Alcohol and healthspan: knowing the risks and how to mitigate them

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Peter Attia December 2, 2025

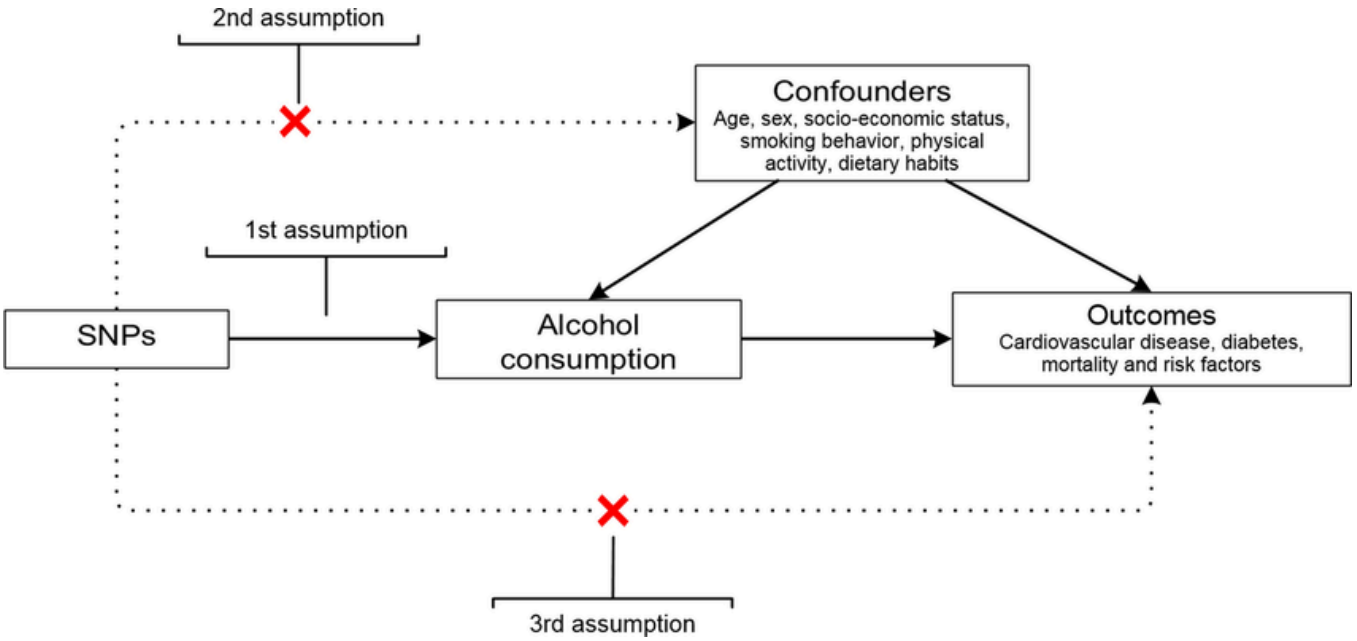

Figure 1: The design and assumptions of Mendelian randomization analyses. From van de Luitgaarden *et al.* 2022.[1]

Alcohol occupies a complex and often contradictory role in society, celebrated for its social and cultural significance yet scrutinized for its potential to harm health. While moderate drinking has long been associated with certain health benefits, recent research increasingly challenges these assumptions, revealing risks even at low levels of consumption. At the same time, societal attitudes toward alcohol are shifting, with growing awareness of its negative impact on long-term health and quality of life. In fact, in 2023, the World Health Organization declared there is *no safe dose* of alcohol consumption. The popular press headlines on the topic are leaving light-to-moderate drinkers asking, “how worried should I be?”

Against this backdrop, we are dedicating this premium article to an examination of alcohol’s effects on health, focusing not just on how it influences *lifespan* (as discussed in a previous premium piece) but also on its more immediate and profound effects on *healthspan*—the portion of life spent in good health. We will explore the relationship between alcohol and the four horsemen of chronic disease, outline day-to-day cumulative impacts of alcohol consumption, present potential disease risk modifiers, and summarize approaches for alcohol harm reduction—including not only strategies for drinking less, but also strategies for making any alcohol you do consume less damaging. In sum, this piece provides a realistic, quantified look at the risks associated with alcohol consumption, allowing you to find a balanced, risk-informed relationship with alcohol.

The (debunked) J-shaped curve theory

Conventional wisdom has long maintained that light-to-moderate alcohol consumption may in fact be *beneficial* for health, a belief that stems in part from observational studies reporting that the relationship between alcohol consumption and all-cause mortality or other health outcomes follows a J-shaped curve. In other words, such research indicates that the lowest risk occurs not with complete alcohol abstention, but instead with *light-to-moderate* alcohol consumption, while higher risk is seen in abstainers and heavier drinkers. (Light-to-moderate is less than one drink per day for women and less than two per day for men.) Unfortunately for wine lovers, this apparent "protection" with light drinking crumbles once you dig into the data. While we covered this subject in depth two years ago in our previous premium article on alcohol, a few broad points bear reiteration, as they highlight some inherent limitations of studying alcohol intake.

Few randomized trials exist on the health effects of alcohol, and those that do exist tend to be limited in cohort size and duration. This is unlikely to change with time due to ethical concerns of randomizing individuals to alcohol consumption, so we're mostly left with observational studies, which suffer from healthy user bias (low-to-moderate drinkers are likely to engage in other healthy behaviors that may confound results) and self-reported alcohol intake data of questionable reliability. (Indeed, even if we assume that participants report their total intake accurately, such questionnaires often miss important nuances such as the distribution of alcohol intake—i.e., whether seven drinks per week represents a single glass of wine each evening or a one-night, seven-shot tequila binge.) Further, *former* drinkers are typically lumped together with life-long abstainers in epidemiology studies, yet those who make the decision to quit drinking entirely after years of alcohol consumption often do so because they have developed alcohol use disorder or chronic health concerns that would be substantially exacerbated or accelerated by alcohol intake (the so called "sick-quitter" bias). This can artificially inflate the apparent risk with alcohol abstention, as many in the "abstainer" group are former drinkers with baseline health problems.

Better data with Mendelian randomization

With the reliability concerns of observational studies and the ethical concerns of randomized trials, are we doomed to remain clueless on the true impact of alcohol on human health? Fortunately, no—we can turn to Mendelian randomization (MR), a methodology that circumvents the problems associated with *both* traditional observational studies and randomized trials. MR uses common genetic variants associated with higher or lower alcohol consumptions as a proxy for alcohol consumption itself. Because these genes are randomly distributed and are fixed at conception, this study design essentially constitutes a *naturally* randomized study, permitting us to make causal inferences regarding the effects of alcohol consumption on health.

