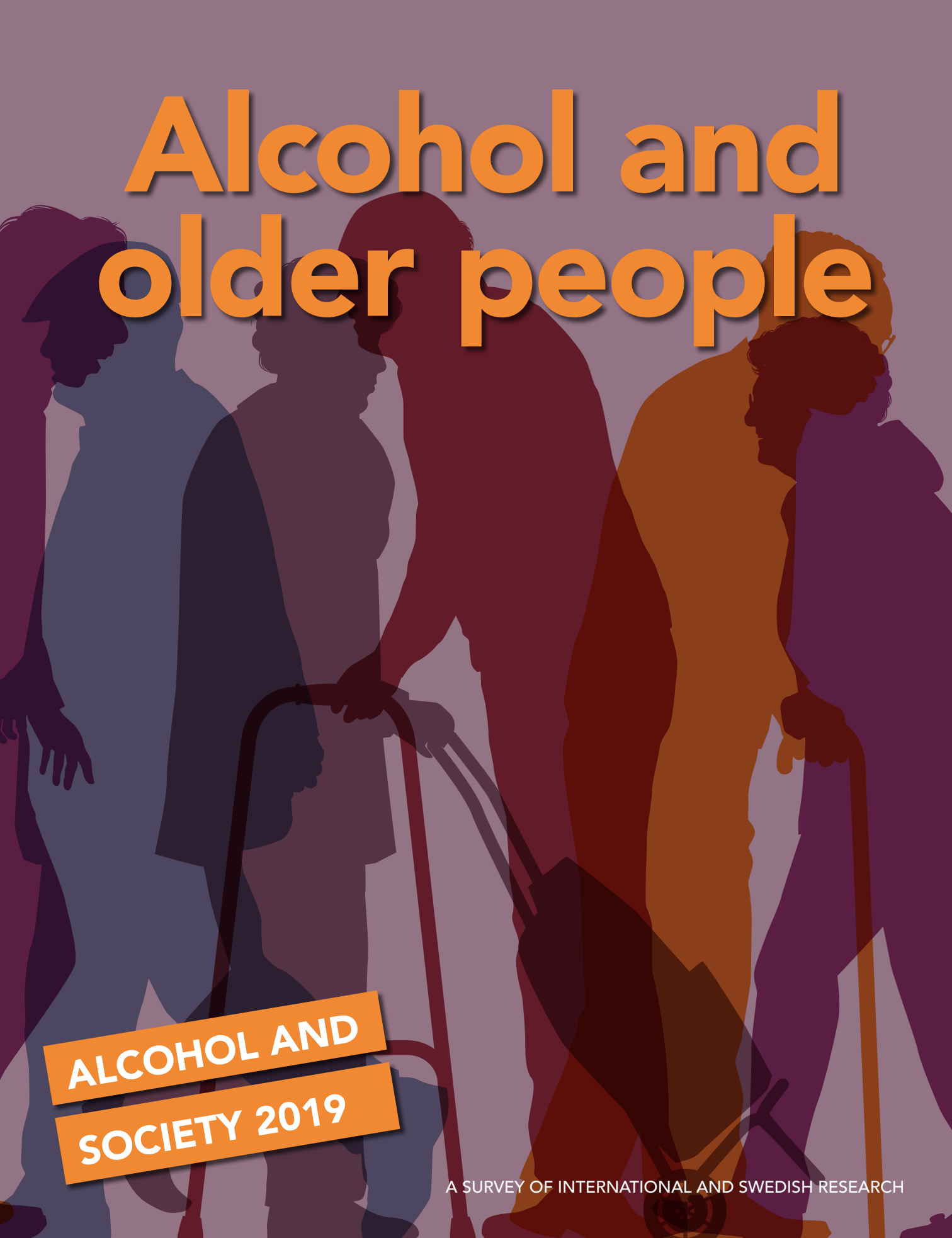
Alcohol and older people



ALCOHOL AND SOCIETY 2019

A SURVEY OF INTERNATIONAL AND SWEDISH RESEARCH

The Swedish Society of Medicine, Swedish Society of Nursing and IOGT-NTO are voluntary organisations independent of commercial interests. The Swedish Society of Medicine is the scientific organisation of the Swedish medical profession and has a broad range of interests across the entire field of medicine. The importance of lifestyle to people's health at both individual and societal level, is a priority issue. The Swedish Society of Nursing is a nonprofit organization and a forum for discussing and developing nursing care by promoting nursing research, ethics, education and quality in nursing. IOGT-NTO focuses on the effects of alcohol and narcotics on individuals and society, but is also engaged in broad social and club activities. CERA is an interdisciplinary and collaborative centre for education and research into hazardous use, abuse and addiction at Gothenburg University – which works to strengthen and develop research and education in the field of addiction, and to disseminate scientific expertise to people working professionally in the field of abuse and addiction, and other interested parties.

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Foreword

Alcohol consumption and alcohol-related harm, whether in the form of chronic disease or acute harm, has increased amongst Sweden's elderly in recent years. The percentage of the population classified as elderly has increased and will continue to do so. Prevention of disease and harm, including alcohol-related disease and harm, is, therefore, very important – both for all those at risk and for the health and medical care sector.

The report addresses the elderly's increased sensitivity to the effects of alcohol which, in combination with the ageing process, may increase the risk of disease and accidents, even at relatively low consumption levels. It describes the relationship between alcohol and various diseases and problems from which the elderly may suffer, such as cardiovascular disease, diabetes, dementia, and cancer. The report also highlights the significance of lifetime lifestyles for health in old age.

The report's authors comprise some of the world's leading, international alcohol researchers, headed by Harold Holder. The researchers have collated and aggregated facts and figures from international studies in the field and evaluated the scientific strength of the results, and both describe the role of alcohol policy measures, and offer recommendations for guideline alcohol consumption levels for the elderly.

The Swedish Society of Medicine, the University of Gothenburg's Center for Education and Research on Addiction (CERA), the Swedish Society of Nursing, the "Ansvar för Framtiden" [Responsibility for the Future] Foundation, and IOGT-NTO issue an annual research report entitled "Alcohol and Society", with the aim of highlighting what science has to teach us about the effects of alcohol consumption on individuals and society. This is the sixth such report. Previous years' reports have focused on such issues as alcohol and young adults, the effects of low dose consumption, alcohol's second-hand harm, alcohol and violence, and alcohol-related cancers. These reports, together with this year's edition, are all available on our respective websites.

Alcohol consumption by the elderly is often unremarked by the health and medical sector. It is our hope that this report can help increase awareness of this issue, both amongst medical and healthcare personnel, and amongst other interested parties, and that it both provokes interest and stimulates discussion.



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Executive summary

In Sweden, as in most of the developed world, older people comprise an increasing proportion of the population. For this subgroup, the aging process in general increases risks to health, safety, and quality of life and, as a result, older persons account for a substantial burden of health care problems and health-related costs in Sweden, as in most countries.

While consuming alcohol can add to the health and safety risks of any age group, these risks are increased for the older population. Typically, older people drink less than younger age people which may lead to the improper conclusion that they have less risk associated with alcohol. However, in reality the interaction of greater susceptibility to alcohol's effects and the greater health risks associated with aging combine to actually increase the risks of alcohol-related harm among older people. For example, decreases in body mass associated with aging can result in higher blood alcohol concentration (BAC) for older persons from consuming a fixed quantity of alcohol. In addition, changes in liver metabolism, slower reaction time and taking multiple chronic medications may further increase both BACs and the risk of experiencing negative alcohol-related effects from a given BAC.

Risks from alcohol-related harms arise from both chronic exposure as the result of cumulative consumption over time (e.g., liver cirrhosis), and acute impairment from heavy drinking episodes (e.g., falls and motor vehicle crashes). Although many think of alcohol-related conditions as being caused only by very heavy drinking or drinking to the point of severe intoxication, there has been a growing recognition that lower levels of consumption, either in aggregate or on a per-occasion basis, can cause health, safety, and social problems, especially for older persons.

- Heavy drinking either on average or per-occasion (i.e. binge drinking) increases the risk of almost all alcohol-related diagnoses, e.g., cardiovascular disease, liver cirrhosis or alcohol use disorder, and certain cancers.

- Even drinking lower amounts of alcohol by older persons has some risk. For example, the risk of some cancers begins to increase with any consumption. Older persons have increased risk of car crashes at very low BAC levels, and are more likely to incur severe injury and death than younger persons.

- In Sweden, alcohol consumption has increased among older persons over the past 14 years in absolute terms and relative to other age groups, and deaths attributed to alcohol have increased among older Swedes.

- Heavy drinking is the strongest modifiable risk factor for dementia onset. Although most non-randomized studies suggest that low-volume drinking may reduce the risk of dementia, higher quality research using advanced medical MRI brain scans, genetic randomization studies, and experimental animal studies suggest that there is likely no protective effect of even low-volume consumption on cognition.

- There are a variety of public policy interventions that can reduce excessive alcohol use and reduce alcohol-related harms. These include maintaining government monopoly systems, increasing the price of alcohol (e.g., taxation, minimum pricing), decreasing the physical availability of alcohol (e.g., limiting the number of outlets), and restricting alcohol advertising.

- The level of alcohol consumption with the lowest health risk is zero. However, the health of most older persons who continue to drink would be improved by reducing their consumption, either overall or during days in which alcohol is consumed. Among those who drink, the lowest level of risk is one standard Swedish drink per day (12 grams of pure alcohol) or less on average and no more than 2 drinks on any one day.

- Those who don't drink or who drink infrequently should not begin to drink, reinitiate drinking, or drink more frequently in order to achieve claimed health benefits. In addition, no consumption is generally preferable among those with liver disease, peptic ulcer disease, who take psychoactive or sedating medications, are driving, have cognitive difficulties, a history of falls or poor balance or cardiac arrhythmias.

- On balance, alcohol is an unhealthy substance in which harms from heavy use is considerable and supported by a robust base of scientific evidence. Even 'moderate' use has some risks, particularly in older persons, and the evidence for health protection has eroded in recent years such that we conclude: *In many ways moderate drinking may be a sign but not a cause of good health.*



While consuming alcohol can add to the health and safety risks of any age group, these risks are increased for the older population.

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1 Alcohol and older people – an introduction

The average age of the world population is rising. It is expected that the percentage of the world's population over 60 years of age will double by 2050.[1] Within Sweden the population older than 65 is expected to rise from 1.9 million in 2015 to 2.4 million in 2030, a 26% increase.[2] Swedish life expectancy has increased by 2.5 years, to 82.2 years, between 2000 and 2015 with women at the age of 65 expected to live 21.5 years longer and men 18.9 years longer.[3]

As a natural consequence of aging, the human body becomes more vulnerable to disease. Furthermore, sight, hearing and cognitive processing all decline. Older people have a higher risk of disability and death from all major diseases, such as heart disease, stroke, chronic respiratory disorders, cancer and dementia. With increased longevity, certain diseases have increased considerably. For example, new cases of cancers in the 65 to 85 year group more than doubled between 1970 and 2016. Expressed as new cancer cases per 100 000, cancer incidence has increased almost 50%.[4]

In addition to chronic illness and health issues the incidence of acute harm among older age groups has increased. [5] Acute harms include falls and other unexpected accidents, violence and mistreatment.Thus, on average, the personal risk of mortality and morbidity rises as a person ages. In Sweden, even as mortality has decreased among the older age groups, years lived with disease has increased in absolute numbers.[5] As a result, the number of older people receiving care from municipalities and counties has substantially increased along with higher medical care costs, constituting a greater challenge for health care and social services.[2]

Against this backdrop of increasing numbers of older people in Sweden requiring more health services, the use of alcohol among older people also calls for special attention. While alcohol consumption mostly is at a lower level than among younger generations, this still is of special concern, given the growing susceptibility of older people to both the acute and chronic effects of alcohol.

