MODERN MEDICINE AND PRACTICE
https://inovatus.es/index.php/ejmmp

EUROPEAN JOURNAL OF MODERN MEDICINE AND PRACTICE
Vol. 4 No. 12 (Dec - 2024) EJMMP ISSN: 2795-921X

ALCOHOL AS A RISK FACTOR FOR A SPECTRUM OF INTERNAL DISEASES: A COMPREHENSIVE REVIEW

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Abstract: Alcohol abuse poses a significant health risk globally. It contributes substantially to the burden of disease, being the primary cause of more than 30 conditions and a contributing factor to 230 others (WHO 2016). These conditions span cancer , neuropsychiatric disorders , cardiovascular diseases, liver and pancreatic diseases, as well as unintentional and intentional injuries. Understanding these risks may promote low-risk drinking practices. Moreover, alcohol's impact extends beyond the individual drinker, causing social harm and adding to overall societal costs. Effective prevention strategies are urgently needed..

Keywords: Alcohol consumption, risk factor, alcohol-related diseases, public health, chronic diseases, alcohol abuse, disease prevention, health impact, epidemiology, alcoholism.

Introduction

Alcohol has been intertwined with human customs and traditions for centuries, playing a central role in social gatherings, celebrations, and everyday life. However, beneath its cultural significance lies a shocking reality: alcohol poses a significant threat to global health. Its widespread consumption is closely linked to a multitude of health problems, ranging from infectious diseases to chronic conditions and disabilities [14,17]. Even it ranks fourth most disabling disease category in low to medium income country and third most disabling in high income country (WHO 2008).

Alcohol is not merely a drink but a catalyst for numerous health issues. It contributes to the development of more than 230 diseases (WHO 2016) , injuries and other health conditions. These health concerns encompass various domains, including cancer, heart diseases, liver ailments, mental health disorders like alcohol use disorders, diabetes, and various injuries, both accidental and intentional.

Given the complexity of alcohol-related health issues, there is a pressing need to delve deeper into the intricate relationship between alcohol consumption and health outcomes. By understanding the underlying mechanisms, we can develop more effective strategies to address this critical public health concern. This review aims to explore the multifaceted nature of alcohol's impact on health.

Alcohol abuse and its impact on worldwide mortality rates

For an average individual, alcohol concentration in blood reaches 80mg/dL after consumption of three standard drinks, about three (12 ounce) bottles of beer, 15 oz of wine, or 4 to 5 oz of 80-proof distilled spirits. Drowsiness occurs at 200 mg/dL, stupor at 300 mg/dL, and coma, with possible respiratory arrest, at higher levels[5].

According to global Status report on alcohol and Health 2018,

In 2016, the harmful use of alcohol resulted in some 3 million deaths (5.3% of all deaths) worldwide and 132.6 million disability-adjusted life years (DALYs) – i.e. 5.1% of all DALYs in that year. Mortality resulting from alcohol consumption is higher than that caused by diseases such as tuberculosis, HIV/AIDS and diabetes. Among men in 2016, an estimated 2.3 million deaths and 106.5 million DALYs were attributable to the consumption of alcohol. Women experienced 0.7 million deaths and 26.1 million DALYs attributable to alcohol consumption[2].

- The age-standardized alcohol-attributable burden of disease and injury was highest in the WHO African Region whereas the proportions of all deaths and DALYs attributable to alcohol consumption were highest in the WHO European Region (10.1% of all deaths and 10.8% of all DALYs) followed by the Region of the Americas (5.5% of deaths and 6.7% of DALYs) [2].
- In 2016, of all deaths attributable to alcohol consumption worldwide, 28.7% were due to injuries, 21.3% due to digestive diseases, 19% due to cardiovascular diseases, 12.9% due to infectious diseases and 12.6% due to cancers. About 49% of alcohol attributable DALYs are due to noncommunicable and mental health conditions, and about 40% are due to injuries [1, 2].

