

The impact of alcohol on health

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Introduction

Apart from being a drug of dependence, alcohol has been known for many years as a cause of some 60 different types of disease and condition, including injuries, mental and behavioural disorders, gastrointestinal conditions, cancers, cardiovascular diseases, immunological disorders, lung diseases, skeletal and muscular diseases, reproductive disorders and pre-natal harm, including an increased risk of prematurity and low birth weight ([Anderson & Baumberg, 2006](#)). In recent years, overwhelming evidence has confirmed that both the volume of lifetime alcohol use and the combination of frequency of drinking and amount drunk per incident increase the risk of alcohol-related harm, largely in a dose-dependent manner ([WHO Regional Office for Europe, 2009](#); [Rehm et al., 2010](#)) with the higher the alcohol consumption, the greater the risk. For some conditions, such as cardiomyopathy, acute respiratory distress syndrome and muscle damage, harm appears only to result from a sustained level of high alcohol consumption, but even at high levels, alcohol increases the risk and severity of these conditions in a dose-dependent manner. The frequency and volume of episodic heavy drinking are of particular importance for increasing the risk of injuries and certain cardiovascular diseases (coronary heart disease and stroke). Although there is a protective effect of light to moderate drinking on ischaemic diseases, overwhelmingly alcohol is toxic to the cardiovascular system.

Alcohol is an intoxicant affecting a wide range of structures and processes in the central nervous system which, interacting with personality characteristics, associated behaviour and sociocultural expectations, are causal factors for intentional and unintentional injuries and harm to both the drinker and others. These injuries and harm include interpersonal violence, suicide, homicide and drink-driving fatalities. Alcohol consumption is a risk factor for risky sexual behaviour, sexually transmitted diseases and HIV infection. Moreover, it is a potent teratogen with a range of negative outcomes to the fetus, including low birth weight, cognitive deficiencies and fetal alcohol disorders. It is neurotoxic to brain development, leading to structural changes in the hippocampus in adolescence and reduced brain volume in middle age. Alcohol is a dependence-producing drug, similar to other substances under international control. The process of dependence occurs through its reinforcing properties and neuroadaptation. It is also an immunosuppressant which increases the risk of communicable diseases, including tuberculosis. Further, alcoholic beverages and the ethanol in them are classified as carcinogens by the International Agency for Research on Cancer.

Alcohol as a carcinogen

In 2007, the International Agency for Research on Cancer concluded that there was a causal link between alcohol and cancer of the oral cavity, pharynx, larynx, oesophagus, liver, colon, rectum and female breast ([Baan et al., 2007](#); [IARC, 2010](#)). All these cancers showed evidence of a dose-response relationship; that is, the risk of cancer increases steadily with greater volumes of drinking ([Rehm et al., 2010](#)). The strength of the relationship to levels of average alcohol consumption varies for different cancers. For example, with regard to female breast cancer, each additional 10 g of pure alcohol per day is associated with an increase of 7% in the relative risk of breast cancer, whereas regular consumption of approximately 50 g of pure alcohol increases the relative risk of colorectal cancer by 10–20%, indicating that the association is stronger for female breast cancer. Conversely, the relationship of average consumption to cancer of the larynx, pharynx and oesophagus is markedly higher than the relationship to both breast and colorectal

cancer (more than a 100% increase for an average consumption of 50 g pure alcohol per day). Among the causal mechanisms that have been indicated for some cancers is the toxic effect of acetaldehyde, which is a metabolite of alcohol.