The purpose of this report is to review the health and social effects of alcohol among older persons generally, and in Sweden in particular. By 'older' persons, we are broadly referring to those aged 55 years and older, although we have a particular focus on those aged 65 or older, which is the more conventional definition of the onset of old age. In addition, for conditions that predominantly affect older persons (e.g., stroke) we draw on studies of the general population. In this report we discuss alcohol use and alcohol-related mortality outcomes among older persons in the Swedish population, describe the physiologic effects of alcohol on older persons, describe relationships between alcohol consumption and a variety of health outcomes in older persons, outline the implications of this increased susceptibility to alcohol's effects among older people, describe prevention and policy responses, and make recommendations for low-risk drinking for older persons who consume alcohol.



2 Increased susceptibility

Physiological changes due to ageing result in older people becoming increasingly susceptible to both the acute and longer term effects of alcohol consumption. These physiological changes include reduced muscle mass and reduced water in the body to dilute the damaging effects of alcohol on human tissue. These changes that occur in the older body result in higher and longer lasting blood alcohol concentrations (BACs) than would occur for the same quantity of ethanol intake by younger drinkers.

2.1 Reduced body water

With increased age muscle mass is reduced. Since muscle is largely constituted of water and alcohol is mainly water soluble, there is less water for alcohol to be distributed in, leading to higher BACs when drinking, and also increased exposure to acetaldehyde, a highly toxic and carcinogenic substance produced in the body when alcohol is metabolised. The tendency for older people to experience higher BACs from a fixed dose of alcohol increases with advancing age and can be responsible for increasing the risks of illness or injury when even relatively small amounts are consumed.

Ageing is usually associated with a reduced capacity of the liver to metabolise alcohol, due to reduced activity of the enzyme alcohol dehydrogenase, which breaks down alcohol. This does not affect blood alcohol levels to the same extent as the changes in muscle mass however.

2.2 Impacts on stress hormones

Exposure to alcohol activates the hypothalamic-pituitary-adrenal (HPA) system, resulting in increased levels of cortisol in a dose-response manner.[6] Cortisol is recognized as the main stress hormone and, when chronically elevated, creates negative metabolic effects such as hypertension, diabetes, osteoporosis and increased susceptibility to infection. Increased levels of cortisol are also associated with some neuropsychological disorders, including depression and Alzheimer's disease, although it remains unclear whether these are causal associations. Thus, alcohol induced exposure to cortisol results in decreased efficiency and resiliency of physiological function

in many organs and an acceleration of the aging process. Given the increased blood alcohol levels from drinking in old age compared to younger ages, these effects are especially pronounced among the elderly.

2.3 Interactions with medication

Improper use of medications is estimated to cause 10 – 15 per cent of all hospitalizations in Sweden. Taking medications along with alcohol does not usually have a significant impact on alcohol metabolism or blood alcohol levels. Alcohol consumption can, however, exert strong effects on the effectiveness and action of medications. Eighty per cent of all people above 65 years of age in Sweden have at least one medication prescribed. Alcohol can interact with many types of medication and increase the risk of side effects through a variety of mechanisms. Examples of medications that may interact with alcohol include those used for the treatment of hypertension, mood disorders (e.g. anxiety and depression), insomnia and pain.[7] Alcohol may also reduce the effectiveness of a wide variety of prescribed medicines.

Interactions of alcohol with other sedative drugs such as strong painkillers (opioids) and antianxiety drugs like benzodiazepines can be particularly dangerous as they can both increase sedative effects and can change blood pressure either upwards or downwards. In extreme cases, alcohol increases the risk of fatal and non-fatal overdoses from opioid drugs, an increasingly common concern. There is evidence that even small quantities of alcohol reduce tolerance to the effects of strong painkillers and so increases risk of overdose.

When alcohol is combined with drugs used to treat hypertension this can result in sudden and potentially serious drops in blood pressure. Alcohol use can also directly impact the ability to maintain blood pressure when standing upright after sitting, a problem that is anyway exacerbated with age and leads to increased risk of falls and other injuries.

Antidepressant drugs are commonly prescribed to older people. Common side effects are drowsiness, memory impairment, confusion and impaired muscle control with increased risk of falls. All these side effects are compounded by alcohol. Another common class of drugs used by elderly include anticoagulants (blood thinning agents) such as warfarin or newer medications such as direct thrombin inhibitors. The metabolism of warfarin can be affected by alcohol and may cause bleeding. Alcohol induced liver disease can also affect the effectiveness of medications used to thin the blood, thereby increasing bleeding risk. Finally, alcohol use may increase the risk of peptic ulcer disease or gastritis and may increase the risk of bleeding for those on aspirin therapy.

As older people are prescribed more and more medications, these problems have increased over time. There is a comprehensive scientific literature dealing with medication effects among the elderly, but the role of alcohol is rarely noted in clinical guidelines. This despite the fact that alcohol is the single most commonly used drug among older people, and that negative effects may occur even at quite low levels of alcohol consumption.

2.4 Social relations

A number of social and lifestyle factors may contribute to potentially hazardous drinking among the elderly. Most important among these appear to be improved purchasing power compared to previous generations, smaller social networks and stressful life events such as loss of a spouse. Increasing social acceptance of drinking among older women also plays an important role. Depression and increased risk of suicide are quite common among elderly people. While depression can lead to increased use of alcohol, this in turn can worsen depressed mood over the longer term and increase risk of acting on suicidal thoughts.[8]

Retirement can be associated with increased alcohol consumption and/or problems. This may result from having high pre-retirement job satisfaction and enforced retirement.[9]

80%

Eighty per cent of all people above 65 years of age in Sweden have at least one medication prescribed. Alcohol can interact with many types of medication and increase the risk of side effects through a variety of mechanisms.

3 Health risks, acute and chronic

Risk from alcohol-related harms can be categorized as those arising from chronic exposure over time (e.g., liver cirrhosis) or those stemming from acute impairment (with or without symptoms of acute intoxication, e.g., motor vehicle crashes). Although many think of alcohol-related conditions as being caused by heavy drinking or drinking to the point of severe intoxication, there has been a growing recognition that lower levels of consumption, either in aggregate or a per-occasion basis, can cause health and social problems.



Even drinking low amounts of alcohol can add to the health and safety risks already present during later life.

This is particularly the case among older people. Alcohol consumption at relatively low doses among the elderly is associated with health problems including atrial fibrillation, a number of gastrointestinal disorders and some cancers. In addition, alcohol consumption can cause declines in cognitive processes, reflexes in response to dangerous situations such as driving, walking and swimming, as well as decrements in other skill-based behaviors. Therefore, the inherent increased risks of chronic and acute harms among the older population are enhanced with the consumption of alcohol. In short, even drinking low amounts of alcohol can add to the health and safety risks already present during later life.

While a relatively small percentage of health and safety outcomes can be attributed to genetic factors, the greater majority of health and safety determinants in older age are problems which could be prevented or delayed by healthy lifestyle in younger years but also with advancing age.[10] This means that the overall quality of life for older persons can be shaped by the physical and social environments in which they live.

Since many of the health and safety conditions that occur during older ages are preventable, environmental strategies for the prevention of disease and declines in capacity are important. As with the general population, the most effective environmental strategies to reduce alcohol consumption include public policies such as increasing the price of alcohol (e.g., increased alcohol taxation and minimum pricing) and reducing the physical availability of alcohol (e.g., limiting the number of outlets, restricting permissible hours of sale). Health promotion recommendations for preventing non-communicable diseases among older persons include increased physical activity, good nutrition, increased social engagement, and reduced use of tobacco and alcohol.

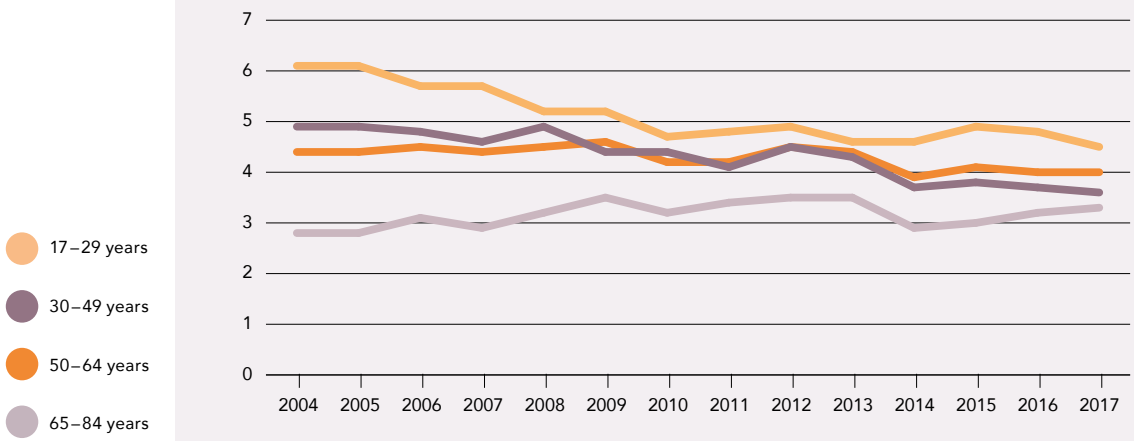


4 Trends in alcohol consumption and related harms in Sweden

Alcohol consumption across different age groups shows some consistent patterns in countries from many regions of the world. Of particular interest here, is the tendency evident in both Sweden and many other countries for older people to reduce their consumption, especially as they become less healthy and frail. For example, a major English study of the ageing process following up a large cohort over time confirmed a pattern of reduced alcohol consumption with age. However, among older people, this decline was less and sometimes consumption even increased among both men and women with higher income and education.[11] This study also provides further evidence that regular alcohol consumption in later life may be an indicator of good health rather than a cause of good health. This limits the interpretation of studies linking level of alcohol consumption to health outcomes among older people, as discussed throughout his report.

In Sweden data on self-reported alcohol consumption are found in the ongoing Monitor study[12] and the Swedish National Public Health Survey.[13] Figure 1 compares trends in average consumption of different age groups using data from the most recent Monitor report. As shown in Figure 1, younger and middle-aged Swedish people are now drink-

FIGURE 1 Self-reported alcohol consumption liters 100 % alcohol per year in Sweden, per age group 2004–2017



Source: Monitor study, from appendix to Befolkningens självrapporterade alkoholvanor 2004–2017 (Self-reported alcohol consumption in Sweden 2004–2017), CAN report 173, 2018. Upper age limit 80 years before 2014.

ing less on average than they were in 2004. However, this trend is not evident among older people and, while there is some variation across the years, there has actually been an overall increase in consumption among people aged between 65 and 84 years since 2004. It is important to note that these data are based on self-report surveys and that the average levels of consumption reported across all age groups are significantly lower than the level of actual consumption of alcohol in Sweden when estimated from official alcohol sales. While the 65 years and older age group in this survey has consistently consumed less than the younger age groups, with an estimated consumption of 3.3 litres of pure alcohol per person per year compared with 4.5 litres for those under 30 years of age, both of these estimates are less than half the actual consumption of the population. It is noteworthy that while older people on average consume a little less than their younger counterparts, this difference has been decreasing over the years and, as discussed in the present report, is at least partly counteracted by an increased susceptibility to alcohol's effects among older people. Indeed, the reduced consumption of alcohol by older people is likely at least partly in response to their increased susceptibility to its effects.

