



# Comprehensive Overview of Alcohol Intoxication: Mechanisms, Effects, and Interventions

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## ABSTRACT

Alcohol intoxication, a reversible state of diminished brain responsiveness due to alcohol consumption, poses significant public health challenges worldwide. This review delineates the biological, psychological, and social mechanisms underlying alcohol intoxication, emphasizing blood alcohol concentration (BAC) thresholds for clinical diagnosis. The prevalence and sociocultural influences on alcohol consumption are examined alongside the pharmacokinetics and pharmacodynamics of ethanol. Acute and chronic health consequences, including impaired motor and cognitive functions, liver disease, cardiovascular effects, and mental health issues, are discussed in detail. Finally, this review explores prevention and treatment strategies, highlighting public health interventions and therapeutic approaches aimed at mitigating the adverse effects of alcohol intoxication. This comprehensive analysis underscores the need for multidisciplinary efforts to address the complex interplay of factors contributing to alcohol intoxication and its widespread impact.

Keywords: Alcohol intoxication, Blood alcohol concentration, Psychotropic effects, Acute and chronic health consequences, Public health interventions, Therapeutic approaches

## INTRODUCTION

Prevalence of Alcohol intoxication is a transient condition caused by the ingestion of alcohol in harmful amounts, characterized by diminished responsiveness and inappropriate behavioral changes [1-3]. This review explores the criteria for clinical diagnosis, societal influences on alcohol consumption, and the prevalence of intoxication, emphasizing the impact of psychosocial factors and legislative measures. Understanding the pharmacokinetics and pharmacodynamics of ethanol is crucial for comprehending its effects on the human body [4-5]. Ethanol absorption, distribution, metabolism, and its interaction with neurotransmitter systems are detailed, providing a foundation for discussing its acute and chronic effects. Impaired Coordination and Motor Skills Alcohol impairs motor skills and coordination, affecting performance in tasks requiring precision and adaptation [6-9]. The review highlights the controversy surrounding alcohol's impact on simple versus novel tasks and its implications for activities such as driving. Cognitive Impairment Alcohol significantly impairs cognitive functions, affecting learning processes, decision-making, and memory. Chronic alcohol consumption leads to liver damage, including fatty liver, hepatitis, and cirrhosis [10-13]. The review discusses the pathophysiological mechanisms and the potential for disease progression, emphasizing the importance of early intervention. Alcohol has multifaceted effects on the cardiovascular system, contributing to hypertension, heart disease, and cerebrovascular events. Alcohol is commonly used as a self-medication for anxiety and depression [14-

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16]. The review examines the bidirectional relationship between alcohol consumption and mental health disorders, highlighting the need for integrated treatment approaches. Alcohol intoxication increases impulsivity and the risk of suicide [17-19]. Effective public health strategies are essential for reducing alcohol-related harm. Managing alcohol intoxication requires a combination of prevention, early intervention, and acute care [20-21]. Alcohol intoxication is a complex, multifaceted issue with significant implications for public health [22-23]. Comprehensive strategies integrating public health policies, clinical interventions, and community efforts are essential for reducing alcohol-related harm. Future research should continue to explore innovative approaches and refine existing strategies to address the global burden of alcohol intoxication effectively [24-26].

### **Alcohol Intoxication**

Alcohol intoxication is defined as a reversible and spontaneous state of diminishing responsiveness to stimulation of the brain due to consumption of alcohol or alcoholic beverages in amounts that recently have been considered as quite harmful [27-28]. It is a result of the toxic effect of alcohol and can be measured. This raises the question of the exact point or value where a medical diagnosis of alcohol intoxication can be made. Biological reactions in healthy human organisms to blood alcohol concentration (BAC) greater than 0.50 g/l are described [29-30]. Legal BAC-zero tolerance level when driving is observed in many countries worldwide. Psychotropic effects of alcoholic beverages, such as relaxation, relief of symptoms, decrease in anxiety and panic reactions, loosening one's inhibitions, and the feeling of well-being, are reflected in a high consumption of ethanol and a popular BAC greater than 8.0‰ in party-like situations [31-33]. However, the lower reliability on brainstem functions seen at increasing BAC values is significant because BAC defense mechanisms of the organism under an anesthesia intoxication double-state are contradictory. Alcohol intoxication, as a high BAC, is associated with health impairments, including temporary or long-term brain damage or damage of the entire organism. The special treatment indication is especially severe in case of the sudden failure of the body temperature regulation. Abstinence syndrome can be triggered by the rapid abolition of alcohol [34-35].

