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Alcohol hangover and cancer risk: The impact of oxidative stress and systemic inflammation

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Abstract

The 2025 US Surgeon General's Advisory on alcohol and cancer risk addresses the important health issue that alcohol consumption increases the risk of developing at least 7 types of cancer. Literature shows that the negative effects of alcohol are not limited to an increased cancer risk but comprise the development of a variety of immune-related chronic diseases. The major proposed mechanisms of action by the Surgeon General's Advisory are increased level of acetaldehyde, oxidative stress, and inflammation. The effects are more pronounced if larger amounts of alcohol are consumed, on more frequent occasions. Most likely these are drinking occasions that may result in the next-day hangovers. Research on the pathology of hangovers shows a clear role of inflammation, and it is hypothesized that experiencing hangovers frequently will result in developing chronic systemic inflammation. Chronic systemic inflammation is at the root of many chronic diseases. It is therefore important to develop interventions and campaigns to reduce alcohol consumption, and to develop effective treatments to prevent hangovers and associated systemic inflammation.

Keywords: alcohol; hangover; cancer; systemic inflammation; acetaldehyde; oxidative stress; chronic disease

Introduction

The 2025 US Surgeon General's Advisory on alcohol and cancer risk addresses the important health issue that alcohol consumption increases the risk of developing at least 7 types of cancer [1]. Alcohol was identified as a leading preventable cause of breast cancer, liver cancer, colon and rectum cancer, esophagus cancer, mouth and throat cancer, and larynx cancer. In 2019, alcohol consumption contributed to nearly 100,000 cancer cases in the United States and about 20,000 cancer deaths each year [2,3]. In 2020, worldwide 741,300 alcohol-related cancer cases were reported [4]. Although increased cancer risk has been associated with any level of alcohol consumption, the risk becomes greater when larger amounts of alcohol are consumed [4]. A total of 83% of cancer deaths was related to alcohol consumption above 2 standardized drinks per day, as recommended upper limit by the 2020-2025 U.S. Dietary Guidelines for Americans [5]. Of concern, research revealed that only 45% of the Americans are aware of the relationship between alcohol consumption and cancer risk [6]. In comparison, a much larger segment of the American population was aware of the cancer risk of asbestos (81%) and smoking (83%).

The US Surgeon General's Advisory mentioned four mechanisms of action that contribute to developing cancer. First, alcohol's breakdown product acetaldehyde may cause cancer by damaging DNA. Second, alcohol produces oxidative stress which increases inflammation and can damage DNA, proteins, and lipids. Currently, these two mechanisms of action are supported by the strongest evidence. In addition to the role of acetaldehyde and inflammation, altered hormone levels, and the co-use of other carcinogens (e.g., smoking tobacco) were mentioned. The Surgeon General's Advisory stressed that current data is related to average daily alcohol consumption. Specific data on occasions with greater alcohol intake, i.e. the frequency of binge drinking occasions and the quantity of alcohol consumed on these occasions, is scarce. Not mentioned in the Surgeon General's Advisory was the possible relationship between hangover frequency and severity, systemic inflammation, and cancer risk.

Alcohol, systemic inflammation, and the development of chronic disease

The negative health effects of alcohol are not limited to increasing cancer risk. In fact, the World Health Organization considers alcohol consumption as one of the major risk factors for developing a variety of chronic diseases [7]. Crucial in health and disease is the concept of immune fitness. Immune fitness is defined as the capacity of the body to respond to health challenges (such as infections) by activating an appropriate immune response, which is essential to maintain health and quality of life and prevent or resolve disease [8]. Reduced immune fitness may be associated with systemic inflammation, i.e., elevated immune-related biomarkers such as cytokines. Chronic systemic inflammation is a major risk factor for developing chronic diseases such as cardiovascular disease, cancer, diabetes, depression, and asthma [9]. Worldwide, these noncommunicable diseases account for 71 % of all deaths [10].

Table 1 summarizes the top 10 causes of death in the US and the top 10 health complaints reported in primary healthcare [11]. Except for accidents (unintentional injuries), the leading causes of death share a common pathology: chronic systemic inflammation [12,13].

Table 1: Top 10 leading causes of death in the USA. Abbreviation: COVID-19 = 2019 corona-virus disease. Data from reference [11].