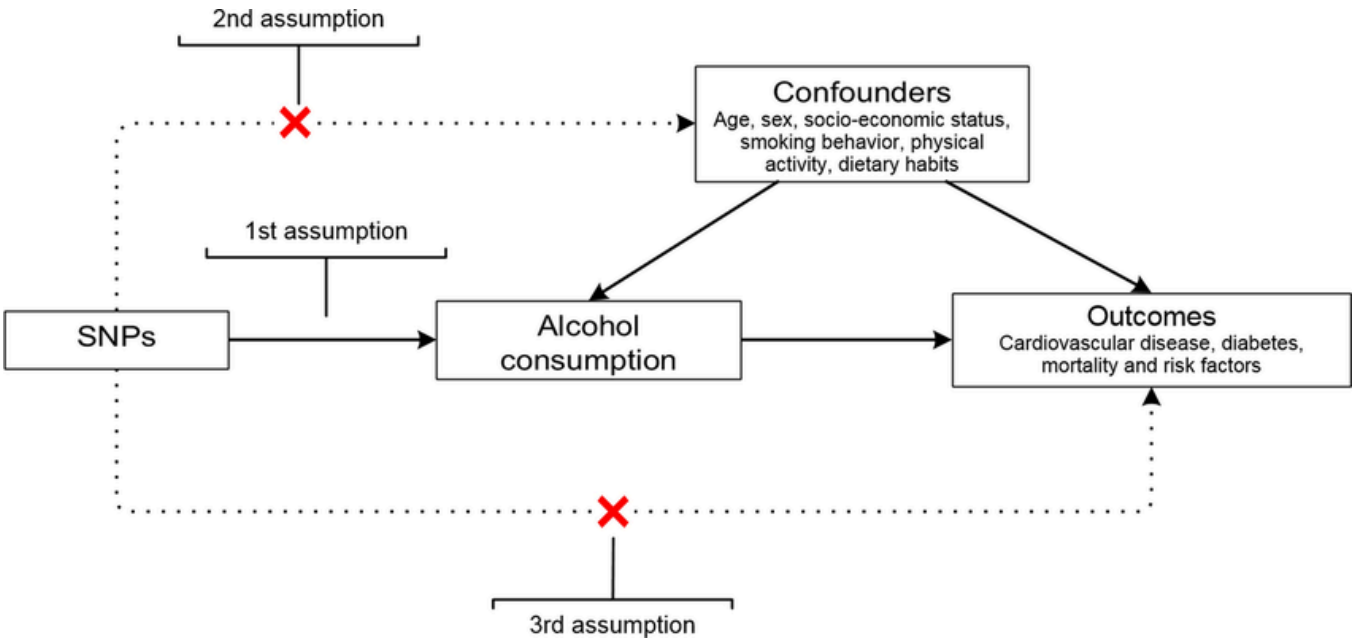

Figure 1: The design and assumptions of Mendelian randomization analyses. From van de Luitgaarden *et al.* 2022.[1]

Indeed, MR analysis on British UK Biobank participants reveals a *linear* increase in all-cause mortality with increasing alcohol intake, with each 8-g/day increase in alcohol intake associated with a 27% higher risk of death by any cause (odds ratio=1.27; 95% CI: 1.16–1.39).[2] Still, while MR analyses have provided eye-opening data that ultimately put to rest the "J-shaped curve" theory of alcohol and mortality, this methodology has not been applied to all health outcomes potentially related to alcohol intake, which range from cancer and cardiovascular disease to accidental deaths or injuries from automotive accidents or falls. Because MR requires the fulfilment of certain validation criteria regarding the relationship between the genetic proxy, exposure of interest (i.e., alcohol consumption), and the outcome of interest (**Figure 1**), not all questions *can* be addressed with this study design. As such, a close look at a *combination* of available data sources—including randomized trials and MR analyses where available, epidemiology studies, and mechanistic research in animals—allows us to come to the most rational conclusions regarding the full spectrum of alcohol's effects on healthspan and lifespan.

Alcohol and the four horseman

Roughly 80% of deaths among non-smokers over 50 years of age can be attributed to what I call the "four horsemen of disease." These include (1) atherosclerotic disease (including cardiovascular disease and cerebrovascular disease), (2) cancer, (3) neurodegenerative disease (with Alzheimer's disease being the most common), and (4) metabolic disease (a spectrum of everything from hyperinsulinemia to insulin resistance to fatty liver disease and type 2 diabetes).

Ethanol—the type of alcohol present in alcoholic drinks—has the potential to impact each and every one of the horsemen, yet various biological systems are not affected equally, nor at equal alcohol doses. Indeed, the adage that "the dose makes the poison" holds just as true for alcohol as for any other substance. So what are the magnitudes of these various risks? And at what level of intake do they become significant concerns?

Cardiovascular disease

One of the clearest risks associated with alcohol consumption concerns cardiovascular health. Epidemiology data indicate that alcohol intake correlates with increases in systolic blood pressure in a dose-dependent manner, with those consuming 12 g/day (about 1 drink) exhibiting an average 1.25-mmHg increase in systolic blood pressure relative to non-drinkers, and those consuming 48 g/day exhibiting a 4.90-mmHg increase.[3]

But again, the strongest evidence regarding alcohol and heart health comes from MR analyses, which have reported a dose-response relationship between alcohol consumption and coronary artery disease. For instance, a 2022 MR study involving nearly 400,000 participants found that each 1–standard-deviation increase in genetically predicted alcohol consumption (for this study, this translates to approximately 7–8 drinks per week) was associated with a 30% higher risk of hypertension and a 40% higher risk of coronary artery disease.[4] Nonlinear MR modeling in this study suggests there may be minimal change in risk at low intakes (on the order of one drink per day or less), but once consumption rises above about two drinks per day, the risk curve becomes steeper.

It bears note that the positive association between alcohol consumption and heart disease is not *exclusively* mediated by the former's effects on blood pressure. The MR analysis described above also revealed a significant, exponential correlation between genetically predicted alcohol intake and circulating levels of low-density lipoprotein cholesterol (LDL-C), a major risk factor for atherosclerosis (**Figure 2**). This means that even for someone who *doesn't* experience changes in blood pressure with alcohol consumption, alcohol may still increase cardiovascular risk through its effects on circulating lipid levels.