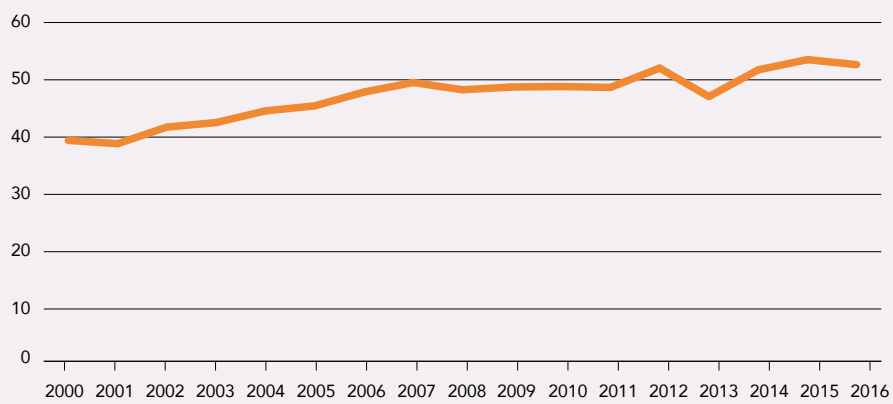
On the surface this reduction in drinking with increasing age suggests a contradiction in concerns about alcohol-related health and safety risks among the elderly, since people typically drink less in general as they get older. That is alcohol-related risks would appear to be less for the older population based upon alcohol consumption alone. However, even if there is a decline, on the average, for alcohol consumption with increased age, the naturally increased risks for health and safety problems associated with aging are added to any increased risk associated with drinking. In short, the risks of alcohol-related problems, even if lower than for younger age groups, can increase overall health and safety risks associated with aging.

A study of changes in drinking habits of Swedish people over the decades found that there were substantial increases in the percentage of 75-year-olds drinking at hazardous levels in 2005/2006 compared with almost 40 years ago, in 1976-1977. Among women, hazardous drinkers increased from less than 1% to 10% of the population. Among men this proportion increased from 19 to 27%.[14]

As further evidence of increasing consumption and related harms, there has been a 30% increase in the rate of alcohol caused deaths of all kinds between 2000 and 2016. The rate of increase for alcohol-related liver disease has been particularly dramatic having increased over this period by more than 100%.[15]

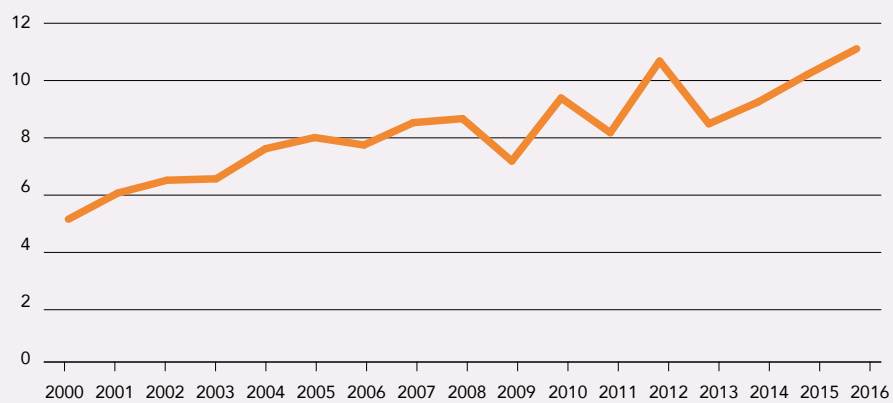
While there have been some variations upwards and downwards over this time period, there have also been significant overall increases in Sweden between 2000 and 2016 for deaths and years lived with a disability specifically from liver cancer and also violence. For example, among those age 65 to 69, years lived with cancer (YLD) increased by 17% per hundred thousand people and by 4% for those aged over 70. Years of life lived with a disability due to violence increased by between 6 and 7% for those aged 65 years and above.[5] These are two outcomes highly associated with alcohol use.

FIGURE 2 **Deaths per 100,000, alcohol index (diagnoses 100 % attributed to alcohol consumption), ages 65–85+, 2000–2016, both sexes**



Source: Swedish National Board of Health and Welfare, Statistical Database, 2018-08-13

FIGURE 3 **Deaths per 100,000, alcoholic liver disease, ages 65-85+, 2000-2016, both sexes**



Source: Swedish National Board of Health and Welfare, Statistical Database, 2018-08-14

5 Methodological considerations: How do we determine risks for older drinkers?



Some of these apparent protective effects do not have biological plausibility.

5.1 Assessing Associations and Causation

Various research approaches have been used to study the potential contribution or even a causal relationship of alcohol to the health and safety of older people. A common task when assessing the extent of alcohol's contribution to different health outcomes is to apply the main criteria for causation in the science of epidemiology i.e. the study of the spread of illnesses in populations. The two major criteria are (i) biological plausibility e.g. evidence from experimental laboratory studies identifying mechanisms of harm (or benefit) from alcohol's impact on human functioning, and (ii) evidence of increased risk of an illness or injury associated with the amount of alcohol an individual consumes – often referred to as evidence of a 'dose-response' relationship.[16] Neither criteria is sufficient on its own: biological plausibility which supports the potential of alcohol effects does not guarantee a significant impact on health outcomes in practice as factors other than alcohol consumption can come into play.

Our confidence is increased in a causal association if there is evidence from well conducted randomised controlled trials (RCTs) of interventions that experimentally add or remove a risk factor in which actual drinking amounts are carefully measured over time including a control group for non-drinking. In practice, it is often practically and ethically challenging to conduct an RCT on a risk factor for disease like alcohol consumption. For example, a planned RCT to investigate whether moderate alcohol use was protective against heart disease had a budget of US$100 million and would have required many thousands of people to be randomised to either abstain from drinking or drink a small quantity each day over several years.[17] In fact, this study was closed down due to ethical and scientific concerns about its design and undue alcohol industry influence.[18, 19]

A research design frequently used in epidemiological research is the study of self-reported alcohol consumption by participants later linked to health or safety outcomes identified in official health archives. These "observational" cohort studies have a number of threats to validity including: selection bias arising from study participants essentially self-selecting themselves into drinker and non-drinker groups; recall bias about drinking which may become worse over time; and under reporting of major changes in drinking levels and patterns which can impact health outcomes.



The majority of observational studies of older people's alcohol use and risk of future illness or death find some evidence of protective effects for people who drink moderately in comparison with those who abstain. Some of these apparent protective effects do not have biological plausibility. For example, a review by Fekjaer (2013)[20] found reduced risk among moderate drinkers for such implausible conditions as deafness, some cancers, liver disease and the common cold. It is hard to explain how such protection is biologically possible. Instead, it is more plausible to suppose that important biases exist in many such studies that create the appearance of health protection. Firstly, many older people who identify as abstainers in these observational studies were drinkers who quit or reduced their consumption for health reasons.[21, 22] Secondly, there is evidence that young adults who are lifetime abstainers are more likely to be disabled, have poor health and low income.[23, 24] Studies comparing abstainers and moderate drinkers also find they differ on a range of lifestyle and personal characteristics unrelated to their drinking which placed them at differential risk of ill-health e.g. bodyweight, nutrition and exercise (Naimi, 1995).[25] There are many "observational" studies linking some exposure to a risk factor for a disease that have been subsequently refuted by RCTs (e.g., hormone replacement therapy and reduced risk for heart disease).

Naimi et al (2017)[26] have shown how biases in long term follow up studies can accumulate with increasing age resulting in the health

profiles of abstainers being increasingly negative and those of drinkers increasingly positive. Furthermore, systematic biases towards continuing drinkers appearing healthier than abstainers will be most pronounced among older people. Such systematic selection bias over the life course is also supported by a meta-analysis of all studies on alcohol use and all-cause mortality by Stockwell et al. (2016).[27] It was also found in another meta-analysis by the same authors that evidence for a J-shaped curve or protection from moderate drinking against coronary heart disease mortality was only present in studies of older cohorts for whom there should be most accumulated bias (people aged over 55 years at intake). Younger cohorts followed up to old age showed no evidence of cardio-protection from moderate drinking.[28] In many ways regular "moderate" drinking may be a sign but not a cause of good health.

One longitudinal study design, referred to as Mendelian randomisation or genetic randomization, is however thought to minimise confounding and reverse causation and be related to lifetime alcohol exposure. This design is applied in studies that identify genetic characteristics which are randomly present in individuals in a way that is not influenced by upbringing and environment. Mendelian randomisation is the basis for studies that compare the risk of disease between individuals with different genetic profiles (e.g. Holmes et al., 2014).[29] Where available we will refer to evidence from both RCTs and Mendelian randomisation studies that relate to alcohol's effect on health. Both of these types of studies are less likely to suffer from bias and confounding than the more frequently conducted observational design studies (i.e. prospective cohort, case-control, case-crossover). In addition, animal studies often have strong experimental designs and can assess alcohol-related exposures that might be considered too risky or unethical in human subjects.

5.2 Identifying and Prioritizing Studies for this Report

In preparing this report we set out to summarise key findings and then draw conclusions from a very large research literature concerning alcohol's effects on physical and mental health as well as safety. There are many thousands of published studies in this area covering different aspects and health conditions. In order to make sense of this complexity, and distil what can be learned, we searched for the most representative and authoritative studies by prioritising a) recent comprehensive and systematic reviews b) major recent studies from official national and international sources c) high-quality studies with strong designs and d) studies of particular relevance to Sweden and Scandinavia. As academic researchers who have contributed to the fields of alcohol epidemiology and policy, we also took account of well-known systematic biases and methodological problems in these literatures in drawing our conclusions.

6 Impact of alcohol on death, disease, injury and disability

6.1 Total mortality

More than 60 conditions have been identified as either partially or entirely caused by exposure to alcohol.[30] There is no doubt that alcohol is a major preventable cause of premature mortality for all ages. Increased risk of death is not limited to high levels of consumption and in fact low level drinking has been identified as a major cause of excess mortality arising from a range of alcohol-related cancers. Past meta-analyses of the observational studies which dominate this literature have nevertheless shown J-shaped curves for all-cause mortality with beneficial effects at low doses. Interestingly, these studies also suggest that the level at which maximum potential benefit may be achieved is probably less than 10 grams of ethanol per day and for women may be as low as 5 grams a day (e.g. Bagnardi et al 2004[31]). In fact, the latest, and largest, report on this subject, from the Global Burden of Disease project, finds that the safest consumption level is 0.[30] What is more, social and economic cost studies based on the supposition that health benefits from alcohol exist, show that the total social and economic costs of harms from alcohol still outweigh the sum of the benefits.[32, 33]

In recent years, with the emergence of novel research approaches that substantially reduce potential for confounding and bias compared to traditional observational study designs (i.e. Mendelian randomization studies), long-standing doubts about the veracity of J-shaped curves for alcohol have been reinforced.[34] Mendelian randomization studies for instance suggest no protective effect for low-dose consumption and coronary heart disease events.[29] This is important because the apparent protective effects for low-volume consumption on total mortality are due to observed protective effects on coronary heart disease, so the veracity of the J-shaped curve and protective associations for low-volume consumption for all-cause mortality remains in doubt. However, based on observational study data, across the general population, including for older persons when assessed, the lowest risk of death is generally found among those drinking less than 10 grams of ethanol daily.[27, 35–39] Furthermore, a Mendelian randomization study of older persons finds reduced all-cause mortality with a genetic variant associated with less alcohol consumption.[40]

60

More than 60 conditions have been identified as either partially or entirely caused by exposure to alcohol. There is no doubt that alcohol is a major preventable cause of premature mortality for all ages.