1	Heart disease
2	Cancer
3	COVID-19
4	Accidents (unintentional injuries)
5	Stroke (cerebrovascular diseases)
6	Chronic lower respiratory diseases
7	Alzheimer's disease
8	Diabetes
9	Chronic liver disease and cirrhosis
10	Kidney disease

It is vital to maintain adequate immune fitness, and point the general public at effective preventive measures they can adopt to prevent or decrease systemic inflammation. The World Health Organization (WHO) lists alcohol consumption as one of the major risk factors for developing non-communicable diseases, next to raised fasting blood glucose, hypertension, raised blood cholesterol, insufficient physical activity, overweight and obesity, and tobacco use [7]. Indeed, systemic inflammation has been associated with having an increased risk of developing a wide range of non-communicable (immune-related) diseases including diabetes, cardiovascular diseases, and pulmonary diseases [9,12,13], and addiction and alcohol use disorder [17]. In addition, poor immune fitness has also been associated with an increased risk of developing communicable diseases such as infections. For example, having a poorer immune fitness showed to be the strongest predictor of the presence and severity of COVID-19 symptoms [18]. Finally, systemic inflammation and reduced immune fitness also increase the prevalence of experiencing relatively minor, immune-related complaints such as fever and the common cold [16,19]. Taken together, preventable risk factors such as alcohol consumption may lead to chronic systemic inflammation and subsequently the development of disease. Reducing alcohol consumption may help maintain adequate immune fitness and prevent disease.

The pathology of the alcohol hangover

The alcohol hangover is defined as the combination of negative mental and physical symptoms which can be experienced after a single episode of alcohol consumption, starting when blood alcohol concentration (BAC) approaches zero [20]. Current research points to the involvement of acetaldehyde, oxidative stress and the immune system in the pathogenesis of the alcohol hangover [21,22]. After consumption of alcoholic beverages, ethanol is converted into acetaldehyde, and subsequently into acetate and water (See Figure 1).

Research has shown that both ethanol and acetaldehyde concentrations in blood and saliva correlate with hangover severity [23,24]. In relation to hangover severity, acetate has not been previously investigated in humans. However, results of an animal study also suggested a role of acetate in the pathogenesis of the alcohol hangover [25]. A controlled experimental study that conducted breathalyzer assessments every 5 minutes after alcohol intake revealed a significant correlation between ethanol breakdown and hangover severity [26]. Thus, a quick breakdown of ethanol and acetaldehyde has been related to experiencing less severe hangovers.

Alcohol consumption, in particular when larger amounts are consumed, can result in oxidative stress. Oxidative stress refers to the imbalance of toxic free radicals and antioxidants. Antioxidants such as glutathione and superoxide dismutase become depleted after consuming larger amounts of alcohol, and in particular during the alcohol hangover state [27]. Reactive oxygen species (e.g., free radicals) are then not removed from the blood and lipid peroxidation can then result in the production of toxic combinations with proteins and molecules (i.e., adducts) such as 8-isoprostane and malondialdehyde. Blood concentrations of biomarkers of oxidative stress such as 8-isoprostane and malondialdehyde have shown to correlate with hangover severity [27]. Malondialdehyde and acetaldehyde combine together with

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proteins to produce toxic protein adducts. These so-called MAA-adducts are capable of producing a strong inflammatory response [28]. Thus, ethanol, acetaldehyde, and oxidative stress together elicit an inflammatory response that is currently regarded as the cause of the alcohol hangover.

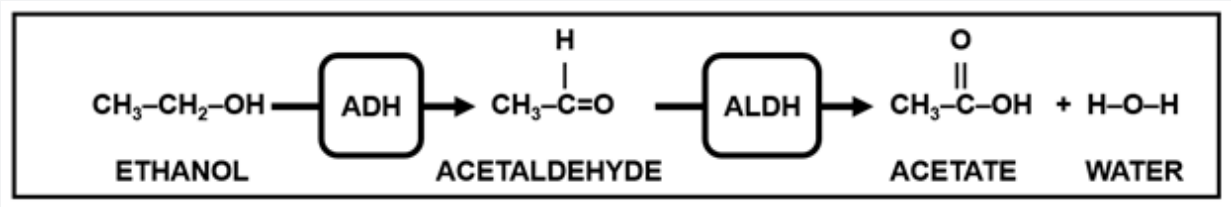

Figure 1: Alcohol metabolism. Ethanol is metabolized to acetaldehyde by alcohol dehydrogenase (ADH). Acetaldehyde is metabolized to acetate and water by aldehyde dehydrogenase (ALDH).

Table 2: Top 10 health complaints in primary care, reported by clinicians and patients. Data from reference [14].