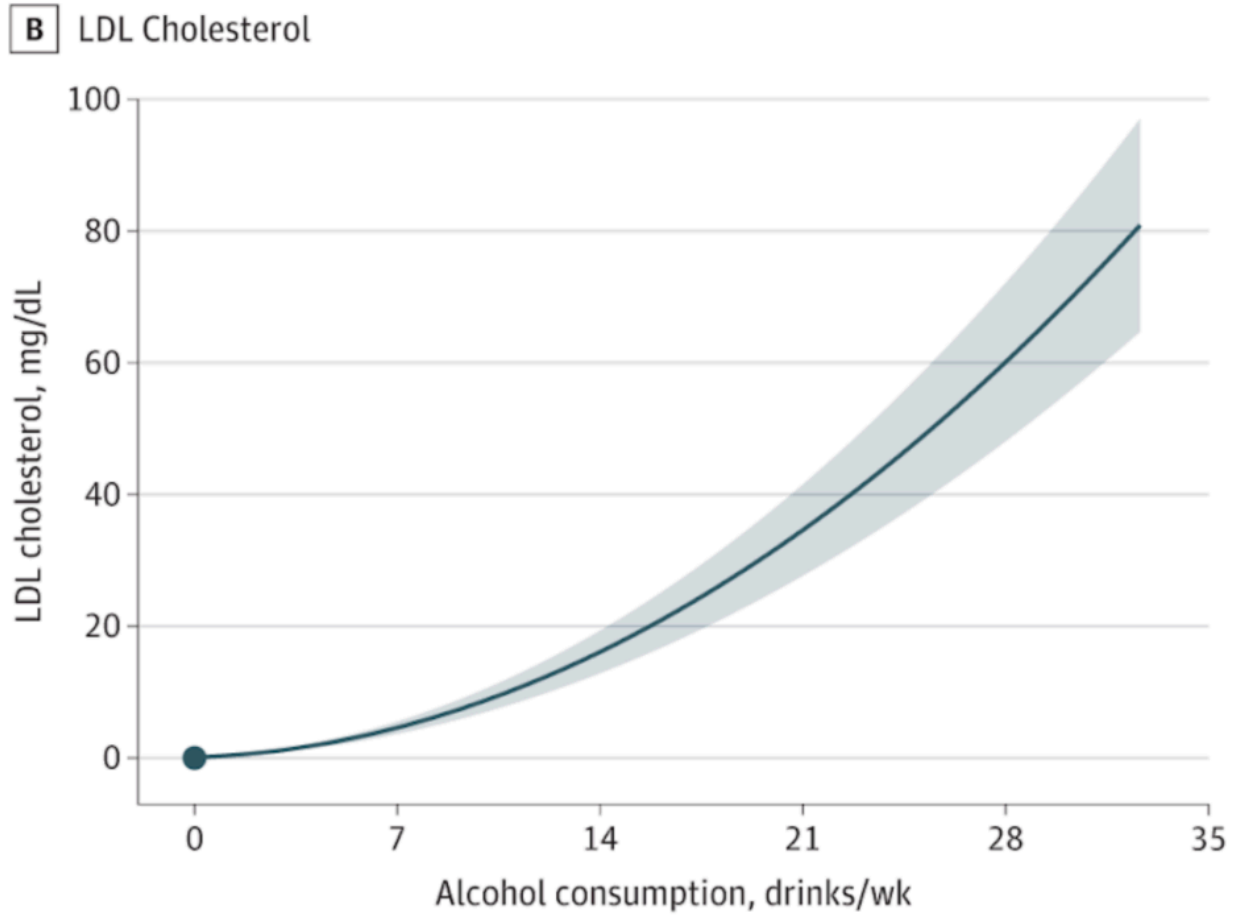

Figure 2: The relationship between genetically predicted alcohol consumption and low-density lipoprotein cholesterol (LDL-C). Mendelian randomization analysis reveals an exponential rise in LDL-C as genetically predicted alcohol consumption increases. From Biddinger *et al.* 2022.[4]

Metabolic disease

When it comes to the health consequences of alcohol consumption, one of the first things that likely comes to mind is liver damage. The liver, which plays a central role in metabolic function and metabolic regulation, is the primary site of alcohol processing and is therefore particularly exposed to the toxic compounds resulting from alcohol metabolism.

Life-threatening in its own right, liver disease also contributes to other serious metabolic derangements, including type 2 diabetes and dyslipidemia, and indeed, alcohol consumption increases risk of all of these conditions, in addition to increasing risk of obesity. For instance, a 2023 Mendelian randomization study involving over 400,000 participants provided evidence of a causal, dose-dependent relationship between alcohol consumption and metabolic health issues. Each one-drink-per-week increase in genetically predicted alcohol intake was found to be associated with an 8% increase in risk of obesity (OR=1.08; 95% CI: 1.06–1.10), and a 10% increase in risk of type 2 diabetes (OR=1.10; 95% CI: 1.06–1.13) relative to non-drinkers, though these differences in risk only became statistically significant at moderate-to-high levels of intake—above at least seven drinks per week.[5] While liver damage is a significant pathway through which alcohol increases risk for these metabolic disorders, these associations may

also arise from alcohol's effects on inflammation, hormone balance, and appetite, as we will discuss in greater detail later in this piece. Further, metabolic dysfunction brought on by alcohol consumption can increase risk for all of the other horsemen of chronic disease.

Neurodegenerative disease

The brain's heavy reliance on healthy blood flow makes it particularly vulnerable to vascular disruption, and thus, given alcohol's demonstrated impact on vascular systems—evident in both cardiovascular and metabolic disease—similar trends in brain health are perhaps unsurprising. Large-scale MR analyses are fairly consistent in demonstrating a linear increase in risk of dementia and cognitive decline with increasing alcohol consumption[6,7] (though it's worth noting that most MR analyses specifically on Alzheimer's disease have found no evidence of a causal link[8,9]). For instance, one of the largest such studies—involving nearly 600,000 adults from the United States and United Kingdom—reported that each standard-deviation increase in genetically predicted alcohol consumption per week was associated with a 15% increase in risk of dementia (OR=1.15; 95% CI: 1.03–1.27) over a mean follow-up of 4.3 years (US cohort) or 12.4 years (UK cohort).[6] Interestingly, this study also found that individuals who developed dementia were also more likely to reduce their alcohol intake, suggesting that the apparent "protective" effect of alcohol consumption reported in many strict observational studies may in fact be due to reverse causality: those who abstain from alcohol may be doing so because they are *already* experiencing health concerns such as cognitive impairment (again, this is the so-called "sick quitter" bias, as discussed in more detail in our last premium article on alcohol).

These associations reported by MR analyses are corroborated by longitudinal neuroimaging studies showing accelerated brain aging and loss of gray and white matter with increasing alcohol intake.[10–12] While these studies are observational in nature and thus may be susceptible to the influence of confounding factors, the fact that the associations scale with the level of alcohol consumption—and their agreement with MR analyses—lends credence to a causal relationship. Further, neuroimaging studies have also shown that alcoholics who abstain from alcohol for a few weeks to months *regain* a degree of brain tissue volume, whereas those who relapse continue to exhibit losses.[13,14] This strengthens the evidence for the neuroinflammatory and neurotoxic effects of alcohol while simultaneously signaling the potential benefits that may come with reducing alcohol intake, even among those with a history of heavy consumption.

Cancer

Recent news has highlighted potential links between alcohol and cancer, and both ethanol and its primary metabolite, acetaldehyde, are indeed classified as Group 1 carcinogens according to the International Agency for Research on Cancer (IARC). Experiments in cell culture and animal models provide strong evidence that acetaldehyde in particular can drive DNA damage while simultaneously disrupting the cell's capacity for DNA repair and DNA synthesis, thus increasing risk of cancer development.[15,16]

Again, we can turn to several MR analyses to validate this link in humans. Such research has generally pointed to a causal relationship between alcohol and incidence of various cancers, including those of the oropharynx (oral cavity and throat), esophagus, colorectum, and liver.[17,18] The strongest associations tend to be with oropharyngeal and esophageal cancers; gene variants that are predictive of higher alcohol intake have been reported to raise risk of these cancer types by as much as *two- or three-fold*, depending on the analysis.[19–21] The heightened risk for these particular cancers likely reflects the direct contact that alcohol has with these areas of the body.

While most MR studies on alcohol and cancer have not examined dose-response relationships, meta-analyses of observational studies indicate that such a relationship exists and follows an exponential trajectory, such that the jump in risk between moderate and heavy drinking, for instance, is greater than the jump in risk between low and moderate drinking.[22,23] (Of note, various epidemiological studies have also reported positive associations between alcohol consumption and cancers of the breast, ovaries, and bladder, though these correlations largely disappear completely in MR analyses, suggesting that they were likely due to common confounding variables such as smoking or obesity.[24,25])

A repeated pattern

Considering evidence from all four horsemen, a pattern repeatedly emerges: the relationship between alcohol and health risk is dose-dependent, and the slope steepens once intake rises above 1–2 drinks per day. Thus, the clearest evidence for alcohol's detrimental long-term effects comes from individuals who drink heavily (particularly in binge-drinking patterns of >4 drinks in a single sitting). However, though low levels of intake might be associated with much smaller increases in risk relative to abstainers, the difference is not zero.