6.2 Cardiovascular diseases and diabetes

Coronary heart disease, stroke, hypertension and diabetes are all more prevalent among older people, and so studies of these outcomes among the general population are relevant to those in later life, even if the cohorts used in those studies are sometimes not restricted to older people. Both congestive heart failure (associated with heavy consumption) and atrial fibrillation (associated with any consumption) are for example far more common among older individuals but will not be discussed in detail in this report.

There has been no randomized study of low-volume alcohol consumption and any morbidity or mortality outcome related to coronary heart disease. Excessive alcohol consumption, including heavy average consumption and binge drinking, are risk factors for coronary heart disease, stroke, hypertension and the development of diabetes or to poor diabetes control.

In terms of low-dose or "moderate" consumption, most observational studies find protective effects for coronary heart disease. Furthermore, protective associations appear to be greater for relatively older persons compared with the general population.[28, 41] However, the concern is that older ages would also tend to magnify sources of bias including the 'sick quitter' phenomenon and the 'healthy survivor' bias, and protective effects attenuate when attempting to account for these biases.[28] In addition, Mendelian randomization studies find no protective effect for low-volume alcohol on coronary heart disease events or coronary calcification. [29, 42, 43]



While randomized trials of the Mediterranean diet find protective effects for the primary and secondary prevention of CHD outcomes[44, 45], those trials were not randomized with respect to alcohol consumption. Although these latter findings do not preclude protective effects of low-dose alcohol consumption, they mean that the apparent benefits of a Mediterranean diet (i.e. one in which the moderate use of alcohol is one small part) may be explained by factors other than alcohol consumption.

Strokes may either be ischemic (in which blood flow is blocked or reduced to a portion of the brain) or hemorrhagic (in which a blood vessel bursts, interrupting flow and also sometimes creating pressure on surrounding brain tissue); ischemic are the most prevalent of the two. In observational studies, high-volume consumption is a risk factor for both stroke types. Low volume alcohol consumption appears protective for ischemic (but not hemorrhagic) strokes among both men and women, though at lower levels for men (less than one drink per day) than for women (about one drink per day).[46, 47] A recent well-designed and large study questions whether even light drinking protects against ischemic stroke[39] while a Mendelian randomization study finds no protection from having a gene related to reduced alcohol consumption on all strokes.[29]

For hypertension, in itself an important risk factor for cardiovascular disease and mortality, most but not all observational studies find a positive relationship between alcohol consumption and both higher blood pressure and incident hypertension across

all levels of consumption, particularly among men.[48] Furthermore, a meta-analysis finds that reductions in alcohol consumption are associated with reductions in blood pressure.[49] A meta-analysis of Mendelian randomization studies also finds positive relationships between alcohol consumption with increased blood pressure and hypertension among men.[50]

In terms of blood glucose and diabetes, observational studies generally find a protective effect of alcohol consumption (e.g. [51, 52]), with the lowest level of risk among those consuming about one drink daily. However, sex stratified results find significant protective effects only among women[53] and even these may be questioned as there are relatively few well designed studies of women. Moreover, in analyses restricted to trials with a never-drinking comparison group (as opposed to all non-current drinkers), alcohol was not associated with any protective effects for either women or men.[53] Mendelian randomization studies also find no significant protective effect of alcohol consumption on diabetes.[29, 54, 55] Finally, a meta-analysis of randomized clinical trials finds no protective effect of low-volume alcohol consumption on blood glucose control among those with diabetes.[56]

In experimental studies, alcohol administration raises HDL cholesterol levels. However, a Mendelian randomization study found no cardio-protection for those carrying a gene variant that raises HDL. Furthermore, after controlling for their effects on LDL, randomized studies of statin drugs have not found a significant relationship between changes in HDL and coronary heart disease. Finally, several drugs that effectively raise HDL have not resulted in reduced coronary heart disease events in clinical trials. For these reasons, although HDL is associated with cardiac outcomes it is not likely causal for heart disease.[57–59] Alcohol consumption does not appear to meaningfully affect levels of LDL (bad) cholesterol.[60]

In summary, taking account of studies of biological plausibility and studies with stronger designs, there is considerable evidence that heavy alcohol use is responsible for worse outcomes and increased risks for cardiovascular diseases and diabetes. In particular, studies of hypertension, a major risk factor for cardiovascular disease, find only negative impacts from the consumption of alcohol even at low doses. In better designed studies there is little or no evidence of protective effects for low-dose alcohol, especially for men. Furthermore, genetic studies using Mendelian randomisation have found no evidence of protective effects from low-dose alcohol use on cardiovascular disease or diabetes for either men or women.

Alcohol has been classified by the World Health Organisation as a Group 1 carcinogen since 1988



6.3 Cancer

Advancing age is the leading risk factor for incident cancer and for cancer mortality, and those over age 65 account for approximately 70% of all cancer deaths. Increased cancer susceptibility among older persons is likely due to a combination of reduced cellular mechanisms (e.g., DNA repair mechanisms) and an accumulation of carcinogenic damage from environmental exposures over the life course (e.g., tobacco, alcohol).[61] In addition, cancer treatment options may be limited or less desirable on the basis of age (e.g., stem cell transplants, aggressive surgical debulking), or available treatments may have greater relative toxicities than among relatively younger persons.

Alcohol has been classified by the World Health Organisation as a Group 1 carcinogen since 1988 when it was concluded that there was sufficient evidence for its causal role in cancers of the oral cavity, pharynx, larynx, esophagus and liver. Since that time, several hundred more epidemiological studies have reported on the association between the consumption of alcoholic beverages and the risk for cancer at various sites. In 2007, the IARC

added cancers of the female breast, colon and rectum to the list of cancers caused by alcohol (IARC, 2007).[62] World Cancer Research Fund (2016)[63] more recently concluded that stomach cancer also was causally related to alcohol consumption. There is also accumulating evidence for causal association between alcohol use and both prostate and pancreatic cancers.[64–66]

The increased risk of these cancers is normally found to be increased even for light or moderate drinkers with no safe level of consumption (e.g. [64]). However, the appearance of a protective effect does occur in some studies, likely due to the same kinds of confounding factors described earlier in relation to the apparent J-shaped curve for alcohol and mortality generally. Most studies of alcohol and cancer outcomes suffer from the same kinds of systematic bias discussed earlier in particular relation to cardiovascular disease. In one meta-analysis of prostate cancer, it was found that when former drinker bias was corrected the estimated risk for moderate drinkers increased from 8% to 22%.[66] This means that the increased risk of cancers from alcohol consumption are likely underestimated when based on meta-analysis of the existing literature.

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For Swedish people 65+ and older we estimated 571 deaths attributable to alcohol in 2014 for the seven cancers currently deemed by WHO to be caused by alcohol.

In one of our previous reports[67], we applied the latest methods used internationally to quantify the burden of cancers attributable to alcohol in Sweden.[68] For Swedish people 65+ and older we estimated 571 deaths attributable to alcohol in 2014 for the seven cancers currently deemed by WHO to be caused by alcohol. This is likely to be a substantial underestimate as there are likely more cancers than these causally related to alcohol use and the risk estimates derived from the existing literature are themselves likely to be underestimated.

6.4 Cognitive function

Alcohol is recognized as a powerful neurotoxin, known to cause or contribute to a wide range of neurological disorders including dementia and fetal alcohol syndrome, among many others. Furthermore, the burden of dementia attributable to alcohol is now recognized as much larger than previously thought. Heavy drinking is strongly associated with the development of Alzheimer's disease. Alcohol use disorder is the strongest modifiable risk factor for dementia onset and is associated with all other independent risk factors for dementia onset.[69]

The areas of cognitive decline and dementia are areas of particular concern for older persons, and have been increasing in developed nations worldwide. Although it is clear that heavy drinking can cause alcoholic dementia, the effects of low-volume consumption on dementia, including Alzheimer's dementia, are more controversial. Summarizing the literature on the effects of low volume drinking on cognition, four lines of research are considered. One of these supports protective effects for cognitive impairment while the other three do not. In the first, the observational studies, a majority of studies find J-shaped curves, where low-volume drinking appears protective of dementia (e.g. [70]). While most of these studies find a J- or U-shaped curve for alcohol consumption and the risk of dementia, some have investigated the effects of the apolipoprotein e4 allele, where they find that carriers of this allele have an increased risk of dementia with increasing alcohol consumption.[71] Nearly every review of observational (i.e., non-randomized) studies however describes methodological problems of underlying studies, such as under-representation of heavy drinkers in population-based cohorts; inconsistent measurement of alcohol use or dementia, or both; insufficient control of potential confounders; and insufficient consideration of sample attrition in patients with alcohol use disorders.

In the second, the structural studies consistently report increased rates of atrophy of the brain, and especially the hippocampus (an area of the brain associated with memory), at all levels of drinking including relatively low levels.[72] In the third, the Mendelian randomization studies, the outcomes are mixed, but overall there is no evidence for protective effects from moderate drinking.[73] In the fourth,



the animal studies, of which some have the advantage of having an experimental design with controls, all levels of alcohol consumption appear to be harmful to the brain and cognitive function.[74]

Some research has shown a relationship between the development of cognitive impairment and lifestyle-related risk factors that are shared with other non-communicable diseases. These risk factors include physical inactivity, obesity, unbalanced diets, tobacco use and harmful use of alcohol as well as diabetes mellitus and mid-life hypertension. Other potentially modifiable risk factors more specific to dementia include mid-life depression, low educational attainment, social isolation and cognitive inactivity.[75] A randomized study of the Mediterranean diet showed protective effects for the development of dementia; however, the study was not randomized with respect to alcohol consumption and so the results were inconclusive.[76]

The scientific controversy is illustrated by conflicting evidence from the Whitehall II study. On the one hand, an observational study on the Whitehall II cohort, on alcohol consumption and dementia, found a J-shaped risk curve in which low-volume drinking appeared to be protective for dementia.[77] On the other hand, the Whitehall II imaging study found increased odds of hippocampal atrophy in a dose dependent fashion, with increased risk starting at low levels of consumption.[78]

A recent Swedish study, based on the Swedish twin registry adds to the growing body of research in which all levels of alcohol consumption appear detrimental to cognitive function.[79] The study used information from a sample of people in the Swedish Twin Registry, who in their midlife (1967) participated in a survey on alcohol intake and 25 years later participated in a longitudinal study on cognitive aging. This study showed that alcohol intake was related to lower cognitive performance in a dose-response manner, starting at low levels.