	Clinician reported	Patient reported
1	Upper respiratory tract infection	Cough
2	Hypertension	Backpain
3	Routine health maintenance	Abdominal symptoms
4	Arthritis	Pharyngitis
5	Diabetes	Dermatitis
6	Depression or anxiety	Fever
7	Pneumonia	Headache
8	Acute otitis media	Leg symptoms
9	Backpain	Unspecified respiratory concerns
10	Dermatitis	Fatigue

Several studies investigated inflammatory biomarkers in relation to the alcohol hangover. Kim et al. [29] found the increase in interleukin (IL)-12 and interferon (IFN)-γ in blood correlated significantly with overall hangover severity. Wiese et al. [30] observed significant correlations between blood C-reactive protein (CRP) concentration and hangover severity. In saliva, van de Loo et al. [31] found significant increases in IL-6 and IL-10 concentrations the morning after heavy drinking. Van de Loo et al. [27] further analyzed data from Kim et al. [32] and Mammen et al. [33] and found significant correlations between hangover severity and blood concentrations of IL-6, tumor necrosis factor-alpha (TNF-α) and CRP. Thus, there is accumulative evidence that the inflammatory response after alcohol consumption is at the root of developing the alcohol hangover.

The alcohol hangover and susceptibility to immune-related disease

Recently, Išerić et al. [22] discussed the relationship between experiencing hangovers frequently, and the increased risk of developing immune-related diseases. As discussed in the previous section, alcohol hangovers are associated with an inflammatory response. In case hangovers are experienced occasionally (See Figure 2a) an inflammatory response occurs, but returns to baseline levels of immune activity after the hangover is resolved. However, if hangovers are experienced more frequently, the inflammatory reaction does not return to baseline immune activity. Instead, chronic systemic inflammation may develop (See Figure 2b). In relation to hangovers, it has been shown that when hangovers are experienced more frequently, they become more severe [34]. Systemic inflammation and reduced immune fitness may account for this reverse tolerance.

Taken together, systemic inflammation is an important alcohol-related health risk that increases the chances of developing diseases. Chronic systemic inflammation may in particular develop when hangovers are frequently experienced. It is therefore crucial to prevent or reduce hangover occasions.

Alcohol hangover and cancer risk

The Surgeon General's Advisory on alcohol and cancer proposes four main mechanisms of action that contribute to increased cancer risk after alcohol consumption. Two of these are clearly related to alcohol hangovers and the development of systemic inflammation. The first mechanism of action comprises the conversion of ethanol into acetaldehyde. Acetaldehyde

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concentrations have been associated with hangover severity and correlate with biomarkers of inflammation that are evident during the hangover state [24]. Further, acetaldehyde contributes to the formation of toxic adducts, that result in oxidative stress and inflammation [28]. The second mechanism of action comprises oxidative stress, which also plays a major role in the pathology of the alcohol hangover. Both acetaldehyde and reactive oxygen species may cause DNA damage contributing to the development of cancer [4].

