-inheritance *sensu* Mameli 2005), and (iii) it could affect the covariance between phenotype and fitness of future generations. All of these are potentially important, but not easily captured by the term 'inheritance'. The diversity of effects on the evolutionary process that stems from ecological inheritance may be representative also for the evolutionary implications of other non-genetic systems of inheritance. Indeed, although the effect on selection is emphasized for the term ecological inheritance, it is important to note that other forms of genetic and non-genetic inheritance also modify covariance between phenotype and fitness of the subsequent generation (Badyaev & Uller 2009). More generally, because what matters in evolution is how fitness of parents relates to the distribution of phenotypes in offspring, any developmental process that influences either fitness or parent-offspring distributions can be evolutionarily consequential (Rice 2004; 2012). Non-genetic mechanisms of inheritance clearly have the capacity to do both.

Heredity and the Extended Evolutionary Synthesis

The development of the Modern Synthesis did not only adopt a genetic definition of heredity and evolution. It also came with a shift in what was considered to be sufficient as an *evolutionary explanation*. Whereas previous debates often centred on how novel adaptive characters can arise from existing characters (e.g., 'the arrival of the fittest'; de Vries 1904), the reduction of heredity to the transmission of genes implied that one can study evolution without reference to development (e.g., Mayr 1961). In fact, phenotypes take on a limited role in this Modern Synthesis view of evolution, which is well exemplified by the redefinition of evolution as cross-generational change in gene frequencies, and not in phenotypes (Dobzhansky 1937). The notion of the genome as a blueprint and Mayr's introduction of the distinction between proximate and ultimate causes (Mayr 1961) further cemented the view that non-genetic mechanisms could not

do any explanatory job in evolutionary biology, but rather should be seen as execution of functions encoded in the genome (see Dawkins 2004; Haig 2007; Dickins & Dickins 2008; Scott-Phillips et al. 2011; Dickins & Rahman 2012 for recent versions of this argument and Mesoudi et al. 2012; Laland et al. 2011,2013 for criticism; see also Bonduriansky 2012; Uller 2013).

These views are at odds with contemporary evolutionary biology that emphasizes the importance of the developmental origin of novel, potentially adaptive, variants, the many processes that may promote their recurrence and spread, and what maintains the ability for further evolution. This includes discussions of the role of plasticity as an initiator of evolutionary change in novel environments (e.g., West-Eberhard 2003), the importance of developmental bias promoting directional and perhaps lineage-specific evolutionary trajectories (e.g., Arthur 2004), how organism-environment interactions contribute to selective dynamics in time and space (e.g., Odling-Smee et al. 2003), and how developmental mechanisms influence evolvability (e.g., Stern 2009). Together these advances in evolutionary thinking have suggested to some authors that there is a new 'extended evolutionary synthesis' emerging (see Pigliucci & Müller 2010 for an attempt to summarize these arguments).

The conceptual structure of such an extended evolutionary theory – if it ever is realised – is debated (Pigliucci & Muller, 2010). However, a fundamental feature is that it is a theory of phenotypes rather than of genotypes. Treating heredity as a process makes it take centre place in evolutionary scenarios that involve developmental plasticity or developmental bias. In fact, we suggest that an extended synthesis necessitates a shifting concept of heredity, away from transmission genetics and towards the reconstruction of life cycles. A consequence of this shift in emphasis is that it makes explicit that non-genetic inheritance – the transference from parents to offspring of developmental resources that contribute to the reconstruction of life cycles – enters evolutionary theory through all three of Lewontin's conditions. Non-genetic inheritance contributes to the origin of variation (condition one) and those variants are inherited because the parents reconstruct the developmental niche for the offspring in recurrent 'cycles of contingency' (Oyama et al. 2001) (condition three), and not only because they transfer genes. This parental transference of developmental resources does not only affect offspring phenotype and its recurrence within populations, it also contributes to the relationship between phenotype and survival or reproductive success