Alcohol abuse claims many more lives than opiates and cocaine abuse. According to a 2017 survey conducted by the National Institute on Alcohol Abuse and Alcoholism, 14 million of adults (over 18 years of age) suffer from alcohol abuse disorder (AUD) in the United States (5.7% of this age group) [9, 18].

Role of Liver in Alcohol metabolism

There are three enzyme systems the liver which metabolize alcohol to acetaldehyde[5]. Those enzymes are : alcohol dehydrogenase, cytochrome P-450 isoenzymes, and catalase.

alcohol dehydrogenase is located in the cytosol of hepatocytes. The microsomal ethanol-oxidizing system also plays an important role at high blood alcohol level. This system involves CYP2E1 isoform of cytochrome P-450 enzymes, located in the smooth endoplasmic reticulum. Induction of P-450 enzymes by alcohol explains the increased susceptibility of alcoholics to other compounds metabolized by the same enzyme system, which include drugs (Metronidazole, sulfonylurea, cefoperazone, acetaminophen, cocaine), anaesthetics, carcinogens, and industrial solvents. Catalase is of minor importance, being responsible for only about 5% of alcohol metabolism. Acetaldehyde produced by these systems is in turn converted by acetaldehyde dehydrogenase to acetate, which is used in the mitochondrial respiratory chain or in lipid synthesis[5].

Several toxic effects result from ethanol metabolism. Few are listed here :

- Acetaldehyde, the direct product of alcohol oxidation is responsible for some of the acute effects of alcohol. The efficiency of alcohol metabolism varies among populations, depending on the expression levels of alcohol dehydrogenase and the presence of genetic variants that alter enzyme activity. About 50% of Asians have very low aldehyde dehydrogenase activity due to the substitution of lysine for glutamine at residue 487 (the normal allele is termed ALDH21, and the inactive variant is designated as ALDH22). The ALDH22 protein has dominant-negative activity, such that even one copy of the ALDH22 allele reduces enzyme activity significantly.

Individuals homozygous for the ALDH22 allele are completely unable to oxidize acetaldehyde and cannot tolerate alcohol, experiencing nausea, flushing, tachycardia, and hyperventilation after its ingestion.

- Alcohol oxidation by alcohol dehydrogenase causes the reduction of nicotinamide adenine dinucleotide (NAD) to NADH, with a consequent decrease in NAD and increase in NADH. NAD is required for fatty acid oxidation in the liver and for the conversion of lactate into pyruvate. Its deficiency is a main cause

of the accumulation of fat in the liver of alcoholics. The increase in the NADH/NAD ratio in alcoholics also causes lactic acidosis.

- ROS generation. Metabolism of ethanol in the liver by CYP2E1 produces ROS, which cause lipid peroxidation of hepatocyte membranes. Alcohol also provokes the release of endotoxin (lipopolysaccharide) from gram negative bacteria in the intestinal flora, which stimulates the production of tumor necrosis factor (TNF) and other cytokines (IL-6 and TGF alpha) from macrophages and Kupffer cells, leading to hepatic injury.
- Impaired proteasome function: Normal function of the ubiquitin-proteasome pathway is to remove irregular and damaged proteins. In alcoholic cirrhosis, the function of proteasome is impaired. inefficient degradation of ubiquitin accumulation of large amounts of ubiquitin in the hepatocytes in the form of Mallory bodies. Impaired proteasome function also causes death of hepatocytes and release cytokines, such as interleukin (IL)-8 and IL-18. IL-8 attracts neutrophils and IL-18 sustains inflammation and causes damage to liver cells.

Alcohol Intoxication

Alcohol intoxication occurs when a person drinks an excess of alcohol at one time. It causes mild to severe physical and behavioural symptoms [25,27].

Severe alcohol intoxication (or alcohol poisoning) — is a very dangerous condition that requires immediate medical attention.