Cardiovascular disease

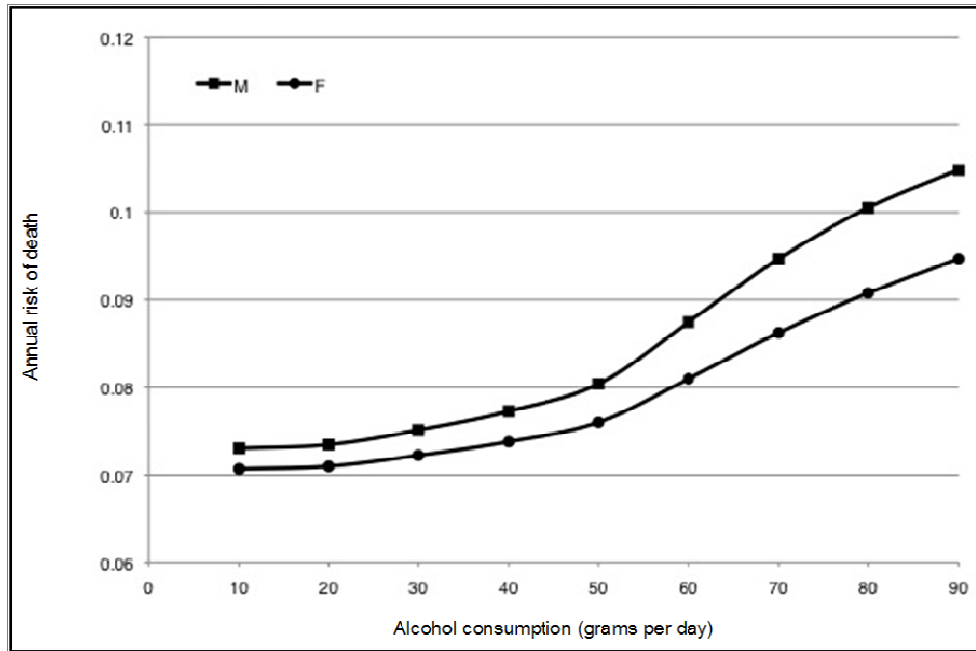
Alcohol use is related overwhelmingly detrimentally to many cardiovascular outcomes, including hypertensive disease (Taylor et al., 2009), haemorrhagic stroke (Patra et al., 2010) and atrial fibrillation (Samokhvalov, Irving & Rehm, 2010). For ischaemic heart disease and ischaemic stroke, the relationship is more complex. Chronic heavy alcohol use has been associated uniformly with adverse cardiovascular outcomes (Rehm & Roerecke, 2011). But, on average, light to moderate drinking has a protective effect on ischaemic diseases (Roerecke & Rehm, in press). This effect is found to be equal for people who just drink beer or who just drink wine (Di Castelnuovo et al., 2002). More and more, however, it is being understood that a large part of this effect is due to confounders (Roerecke & Rehm, 2010), with low to moderate alcohol use being a proxy for better health and social capital (Hansel et al., 2010). In any case, the protective effect totally disappears when drinkers report at least one heavy drinking occasion per month (Roerecke & Rehm, 2010); there is no protective effect for younger people, for whom any dose of alcohol increases the risk of ischaemic events (Juonala et al., 2009); and, in older people, a greater reduction in death from ischaemic heart disease can be more effectively obtained by being physically active and eating a healthier diet than by drinking a low dose of alcohol (Mukamal et al., 2006). The detrimental effects of heavy drinking occasions on ischaemic diseases are consistent with the physiological mechanisms of increased clotting and a reduced threshold for ventricular fibrillation which occur following heavy drinking (Rehm et al., 2010).

Death

It is mostly the middle-aged (and men in particular) who die from alcohol (Jones et al., 2009; Rehm, Zatonski & Taylor, 2011). Taking into account a lifecourse view, however, the adolescent brain is particularly susceptible to alcohol, and the longer the onset of consumption is delayed, the less likely that alcohol-related problems and alcohol dependence will emerge in adult life (Norberg, Bierut & Grucza, 2009). The absolute real risk of dying from an adverse alcohol-related condition increases linearly with the amount of alcohol consumed over a lifetime, with no safe level (Rehm, Zatonski & Taylor, 2011). In many societies there is no difference in the risks between men and women. Australians who regularly drink six drinks (60 g of alcohol) a day over their lives as adults have a 1 in 10 chance of dying from alcohol (National Health and Medical Research Council, 2009).