(Lewontin's second condition). As ecological inheritance in particular makes explicit, selection, or an absence of selection, partly arises from the actions of ancestors (Odling-Smee et al. 2003). It is a major focus of the extended evolutionary synthesis to establish how selection arises through the interactions between organism and environment. Both genetic and non-genetic inheritance will affect what, and in what form, phenotypic accommodations to novel genetic and environmental input are recurrent down generations. Because induction of phenotypic variation in offspring through non-genetic inheritance has been channelled through a responsive phenotype (i.e., the parent), this may further enhance the functionality of such variation (Badyaev 2009). Once expressed, parental transference of developmental resources can facilitate or increase transgenerational persistence of induced phenotypes, for example via behavioural mechanisms of inheritance (Badyaev & Uller 2009; Uller 2009, 2012;). For example, cross-fostering of great and blue tit chicks suggests that a suite of species differences in sexual preferences and foraging behaviour may have originated, spread and been maintained as culturally inherited phenotypes (Slagsvold & Wiebe 2011). In the extended evolutionary synthesis view on evolution, therefore, some adaptive phenotypes may initially be inherited through mechanisms with low fidelity, and hence exhibit a low parent-offspring covariance, and only gradually become stably inherited as combinations of genes and non-genetic components of parent-offspring interactions that increase this covariance are accumulated under stabilizing selection (West-Eberhard, 2003; Badyaev 2009,2011).

Summary and Outlook

Heredity is fundamental to evolution. We have argued that it cannot be reduced to genetic inheritance and that the causal-mechanistic perspective offered by heredity-as-developmental-process is the only of the four concepts of heredity that we have discussed that also is causally and explanatory sufficient in evolutionary biology. This perspective recognises that recurrence of phenotypes within lineages, and differences among lineages, requires reference to the recurrence of both genetic and non-genetic causes of *development*. Heredity occurs precisely because parents transfer a variety of developmental resources that enable reconstruction of life cycles and hence phenotypes. Non-genetic inheritance refers to this transference, which is mediated through a variety of epigenetic, physiological, and behavioural mechanisms. These

concepts of heredity and inheritance avoid the problems inherent in a gene-centric perspective and allows for a richer understanding of the reasons for why offspring resemble their parents. Importantly, it shows that mechanisms of non-genetic inheritance contributes to all three of Lewontin's (1970, 1985) conditions for evolution by natural selection.

Treating heredity as a developmental process makes non-genetic inheritance fundamental to a phenotype-oriented evolutionary framework. What ultimately matters for phenotypic evolution is the relationship between the fitness of parents and the phenotypes of their offspring (Rice 2012). All the mechanisms by which parents contribute to the reconstruction of life cycles can potentially affect the origin, fitness, and inheritance of phenotypes. The Price Equation helps to establish how genetic and non-genetic inheritance contributes to intergenerational phenotypic change (Day & Bonduriansky 2011; Uller & Helanterä 2013). Non-genetic mechanisms of inheritance can affect the regression of offspring phenotype on parental phenotype (i.e., heritability). But we have seen that this is not all there is to transgenerational phenotypic change. Non-genetic inheritance can cause transmission bias and spurious responses to selection, partly by causing non-linear parent-offspring relationships. Thus, 'parental effects' should not be treated as confounding environmental noise, but instead as a real cause for parent-offspring resemblance that can have evolutionary consequences at both short and long time scales. Non-genetic inheritance can also link the phenotypes in one generation with selection in future generations (Odling-Smee et al. 2003), which makes the covariance between phenotype and fitness dynamic and evolving rather than a static property This is the fundamental point of niche construction theory and shows that niche construction is essentially a developmental process occurring in an ecological context (Odling-Smee 2010; Odling-Smee et al. 2013). Although these complexities can make the mathematics complicated, recent research has shown that non-genetic inheritance (and development more generally) can be incorporated into formal models of phenotypic evolution (e.g., Feldman & Cavalli-Sforza, 1976; Rice 2004, Ch. 8; Rice 2008a,b; Day & Bonduriandsky 2011).

Does this mean that we should abandon transmission genetics in our evolutionary models? We believe not. It remains entirely valid to use abstraction in theoretical model building. Reducing the complexity of heredity to transmission of genes will remain a useful way to model the evolutionary process. This is not surprising

781 considering that DNA has features that make it particularly well suited for long-run
782 heredity. But it is an abstraction that leaves out some important features that can affect
783 evolutionary dynamics. One therefore needs to be aware that by reducing inheritance to
784 transmission genetics, one leaves out potentially important evolutionary processes.
785 This is not unusual in evolutionary biology. For example, it is acknowledged that
786 phenotypic optimality models do not account for the underlying genetics (Grafen 1984),
787 which makes it necessary to treat them with caution. Confusing the biological
788 mechanisms of heredity with a heuristic abstraction, i.e., transmission genetics, may
789 have delayed recognition of the importance of development (including non-genetic
790 inheritance) in evolutionary theory (Rice 2012). Non-genetic inheritance is not a just a
791 proximate mechanisms of no evolutionary significance on its own, it is an essential part
792 of the reconstruction of life cycles on which evolution relies (Badyaev & Uller 2009;
793 Griffiths & Stotz 2013).