Different symptoms occur in different stages of alcohol consumption.

Table. 1 below shows common symptoms at each level of alcohol intoxication.

This data is obtained from the National Institute on Alcohol Abuse and Alcoholism. It includes information about blood alcohol concentration or content (BAC) — BAC refers to how much alcohol is in the bloodstream

Table.1 [9]

Intoxication Stage	BAC	Symptoms
Mild	0.00% to 0.05%	mild impairments to speech and memory mild impairments to balance and coordination mild impairments to attention initial sleepiness perceived beneficial effects, such as relaxation
Moderate	0.06% to 0.15%	increased impairments to speech and attention increased impairments to balance and coordination moderate memory impairments increased risk of aggression, in some people increased risk of injury to self and others significant impairments to skills necessary for driving increase in perceived beneficial effects of alcohol, such as relaxation
Severe	0.16% to 0.30%	significant impairments to speech and memory significant impairments to coordination and balance significant impairments to judgment and reaction time dangerous impairments to skills necessary for driving

		vomiting blackouts (amnesia) loss of consciousness
Life threatening	0.31% to 0.45%	loss of consciousness danger of a life threatening alcohol overdose suppression of vital functions, leading to a significant risk of death

Alcohol as Primary Cause of disease

WHO's International Classification of Diseases, 10th Edition mentions the term "alcohol" in their name or definition, indicating that alcoholism is a primary cause underlying more than 30 conditions and according to The Global Health Observatory data, alcohol proves to be risk factor in 230 diseases.

The Table. 2 contains list of diseases directly associated with alcohol abuse [4]

Table. 2

- Alcohol-induced pseudo-Cushing's syndrome
- Mental and behavioural disorders attributed to use of alcohol
- Dependence syndrome
- Withdrawal state with delirium
- Psychotic disorder
- Degeneration of nervous system attributed to alcohol
- Alcoholic polyneuropathy
- Alcoholic myopathy
- Alcoholic cardiomyopathy
- Alcoholic gastritis
- Alcoholic liver disease
- Alcoholic fatty liver
- Alcoholic hepatitis
- Alcoholic fibrosis and sclerosis of liver
- Alcoholic cirrhosis of liver
- Alcoholic hepatic failure
- Alcoholic liver disease, unspecified
- Alcohol-induced acute pancreatitis
- Alcohol-induced chronic pancreatitis
- Maternal care for (suspected) damage to fetus from alcohol
- Fetus and newborn affected by maternal use of alcohol
- Fetal alcohol syndrome (dysmorphic)
- Accidental poisoning by and exposure to alcohol

Alcohol as Risk Factor of disease

Alcohol plays a significant role in the development of many diseases (refer to Table. 3), including those that rank among the top 10 leading causes of death worldwide[4]

Table.3

• Liver disease • pancreas disease; • Cardiovascular disease; • Gastrointestinal disease; • Infectious disease; • Cancer; • Diabetes; • Neuropsychiatric disease; • Unintentional and intentional injury

Let's explore the ways in which alcohol contributes to the development and progression of various diseases. Alcohol's involvement in the pathogenesis of these conditions is multifaceted. By understanding the specific mechanisms through which alcohol exerts its harmful effects on the body, we can gain insight into how it contributes to the onset and progression of diseases.

Alcoholic Liver Disease

Alcoholic liver disease Includes:

1. Hepatic steatosis

2. Alcoholic hepatitis

3. Alcoholic cirrhosis.

Chronic and excessive alcohol (ethanol) consumption is one of the major causes of liver disease. Alcoholic liver disease (ALD) constitutes a spectrum of disorders directly related to the excessive alcohol use [5,7].