### **Prevalence**

The criteria for alcohol intoxication are rather evidence-based in contrast, encompassing a transient condition brought on by excessive ingestion of alcohol, characterized by inappropriate behavioral changes, such as impaired social judgment, decreased motor skills, and slurred speech; increased sociability, and lack of coordination [36-37]. The latter term of alcoholic poisoning, encompassing the state of physical silence that occurs following extreme consumption of either alcohol or alcohol-containing beverages, covers the more severe level of intoxication that can occur [38]. Based on these strict criteria, prevalence varies and is influenced by societal views, religious convictions, and cultural practices. Family, community, and peer influence, as well as legislation, has been seen as having an appreciable impact on the rate and the amount of alcohol consumed. The intoxication process is facilitated by any number of psychosocial factors and the situation has been seen as reinforcing the pharmacological effect of alcohol. Accidents are the most notable public health hazards associated with alcohol intoxication. Their frequency reflects their conditions which are often used as a measure of the problem. Various individuals react in various ways when under the influence of alcohol. The alcohol ingested may leave the individual quiet, unobtrusive, and inattentive or, indeed, it may transform an otherwise upstanding citizen into a belligerent, non-reflective vandal. Other intoxicated individuals may resort to more flamboyant, attention-seeking behavior as they slur their words and stumble around. The last type constitutes what may be referred to as the typically visible, typically moderate, single-episode intoxicated individual [30-35].

The term "alcohol intoxication" is often used interchangeably with "alcohol abuse" and "alcoholic." However, pathophysiologically, the three vary greatly. Alcohol abuse is a recurrent pattern of alcohol consumption that results in one or more of the following within a 12-month period: failure to fulfill major work, school, or home responsibilities, use of alcohol in situations that are physically hazardous, recurrent alcohol-related legal problems, and having frequent alcohol-related social or relationship problems. Alcoholism, on the other hand, or alcohol dependence as it is otherwise referred to, is characterized by the presence of a number of distinct signs and symptoms including having withdrawal symptoms when not further under the influence (shakiness, nausea, the sensation of feeling sick) and taking alcohol in larger amounts and over a longer period than was intended. These individuals may also continuously try to cut down and not succeed, use a lot of time to obtain alcohol, give up important social activities because of alcohol use, and continue to use the substance despite its knowledge of some problems that are caused or are exacerbated by it [20-24].

### **Pharmacology of Alcohol**

The rate of metabolism for a given quantity of ethanol is remarkably constant, averaging about 0.015 blood alcohol concentration (BAC) per hour at all BAC levels. This means that the more alcohol the individual drinks, the greater the quantity of unabsorbed alcohol circulating. Although ethanol behaves like a depressant at the central level, the effect of alcohol on brain function is not limited to the cell membrane and action potential channels. Alcohol can depress more rapidly some areas of neurotransmission, including the spike of the hippocampal pyramidal neurons in granular layers and the septohippocampal cholinergic pathway assessed by means of in vivo microdialysis [12-16]. Alcohol affects many different neurotransmitter systems. Concerning its effect on gene expression, it has been shown, using RT-PCR, that subchronic exposure to alcohol can significantly increase the expression of NMDA receptor NR2B subunit. To discuss alcohol intoxication, it is important to appreciate the pharmacology of ethanol and its acute and chronic metabolic effects. Ethanol is rapidly absorbed throughout the gastrointestinal tract, reaching maximal concentration in the blood within 30 to 90 minutes after ingestion. The rate of absorption is influenced by many factors including the volume consumed, the type of beverage, and the concurrent consumption of food. After absorption, ethanol quickly distributes throughout the body, as a result of its high solubility at the cellular level [23-25]. The volume of distribution is approximately 0.6 kg/l (range 0.5-0.8), approximating total body water. Alcohol is metabolized in the liver, primarily by the enzymes alcohol dehydrogenase and aldehyde dehydrogenase to acetaldehyde and acetic acid, respectively.