6.5 Injury

Injuries may result from a wide range of external causes such as road crashes, falls, near drowning, burns, or violence. Here we have considered the research evidence for three

major forms of injury, road crashes, falls and physical/psychological abuse. Several meta-analyses find significant associations between alcohol use and risk of injury within six hours of drinking compared to abstainers for all ages.[80–82] For older drinkers specifically, risk of injury is also likely to be higher immediately after alcohol exposure but may also be compounded by the onset of physiological changes that occur with aging including reduced bone density, decline in balance, coordination and reaction time, vision and hearing problems and medication use.[83]



There is evidence that older drivers have higher risk of crashes than younger drivers at the same BAC levels.

6.6 Road crashes

It is well established that in relation to motor vehicle injury, alcohol use, even at relatively low levels, is a risk factor for drivers of all ages.[84, 85] Road crashes are of concern in most industrialized societies and as increased wealth increases the availability of private automobiles for personal transportation, the risk of traffic crashes and resulting injuries and fatalities becomes increasingly serious across the world. Increased wealth also leads to increased resources to traffic safety work which in the longer run decreases the risks for road crashes.[86] Since safe driving is dependent upon both training and personal driver skills and ability to operate complex tasks, the aging process may affect some older driver abilities and also increase driving risks due to changes in vision and cognitive functioning (ability to reason and remember), as well as physical changes.[87] In 2002, internationally more than 193,000 traffic deaths traffic were registered among people who were 60 years and older. The mortality rate for such accidents for 100,000 persons in this age population was the highest compared to other age groups based upon a systematic review of scientific papers published between 2003 and 2013.[88] The U.S. Centers for Disease Control in reviewing national crash records for all of the U.S. found that involvement in fatal crashes per mile travelled begins increasing among drivers ages 70 – 74 years and are highest among drivers aged 85 and older in the U.S. This later trend has been attributed more to an increased susceptibility to injury and medical complications among older drivers rather than an increased risk of crash involvement.[89]

While driving and drinking alcohol increases crash risk for all ages, there is evidence that older drivers have higher risk of crashes than younger drivers at the same BAC levels. This has been confirmed by one controlled laboratory study of driving simulation in which participants were given alcohol prior to the simulation. The study found in general that older adults performed more poorly with driving precision and impairment as a result of alcohol consumption measured by steering rate and the ability to maintain a constant speed than younger drivers. The study also found that one serving of alcohol was enough to affect seniors' driving abilities in contrast to younger age groups with the same level of alcohol consumption.[90] A national study in the U.S. based upon fatal crash records examined BAC levels for drivers and found that among fatal crashes in which drivers had BAC levels less than 0.08% there was a much higher proportion of older drivers compared with the youngest aged driver group, despite the latter also being a sub-population at high crash risk.[91]

In short, not only are older drivers of greater risk of injury and death than all younger ages, but this accident risk is increased with the consumption of alcohol, even at very low dosages. Moreover, there is the additional risk of interaction between alcohol and psychoactive medications taken by the older driver. While younger drivers are at greater risk for crashes at a given BAC, the risk of serious outcomes is greater for older drivers at similar BAC.

6.7 Falls

For the general population research evidence on the association between falls and episodic alcohol use generally shows a linear dose-response relationship such that the more alcohol that is consumed the greater the risk of falling.[81] Older people fall more often than their younger counterparts often as a consequence of chronic health conditions, impaired vision, ear problems, muscle weakness or possibly medication use. They may also have an increased fear of falling as the consequences can be more serious, long lasting and occur at times and places where they are alone.[83]

One of the few systematic reviews of alcohol's effect on risk of falling among older drinkers specifically (65+ years) was conducted by Ridolfo and Stevenson (1998).[92] They noted that one of the problems with studies in this area is that they often recruit participants from nursing homes and that both self-reported alcohol use and the nature of falls among those residing in institutional settings will differ to individuals living in unsupervised situations. People living in residential aged care are likely to be more frail, take more medications and be at higher risk of falling but also less likely to consume significant quantities of alcohol. They concluded that for people aged 65 years and older, acute intoxication is a cause of 12% of male and 4% of female falls. These findings matched results from a recent experimental study showing that even low level alcohol use adversely affects postural stability among persons over 65 years, especially among those who already have poor balance.[93]

However, some recent studies have produced mixed findings with some showing no association at all (e.g. [94]) and others showing a protective effect for falls at low doses (e.g. [95]). Research on hip fracture, osteoporosis and low bone mass density also suggests that compared to abstainers, those who regularly drink small amounts of alcohol (between 0.5 to 1 drink per day) have a lower risk of developing these conditions.[96–99] At least two studies suggest a J-shaped curve where older drinkers, particularly women, who report drinking lower amounts of wine have a significantly lower risk of falls and hip fracture compared to non-drinkers[100, 101] but that risk increases at 2 or more drinks per day.[96] However, these observational studies are subject to the same kinds of design limitations described in earlier and apparent protective effects at low doses are likely to be due to uncontrolled confounding and bias.

6.8 Physical and psychological abuse of older people

Physical and/or psychological abuse of older people by others ('elder abuse') has gained attention as a significant societal problem in more recent years. A large meta-analysis of data collected from 28 countries estimated that about one in six people aged 60 years or older had experienced elder abuse of some form including psychological, financial, neglect, physical and sexual.[102] Elder abuse may be even higher among vulnerable dementia patients with estimates ranging from about 28% to 52%.[103] As average population age continues to increase worldwide, the prevalence of elder abuse is also likely to grow rapidly.





Alcohol use by either the victim or the abuser or both has been repeatedly identified as a key risk factor in elder abuse. A 2012 Swedish survey of 20,000 men and women aged between 60 and 74 years found that 4.2% of participants (5.8% of women and 2.7% of men) had been a victim of violence in the past year, often from a current partner or family member. Older people who drank at an 'at-risk level' (according to the AUDIT) were more than four times more likely to have been a victim of violence compared to low risk drinkers.[104]

A recent review by Warmling et al (2017) identified a range of risk factors for intimate partner violence among older people that included alcohol use, depression, low income, functional impairment and previous exposure to violence.[105] A systematic review of types of physical injuries among general elder abuse cases identified alcohol and/or drug abuse by perpetrators as a major factor and suggested that physical elder abuse occurs more commonly in the evenings and on weekends when greater social interaction and increased perpetrator alcohol occurs.[106]

Analysts have nevertheless observed that few studies consider alcohol use among abused older persons as a risk factor. It is plausible that this reflects societal stereotypes of older people that make alcohol consumption less likely to be taken into consideration. Ahnlund and colleagues have recommended that workers in the caring professions should routinely inquire into the experiences of violence among older women and men.[104]

6.9 Depression and suicide

Heavy alcohol use is linked to the development of depression; in addition, alcohol use can exacerbate depression or complicate the treatment of depression. Depression, in turn, is closely linked with suicide attempts and completed suicides. Acute impairment from alcohol can induce transient dysphoria, a loss of impulse control and a weakening of executive functioning, all of which can increase the likelihood of attempting suicide among persons who may be depressed or despondent. Rates of suicide have risen in many developed countries in recent years. Approximately 20% of those aged 65 years or older also have an AUD disorder[107], and AUD is associated with a doubling of risk for suicidal ideation

or attempts.[108] Among older persons who completed or attempted suicide, 10-20% have alcohol in their bloodstream at the time of the episode.[108, 109]

6.10 Alcohol use disorders

Alcohol use disorders (AUD) are the most important neuropsychiatric conditions caused by alcohol. Prolonged heavy use of alcohol is the leading risk factor for alcohol dependence. Alcohol has adverse effects on all organ systems, including a number of functions of the brain underlying the development of dependence. The prevalence of AUD is reduced with increasing age, most likely as a result of reduced heavy drinking. Among those who continue to drink however, or increase their consumption, the risk of AUD may be increased as older people are more vulnerable to alcohol related harm from a given volume of alcohol than other age groups. This increased vulnerability may be related to the age-related increased stress produced by alcohol.[110–112]

6.11 Other conditions

There is a range of other chronic conditions that occur more frequently with age, often diminishing quality of life, that are partially attributable to or exacerbated by alcohol use. Respiratory diseases such as pneumonia are common causes of morbidity and mortality among older people.[113] Due to associated cardiopulmonary disease and impaired immunity, pneumonia in particular, is more likely to be fatal or have severe consequences when contracted by older compared to younger people.[114] A systematic review by Samokhvalov (2010) reported a dose-response relationship between chronic alcohol use and risk of community acquired pneumonia.[115] This review also reported that people with alcohol use disorder had more than eight times higher risk of contracting pneumonia compared to non-drinkers.

Gout is a relatively common and debilitating condition and prevalence may be rising, particularly in developed countries, as a result of rising obesity and hypertension.[116, 117] Two reviews identify a positive relationship between regular alcohol use and gout, with particularly large increases in risk at higher levels of use.[118, 119] People with gout are also more likely to suffer from osteoarthritis.[120] Osteoarthritis is a health concern common to older people characterized by painful, progressive degeneration of joint cartilage (e.g. knees, hips). Although we were unable to identify any systematic reviews on whether alcohol use increases the risk of developing osteoarthritis or worsening of the condition, there is growing evidence that disease progression is correlated with oxidative stress and inflammatory processes. An experimental study exposed mice to low/moderate doses of alcohol for eight weeks and found a pathological role for chronic alcohol exposure in the development of osteoarthritis in knee joints compared to control mice.[121] Although these results need to be confirmed in human clinical trials they suggest that for osteoarthritis sufferers, avoiding environmental factors, such as alcohol, that potentially promote oxidative stress and inflammation may be helpful.

Parkinson's Disease is neurodegenerative disease occurring in the brain that is more common among older ages. Parkinson's has severely debilitating symptoms and often results in reduced life quality and life expectancy.[122, 123] A review of 16 studies found a weak protective association between alcohol use and Parkinson's disease but as noted elsewhere in this report, in terms of strength of design this literature is particularly weak and the association may be due to uncontrolled confounding, selection and recall bias.[124]

Sleep disorders are common among the general population and may become more noticeable or bothersome as people age.[125] Alcohol at all levels reduces sleep onset and may also disrupt sleep-related physiology and exacerbate day time sleepiness. Alcohol consumed before bedtime has an adverse effect on sleep overall by reducing sleep quality, even if it does help some people to fall asleep.[126–128]

20%

Approximately 20% of those aged 65 years or older also have an AUD disorder, and AUD is associated with a doubling of risk for suicidal ideation or attempts.

7 The role of alcohol policies



The study suggests that stronger policies on alcohol at the present time will pay dividends for older people in the future.

There is specific evidence for Sweden that older people's drinking reflects the kinds of alcohol policies that were in place during their younger, more formative years. An analysis of continuous population surveys conducted monthly for the 12 years up to 2013 and including 127,480 Swedes born between 1951 and 1989 supports this conclusion.[129] Within that sample, cohorts who grew up during more restrictive alcohol policy years (turning 15 between 1978 and 1991) had significantly lower alcohol consumption than those born during more liberal eras (turning 15 between 1966 and 1977 or between 1992 and 2004). These findings apply to both men and women and support the general idea that older people's alcohol use and hence health outcomes can be influenced by strong alcohol policies. In particular, this important study suggests that stronger policies on alcohol at the present time will pay dividends for older people in the future.