The day-to-day costs behind the chronic risks

The long-term disease risks don't arise from nowhere and aren't attributable to any single drink you might have over your lifetime. Rather, they represent an accumulation of small, day-to-day impacts on both the cellular level and the level of whole-body physiology, from effects on DNA and inflammation to effects on sleep, psychological stress, and physical recovery. While we won't delve into every acute disruption and mechanism through which alcohol may impact disease risk, a summary of key points helps to *understand* the long-term risks and provides a necessary foundation for our subsequent discussion of risk mitigation strategies. By knowing the mechanisms through which alcohol can lead to chronic disease, we can better understand how to *counteract* these negative effects.

Inflammation & immune effects

Alcohol and acetaldehyde trigger oxidative stress, especially in chronic heavy drinkers,[26] resulting in pro-inflammatory signals throughout the body. In binge drinking studies, this shows up as measurable spikes in inflammatory molecules like TNF-α and IL-6 the next day, which contribute to hangover symptoms such as headache and malaise.[27] Additionally, because

alcohol can damage the lining of the gastrointestinal tract, alcohol consumption is thought to increase permeability of the gut to bacteria, which in turn may trigger systemic immune activation.[28]

At the same time, alcohol actually *suppresses* some immune defenses. White blood cells involved in the body's immune response become less effective at fighting pathogens after intoxication.[29] The result is an immune system that is both chronically activated systemically *and* impaired for handling acute insults.

Because the pro-inflammatory effects of alcohol are chronic and systemic, they have the potential to impact many different tissues throughout the body, and because chronic, systemic inflammation has been implicated in the pathogenic process for all four horsemen, alcohol thus increases risk of chronic disease *across the board*. Counterintuitively, the immune suppressive effects of alcohol may also increase chronic inflammation, as a reduced ability to fight acute infections can result in more severe episodes of infectious disease and associated tissue damage.

Hormone and growth effects

A key mechanism through which alcohol exerts negative health effects is through its ability to activate the hypothalamic-pituitary-adrenal (HPA) axis, increasing the stress hormone cortisol and stimulating the "fight-or-flight" sympathetic nervous system. Indeed, as discussed in our previous premium article on cardiovascular fitness metrics, one of the most predictable and easy-to-monitor consequences of alcohol consumption is an increase in heart rate and decrease in heart rate variability—metrics that are indicative of autonomic balance and increase when the sympathetic nervous system is activated.

This stress signal carries throughout the night, where alcohol fragments sleep architecture. We discuss sleep more with Matt Walker on the podcast, but in brief: even one or two drinks within three hours of bed time can have significant detrimental impacts on sleep. The most consistent finding is a suppression of rapid eye movement (REM) sleep in the first half of the night, along with lighter, more disrupted sleep throughout the night. This is evident the next day, when even a few drinks can lead to higher resting heart rates and lower heart rate variability. These are signs that the body never fully settled overnight.

These disruptions can extend into hormone signaling pathways beyond the HPA axis, particularly where anabolic hormones are concerned. Alcohol consumption suppresses the body's primary anabolic hormones—testosterone, growth hormone (hGH) and IGF-1.[30,31] While a single drink is not going to cause major shifts, the nightly presence of alcohol even at modest doses will activate these systems, leaving the body less anabolic overall, and indeed, a downstream consequence of this state is impaired muscle and bone formation and repair. In a controlled study, consuming binge levels of alcohol after exercise (about 12 standard drinks) reduced muscle protein synthesis by 24%, even when combined with protein intake.[32] Ultimately, these effects mean that alcohol is counterproductive to optimizing growth, recovery, and repair.

As with inflammatory effects, these hormonal impacts can ultimately increase risk of all four horsemen, though heightened stress and poor sleep are particularly strong risk factors for neurodegenerative disease, metabolic disease, and atherosclerosis. (Refer to the series of podcast discussions with Matt Walker covering sleep and chronic disease in episodes #47, #48, and #49 for more detail.)

Appetite and calorie intake effects

Alcohol is a source of calories, with alcohol itself providing seven calories per gram and many alcoholic beverages containing sugars that further increase calorie load (e.g., a 12-oz beer typically provides 150–200 calories). Despite this fact, alcohol consumption does *not* appear to attenuate appetite or result in any reduction in food intake to compensate for the added calories from alcohol. Indeed, randomized trials on alcohol and food intake attest to the *opposite* effect: consumption of alcoholic beverages *potentiates* appetite and leads to *greater* calorie intake in a subsequent meal in a dose-dependent manner.[33,34]

This effect on food intake may be partially explained by the impulsivity and disinhibition that accompany alcohol intake,[35] yet mechanistic investigations have shown that alcohol can also promote appetite more directly by activating pro-feeding neural circuits in the hypothalamus (i.e., AgRP neurons).[36] Thus, while studies have shown this appetitive effect to continue for up to one hour after alcohol intake, it likely extends as long as ethanol remains in the system.[37]

Although no randomized trials have examined these appetitive effects beyond acute timescales, it's certainly conceivable (if not very probable) that frequent episodes of caloric excess stimulated by alcohol consumption would translate to a gradual accrual of fat mass—a phenomenon recognized colloquially as a "beer belly." This increase in fat mass could of course result in obesity and metabolic dysfunction, which secondarily increase risk of the three other horsemen—cardiovascular disease, neurodegenerative disease, and many forms of cancer.

Additional risks

Not all consequences of drinking need a lab test to confirm. Alcohol impairs coordination, judgement, and impulse control, which is why binge episodes carry such disproportionate risk for accidents, falls, violence, and unsafe sex. These (along with a hangover) are the immediate, tangible risks most people associated with a night of heavy drinking.

When you line up all of the consequences of a single night of heavy drinking—stress, sleep, immune function, hormone signalling, appetite, and the overt risks of intoxication—the pattern is clear. Alcohol consistently works against the body's recovery process. Of course, we have used extreme binge-level evidence to illustrate the point, but the mechanisms underlying the damage are the same and may accumulate over time. Occasional light drinking—one or two drinks, once or twice a week—are far less concerning, but as drinking becomes more frequent or concentrated, alcohol becomes a potent anti-recovery agent. This doesn't mean a glass of

wine at dinner will wreck your physiology. But for people truly optimizing performance, longevity, or simply feeling sharp day to day, that's an important cost of alcohol—not just what it does over decades, but how it interferes with recovery tonight and tomorrow morning.

Importantly, for both the day-to-day consequences and the long term risk of alcohol consumption, the impact of alcohol may be greater for some than others.

Risk modifiers

Thus far, we have discussed risks associated with alcohol intake in a blanket sense, but it's critical to understand that these risks are *not uniform* across all individuals and all situations. Certain risk modifiers can make the *same* dose more problematic for some than for others. These modifiers can fall into two broad categories: 1) factors that contribute to *higher relative concentrations* of alcohol in your blood for the same amount ingested (in turn exposing you to more risk), and 2) factors that may make your body less *resilient* to alcohol at *any* given blood concentration. For both of these categories, many factors fall outside of our ability to control. We will start by discussing these non-modifiable risk factors before moving on to guidance on how we can take conscious steps toward reducing risk through factors more within our control.