### **Short-Term Effects of Alcohol Intoxication**

The short-term effects of alcohol intoxication depend on the dose, mode of administration, overall state of physical health, age of the drinker, gender, and prior pharmacological history of alcohol exposure as well as individual expectations and social interactions. Personality, mood, and situation can influence these expectations. Manic or impulsive reactions can result from the disinhibitory effects of alcohol [6-9]. Mentally alert, intelligent persons may stagger and slur their words due to ethanol's effects on the cerebellum and cerebrum, while displaying judgment and decision-making defects due to its effects on the cortex. Ethanol's ability to help sufferers forget past pain can then lead these persons to return to alcohol again as a method of forgetting present woes. The health implications of alcohol intoxication are vast, warranting the attention and intervention of healthcare professionals. Acute intoxication, caused by ingesting large quantities of alcohol in one drinking session, can result in symptoms of lowered inhibition, slurred speech, unsteady gait, loss of judgment and memory, even loss of consciousness, coma, or death. These symptoms result from the sedative effect of ethanol, involving potentiation of the inhibitory neurotransmitter  $\gamma$ -aminobutyric acid (GABA), activation of the inhibitory ion channel glycine, blockade of excitatory glutamate NMDA receptors, and functionally antagonistic, euphorogenic stimulation of many additional neurotransmitter systems. In addition, the direct and indirect metabolic effects of ethanol contribute to intoxication-related cellular damage, notably in the liver, immune system, brain, and gastrointestinal tract, with resultant dysfunction in each of these organs [10-12].

### **Impaired Coordination and Motor Skills**

The effect of alcohol intoxication on skillful performance has also been explored using models for observing adaptation. There is controversy as to whether a small or moderate quantity of alcohol improves performance in simple, familiar tasks in which adaptation is not essential, such as driving a motor vehicle [13]. It is generally accepted, however, that alcohol impairs the capacity to perform novel or unpracticed tasks. The difficulty in recognizing heel prints further suggests that a drunk individual may sometimes be more perplexed than one suspects by his inability to perform adequately. The most conspicuous behavioral impairments caused by acute alcohol intake are the disturbances in coordinative adjustments and apparent decrements in response speed. These changes exhibit the typical characteristics of a state of psychic and motor retardation. The influence of alcohol on attentiveness is marked also by slowing of various reaction times. The upper extremities react in less complex modes than under normal conditions [15].

### **Cognitive Impairment**

In the next place - indeed, in any other - there won't be a need to try to tailor the testing to the "partly irregularly" impaired process. Although many drugs impair non-conscious and conscious learning processes to the same degree that they impair conscious cognitive processes, there are predictions that drugs such as alcohol and general anesthesia will act twice: progressively impairing conscious abilities remaining when the subject takes in information unconsciously, in the middle, and reverting to a simple concentration-response effect later, when the subject has had time to learn how quickly to do such things as run a maze or react by unconscious training [8]. This is because alcohol may be partially

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concentrating, as well as reducing the rate of change of the flow of information reaching consciousness. The idea that alcohol impairs cognitive functioning is probably the most widespread of folk myths, and certainly one of the most firmly established facts of general pharmacology. Since we are ultimately a civilization built on our brains rather than our brawn, a drug that impairs cognitive functioning should always be approached with the reverence appropriate to a dangerous cutting tool. The concept of cognitive impairment is so broad that we would probably have to measure almost every individual aspect of the mind and its operation in order to have a clear picture of exactly how alcohol acts to impair cognition. We certainly are aware that the traditional mental game of "let's look at your favorite brain operation" shows that a few bottles of alcohol "shrink down" the observed process. Skill at any informational task that we are not aware of or do not claim to understand will obviously not be impaired [9].