794 Our discussion also sheds doubt on the value of interpreting all forms of
795 inheritance as transmission of information. Information emerges through the
796 contingencies of development and evolution. That inheritance mechanisms carry
797 information is therefore a derived state, a consequence of the adaptive evolution of life
798 cycles. This can make it useful to explicitly link inheritance and information because it
799 provides a condition (or maximand) for evaluating the adaptive value of different
800 mechanisms of inheritance[12]. However, it may also detract from the importance of non-
801 genetic inheritance in the origin and recurrence of novel characters through
802 developmental plasticity.

803 These final points suggest to us that treating heredity as a developmental
804 process invites a pluralistic stance with respect to how heredity is treated in formal
805 models. But it also implies that non-genetic inheritance cannot be fully integrated into
806 evolutionary theory without an integration of development and evolution. In fact, we
807 suggest that a wider concept of inheritance is a necessary and fundamental component
808 of the extended evolutionary synthesis. At the very least, as our understanding of the
809 role of developmental processes in evolution becomes more sophisticated, the part of

[12] English et al. (in review) shows how adaptive evolution of developmental switches tends to maximize mutual information between phenotype and environment. This maximization can occur through differential response to inherited genes, non-genetic inheritance, or direct environmental input, which are all on a par in terms of their effect on development even if the processes that causes them to carry information differs. See also Shea et al. 2011.

those processes that underlie heredity should take on a more central role in evolutionary theory (Badyaev & Uller 2009; Odling-Smee 2010; Rice 2012; Griffiths & Stotz 2013).

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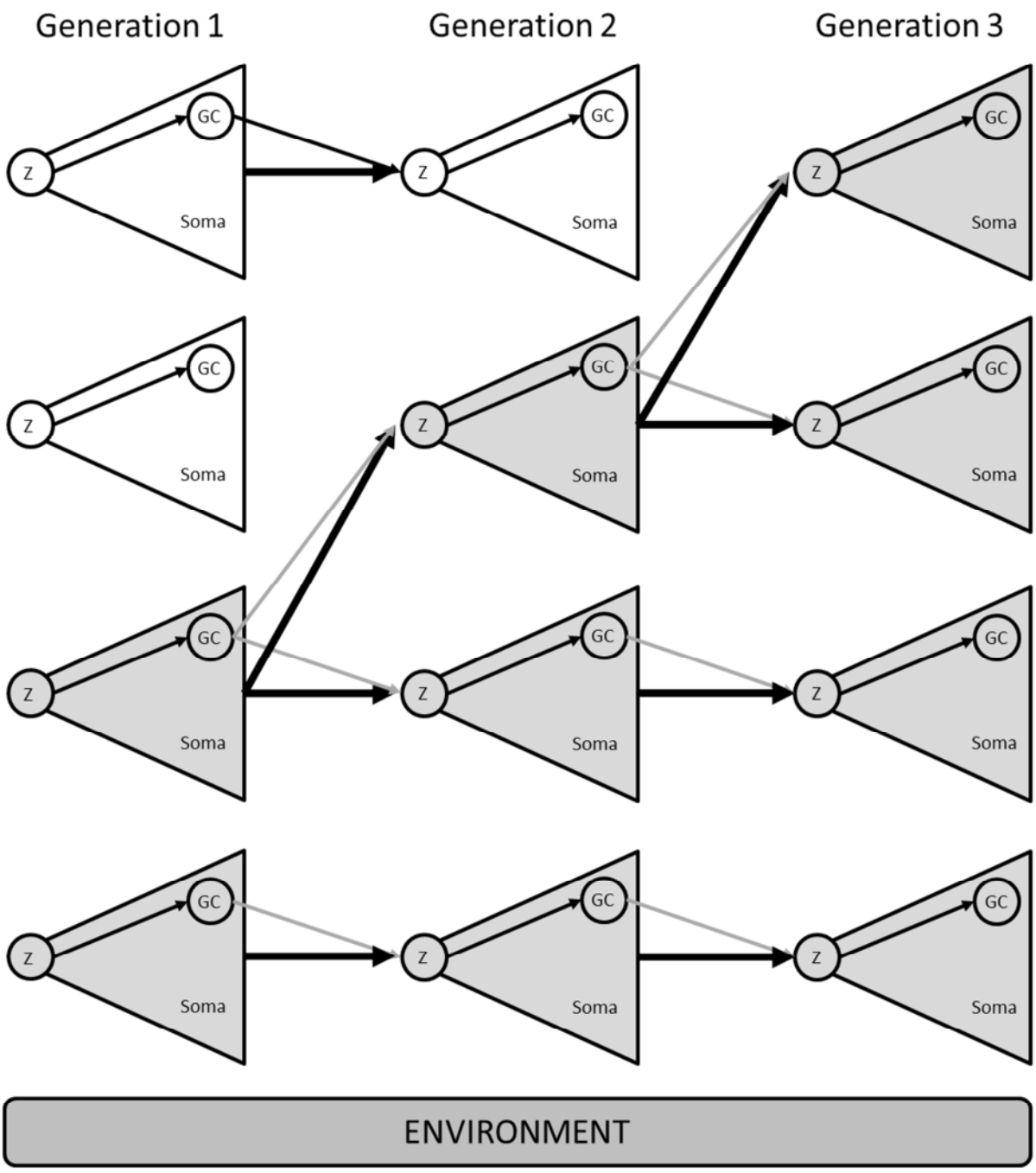
Generation 1
Generation 2
Generation 3
GC
Z
Soma
ENVIRONMENT