Table. 4 Signs and symptoms of alcohol-related liver disease (ALD) [22]

Signs and symptoms of ALD	Signs of chronic alcoholism
Nausea/Vomiting	Spider veins
Abdominal pain (right upper quadrant)	Gynaecomastia
Fatigue	Parotid hypertrophy
Weakness	Palmar erythema
Anorexia	Collateral circulation
Jaundice	Dupuytren's disease
Fever	Fetor hepaticus
Abdominal distension/increased abdominal girth with ascites	
Smooth hepatomegaly	
Oedemas in lower extremities	

Consumption of moderate amounts of alcohol is usually not injurious, but excessive amounts causes damage.

- Short-term ingestion of about 80 g of alcohol (six beers or 8 ounces of 80-proof liquor) over one to several days produces mild, reversible hepatic steatosis.
- Daily intake of 80 g or more of ethanol increases the risk for severe hepatic injury.
- Daily consumption of 160 g or more for 10–20 years severe injury.

Alcohol is a direct hepatotoxic and its metabolism in the liver initiates several pathogenic process. About 10–15% of alcoholics develop cirrhosis.

Alcohol increases the catabolism of fat in the peripheral tissues (lipolysis) and increases delivery of free fatty acids to the liver. It increases the synthesis of fatty acid in the liver. It decreases the oxidation of fatty acids by mitochondria. It Increases the production of triglycerides, impairs the assembly and secretion/release of lipoproteins, which contribute to development of hepatic steatosis.

One of the characteristic features of cirrhosis is fibrosis. Alcohol activates hepatic stellate cell transformed into highly fibrogenic cells with myofibroblast-like contractile property which produce collagen (fibrosis) [5,10,11].

Acute alcoholic hepatitis and liver cirrhosis are associated with high mortality (which can reach 50% in acute alcohol hepatitis), and the median survival time of patients with advanced liver cirrhosis can be as low as 1–2 years. Health systems may face a significant and increasing treatment demand for alcohol liver diseases.

Liver cirrhosis, are becoming a more frequent cause of mortality and morbidity globally, especially in developed countries .Alcohol is causally related to an increase in the risk of both liver cirrhosis and pancreatitis , causing an estimated 637 000 digestive disease deaths and 23.3 million digestive disease DALYs in 2016. Within the burden of alcohol-attributable digestive diseases, alcohol-attributable liver cirrhosis caused 607 000 deaths and 22.2 million DALYs, while alcohol-attributable pancreatitis resulted in 30 000 deaths and 1.1 million DALYs [1,2].

Cardiovascular Diseases

The role of alcohol in atherosclerosis, myocardial infarction (MI), and coronary artery disease (CAD) is complex[5, 26].

Alcohol consumption, particularly heavy or chronic drinking, can contribute to the development and progression of atherosclerosis, a condition characterized by the buildup of plaque in the arteries. Alcohol can disrupt lipid metabolism, promote inflammation, impair endothelial function, and increase oxidative stress, all of which contribute to the formation of atherosclerotic plaques.

Coronary artery disease is a common consequence of atherosclerosis, characterized by the narrowing and hardening of the coronary arteries. Chronic alcohol consumption can exacerbate CAD by promoting plaque formation, endothelial dysfunction, and inflammation within the coronary arteries. Additionally, alcohol-related hypertension and dyslipidaemia can further increase the risk of CAD.

While moderate alcohol consumption may have some protective effects against MI and diabetes [14,15,16], by increasing levels of high-density lipoprotein (HDL) cholesterol and improving insulin sensitivity, excessive or heavy drinking can increase the risk of MI. Chronic alcohol consumption is associated with hypertension, cardiac arrhythmias, cardiomyopathy, and thrombosis, all of which can contribute to the development of MI.