Non-modifiable factors affecting blood alcohol concentration

For a given amount of alcohol ingested, different individuals can wind up with different blood alcohol concentrations (BAC). While several modifiable factors play a role, the most important *non-modifiable* factors that impact how much alcohol actually ends up in your bloodstream for each drink include body size, body composition, age, and sex. Indeed, these variables share a common thread: body water.

Because alcohol dissolves in water, BAC is heavily dependent upon how much water is present in the body—if alcohol can be diluted in a larger water volume, BAC won't rise as high as it would in a smaller volume. Total body water content is related to body size (larger bodies have more body water), as well as to body composition (lean mass is associated with more water than fat mass). Therefore, individuals who are relatively small and have lower relative lean mass will experience a greater increase in BAC for a given amount of alcohol than someone who is larger and more muscular.

This also accounts in part for the fact that older adults and women tend to be more strongly impacted by alcohol than younger adults and men. Both older individuals and women have less lean mass and body water, so they reach higher BACs at the same dose compared with their young or male counterparts. (Of note, this isn't the only mechanism by which sex impacts BAC changes per drink: women also have reduced stomach alcohol dehydrogenase activity compared to men, which means less "first-pass" breakdown of alcohol before it enters circulation.[38] Refer to our previous newsletter on this topic for more detail.) Indeed, women show faster progression to organ damage with alcohol intake—for example, at about three drinks per day (about 40 g alcohol), women face a nine-fold higher risk (RR=9.35) of cirrhosis compared with abstainers, versus a three-fold risk in men (RR=2.82).[39]

Put simply: two people can drink the same number of glasses, but the smaller, older, or female drinker will have more alcohol in their blood—and therefore greater exposure of organs like the brain and liver to its effects.

Non-modifiable factors affecting alcohol resilience

Even when two people reach the same BAC, their bodies may not respond equally. Data from rodent studies indicate that older adults are more sensitive to alcohol's sedative and motor-impairing effects than younger adults,[40,41] suggesting that the elderly may be at higher risk for falls, car crashes, or other accidents at a given level of intake. On the other end of the age spectrum, children and adolescents are uniquely vulnerable to alcohol due to the continued development of the brain during these phases of life, and alcohol consumption at these ages can disrupt gray- and white-matter growth.[42]

Certain health conditions or medications can also increase the risks associated with any given BAC. For individuals with cardiac arrhythmias such as atrial fibrillation, even low-to-moderate drinking can trigger recurrence of acute arrhythmia episodes,[43] and those on sedative medications for anxiety or pain management (e.g., benzodiazepines, opioids) face additive sedation and respiratory suppression with alcohol—resulting in increased risk of overdose or death even when taking medications as prescribed.[44]

Indeed, mental health conditions deserve special attention here for their impact on risks with alcohol. Beyond potential medication interactions, mood and anxiety disorders can greatly raise the likelihood of alcohol *dependence*. For instance, those with generalized anxiety disorder are 3–5 times more likely to develop alcohol use disorder (AUD), and people with social anxiety often find alcohol's temporary ease particularly reinforcing.[45]

A big driver of these associations is self-medication. Alcohol is often viewed as a source of quick relief, as drinking dampens anxiety by acting on calming neurological pathways and can briefly boost mood, and roughly one in four people with mood or anxiety disorders report using alcohol to manage symptoms.[46] But when the effects wear off, the rebound often involves *worse* anxiety ("hangxiety"), poor sleep, and lower mood. This creates a self-perpetuating cycle, in addition to delaying healthier coping strategies and making psychiatric treatments less effective.[45,47]

A note on alcohol use disorder

We would be remiss not to touch a bit more on alcohol use disorder (AUD)—sometimes referred to as alcohol dependence, abuse, addiction, or alcoholism—which many people might imagine as a life that is unraveling: missed work, broken relationships, daily drinking. But in reality, the threshold is much lower and is defined not only by *how much* you drink, but also *how and why*. Psychiatry's diagnostic manual (DSM-5) defines AUD as meeting two out of the eleven symptomatic criteria within a year—such as craving alcohol, drinking more than intended, needing more to feel the same effect, or continuing despite harm. Nearly 30% of U.S.

adults meet criteria at some point in their lives, though few recognize it or seek help.[48] Paying attention to your urges, patterns, and the role alcohol plays in your life is the first step toward reducing risk and making meaningful change.

Importantly, the advice contained within this article is *not* intended for those with clear, problematic AUD. Such cases typically warrant targeted therapeutic interventions, including talk therapy, support groups like Alcoholics Anonymous, or medications such as naltrexone, which has been shown to reduce both drinking days and heavy-drinking episodes by 1–2 days per month.[49] Ultimately, harm reduction may require a combination of approaches, and as with any form of addiction, willpower alone often isn't enough.

In short, if you are concerned about your relationship with alcohol, reach out to a mental health professional, even if you haven't hit "rock bottom." The sooner you address the problem, the less damage it will do to your health—though it's important to note that it's never "too late." *Anyone* who reduces or completely eliminates alcohol consumption can see health benefits, even after years or decades of alcohol abuse.

Reducing alcohol intake reduces risk

Reducing alcohol intake, even modestly and/or after years of heavy consumption, can still lead to profound reductions in risk for several chronic diseases. For example, within 5–10 years after quitting alcohol completely, the risk of developing cancers of the head and neck falls by around 15%, approaching those of never-drinkers after around two decades of abstinence.[50,51] Likewise, older adults who quit drinking for 5–9 years have been shown to have a 21% lower risk of developing cognitive impairment compared to those who continued drinking (HR=0.79; 95% CI: 0.66–0.96)].[52] These findings reinforce that it's never too late to see benefits, even in later life.

Importantly, significant risk reduction can still be achieved without complete abstinence. For example, a Korean cohort of individuals who chronically drank heavily (defined as >4 drinks per day for males or >3 drinks per day for females) found that those who reduced their intake simply to moderate levels (up to 4 drinks per day for males or up to 3 drinks per day for females) had a 23% lower incidence of major adverse cardiovascular eventscompared to those who remained heavy drinkers (we covered this study in more detail in a previous newsletter).[53]

It is clear that cutting out alcohol reduces risk across multiple domains—cancer, cardiovascular health, and likely cognition. While many will find complete abstinence to be too difficult or impractical, even modest reductions can have substantial benefits. Further, even for a given amount of alcohol intake, we have strategies at our disposal for reducing the long- and short-term *impacts* of alcohol when you do choose to drink.

Modifiable factors for reducing alcohol intake and harm

As we've seen, the risks associated with alcohol are not uniform across the population, and many risk modifiers—such as age, sex, and body size—are out of our control. However, several *modifiable* factors affect the health impacts of alcohol consumption, too. Of course, the

most obvious modifiable variables are the amount of alcohol you choose to consume and the frequency with which you consume it, but again, even keeping total alcohol consumption *constant*, there are ways to mitigate negative consequences. These strategies largely revolve around reducing the rate of alcohol absorption and staying hydrated as much as possible while drinking and in the following hours.

Beyond decreasing the amount of alcohol you drink, the best way to avoid large increases in BAC is to reduce the rate at which alcohol is absorbed into your system. One critical way to accomplish this is to slow the rate at which you're consuming alcohol in the first place, both by controlling your drinking pace and choosing lower alcohol-by-volume (ABV) drink options. Avoid taking shots or participating in drinking games that force rapid ingestion of large quantities of alcohol, and aim for no more than one standard drink per hour (keeping in mind that many mixed drinks will contain more than one standard drink). Further, because drinking pace tends to accelerate as one becomes more intoxicated,[54] limiting your total number of drinks throughout the night also helps with the goal of keeping drinking pace in check.