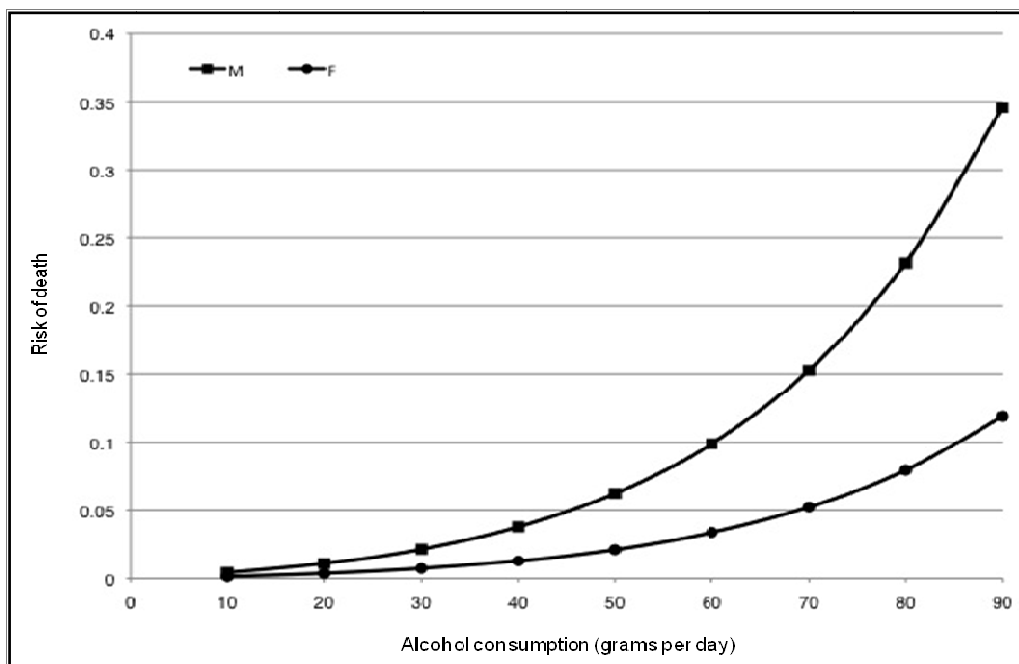
The annual absolute risk of dying from an alcohol-related disease (accounting for the protective effect of ischaemic diseases) for people aged over 15 years across the population of the WHO European Region is shown in Fig. 1. The risks increase from the consumption of 10 g alcohol a day (one drink, the lowest data point) so that at a consumption of 60 g/day, men have a just under 9% annual risk of dying from an alcohol-related disease and women an 8% risk. At any given level of alcohol consumption, men are at greater risk than women. The lifetime risk of dying from an alcohol-related injury across the total population aged over 15 years rises exponentially with an increasing daily alcohol consumption beyond 10 g of alcohol per day, the first data point (Fig. 2). At any given level of alcohol consumption, the risks are much higher for men than for women.

Fig. 1. Absolute annual risk of death from alcohol-related diseases^a



^a Absolute annual risk of death from alcohol dependence, liver cirrhosis and alcohol-related cancers and cardiovascular diseases, net of protective effects, from drinking a certain average amount of alcohol daily from 10 g alcohol/day to 90 g/day, age-standardized for adults aged over 15 years for the WHO European Region (Source: Taylor, Rehm & Anderson, 2010, personal information).

Fig. 2. Life-time risk of death from alcohol-related injuries^a



^a Absolute lifetime risk of death from alcohol-related intentional and unintentional injuries from drinking a certain average amount of alcohol daily from 10 g alcohol/day to 90 g/day, age-standardized for adults aged over 15 years for the WHO European Region (Source: Taylor, Rehm & Anderson, 2010, personal information).

Social circumstances

People who are socially disadvantaged people or who live in socially disadvantaged areas experience more harm per gram of alcohol consumed than the better-off (Rehm et al., 2009). In Finland, areas with higher levels of manual workers or of unemployment and areas with lower

social cohesion had higher levels of alcohol-related mortality among men aged 25–64 years (Blomgren, Martikainen & Makela, 2004). In the same way, social networks matter. Changes in alcohol consumption among a person's social network have a significant effect on that person's subsequent behaviour, in terms of not drinking (when more of the network abstain) or of drinking heavily (when more of the network drink heavily) (Rosenquist, Murabito & Fowler, 2010).

Conclusions for policy and practice

The following conclusions should be helpful for policy and practice.

- The risk of death from an alcohol-related illness or injury rises with increasing alcohol consumption.
- At 20 g of alcohol consumed on average per day or per drinking occasion per day (at least for the Australian population), the lifetime risk of death from an acute or chronic condition is less than 1 in 100.
- For a given level of alcohol consumption, people from lower socioeconomic groups are at increased risk of an alcohol-related death, compounded by living in areas with a higher degree of disadvantage.
- Incentives need to be implemented (Anderson et al., 2011) that make it easier for individuals to drink less alcohol per day and per occasion (Anderson, Harrison & Cooper, 2011).

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