CVDs are the leading cause of mortality globally, causing 17.9 million deaths (31.6% of all deaths) and 413.2 million DALYs (15.9% of all DALYs). Globally in 2016, alcohol caused an estimated net CVD burden of 593 000 deaths (3.3% of all CVD deaths) and 13 million CVD DALYs (3.2% of all CVD DALYs). CVDs were responsible for 19.8% and 9.8% of all alcohol-attributable deaths and DALYs lost respectively. Geographically, the age-standardized burden of alcohol-attributable CVD deaths and DALYs was highest in WHO's European Region (22.8 deaths and 541 DALYs per 100 000 people). Similarly, the role of alcohol as a contributory cause of CVDs also varied by region and was highest in the European Region (where alcohol was responsible for 10.5% of all CVD deaths and 11.0% of CVD DALYs) followed

by the ischaemic heart disease, responsible for 47.5% and 42.5% of all alcohol-attributable CVD deaths respectively, and for 56.2% and 33.2% of all alcohol-attributable CVD DALYs [2].

Although not included in this analysis, alcohol increases the risk of atrial fibrillation and flutter .

Malignant neoplasms

Cancer is a leading cause of death in both low-income and high-income countries, and the burden of cancer is expected to increase, especially in developing countries (where the majority of the world's population lives) due to population ageing as well as shifts in lifestyle and environmental risks as countries develop .The International Agency for Research on Cancer (IARC) has determined that alcohol consumption is causally related to oral cavity, oropharyngeal, hypopharyngeal, oesophageal (squamous cell carcinoma), colon, rectal, laryngeal, liver [24].

Colorectal, liver and oesophageal cancers were the largest contributors to the alcohol attributable cancer burden, responsible for 90 000, 84 000 and 73 000 alcohol-attributable cancer deaths respectively. Furthermore, alcohol had the largest impact on cancers of the upper aerodigestive tract, being responsible for 26.4% of all lip and oral cavity cancers, 30.5% of all other pharyngeal cancers (excluding nasopharyngeal cancers), 21.6% of all laryngeal cancers, and 16.9% of all oesophageal cancers[2].

Alcohol consumption has been associated with an increased risk of various types of cancer, with multiple mechanisms underlying its carcinogenic effects.

Alcohol metabolism primarily occurs in the liver, where alcohol is converted into acetaldehyde by the enzyme alcohol dehydrogenase (ADH). Acetaldehyde is a toxic compound that can damage DNA and proteins, leading to cellular mutations and carcinogenesis. The gene responsible for encoding ADH is primarily ADH1B, with variations in this gene affecting an individual's ability to metabolize alcohol and their susceptibility to alcohol-related cancers [5,6].

Chronic alcohol consumption can lead to the production of reactive oxygen species (ROS) and free radicals during alcohol metabolism. These reactive molecules can induce oxidative stress and DNA damage, contributing to the initiation and progression of cancer. Genes involved in DNA repair pathways, such as BRCA1, BRCA2, and TP53, play crucial roles in repairing alcohol-induced DNA damage. Mutations or dysregulation of these genes can increase the risk of cancer development.

Alcohol consumption can disrupt hormone levels in the body, particularly estrogen and testosterone, which are involved in the growth and proliferation of hormone-sensitive cancers such as breast and prostate cancer. Genes involved in hormone metabolism and signalling pathways, such as estrogen receptor genes (ESR1 and ESR2) and androgen receptor gene (AR), may modulate the carcinogenic effects of alcohol by influencing hormone levels and receptor activity.

Excessive alcohol consumption can weaken the immune system, impairing the body's ability to detect and destroy cancerous cells. Genes involved in immune function and inflammation, such as cytokine genes (e.g., TNF-alpha, IL-6) and immune checkpoint genes (e.g., PD-1, CTLA-4), may influence an individual's susceptibility to alcohol-related cancers by regulating immune responses and tumor surveillance.

Alcohol consumption can induce changes in epigenetic modifications, such as DNA methylation and histone acetylation, which can alter gene expression patterns and contribute to cancer development. Genes involved in epigenetic regulation, such as DNA methyltransferases (DNMTs) and histone deacetylases (HDACs), may be dysregulated in response to alcohol exposure, leading to aberrant gene expression and cellular transformation [5, 6].