There is a large literature examining evidence for the effectiveness of alternative alcohol policies. There is consensus across multiple comprehensive and systematic reviews (e.g. [130, 131]) that the most effective alcohol policies are those which a) reduce the affordability of alcohol, especially of cheap high-strength alcohol b) restrict its physical availability in terms of number of outlets and hours of sale and c) restrict promotions, advertising and marketing. Government monopolies on alcohol production and retail sale are particularly suited to using these policy mechanisms to improve public health. There may be a concern that some older people are on reduced incomes and that there may be negative effects of strict alcohol policies on a vulnerable segment of the population. However, there is specific evidence that heavier drinkers on lower incomes are especially likely to respond to price increases by spending less on alcohol, drinking less and having fewer alcohol-related harms.[132, 133]

It is also important to have effective policies to deter impaired driving among older people given their increased susceptibility to alcohol's effects. Low legal BAC limits and random roadside screening been shown to be particularly effective at deterring alcohol consumption before driving a motor vehicle (e.g. [134]).



8 Examples of drinking guidelines

A number of countries recommend older people to be more cautious with their use of alcohol because of additional susceptibility both to injury and chronic disease as a result of drinking. The U.S. Dietary Guidelines do not have special recommendations based on age, but suggest limiting consumption to no more than 2 drinks in a day for men or 1 drink in a day for women. Non-drinkers are advised not to begin drinking on the basis of health considerations.[135] Australian guidelines recommend no more than two standard drinks (=10g ethanol each) on any day for all healthy adult men and women and additionally suggest that older individuals consult their physician about the most appropriate level of drinking for their health.[136] Italy, Slovenia, Finland and Portugal each recommend no more than one standard drink per day for persons aged over 65 years (= 10 to 12 g ethanol each). Spain also recommends high levels for older people, still also slightly lower than those recommended for the general population.[137] The Canadian low risk drinking guidelines also recommend greater caution among older people and recommend a lower maximum per day than for adults in general. Nonetheless, these are somewhat higher with recommended limits up to 3 drinks (=13.45 g ethanol) per day for men and 2 for women (Butt et al, 2008).[138] Some of these guidelines also advise abstinence from alcohol in older people if they have particular additional vulnerabilities e.g. mental or physical illness, taking prescribed medications.

9 Our Recommendation for Drinking Among Older Persons

On balance alcohol is an unhealthy substance. The level of alcohol consumption with the lowest health risk is zero. Even if we take observational studies at face value, among drinkers the lowest level of risk for death from any cause is generally associated with consumption of less than 1 standard Swedish drink per day. Given that older persons are more vulnerable to alcohol-related effects generally and at relatively low volumes, the health of many older drinkers would be improved if they drank less, both overall and on days they drink.

For older persons who choose to drink, to reduce health-related risks we suggest a limit of one drink per day or less on average, and no more than 2 drinks on any day. Those who don't drink or who drink infrequently should not begin to drink, reinitiate drinking, or drink more frequently on the basis of health considerations. In addition, no consumption is generally preferable among those with certain conditions including liver disease, peptic ulcer disease, cardiac arrhythmias, are driving, have cognitive difficulties, a history of falls or poor balance, or who take psychoactive or sedating medications.

References

1. WHO (2017) *Global Strategy and Action Plan on Ageing and Health*, World Health Organisation.
2. Socialstyrelsen (2018) *Vård Och Omsorg Om Äldre – Lägesrapport 2018 (Care of the Elderly – Status Report 2018)*, Socialstyrelsen.
3. Folkhälsomyndigheten (2018) *Folkhälsans Utveckling – Årsrapport 2018 (Public Health in Sweden 2018)*, Folkhälsomyndigheten.
4. Socialstyrelsens statistikdatabas Available at: https://www.socialstyrelsen.se [Accessed July 4, 2018].
5. Institute for Health Metrics and Evaluation (IHME) GBD Compare Data Visualization Seattle, WA: IHME, University of Washington, 2017 Available at: http://vizhub.healthdata.org/gbd-compare [Accessed July 4, 2018].
6. Badrick E, Bobak M, Britton A, Kirschbaum C, Marmot M, Kumari M (2008) The relationship between alcohol consumption and cortisol secretion in an aging cohort. *J Clin Endocrinol Metab* 93, 750–7.
7. Moore AA, Whiteman EJ, Ward KT (2007) Risks of combined alcohol/medication use in older adults. *Am J Geriatr Pharmacother* 5, 64–74.
8. Schneider B (2009) Substance use disorders and risk for completed suicide. *Archives of suicide research* 13, 303–16.
9. Kuerbis A, Sacco P (2012) The impact of retirement on the drinking patterns of older adults: a review. *Addict Behav* 37, 587–95.
10. WHO (2015) *World Report on Ageing and Health*, World Health Organization.
11. Holdsworth C, Frisher M, Mendonça M, DE Oliveiria C, Pikhart H, Shelton N (2017) Lifecourse transitions, gender and drinking in later life. *Ageing Soc* 37, 462–94.
12. Guttormson U, Gröndahl M (2018) *Befolkningens Självrapporterade Alkoholvanor 2004–2017 (Self-Reported Alcohol Consumption in Sweden 2004–2017)*, CAN.
13. Nationella folkhälsoenkäten (National Public Health Survey) Available at: https://www.folkhalsomyndigheten.se/folkhalsorapportering-statistik/statistikdatabaser-och-visualisering/nationella-folkhalsoenkaten/.
14. Waern M, Marlow T, Morin J, Ostling S, Skoog I (2014) Secular changes in at-risk drinking in Sweden: birth cohort comparisons in 75-year-old men and women 1976–2006. *Age Ageing* 43, 228–34.
15. Dödsorsaker [internet] (Causes of death) Available at: https://www.socialstyrelsen.se/statistik/statistikdatabas.
16. Gordis L (2014) *Epidemiology (5th Ed.)*, Philadelphia, PA: Elsevier/Saunders.
17. Moderate Alcohol and Cardiovascular Health Trial (MACH15) Available at: https://clinicaltrials.gov/ct2/show/NCT03169530 [Accessed August 7, 2018].
18. NIH to end funding for Moderate Alcohol and Cardiovascular Health trial Available at: https://www.nih.gov/news-events/news-releases/nih-end-funding-moderate-alcohol-cardiovascular-health-trial [Accessed August 7, 2018].
19. NIH Advisory Committee To The Director (2018) *ACD Working Group for Review of the Moderate Alcohol and Cardiovascular Health Trial* Available at: https://acd.od.nih.gov/documents/presentations/06152018Tabak-B.pdf.
20. Fekjaer HO (2013) Alcohol-a universal preventive agent? A critical analysis. *Addiction* 108, 2051–7.
21. Fillmore KM, Stockwell T, Chikritzhs T, Bostrom A, Kerr W (2007) Moderate alcohol use and reduced mortality risk: systematic error in prospective studies and new hypotheses. *Ann Epidemiol* 17, S16–23.
22. Caldwell TM, Rodgers B, Power C, Clark C, Stansfeld SA Drinking histories of self-identified lifetime abstainers and occasional drinkers: findings from the 1958 British Birth Cohort Study. *Alcohol Alcohol* 41, 650–4.
23. Ng Fat L, Shelton N (2012) Associations between self-reported illness and non-drinking in young adults. *Addiction* 107, 1612–20.
24. Staff J, Maggs J (2017) Alcohol and Cigarette Use From Ages 23 to 55: Links With Health and Well-Being in the Long-Term National Child Development Study. *J Stud Alcohol Drugs* 78, 394–403.
25. Naimi TS, Brown DW, Brewer RD, Giles WH, Mensah G, Serdula MK, Mokdad AH, Hungerford DW, et al (2005) Cardiovascular risk factors and confounders among nondrinking and moderate-drinking U.S. adults. *Am J Prev Med* 28, 369–73.
26. Naimi TS, Stockwell T, Zhao J, Xuan Z, Dangardt F, Saitz R, Liang W, Chikritzhs T (2017) Selection biases in observational studies affect associations between "moderate" alcohol consumption and mortality. *Addiction* 112, 207–14.

27. Stockwell T, Zhao J, Panwar S, Roemer A, Naimi T, Chikritzhs T (2016) Do "Moderate" Drinkers Have Reduced Mortality Risk? A Systematic Review and Meta-Analysis of Alcohol Consumption and All-Cause Mortality. *J Stud Alcohol Drugs* 77, 185–98.

28. Zhao J, Stockwell T, Roemer A, Naimi T, Chikritzhs T (2017) Alcohol Consumption and Mortality From Coronary Heart Disease: An Updated Meta-Analysis of Cohort Studies. *J Stud Alcohol Drugs* 78, 375–86.

29. Holmes MV, Dale CE, Zuccolo L, Silverwood RJ, Guo Y, Ye Z, Prieto-Merino D, Dehghan A, et al (2014) Association between alcohol and cardiovascular disease: Mendelian randomisation analysis based on individual participant data. *BMJ* 349, g4164.

30. GBD 2016 Alcohol Collaborators (2018) Alcohol use and burden for 195 countries and territories, 1990-2016: a systematic analysis for the Global Burden of Disease Study 2016. *Lancet* 392, 1015–35.

31. Bagnardi V, Zambon A, Quatto P, Corrao G (2004) Flexible meta-regression functions for modeling aggregate dose-response data, with an application to alcohol and mortality. *Am J Epidemiol* 159, 1077–86.

32. Collins D, Lapsley H (2008) *The Costs of Tobacco, Alcohol and Illicit Drug Abuse to Australian Society in 2004/05.*, Canberra: Australian Department of Health and Ageing.

33. Laslett A-M, Catalano P, Chikritzhs Y, Dale C, Doran C, Ferris J, Jainullabudeen T, Livingston M, et al (2010) *The Range and Magnitude of Alcohol's Harm to Others.,* Fitzroy, Victoria: AER Centre for Alcohol Policy Research, Turning Point Alcohol and Drug Centre, Eastern Health.

34. Chikritzhs TN, Naimi TS, Stockwell TR, Liang W (2015) Mendelian randomisation meta-analysis sheds doubt on protective associations between "moderate" alcohol consumption and coronary heart disease. *Evid Based Med* 20, 38.

35. Colpani V, Baena CP, Jaspers L, van Dijk GM, Farajzadegan Z, Dhana K, Tielemans MJ, Voortman T, et al (2018) Lifestyle factors, cardiovascular disease and all-cause mortality in middle-aged and elderly women: a systematic review and meta-analysis. *Eur J Epidemiol*. doi:10.1007/s10654-018-0374-z.

36. Di Castelnuovo A, Costanzo S, Bagnardi V, Donati MB, Iacoviello L, de Gaetano G (2006) Alcohol dosing and total mortality in men and women: an updated meta-analysis of 34 prospective studies. *Arch Intern Med* 166, 2437–45.

37. Ferrari P, Licaj I, Muller DC, Kragh Andersen P, Johansson M, Boeing H, Weiderpass E, Dossus L, et al (2014) Lifetime alcohol use and overall and cause-specific mortality in the European Prospective Investigation into Cancer and nutrition (EPIC) study. *BMJ Open* 4, e005245.

38. Kunzmann AT, Coleman HG, Huang W-Y, Berndt SI (2018) The association of lifetime alcohol use with mortality and cancer risk in older adults: A cohort study. *PLoS medicine* 15, e1002585.