Meal timing relative to alcohol intake also constitutes a key determinant of alcohol absorption rate. Having alcohol with or shortly after a meal slows absorption while also reportedly increasing rates of alcohol elimination.[55,56] It has also been suggested that taking a dose of psyllium fiber (e.g., Metamucil) can be enough to slow the absorption of alcohol in your GI system and reduce inflammatory markers associated with hangover.[57] In other words, the takeaway is tried-and-true advice: don't drink on an empty stomach.

In addition to food, hydration is also important for mitigating the negative effects of alcohol. As we noted earlier, lower body water corresponds to higher spikes in BAC for a given volume of alcohol consumed. While body water is in part determined by non-modifiable variables like body size and age, it is also subject to our control to some degree based on how we balance how much fluid we're consuming versus how much fluid we're losing (e.g., through urine or sweat). Indeed, alcohol itself increases urine output, which can lead to dehydration and the associated loss of electrolytes and vitamins. We've covered hydration, the many health consequences of dehydration, and factors that affect fluid balance in depth in AMA #33, but for our current purposes, let it suffice to say that consuming plenty of water before, during, and after you drink—especially when combined with electrolytes—can help to limit the rise in BAC for a given amount of alcohol, in addition to mitigating the effects of alcohol-induced dehydration itself.

Cures for hangovers?

While on the subject of hydration and alcohol risk mitigation, it's worth taking a moment to touch on the unpleasant after-effects of a heavy night of drinking—a set of symptoms that includes headaches, nausea, fatigue, dehydration, difficulty concentrating, and irritability, which we collectively know as a hangover. These symptoms arise from a combination of inputs from multiple disrupted physiological symptoms—dehydration, acetaldehyde toxicity, inflammation, GI irritation, and so on—and there is certainly no shortage of old wives' tales about alleged "cures." But are any truly valid?

Unfortunately, the truth is that you must wait for the acetaldehyde to leave your system and for your body to repair the damage done, and there are no short-cuts for these processes. Scientific evidence does not uphold any alleged hangover cures, so you must simply nurse the symptoms. While some of these symptoms relate to dehydration and may thus be partially alleviated with fluid intake, many of the more severe hangover symptoms are believed to simply *co-occur* with dehydration rather than being *caused* by dehydration, so loading up on water offers only modest relief.[58] (Unless you can't keep water down due to vomiting, IV bags are not necessary for rehydration.) Painkillers such as NSAIDs (e.g., ibuprofen) and acetaminophen (Tylenol) can help with headaches, but like alcohol, these medications are also processed by the liver, so frequent use of painkillers to combat hangovers may exacerbate liver damage from the alcohol itself. For nausea, it's mostly a game of waiting for the symptom to go away on its own, though some over-the-counter treatments may help, and for severe symptoms, you could consider the prescription medication ondansetron (Zofran).

The best route to avoid hangovers—and to avoid the long-term health effects of alcohol—is to drink less and drink less frequently. Yet for many, cutting down on alcohol is about more than the alcohol itself. Sitting out on happy hour with friends or giving up on game-day beer can feel like a compromise on quality of life. For such situations, non-alcoholic (NA) options may provide a way to engage in many of the joys associated with alcohol—without the alcohol itself.

An aside on non-alcoholic options

There's no question that the market for non-alcoholic versions of traditionally alcoholic drinks has exploded in recent years, though not all of these products have quite reached a level of sophistication where they can serve as satisfying substitutes for their alcoholic alternatives. Specifically, NA wine and spirits are still lagging behind the impressive strides made with NA beers, which in many cases have developed to a point of tasting just as good as their alcoholic counterparts. While wine and spirits may eventually catch up, it turns out that there are clear reasons why beer has been uniquely suited to the NA revolution, and these reasons come down to chemistry, consumer expectations, production challenges, and historical investment.

Beer's flavor complexity doesn't rely heavily on ethanol. Much of what we enjoy in beer comes from hops, malt, carbonation, and yeast byproducts like esters and phenols. Unlike beer, wine and spirits depend heavily on ethanol for body, mouthfeel, aroma volatility, and balance. Simply removing ethanol disrupts the balance of the drink in a way that is difficult to replace. This is especially true considering the consumer expectations for these products. Beer drinkers are more open to "sessionable" or light products, so NA beer fits well in this science. On the other hand, wine and spirits drinkers expect more elegance, complexity, and balance—and they notice when those elements are missing.

Production methods also favor beer—NA beer uses techniques like arrested fermentation (halting the fermentation process early) or vacuum distillation (removal of alcohol by heating; doing so under vacuum pressure lowers the boiling point of the beer and allows it to be heated at a lower temperature to maintain flavor better). For wine, the dealcoholization process can

strip volatile aromatics that contribute to the taste. For spirits, which are mostly ethanol and flavor compounds, it simply doesn't make sense to remove the ethanol. A completely new alternative must be created from the ground up.

Perfecting these methods takes time, and the NA beer market has had decades of development (since Prohibition really, or in Europe where they used it as an alternative to water) invested heavily in improving flavor and technique. NA wine and spirits have less investments. Even though the tech is improving, they are still 5–10 years behind the NA beer market.

Not all NA beer is truly 100% alcohol free. In the United States, a beverage can be marketed as NA so long as it contains less than 0.5% alcohol by volume. This is very low (approaching the insignificant levels of alcohol naturally present in some fruits and juices), but those completely abstaining from alcohol should read labels carefully.

The bottom line

Although alcohol consumption has long been entrenched in cultures and societies across the globe, it comes at a cost with respect to health. Alcohol taxes our bodies in numerous ways, from DNA damage to poor sleep, and ultimately increases risk of *all* of humanity's deadliest diseases in a dose-dependent manner. Contrary to popular narratives about a "J-shaped curve," there is truly *no safe dose* of alcohol consumption, though for most health outcomes, light-to-moderate drinking does not increase risk with a large effect size, and risks are most pronounced with higher intake levels.

Despite the risks, most of us still choose to imbibe at least occasionally, so understanding the factors that *modify* risk—those both within and outside of our control—can help to inform our decisions regarding our personal relationships with alcohol and the level of risk we can tolerate. Smaller, older, and female drinkers reach higher BACs per drink and can therefore incur more harm for the same level of intake, but hydration, food intake, and slowing alcohol consumption can at least partially reduce the potential damage of any given drinking bout.

But ultimately, the greatest gains will come with cutting down—or cutting out—alcohol entirely. The growing availability of high-quality NA options opens doors for social "drinking," sans alcohol, and indeed, the decision to abstain entirely is becoming far more socially acceptable—and even admirable—than it had been in previous decades. Choose the dose and pattern that align with your goals—and remember that drinking less (or not at all) always buys back healthspan.