Neuropsychiatric Disorders

Table. 5 Neuropsychiatric manifestations of alcohol intoxication [8,12,19,30].

Blood alcohol, in mg/dl	Effects
<50	Difficulty in performing tasks that require skill, euphoria, loquacity
>100	Loss of self-control, loss of coordination, mental slowness, mild dysarthria, ataxia, altered perception
>200	Amnesia, confusion, diplopia, dysarthria, hypothermia, nausea, vomiting
>400	Stupor, respiratory depression, coma

Table. 6. Neurological manifestations secondary to excessive alcohol consumption [8, 19,20].

Central nervous system	Peripheral nervous system
Acute	
Acute intoxication Withdrawal syndrome and delirium tremens Wernicke's encephalopathy	Acute alcoholic neuropathy Alcohol-related compressive neuropathy
Chronic	
Korsakoff syndrome Alcohol-related dementia Marchiafava–Bignami disease Cerebellar degeneration	Chronic alcoholic neuropathy Disulfiram neuropathy

Gastrointestinal Disorders

Alcohol, or ethanol, is an aggressive factor for the gastrointestinal tract (GI). Alcohol may regulate the function and structure of gastrointestinal segments. In the stomach, alcohol modulates the gastric acid secretion and the activity of muscles surrounding the stomach. High concentration of alcohol erodes the gastric mucosa, thus excessive alcohol consumption may induce gastrointestinal dysfunction, chronic/atrophic gastritis, and gastric carcinoma in rare cases. induce vascular endothelium injury of the gastric mucosa, which becomes edematous and congestive; present point and scattered bleeding lesions, focal haemorrhage, necrosis, and giant and deep ulcers. Principal and parietal cells become swollen and diminished due to alcohol exposure. Principal and parietal cells are rich in mitochondria, which is easily injured as mtDNA is the major target of ethanol-associated intracellular oxidative stress.This disturbs the morphology (becomes swollen, disaggregated, and cristae are dissolved/disappeared) and function of the gastric mucosa. Mitochondrial dysfunction disturbs ATP synthesis and the lack of ATP may lead to metabolic acidosis, cellular edema, intracellular calcium overload, and further damage to the gastric mucosa cells. Mucosa is rich in protein sulfhydryl groups, which may be the target of ROS. Oxidized protein sulfhydryl groups lead to protein denaturation or enzyme inactivation and receptor damage or modification of the cell membrane, thus contributing to mucosal injury[5].

The metabolic product of alcohol-aldehyde is a well-known carcinogen and plays a major role in alcohol-induced gastritis.

Estimated 637 000 digestive disease deaths and 23.3 million digestive disease DALYs in 2016 [2].

Alcohol in Pregnancy

Alcohol abuse in pregnancy is associated with an increased risk of miscarriage, stillbirth, prematurity, and sudden infant death syndrome (SIDS), as well as fetal alcohol spectrum disorders (FASD)[7].

FASD is a term which refers of lifelong conditions that can occur in individuals who were exposed to alcohol before birth and often lead to disability.

Alcohol and drug interactions

Types of alcohol-medication interactions

There are three main ways alchol can interact with medication[6]:

Alcohol can alter the metabolism of a medication, speeding up or slowing down the clearance of the medication from the body and thus lowering or raising the blood levels of the medication.

A medication can alter the rate and extent of the absorption and metabolism of alcohol, potentially resulting in higher blood alcohol concentrations.

Alcohol can alter the pharmacological effects of the medication, which can increase or decrease the effect of the medication on the body.

There is high risk of toxicity from alcohol combined with opioids or benzodiazepines. Alcohol plays a role in about 1 in 5 overdose deaths related to both prescription opioids (22.1%) and benzodiazepines (21.4%) each year [21].