39. Wood AM, Kaptoge S, Butterworth AS, Willeit P, Warnakula S, Bolton T, Paige E, Paul DS, et al (2018) Risk thresholds for alcohol consumption: combined analysis of individual-participant data for 599 912 current drinkers in 83 prospective studies. *Lancet* 391, 1513–23.

40. Almeida OP, McCaul K, Hankey GJ, Yeap BB, Golledge J, Flicker L (2017) Excessive alcohol consumption increases mortality in later life: a genetic analysis of the health in men cohort study. *Addict Biol* 22, 570–8.

41. Knott CS, Coombs N, Stamatakis E, Biddulph JP (2015) All cause mortality and the case for age specific alcohol consumption guidelines: pooled analyses of up to 10 population based cohorts. *BMJ* 350, h384.

42. Au Yeung SL, Jiang C, Cheng KK, Cowling BJ, Liu B, Zhang W, Lam TH, Leung GM, Schooling CM (2013) Moderate alcohol use and cardiovascular disease from Mendelian randomization. *PLoS ONE* 8, e68054.

43. Yun KE, Chang Y, Yun S-C, Davey Smith G, Ryu S, Cho S-I, Chung EC, Shin H, Khang Y-H (2017) Alcohol and coronary artery calcification: an investigation using alcohol flushing as an instrumental variable. *Int J Epidemiol* 46, 950–62.

44. De Lorgeril M, Salen P, Martin JL, Monjaud I, Delaye J, Mamelle N (1999) Mediterranean diet, traditional risk factors, and the rate of cardiovascular complications after myocardial infarction: final report of the Lyon Diet Heart Study. *Circulation* 99, 779–85.

45. Estruch R, Ros E, Salas-Salvadó J, Covas M-I, Corella D, Arós F, Gómez-Gracia E, Ruiz-Gutiérrez V, et al (2013) Primary prevention of cardiovascular disease with a Mediterranean diet. *N Engl J Med* 368, 1279–90.

46. Patra J, Taylor B, Irving H, Roerecke M, Baliunas D, Mohapatra S, Rehm J (2010) Alcohol consumption and the risk of morbidity and mortality for different stroke types-a systematic review and meta-analysis. *BMC Public Health* 10, 258.

47. Rehm J, Shield KD, Roerecke M, Gmel G (2016) Modelling the impact of alcohol consumption on cardiovascular disease mortality for comparative risk assessments: an overview. *BMC Public Health* 16, 363.

48. Taylor B, Irving HM, Baliunas D, Roerecke M, Patra J, Mohapatra S, Rehm J (2009) Alcohol and hypertension: gender differences in dose-response relationships determined through systematic review and meta-analysis. *Addiction* 104, 1981–90.

49. Roerecke M, Kaczorowski J, Tobe SW, Gmel G, Hasan OSM, Rehm J (2017) The effect of a reduction in alcohol consumption on blood pressure: a systematic review and meta-analysis. *Lancet Public Health* 2, e108–e120.

50. Chen L, Smith GD, Harbord RM, Lewis SJ (2008) Alcohol intake and blood pressure: a systematic review implementing a Mendelian randomization approach. *PLoS Med* 5, e52.

51. Baliunas DO, Taylor BJ, Irving H, Roerecke M, Patra J, Mohapatra S, Rehm J (2009) Alcohol as a risk factor for type 2 diabetes: A systematic review and meta-analysis. *Diabetes Care* 32, 2123–32.

52. Kerr WC, Ye Y (2010) Relationship of life-course drinking patterns to diabetes, heart problems, and hypertension among those 40 and older in the 2005 U.S. National Alcohol Survey. *J Stud Alcohol Drugs* 71, 515–25.

53. Knott C, Bell S, Britton A (2015) Alcohol Consumption and the Risk of Type 2 Diabetes: A Systematic Review and Dose-Response Meta-analysis of More Than 1.9 Million Individuals From 38 Observational Studies. *Diabetes Care* 38, 1804–12.

54. Cho Y, Shin S-Y, Won S, Relton CL, Davey Smith G, Shin M-J (2015) Alcohol intake and cardiovascular risk factors: A Mendelian randomisation study. *Sci Rep* 5, 18422.

55. Lawlor DA, Nordestgaard BG, Benn M, Zuccolo L, Tybjaerg-Hansen A, Davey Smith G (2013) Exploring causal associations between alcohol and coronary heart disease risk factors: findings from a Mendelian randomization study in the Copenhagen General Population Study. *Eur Heart J* 34, 2519–28.

56. Hirst J, Aronson J, Feakins B, Ma C, Farmer A, Stevens R (2017) Short-and medium-term effects of light to moderate alcohol intake on glycaemic control in diabetes mellitus: a systematic review and meta-analysis of randomized trials. *Diabetic Medicine* 34, 604–11.

57. Briel M, Ferreira-Gonzalez I, You JJ, Karanicolas PJ, Akl EA, Wu P, Blechacz B, Bassler D, et al (2009) Association between change in high density lipoprotein cholesterol and cardiovascular disease morbidity and mortality: systematic review and meta-regression analysis. *BMJ* 338, b92.

58. Schwartz GG, Olsson AG, Abt M, Ballantyne CM, Barter PJ, Brumm J, Chaitman BR, Holme IM, et al (2012) Effects of dalcetrapib in patients with a recent acute coronary syndrome. *N Engl J Med* 367, 2089–99.

59. Voight BF, Peloso GM, Orho-Melander M, Frikke-Schmidt R, Barbalic M, Jensen MK, Hindy G, Hólm H, et al (2012) Plasma HDL cholesterol and risk of myocardial infarction: a mendelian randomisation study. *Lancet* 380, 572–80.

60. Brien SE, Ronksley PE, Turner BJ, Mukamal KJ, Ghali WA (2011) Effect of alcohol consumption on biological markers associated with risk of coronary heart disease: systematic review and meta-analysis of interventional studies. *BMJ* 342, d636.

61. Berger NA, Savvides P, Koroukian SM, Kahana EF, Deimling GT, Rose JH, Bowman KF, Miller RH (2006) Cancer in the elderly. *Trans Am Clin Climatol Assoc* 117, 147–55; discussion 155–6.

62. IARC (2010) *Alcohol Consumption and Ethyl Carbamate. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. (Vol. 96)*, Lyon, France: IARC.

63. World Cancer Research Fund International/American Institute for Cancer Research (2016) *Continuous Update Project Report: Diet, Nutrition, Physical Activity and Stomach Cancer*. London: WCRF.

64. Bagnardi V, Rota M, Botteri E, Tramacere I, Islami F, Fedirko V, Scotti L, Jenab M, et al (2015) Alcohol consumption and site-specific cancer risk: a comprehensive dose-response meta-analysis. *Br J Cancer* 112, 580–93.

65. Michael J, Howard LE, Markt SC, De Hoedt A, Bailey C, Mucci LA, Freedland SJ, Allott EH (2018) Early-Life Alcohol Intake and High-Grade Prostate Cancer: Results from an Equal-Access, Racially Diverse Biopsy Cohort. *Cancer Prev Res (Phila)* 11, 621–8.

66. Zhao J, Stockwell T, Roemer A, Chikritzhs T (2016) Is alcohol consumption a risk factor for prostate cancer? A systematic review and meta-analysis. *BMC Cancer* 16, 845.

67. Andreasson S, Chikritzhs T, Dangardt F, Holder H, Naimi T, Stockwell T (2016) *Alcohol and Society: Alcohol and Cancer*, Stockholm: Swedish Society of Medicine & IOGT-NTO.

68. Sherk A, Stockwell T, Rehm J, Dorocicz J, Shield KD (2017) *The International Model of Alcohol Harms and Policies (InterMAHP): A Comprehensive Guide to the Estimation of Alcohol-Attributable Morbidity and Mortality.*, Canadian Institute for Substance Use Research, University of Victoria, British Columbia, Canada. Available at: www.intermahp.cisur.ca.

69. Schwarzinger M, Pollock BG, Hasan OSM, Dufouil C, Rehm J (2018) Contribution of alcohol use disorders to the burden of dementia in France 2008-13: a nationwide retrospective cohort study. *Lancet Public Health* 3, e124–e132.

70. Winblad B, Amouyel P, Andrieu S, Ballard C, Brayne C, Brodaty H, Cedazo-Minguez A, Dubois B, et al (2016) Defeating Alzheimer's disease and other dementias: a priority for European science and society. *Lancet Neurol* 15, 455–532.

71. Downer B, Zanjani F, Fardo DW (2014) The relationship between midlife and late life alcohol consumption, APOE e4 and the decline in learning and memory among older adults. *Alcohol Alcohol* 49, 17–22.

72. Verbaten MN (2009) Chronic effects of low to moderate alcohol consumption on structural and functional properties of the brain: beneficial or not? *Hum Psychopharmacol* 24, 199–205.

73. Almeida OP, Hankey GJ, Yeap BB, Golledge J, Flicker L (2014) Alcohol consumption and cognitive impairment in older men: a mendelian randomization study. *Neurology* 82, 1038–44.

74. Kroenke CD, Rohlfing T, Park B, Sullivan EV, Pfefferbaum A, Grant KA (2014) Monkeys that voluntarily and chronically drink alcohol damage their brains: a longitudinal MRI study. *Neuropsychopharmacology* 39, 823–30.

75. WHO (2017) *Global Action Plan on the Public Health Response to Dementia 2017–2025*. Geneva: World Health Organization; 2017.

76. Martínez-Lapiscina EH, Clavero P, Toledo E, Estruch R, Salas-Salvadó J, San Julián B, Sanchez-Tainta A, Ros E, et al (2013) Mediterranean diet improves cognition: the PREDIMED-NAVARRA randomised trial. *J Neurol Neurosurg Psychiatry* 84, 1318–25.

77. Sabia S, Fayosse A, Dumurgier J, Dugravot A, Akbaraly T, Britton A, Kivimäki M, Singh-Manoux A (2018) Alcohol consumption and risk of dementia: 23 year follow-up of Whitehall II cohort study. *BMJ* 362, k2927.

78. Topiwala A, Ebmeier KP (2018) Effects of drinking on late-life brain and cognition. *Evid Based Ment Health* 21, 12–5.

79. Hassing LB (2018) Light Alcohol Consumption Does Not Protect Cognitive Function: A Longitudinal Prospective Study. *Front Aging Neurosci* 10, 81.

80. Cherpitel CJ (2007) Alcohol and injuries: a review of international emergency room studies since 1995. *Drug Alcohol Rev* 26, 201–14.

81. Taylor B, Irving HM, Kanteres F, Room R, Borges G, Cherpitel C, Greenfield T, Rehm J (2010) The more you drink, the harder you fall: a systematic review and meta-analysis of how acute alcohol consumption and injury or collision risk increase together. *Drug Alcohol Depend* 110, 108–16.

82. Zeisser C, Stockwell TR, Chikritzhs T, Cherpitel C, Ye Y, Gardner C (2013) A systematic review and meta-analysis of alcohol consumption and injury risk as a function of study design and recall period. *Alcohol Clin Exp Res* 37 Suppl 1, E1–8.

83. Dionyssiotis Y (2012) Analyzing the problem of falls among older people. *Int J Gen Med* 5, 805–13.

84. Fell JC, Scherer M (2017) Estimation of the Potential Effectiveness of Lowering the Blood Alcohol Concentration (BAC) Limit for Driving from 0.08 to 0.05 Grams per Deciliter in the United States. *Alcohol Clin Exp Res* 41, 2128–39.