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References

1. van de Luitgaarden IAT, van Oort S, Bouman EJ, et al. Alcohol consumption in relation to cardiovascular diseases and mortality: a systematic review of Mendelian randomization studies. Eur J Epidemiol. 2022;37(7):655-669. doi:10.1007/s10654-021-00799-5
2. Kassaw NA, Zhou A, Mulugeta A, Lee SH, Burgess S, Hyppönen E. Alcohol consumption and the risk of all-cause and cause-specific mortality-a linear and nonlinear Mendelian randomization study. Int J Epidemiol. 2024;53(2). doi:10.1093/ije/dyae046
3. Di Federico S, Filippini T, Whelton PK, et al. Alcohol intake and blood pressure levels: A dose-response meta-analysis of nonexperimental cohort studies. Hypertension. 2023;80(10):1961-1969. doi:10.1161/HYPERTENSIONAHA.123.21224
4. Biddinger KJ, Emdin CA, Haas ME, et al. Association of habitual alcohol intake with risk of cardiovascular disease. JAMA Netw Open. 2022;5(3):e223849. doi:10.1001/jamanetworkopen.2022.3849
5. Lu T, Nakanishi T, Yoshiji S, Butler-Laporte G, Greenwood CMT, Richards JB. Dose-dependent association of alcohol consumption with obesity and type 2 diabetes: Mendelian randomization analyses. J Clin Endocrinol Metab. 2023;108(12):3320-3329. doi:10.1210/clinem/dgad324
6. Topiwala A, Levey DF, Zhou H, et al. Alcohol use and risk of dementia in diverse populations: evidence from cohort, case-control and Mendelian randomisation approaches. BMJ Evid Based Med. Published online September 23, 2025. doi:10.1136/bmjebm-2025-113913
7. Zheng L, Liao W, Luo S, et al. Association between alcohol consumption and incidence of dementia in current drinkers: linear and non-linear mendelian randomization analysis. EClinicalMedicine. 2024;76(102810):102810. doi:10.1016/j.eclinm.2024.102810
8. Campbell KA, Fu M, MacDonald E, Zawistowski M, Ware EB, Bakulski KM. Relationship between alcohol consumption and dementia with Mendelian randomization approaches among older adults in the United States. Alzheimers Dement. 2022;18(S11). doi:10.1002/alz.062756
9. Larsson SC, Traylor M, Malik R, et al. Modifiable pathways in Alzheimer's disease: Mendelian randomisation analysis. BMJ. 2017;359:j5375. doi:10.1136/bmj.j5375
10. Daviet R, Aydogan G, Jagannathan K, et al. Associations between alcohol consumption and gray and white matter volumes in the UK Biobank. Nat Commun. 2022;13(1):1175. doi:10.1038/s41467-022-28735-5
11. Angebrandt A, Abulseoud OA, Kisner M, et al. Dose-dependent relationship between social drinking and brain aging. Neurobiol Aging. 2022;111:71-81. doi:10.1016/j.neurobiolaging.2021.11.008
12. Pfefferbaum A, Lim KO, Zipursky RB, et al. Brain gray and white matter volume loss accelerates with aging in chronic alcoholics: a quantitative MRI study. Alcohol Clin Exp Res. 1992;16(6):1078-1089. doi:10.1111/j.1530-0277.1992.tb00702.x
13. Shear PK, Jernigan TL, Butters N. Volumetric magnetic resonance imaging quantification of longitudinal brain changes in abstinent alcoholics. Alcohol Clin Exp Res. 1994;18(1):172-176. doi:10.1111/j.1530-0277.1994.tb00899.x

14. Pfefferbaum A, Sullivan EV, Mathalon DH, Shear PK, Rosenbloom MJ, Lim KO. Longitudinal changes in magnetic resonance imaging brain volumes in abstinent and relapsed alcoholics. Alcohol Clin Exp Res. 1995;19(5):1177-1191. doi:10.1111/j.1530-0277.1995.tb01598.x
15. Garaycoechea JI, Crossan GP, Langevin F, et al. Alcohol and endogenous aldehydes damage chromosomes and mutate stem cells. Nature. 2018;553(7687):171-177. doi:10.1038/nature25154
16. Seitz HK, Stickel F. Acetaldehyde as an underestimated risk factor for cancer development: role of genetics in ethanol metabolism. Genes Nutr. 2010;5(2):121-128. doi:10.1007/s12263-009-0154-1
17. Bouajila N, Domenighetti C, Aubin HJ, Naassila M. Alcohol consumption and its association with cancer, cardiovascular, liver and brain diseases: a systematic review of Mendelian randomization studies. Front Epidemiol. 2024;4:1385064. doi:10.3389/fepid.2024.1385064
18. Im PK, Yang L, Kartsonaki C, et al. Alcohol metabolism genes and risks of site-specific cancers in Chinese adults: An 11-year prospective study. Int J Cancer. 2022;150(10):1627-1639. doi:10.1002/ijc.33917
19. Jee Y, Ryu M, Sull JW. Alcohol consumption and cancer risk: Two sample Mendelian randomization. Epidemiologia (Basel). 2024;5(3):618-626. doi:10.3390/epidemiologia5030043
20. Lewis SJ, Smith GD. Alcohol, ALDH2, and esophageal cancer: a meta-analysis which illustrates the potentials and limitations of a Mendelian randomization approach. Cancer Epidemiol Biomarkers Prev. 2005;14(8):1967-1971. doi:10.1158/1055-9965.EPI-05-0196
21. Gormley M, Dudding T, Sanderson E, et al. A multivariable Mendelian randomization analysis investigating smoking and alcohol consumption in oral and oropharyngeal cancer. Nat Commun. 2020;11(1):6071. doi:10.1038/s41467-020-19822-6
22. Jun S, Park H, Kim UJ, et al. Cancer risk based on alcohol consumption levels: a comprehensive systematic review and meta-analysis. Epidemiol Health. 2023;45:e2023092. doi:10.4178/epih.e2023092
23. Yu X, Chen J, Jiang W, Zhang D. Alcohol, alcoholic beverages and risk of esophageal cancer by histological type: A dose-response meta-analysis of observational studies. Alcohol Alcohol. 2020;55(5):457-467. doi:10.1093/alcalc/agaa047
24. Zhu J, Jiang X, Niu Z. Alcohol consumption and risk of breast and ovarian cancer: A Mendelian randomization study. Cancer Genet. 2020;245:35-41. doi:10.1016/j.cancergen.2020.06.001
25. Zhou X, Yu L, Wang L, et al. Alcohol consumption, blood DNA methylation and breast cancer: a Mendelian randomisation study. Eur J Epidemiol. 2022;37(7):701-712. doi:10.1007/s10654-022-00886-1
26. Tsermpini EE, Plemenitaš Ilješ A, Dolžan V. Alcohol-induced oxidative stress and the role of antioxidants in alcohol use disorder: A systematic review. Antioxidants (Basel). 2022;11(7):1374. doi:10.3390/antiox11071374
27. Crotty K, Anton P, Coleman LG, et al. A critical review of recent knowledge of alcohol's effects on the immunological response in different tissues. Alcohol Clin Exp Res (Hoboken). 2023;47(1):36-44. doi:10.1111/acer.14979