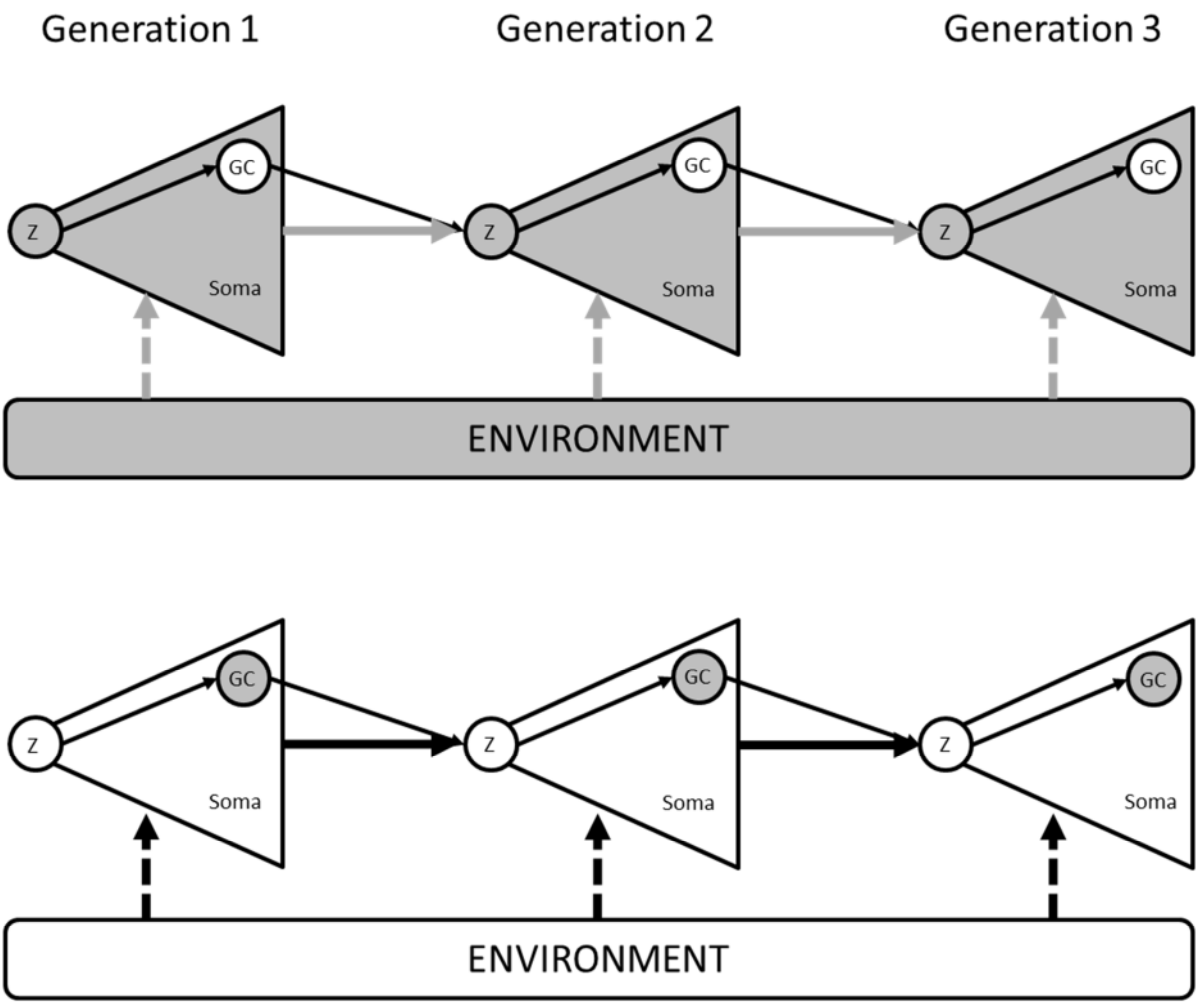

Figure 1. Illustration of the difference between selection-based and detection-based information transmission between generations. Development begins with a zygote (z), which gives rise to germ cells (GC) and Soma in the next generation. Both the germ cells and the soma of the parent contribute to the zygote of the next generation. White and grey colours denote different phenotypes that are caused by corresponding differences in germ cells or soma and that affect the development of the subsequent generation (black vs grey arrows). A) *Selection-based information transmission.* Stably inherited differences, here in germ cells (GC; e.g., DNA or epigenetic variation), generate differences in phenotypes (grey vs. white). Grey phenotypes have higher fitness, which causes grey germ cells to increase in frequency and hence establish a correlation

between germ cell type and environment. B) *Detection-based information transmission*. Individual responses to the environment result in correlations between the phenotype and the selective context, independently of the colour of the germ cells. These differences in parental phenotypes can be exploited by development through non-genetic mechanisms of inheritance, resulting in transgenerationally stable phenotypes within environments.