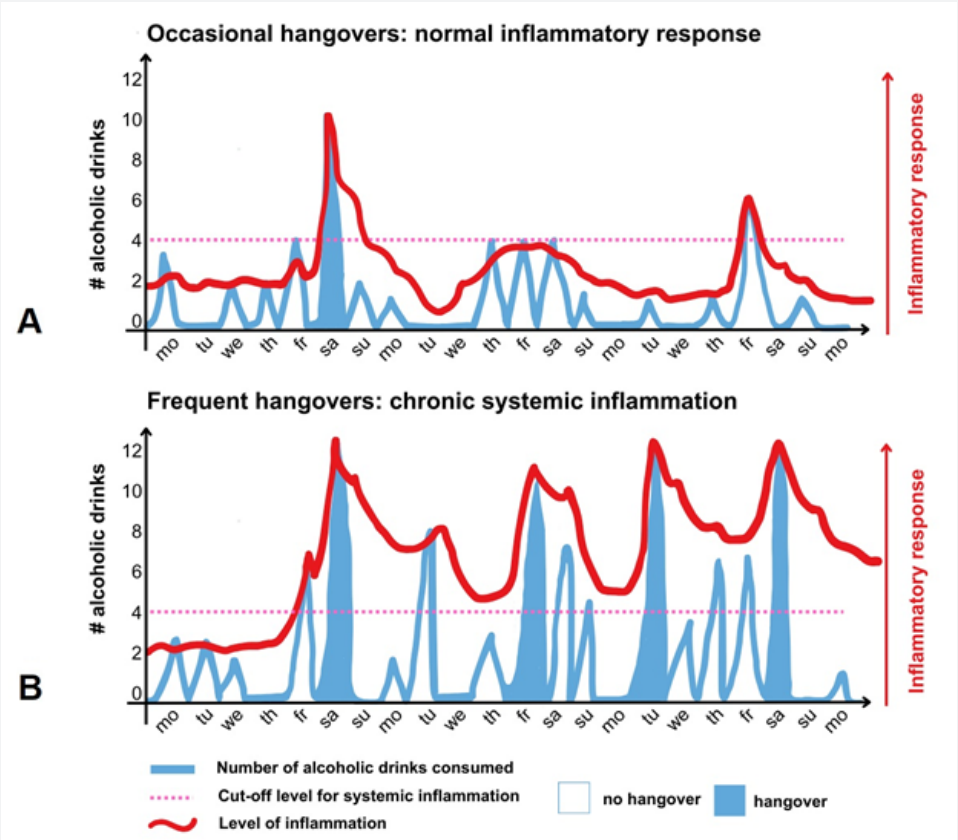

Figure 2: Hangovers and the development of systemic inflammation. Hypothetical data is shown. Figure (A) shows a subject with occasional hangovers, maintaining a normal inflammatory response to discrete events of increased alcohol consumption. Figure (B) shows a subject with more frequent hangovers and greater alcohol intake, developing chronic systemic inflammation. Abbreviations: fr = Friday; mo = Monday; sa = Saturday; su = Sunday; th = Thursday; tu = Tuesday; we = Wednesday. Figure reproduced with permission from reference [22].

The third mechanism of action comprises alterations in hormone levels (e.g., estrogens) due to alcohol consumption. Ylikhari et al. [35-37] investigated the possible relationship between hormonal changes during the hangover state and hangover severity. They found a decreased concentration of growth hormone and inhibited prolactin secretion. No changes were found for thyroid-stimulating hormone, testosterone, follicle-stimulating hormone, and luteinizing hormone. Adrenaline and noradrenalin concentrations were also unaltered during the hangover state [38,39]. It is unlikely that hormonal effects contribute to the hangover state, as none of these studies found any significant correlation between hormonal changes and hangover severity. Increased plasma concentration of vasopressin, aldosterone, cortisol and renin were also found during the hangover state [40-43], but these dehydration-related effects also did not correlate with hangover severity.

The fourth mechanism of action comprises the fact that alcohol consumption makes it easier for other carcinogens to be dissolved in alcohol and absorbed into the body. In particular, tobacco was mentioned. Indeed, drinking and smoking often go hand in hand [44,45], thereby increasing cancer risk. Research has shown that smoking while drinking can aggravate hangover severity [46], likely by increasing oxidative stress and inflammation.

The inflammatory effects are temporary and modest after occasional intake of small amounts of alcohol. However, it can become impactful and chronic after consuming larger amounts of alcohol more frequently, which is the case on drinking occasions that result in a next-day hangover. Taken together, the proposed mechanisms action by the Surgeon General's Advisory on

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alcohol and cancer are in line with the current knowledge on the pathology of the alcohol hangover, and support the hypothesis that frequently experiencing hangovers and more severe hangovers increases cancer risk.

Preventing systemic inflammation: Moderate alcohol consumption by educational efforts

The simplest way to prevent hangovers and systemic inflammation is to moderate alcohol consumption. Educational campaigns should therefore continue to inform the general public about the importance of attaining a healthy lifestyle and maintaining adequate immune fitness by moderating their alcohol intake. In line, the US Surgeon General's Advisory suggests strengthening and expanding education efforts to increase the general awareness that alcohol consumption increases cancer risk [1]. It is further advised to reassess the recommended limits for alcohol consumption, and to incorporate effective alcohol reduction strategies into cancer prevention campaigns. These efforts are important to inform the general population. However, research has shown that mass-media campaigns often have limited impact on alcohol consumption [47].

Finally, the Surgeon General's Advisory advises to update the existing Surgeon General's health warning label on alcohol-containing beverages by including a warning about the risk of cancer associated with alcohol consumption. In order to increase awareness among consumers, the label characteristics should be changed to make the label more visible on beverages. The latter is important as research revealed that health warning labels often go unnoticed or are not understood [48]. In addition, there is little scientific evidence that health warning labels are effective per se [48].

Preventing systemic inflammation: Developing effective hangover treatments

There is ongoing debate regarding the need for hangover treatments [49]. A major concern comprises that the expectation of not having a hangover might lead to more frequent drinking occasions or greater alcohol intake, which could contribute to developing systemic chronic inflammation. There is however little evidence that supports this concern. In fact, research has shown that the great majority of drinkers will not increase their alcohol consumption once an effective hangover treatment becomes available [50]. Consumers are however interested in buying such a product, as they wish to wake up refreshed after an evening of alcohol consumption and participate fully in planned activities (e.g., work, school, social activities), unhindered by hangover symptoms [50]. In this context, a study revealed that the estimated costs of alcohol hangover for the 2019 Dutch economy due to absenteeism (not go to work due to having a hangover) and presenteeism (attend at work with a hangover) equaled €2.7 billion. On presenteeism days, a productivity loss of 24.9% was reported [51].