Alcohol, opioids, and benzodiazepines each suppress activity in respiratory circuits in the brainstem through actions on different receptor systems: opioids via mu-opioid receptors,8,9 benzodiazepines via GABA-A receptors,10–12 and alcohol via GABA-A and NMDA receptor[6].

Antidepressants: In patients being treated for depression, even low levels of drinking may be problematic because alcohol may reduce antidepressant response and decrease patient adherence, while promoting impulsivity, all of which may potentiate suicide risk.

It is not advised to drink alcohol before or while taking “Z-drug” insomnia medications such as eszopiclone, zaleplon, and zolpidem because of the increased risk of side effects. For example, on its own, zolpidem can impair motor coordination and increase fall risk, produce memory impairments, including memory blackouts, and promote behaviors during sleep for which patients have no recall, such as driving. Combining zolpidem with alcohol could increase these risks. Zolpidem overdose is linked with alcohol consumption and often warrants admission to intensive care in the emergency department.

Verapamil is a calcium channel antagonist used to treat arrhythmia, high blood pressure, and angina. Verapamil significantly inhibits alcohol metabolism, which leads to prolonged elevated alcohol level in blood when alcohol and verapamil are consumed together.

Propranolol is a beta blocker used to treat hypertension and other cardiac conditions. Alcohol consumption may increase plasma levels of propranolol and increase the side effects including dizziness, lightheadedness, fainting, and changes in heart rate. In addition, acute alcohol ingestion can cause an initial drop in blood pressure, which could add to the blood-pressure-lowering effects of propranolol.

Antimicrobials with alcohol: Reduced efficacy, liver toxicity, and flushing reactions may occur.

Erythromycin and doxycycline—Reduced efficacy: Erythromycin may be less effective in people who drink alcohol and may increase BACs. Doxycycline may have reduced efficacy in those with long-term, heavier drinking levels.

Ketoconazole, griseofulvin, isoniazid—Liver toxicity: Alcohol should be avoided by patients taking these drugs because of an additive potential for liver toxicity.

Cefotetan, ceftriaxone—Disulfiram-like reactions: Alcohol should be avoided when taking certain cephalosporins (those with a methylthiotetrazole side chain, such as cefotetan, or a methylthiodioxotriazine ring, such as ceftriaxone), and as noted above, when taking ketoconazole and griseofulvin. Disulfiram-like reactions have also been associated with metronidazole and trimethoprim-sulfamethoxazole, but supporting evidence is limited[6].

Preventive Measures

The World Health Organization (WHO), in collaboration with international partners, launched the SAFER initiative in 2018 [3, 28 , 29, 31].

Thus SAFER will be focusing on the most cost-effective priority interventions ("best buys") using a set of WHO tools and resources to prevent and reduce alcohol-related harm:

Strengthen restrictions on alcohol availability

Advance and enforce drink driving countermeasures

Facilitate access to screening, brief interventions, and treatment

Enforce bans or comprehensive restrictions on alcohol advertising, sponsorship, and promotion

Raise prices on alcohol through excise taxes and pricing policies

Conclusion

The review has illuminated the profound role of alcohol as a risk factor to a myriad of internal diseases. Through a thorough examination of statistical data, mechanisms, and clinical observations, it becomes apparent that alcohol intake influences a broad spectrum of health conditions.

The evidences presented underscores the impact of alcohol on internal health and emphasizes the critical need for understanding and addressing this modifiable risk factor. While moderate alcohol consumption may offer some health benefits, the distinction between moderate and excessive drinking remains ambiguous, with the potential detriments outweighing any potential advantages [32].

Strategies focusing on prevention, early intervention, and treatment are essential in alleviating the burden of alcohol-related diseases on both individuals and society. Sustained research endeavours, educational campaigns, and public health initiatives are indispensable in fostering awareness, and promoting healthier lifestyles. By addressing the profound influence of alcohol on internal diseases, we can aspire to a future where individuals are empowered to make informed choices regarding alcohol consumption, leading to improved health outcomes for all.

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