Figure 2

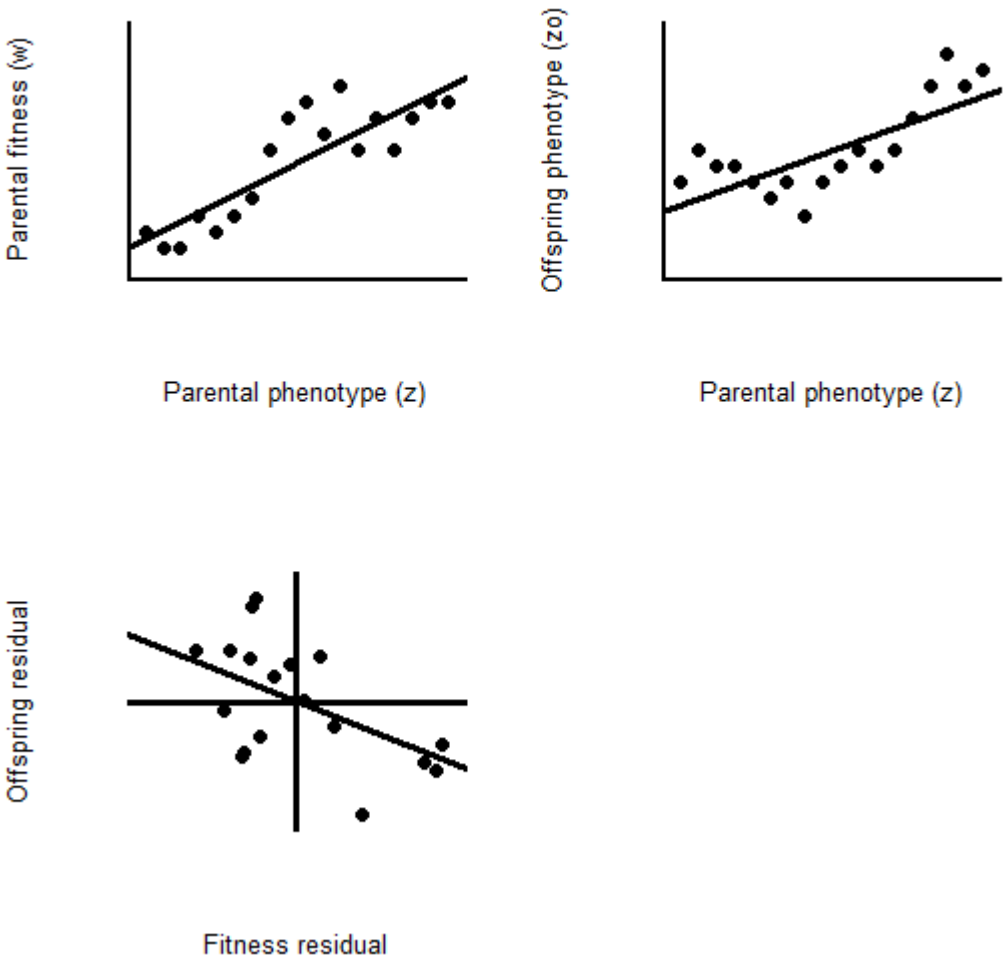

Figure 2. Illustration of a situation that gives rise to a 'spurious response to selection'. The relationship between parental phenotype and fitness (top left) and the relationship between parental and offspring phenotype (top right) are non-linear. As a result, the residuals for the best fitting linear regressions are non-random. This results in a negative covariance between the residuals (bottom graph), which is the spurious response to selection, $Cov(w, z^0 \mid z)$, in Equation 3. Figure from Uller & Helanterä (2013), originally adopted with modifications from Heywood (2005) and Lynch & Walsh (2013).

872 Table 1. Contemporary dictionary definitions of heredity*.

Definition	**Source**
A. The sum of the characteristics and potentialities genetically derived from one's ancestors B. The transmission of such qualities from ancestor to descendant through the genes	**Merriam-Webster Dictionary (**http://www.merriam-webster.com/dictionary)
The passing on of physical or mental characteristics genetically from one generation to another	**Oxford Dictionary (**http://oxforddictionaries.com)
A. The transmission from one generation to another of genetic factors that determine individual characteristics: responsible for the resemblances between parents and offspring B. The sum total of the inherited factors or their characteristics in an organism	**Collins English Dictionary (**http://www.collinsdictionary.com)
A. The genetic transmission of characteristics from parent to offspring, B. The sum of characteristics and associated potentialities transmitted genetically to an individual organism.	**Free Online Dictionary (**http://www.thefreedictionary.com)
A familial phenomenon wherein biological traits appear to be transmitted from one generation to another. [..] heredity results from the transmission of genes from parents to offspring [and] offspring therefore tend to resemble their parents […] rather than unrelated individuals	**King, R.C., Mulligan, P.K. & Stansfield, W.D. 2013. *A dictionary of genetics*, 8th ed. Oxford University Press.**
The transmission of characteristics from parents to offspring via the chromosomes.	***Oxford Dictionary of Biology*, 5th ed. 2005. Oxford University Press.**