A 2019 search on the US Amazon website revealed that many products are marketed to prevent or reduce the alcohol hangover [52]. These products were all supplements, with vitamins, minerals, and natural compounds as key ingredients. Of note, no experimental studies in humans have been published that demonstrate the efficacy or safety of these products [52]. The alcohol hangover is recently included as a separate child entity of alcohol intoxication in the 11th International Classification of Diseases (ICD-11) [49]. As such, the US Food and Drug Administration (FDA) treats the alcohol hangover as a disease, implying that it may only be treated by FDA-approved medicines. In the USA, at this moment there are no FDA-approved medicines to treat or prevent alcohol hangovers.

Effective hangover treatments should accelerate ethanol and acetaldehyde metabolism, limit oxidative stress, and prevent or reduce the inflammatory response observed after alcohol consumption. However, research on new or existing hangover products is limited. Preclinical studies in rats and mice have shown that several potential products were capable of accelerating the breakdown of ethanol or acetaldehyde, reducing oxidative stress or inflammatory biomarker concentrations [53-57]. However, these products have never been tested in double-blind, placebo-controlled clinical trials in humans to demonstrate their efficacy in reducing or preventing hangovers. A search on www.clinicaltrials.gov revealed that only one study is currently recruiting participants (NCT05757089). The hangover product under investigation is a dietary supplement comprising a combination of lignin, glycine, dihydromyricetin (DHM), and vitamin B1. Previous research revealed that DHM was not effective in alleviating hangover symptoms [58].

SJP-001 is a promising hangover product that is currently in development. SJP-001 is a combination of the non-steroid anti-inflammatory Drug (NSAID) naproxen and the antihistamine drug fexofenadine. A pilot study in n=5 healthy volunteers revealed that SJP-001 significantly reduced overall hangover severity [59]. The proposed mechanism of action of SJP-001 is to reduce or prevent oxidative stress and the inflammatory response to alcohol. Naproxen and fexofenadine are marketed over-the-counter as individual drugs for many years, and are proven to be effective and safe. Both naproxen and fexofenadine have anti-inflammatory properties [60,61]. In addition, it has been shown that antihistamine drugs can significantly reduce oxidative stress, and for example reduce the risk of developing cancer [62]. To further investigate the efficacy of SJP-001 a large sample double-blind, placebo-controlled trial is planned for 2025, including the assessment of biomarkers to confirm the proposed mechanism of action of SJP-001.

Taken together, research to develop effective hangover treatments is limited. It is important to intensify research on both the pathology and potential treatments of the alcohol hangover. This will increase the understanding on how alcohol consumption can lead to hangovers, the associated systemic inflammation and oxidative stress, and its relation to increased disease risk, including cancer.

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Conclusions

The 2025 US Surgeon General's Advisory on alcohol and cancer risk addresses the important health issue that alcohol consumption increases the risk of developing at least 7 types of cancer. Literature shows that the negative effects of alcohol are not limited to an increased cancer risk but comprise the development of a variety of immune-related chronic diseases. The major proposed mechanisms of action by the Surgeon General's Advisory are increased level of acetaldehyde, oxidative stress, and inflammation. The effects are more pronounced if larger amounts of alcohol are consumed, on more frequent occasions. Most likely these are drinking occasions that may result in the next-day hangovers. Research on the pathology of hangovers shows a clear role of inflammation, and it is hypothesized that experiencing hangovers frequently will result in developing chronic systemic inflammation. Chronic systemic inflammation is at the root of many chronic diseases. It is therefore important to develop interventions and campaigns to reduce alcohol consumption, and to develop effective treatments to prevent hangovers and associated systemic inflammation.

Conflicts of Interest

Over the past 3 years, J.V. has acted as a consultant/advisor for Eisai, KNMP, Med Solutions, Mozand, Red Bull, Sen-Jam Pharmaceutical, and Toast!. J.V. owns stock from Sen-Jam Pharmaceutical. J.V., E.I., E.O., and L.T. received travel support from Sen-Jam Pharmaceutical. J.I. is founder and Head of Clinical Development of Sen-Jam Pharmaceutical. S.R. has nothing to declare.

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