85. Taylor B, Rehm J (2012) The relationship between alcohol consumption and fatal motor vehicle injury: high risk at low alcohol levels. *Alcohol Clin Exp Res* 36, 1827–34.

86. Dadgar I, Norström T (2017) Short-term and long-term effects of GDP on traffic deaths in 18 OECD countries, 1960-2011. *J Epidemiol Community Health* 71, 146–53.

87. Martin TL, Solbeck PAM, Mayers DJ, Langille RM, Buczek Y, Pelletier MR (2013) A review of alcohol-impaired driving: the role of blood alcohol concentration and complexity of the driving task. *J Forensic Sci* 58, 1238–50.

88. Santos AMR dos, Rodrigues RAP, Diniz MA (2015) Trauma in the elderly caused by traffic accident: integrative review. *Rev Esc Enferm USP* 49, 162–72.

89. IIHS (2016) Fatality facts 2015, Older people. Available at: http://www.iihs.org/iihs/topics/t/older-drivers/fatalityfacts/older-people/2015 [Accessed August 14, 2018].

90. Sklar AL, Boissoneault J, Fillmore MT, Nixon SJ (2014) Interactions between age and moderate alcohol effects on simulated driving performance. *Psychopharmacology (Berl)* 231, 557–66.

91. Scheetz LJ (2015) One for the Road: A Comparison of Drinking and Driving Behavior Among Younger and Older Adults Involved in Fatal Crashes. *J Trauma Nurs* 22, 187–93.

92. Ridolfo B, Stevenson C (2001) *The Quantification of Drug-Caused Mortality and Morbidity in Australia, 1998*, Australian Institute of Health and Welfare.

93. Wu HZ, Barry LC, Duan Y, Bohannon RW, Covault JM, Grady JJ (2017) Acute Effects of Moderate Alcohol Consumption on Postural Stability in Older Adults. *Percept Mot Skill*s 124, 912–31.

94. Li Y, Jensen GA (2012) Effects of drinking on hospital stays and emergency room visits among older adults. *J Aging Health* 24, 67–91.

95. Tait RJ, French DJ, Burns RA, Byles JE, Anstey KJ (2013) Alcohol, hospital admissions, and falls in older adults: a longitudinal evaluation. *Int Psychogeriatr* 25, 901–12.

96. Berg KM, Kunins HV, Jackson JL, Nahvi S, Chaudhry A, Harris KA, Malik R, Arnsten JH (2008) Association between alcohol consumption and both osteoporotic fracture and bone density. *Am J Med* 121, 406–18.

97. Drake MT, Murad MH, Mauck KF, Lane MA, Undavalli C, Elraiyah T, Stuart LM, Prasad C, et al (2012) Clinical review. Risk factors for low bone mass-related fractures in men: a systematic review and meta-analysis. *J Clin Endocrinol Metab* 97, 1861–70.

98. Papaioannou A, Kennedy CC, Cranney A, Hawker G, Brown JP, Kaiser SM, Leslie WD, O'Brien CJM, et al (2009) Risk factors for low BMD in healthy men age 50 years or older: a systematic review. *Osteoporos Int* 20, 507–18.

99. Waugh EJ, Lam M-A, Hawker GA, McGowan J, Papaioannou A, Cheung AM, Hodsman AB, Leslie WD, et al (2009) Risk factors for low bone mass in healthy 40-60 year old women: a systematic review of the literature. *Osteoporos Int* 20, 1–21.

100. Kubo JT, Stefanick ML, Robbins J, Wactawski-Wende J, Cullen MR, Freiberg M, Desai M (2013) Preference for wine is associated with lower hip fracture incidence in post-menopausal women. *BMC Womens Health* 13, 36.

101. Ortolá R, García-Esquinas E, Galán I, Guallar-Castillón P, López-García E, Banegas JR, Rodríguez-Artalejo F (2017) Patterns of alcohol consumption and risk of falls in older adults: a prospective cohort study. *Osteoporos Int* 28, 3143–52.

102. Yon Y, Mikton C, Gassoumis Z, Wilber K (2017) Elder abuse prevalence in community settings: a systematic review and meta-analysis. *Lancet Glob Health* 5, e147–e156.

103. Pillemer K, Burnes D, Riffin C, Lachs MS (2016) Elder Abuse: Global Situation, Risk Factors, and Prevention Strategies. *Gerontologist* 56 Suppl 2, S194–205.

104. Ahnlund P, Andersson T, Snellman F, Sundström M, Heimer G (2017) Prevalence and Correlates of Sexual, Physical, and Psychological Violence Against Women and Men of 60 to 74 Years in Sweden. *J Interpers Violence*, 886260517696874.

105. Warmling D, Lindner SR, Coelho EBS (2017) Intimate partner violence prevalence in the elderly and associated factors: systematic review. *Cien Saude Colet* 22, 3111–25.

106. Murphy K, Waa S, Jaffer H, Sauter A, Chan A (2013) A literature review of findings in physical elder abuse. *Can Assoc Radiol J* 64, 10–4.

107. Caputo F, Vignoli T, Leggio L, Addolorato G, Zoli G, Bernardi M (2012) Alcohol use disorders in the elderly: a brief overview from epidemiology to treatment options. *Exp Gerontol* 47, 411–6.

108. Borges G, Loera CR (2010) Alcohol and drug use in suicidal behaviour. *Current Opinion in Psychiatry* 23, 195–204.

109. Blow FC, Brockmann LM, Barry KL (2004) Role of alcohol in late-life suicide. *Alcohol Clin Exp Res* 28, 48S–56S.

110. Hilton ME (1987) Demographic characteristics and the frequency of heavy drinking as predictors of self-reported drinking problems. *Br J Addict* 82, 913–25.

111. Midanik LT, Clark WB (1995) Drinking-related problems in the United States: description and trends, 1984-1990. *J Stud Alcohol* 56, 395–402.

112. Mäkelä K, Mustonen H (2000) Relationships of drinking behaviour, gender and age with reported negative and positive experiences related to drinking. *Addiction* 95, 727–36.

113. Jacobs LG, Berman A (2003) Respiratory disease in the elderly. *Clinics in Geriatric Medicine* 19, xi – xii .

114. Cunha BA (2001) Pneumonia in the elderly. *Clinical Microbiology and Infection* 7, 581 – 588.

115. Samokhvalov AV, Irving HM, Rehm J (2010) Alcohol consumption as a risk factor for pneumonia: a systematic review and meta-analysis. *Epidemiol Infect* 138, 1789–95.

116. Kuo C-F, Grainge MJ, Zhang W, Doherty M (2015) Global epidemiology of gout: prevalence, incidence and risk factors. *Nat Rev Rheumatol* 11, 649–62.

117. Zhu Y, Pandya BJ, Choi HK (2011) Prevalence of gout and hyperuricemia in the US general population: the National Health and Nutrition Examination Survey 2007-2008. *Arthritis Rheum* 63, 3136–41.

118. Singh JA, Reddy SG, Kundukulam J (2011) Risk factors for gout and prevention: a systematic review of the literature. *Curr Opin Rheumatol* 23, 192–202.

119. Wang M, Jiang X, Wu W, Zhang D (2013) A meta-analysis of alcohol consumption and the risk of gout. *Clin Rheumatol* 32, 1641–8.

120. Roddy E, Zhang W, Doherty M (2007) Are joints affected by gout also affected by osteoarthritis? *Ann Rheum Dis* 66, 1374–7.

121. Kc R, Voigt R, Li X, Forsyth CB, Ellman MB, Summa KC, Turek FW, Keshavarzian A, et al (2015) Induction of Osteoarthritis-like Pathologic Changes by Chronic Alcohol Consumption in an Experimental Mouse Model. *Arthritis & rheumatology (Hoboken, NJ)* 67, 1678–80.

122. Batista P, Pereira A (2016) Quality of Life in Patients with Neurodegenerative Diseases.

123. Ishihara LS, Cheesbrough A, Brayne C, Schrag A (2007) Estimated life expectancy of Parkinson's patients compared with the UK population. *J Neurol Neurosurg Psychiatr* 78, 1304–9.

124. Bettiol SS, Rose TC, Hughes CJ, Smith LA (2015) Alcohol Consumption and Parkinson's Disease Risk: A Review of Recent Findings. *J Parkinsons Dis* 5, 425–42.

125. Dufour MC, Archer L, Gordis E (1992) Alcohol and the elderly. *Clin Geriatr Med* 8, 127–41.

126. Ebrahim IO, Shapiro CM, Williams AJ, Fenwick PB (2013) Alcohol and sleep I: effects on normal sleep. *Alcohol Clin Exp Res* 37, 539–49.

127. Roehrs T, Roth T (2001) Sleep, sleepiness, sleep disorders and alcohol use and abuse. *Sleep Med Rev* 5, 287–97.

128. Stein MD, Friedmann PD (2005) Disturbed sleep and its relationship to alcohol use. *Subst Abus* 26, 1–13.

129. Raninen J, Härkönen J, Landberg J (2016) Long-term effects of changes in Swedish alcohol policy: can alcohol policies effective during adolescence impact consumption during adulthood? *Addiction* 111, 1021–6.

130. Babor TF, Babor T, Caetano R, Casswell S, Edwards G, Giesbrecht N (2010) *Alcohol: No Ordinary Commodity: Research and Public Policy*, Oxford University Press.

131. Nelson TF, Xuan Z, Babor TF, Brewer RD, Chaloupka FJ, Gruenewald PJ, Holder H, Klitzner M, et al (2013) Efficacy and the strength of evidence of U.S. alcohol control policies. *Am J Prev Med* 45, 19–28.

132. Holmes J, Meng Y, Meier PS, Brennan A, Angus C, Campbell-Burton A, Guo Y, Hill-McManus D, Purshouse RC (2014) Effects of minimum unit pricing for alcohol on different income and socioeconomic groups: a modelling study. *Lancet* 383, 1655–64.

133. Zhao J, Stockwell T (2017) The impacts of minimum alcohol pricing on alcohol attributable morbidity in regions of British Colombia, Canada with low, medium and high mean family income. *Addiction* 112, 1942–51.

134. Brady JE, Baker SP, Dimaggio C, McCarthy ML, Rebok GW, Li G (2009) Effectiveness of mandatory alcohol testing programs in reducing alcohol involvement in fatal motor carrier crashes. *Am J Epidemiol* 170, 775–82.

135. US Dietary Guidelines 2015-2020, appendix 9, Alcohol (2015) Available at: https://health.gov/dietaryguidelines/2015/guidelines/appendix-9/ [Accessed August 15, 2018].

136. National Health and Medical Research Council (2009) *Australian Guidelines to Reduce Health Risks from Drinking Alcohol.*, Canberra: Commonwealth of Australia.

137. Scafato E, Galluzzo L, Ghirini S, Gandin C, Martire S Low risk drinking guidelines in Europe: overview of RARHA survey results. Available at: http://www.rarha.eu/Resources/Guidelines/Documents/Overview%20WP5_T1%20LowRiskDrinkingGuidelines_FINAL.pdf [Accessed August 14, 2018].

138. Butt P, Beirness D, Cesa F, Gliksman L, Paradis C, Stockwell T (2011) *Alcohol and Health in Canada: A Summary of Evidence and Guidelines for Low-Risk Drinking*, Canadian Centre on Sustance Abuse.

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