28. Kuo CH, Wu LL, Chen HP, Yu J, Wu CY. Direct effects of alcohol on gut-epithelial barrier: Unraveling the disruption of physical and chemical barrier of the gut-epithelial barrier that compromises the host-microbiota interface upon alcohol exposure. J Gastroenterol Hepatol. 2024;39(7):1247-1255. doi:10.1111/jgh.16539
29. Stadlbauer V, Horvath A, Komarova I, et al. A single alcohol binge impacts on neutrophil function without changes in gut barrier function and gut microbiome composition in healthy volunteers. PLoS One. 2019;14(2):e0211703. doi:10.1371/journal.pone.0211703
30. Smith SJ, Lopresti AL, Fairchild TJ. The effects of alcohol on testosterone synthesis in men: a review. Expert Rev Endocrinol Metab. 2023;18(2):155-166. doi:10.1080/17446651.2023.2184797
31. Bianco A, Thomas E, Pomara F, et al. Alcohol consumption and hormonal alterations related to muscle hypertrophy: a review. Nutr Metab (Lond). 2014;11(1):26. doi:10.1186/1743-7075-11-26
32. Parr EB, Camera DM, Areta JL, et al. Alcohol ingestion impairs maximal post-exercise rates of myofibrillar protein synthesis following a single bout of concurrent training. PLoS One. 2014;9(2):e88384. doi:10.1371/journal.pone.0088384
33. Caton SJ, Ball M, Ahern A, Hetherington MM. Dose-dependent effects of alcohol on appetite and food intake. Physiol Behav. 2004;81(1):51-58. doi:10.1016/j.physbeh.2003.12.017
34. Kwok A, Dordevic AL, Paton G, Page MJ, Truby H. Effect of alcohol consumption on food energy intake: a systematic review and meta-analysis. Br J Nutr. 2019;121(5):481-495. doi:10.1017/S0007114518003677
35. Caton SJ, Nolan LJ, Hetherington MM. Alcohol, appetite and loss of restraint. Curr Obes Rep. 2015;4(1):99-105. doi:10.1007/s13679-014-0130-y
36. Cains S, Blomeley C, Kollo M, Rácz R, Burdakov D. Agrp neuron activity is required for alcohol-induced overeating. Nat Commun. 2017;8(1):14014. doi:10.1038/ncomms14014
37. Yeomans MR. Effects of alcohol on food and energy intake in human subjects: evidence for passive and active over-consumption of energy. Br J Nutr. 2004;92 Suppl 1(S1):S31-S34. doi:10.1079/bjn20041139
38. Frezza M, di Padova C, Pozzato G, Terpin M, Baraona E, Lieber CS. High blood alcohol levels in women. The role of decreased gastric alcohol dehydrogenase activity and first-pass metabolism. N Engl J Med. 1990;322(2):95-99. doi:10.1056/NEJM199001113220205
39. Zhang Z, Xu CM, Chen W, Yao KT, Sun T, Wang JH. Global, regional, and national burdens of alcohol-related cirrhosis among women from 1992 to 2021 and its predictions. Sci Rep. 2025;15(1):10959. doi:10.1038/s41598-025-95563-0
40. Ornelas LC, Novier A, Van Skike CE, Diaz-Granados JL, Matthews DB. The effects of acute alcohol on motor impairments in adolescent, adult, and aged rats. Alcohol. 2015;49(2):121-126. doi:10.1016/j.alcohol.2014.12.002
41. Novier A, Diaz-Granados JL, Matthews DB. Alcohol use across the lifespan: An analysis of adolescent and aged rodents and humans. Pharmacol Biochem Behav. 2015;133:65-82. doi:10.1016/j.pbb.2015.03.015
42. Welch KA, Carson A, Lawrie SM. Brain structure in adolescents and young adults with alcohol problems: systematic review of imaging studies. Alcohol Alcohol. 2013;48(4):433-444. doi:10.1093/alcalc/agt037

43. Wong CX, Tu SJ, Marcus GM. Alcohol and arrhythmias. JACC Clin Electrophysiol. 2023;9(2):266-279. doi:10.1016/j.jacep.2022.10.023
44. Gudin JA, Mogali S, Jones JD, Comer SD. Risks, management, and monitoring of combination opioid, benzodiazepines, and/or alcohol use. Postgrad Med. 2013;125(4):115-130. doi:10.3810/pgm.2013.07.2684
45. Smith JP, Randall CL. Anxiety and alcohol use disorders: comorbidity and treatment considerations. Alcohol Res. 2012;34(4):414-431. doi:10.35946/arcr.v34.4.06
46. Turner S, Mota N, Bolton J, Sareen J. Self-medication with alcohol or drugs for mood and anxiety disorders: A narrative review of the epidemiological literature. Depress Anxiety. 2018;35(9):851-860. doi:10.1002/da.22771
47. Li J, Wang H, Li M, et al. Effect of alcohol use disorders and alcohol intake on the risk of subsequent depressive symptoms: a systematic review and meta-analysis of cohort studies. Addiction. 2020;115(7):1224-1243. doi:10.1111/add.14935
48. Grant BF, Goldstein RB, Saha TD, et al. Epidemiology of DSM-5 alcohol use disorder: Results from the National Epidemiologic Survey on alcohol and Related Conditions III. JAMA Psychiatry. 2015;72(8):757-766. doi:10.1001/jamapsychiatry.2015.0584
49. Murphy CE 4th, Wang RC, Montoy JC, Whittaker E, Raven M. Effect of extended-release naltrexone on alcohol consumption: a systematic review and meta-analysis. Addiction. 2022;117(2):271-281. doi:10.1111/add.15572
50. Rehm J, Patra J, Popova S. Alcohol drinking cessation and its effect on esophageal and head and neck cancers: a pooled analysis. Int J Cancer. 2007;121(5):1132-1137. doi:10.1002/ijc.22798
51. Ahmad Kiadaliri A, Jarl J, Gavriilidis G, Gerdtham UG. Alcohol drinking cessation and the risk of laryngeal and pharyngeal cancers: a systematic review and meta-analysis. PLoS One. 2013;8(3):e58158. doi:10.1371/journal.pone.0058158
52. Zhang XC, Gao X, Lyu YB, et al. Alcohol cessation in late life is associated with lower risk of cognitive impairment among the older adults in China. Biomed Environ Sci. 2021;34(7):509-519. doi:10.3967/bes2021.071
53. Kang DO, Lee DI, Roh SY, et al. Reduced Alcohol Consumption and Major Adverse Cardiovascular Events Among Individuals With Previously High Alcohol Consumption. JAMA Netw Open. 2024;7(3):e244013-e244013. doi:10.1001/jamanetworkopen.2024.4013
54. Groefsema M, Kuntsche E. Acceleration of drinking pace throughout the evening among frequently drinking young adults in the Netherlands. Addiction. 2019;114(7):1295-1302. doi:10.1111/add.14588
55. Ramchandani VA, Kwo PY, Li TK. Effect of food and food composition on alcohol elimination rates in healthy men and women. J Clin Pharmacol. 2001;41(12):1345-1350. doi:10.1177/00912700122012814
56. Jones AW, Jönsson KA. Food-induced lowering of blood-ethanol profiles and increased rate of elimination immediately after a meal. J Forensic Sci. 1994;39(4):1084-1093. doi:10.1520/jfs13687j
57. Yang K, Ryu T, Chung BS. Psyllium fiber improves hangovers and inflammatory liver injury by inhibiting intestinal drinking. Front Pharmacol. 2024;15:1378653. doi:10.3389/fphar.2024.1378653

58. Mackus M, Stock AK, Garssen J, Scholey A, Verster JC. Alcohol hangover versus dehydration revisited: The effect of drinking water to prevent or alleviate the alcohol hangover. Alcohol. 2024;121:9-18. doi:10.1016/j.alcohol.2024.07.006