A. The genetic constitution of an individual B. The transmission of genetically-based characteristics from parents to offspring	**Lawrence, E. (ed). 2008. *Henderson's dictionary of biology*, 14th ed.Pearson Education Ltd.**

873 * The *Dictionary of developmental biology and embryology* (2nd ed., Dye, F.J. 2012) does not include
874 heredity or inheritance. However, it refers to inheritance of acquired characteristics as 'the hypothesis
875 that phenotypic changes in the parental generation can be passed on, intact, to the next generation; may
876 have a mechanism if the inherited morphological alteration can be mediated by epigenetic changes in the
877 DNA methylation of germ cells.

878 Table 2. Summary of the four different perspectives on heredity in evolutionary biology discussed in this chapter. NGI = Non-genetic
879 inheritance.

	Heredity as transmission genetics	Heredity as parent-offspring covariance	Heredity as intergenerational information transfer	Heredity as developmental process
Representative research community/approach:	Evolutionary population genetics	Evolutionary quantitative genetics	Behavioural ecology	Developmental systems theory
NGI is considered as:	Parentally transferred instructions for development under genetic control	Source of variation in offspring phenotype ('parental effects')	Cues or resources that enable adaptive transfer of information across generations	Components of the reconstruction of life cycles that contribute to transgenerational stability and variation of phenotypes
Research emphasis concerning NGI:	A*daptive evolution of* non-genetic inheritance	E*volution of traits subject to* non-genetic inheritance	A*daptive evolution of* non-genetic inheritance	E*volutionary transitions between variation – retention- stabilization of* life cycles

Primary role of NGI in adaptive evolution:	None or as a genetic adaptation	Affect the response to selection by affecting parent-offspring covariance	Mechanism that facilitates adaptation to heterogeneous environments	Contribute to the development, selection, and heredity of phenotypes

880

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