### **Long-Term Health Consequences**

The effect of alcohol on the developing fetus is also an important concern. Alcohol freely crosses the placenta, and at high enough blood alcohol concentrations may cause malformations. But at lower levels, the baby can have long-term structural and functional deficits. Postnatally, the fetus can be affected should breast milk contain alcohol, which at low levels can interfere with the milk ejection reflex. In conclusion, alcohol, like many chronic health conditions, has a multitude of pathways in which it can wreck the body. It is not called the chronic toxin for nothing, and early abstinence from chronic high levels is required to prevent or reduce the damage [11]. To say that heavy drinking is bad for your health is stating the obvious. Alcoholic liver disease occurs in up to 25% of heavy drinkers. This ranges from simple fatty liver, through alcoholic hepatitis, to liver cirrhosis. Stopping drinking can prevent its progress to a certain extent, particularly if caught early. Pancreatitis results from heavy alcohol intake, hence the term 'barman's pancreatitis.' It is generally minimal with lower levels of alcohol intake. Reduced bone mineral density and osteoporosis can be seen in heavy drinkers, particularly women. Alcoholic myopathy can be seen in chronic drinkers and can present with changes characteristic of peripheral neuropathy. Even more concerning is alcohol's effect on the heart, mainly when consistently drunk at high levels. Alcoholic cardiomyopathy can lead to heart failure [12].

### **Liver Disease**

Aside from inducing specific hepatic malfunctions, chronic alcohol consumption inhibits hepatic protein synthesis and reduces the glucose content of the blood by promoting glycogenolysis and gluconeogenesis. The activity of alcohol dehydrogenase, together with hepatic concentration and rate of breakdown of NAD, are main factors in the pathophysiology of liver congestion [8]. The deteriorative and nutritional side effects of hepatic disease may be aggravated by the malnutrition accompanying chronic alcoholism. Despite evidence so far attesting to dolichol-linked glycosylation of the liver in ACKR1+ individuals with hepatic atrophy, impaired retinol dressing, and carbohydrate-deficient glycoproteins, the existing data do not permit definitive conclusions about the structural basis of this abnormal glycosylation. It also is not yet clear whether or not hepatic glycosylation in ACKR1+ persons with hepatic atrophy depends on severity and duration of the disease or on a profound disturbance of hepatic function [12]. The liver plays a principal role in alcohol metabolism. For this reason, it is likely to be the liver, the organ most affected when high doses of alcohol are ingested. Fatty liver is the most frequent damage to the liver observed in alcoholics. It is thought to result from interference of alcohol metabolism with the oxidation of fatty acids. Hepatitis is another liver disease frequently diagnosed in alcoholic individuals. It may be acute or chronic, and its symptoms are similar to those found in hepatitis caused by other diseases. A subgroup of chronic hepatitis of unknown etiology has been recognized in patients who abuse alcohol. This hepatic condition has been named alcohol steatohepatitis. It is characterized by fatty change, ballooning degeneration, and lymphocyte infiltration around Mallory bodies. In addition to fatty liver and the different forms of alcoholic hepatitis, chronic alcohol intake is associated with chronic hepatic congestion, usually mixed with cirrhosis [12-17].

### **Cardiovascular Effects**

Furthermore, evidence has been provided for an acute and a carryover effect of alcohol-induced hypertension irrespective of an above-risk consumption of alcohol. According to research, the high intoxication levels often reached during celebrations may play a part in the increased incidence of coronary artery disease observed at these times. Indeed, studies reported pronounced increases in systolic and diastolic blood pressure during memory-based tests, partly within the interval necessary for the performance of maximal loading operations. In a controlled office environment, comparable exercise performance led to a small increase in systolic pressure and a decrease in diastolic pressure. However, diastolic pressure in a simulated vehicle-driving supervision increased solely after alcohol consumption.

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Alcohol consumption has multimodal effects on the cardiovascular system [8-9]. There are acute hemodynamic and neurohumoral effects and long-term effects such as induction of liver diseases and hypertension, both playing prominent parts in the clinical spectrum of alcohol-related cerebrovascular events. Acute effects of alcohol are mediated by its anesthetic action, by fluid shifts due to its vasodilatory properties, and by an increase in left-ventricular stroke volume. High concentrations may cause an acute increase in mean arterial blood pressure. Not only phenomena of chronic alcohol consumption, such as peripheral resistance and volume regulation, have contributed to the hypothesis that alcohol may preferably induce its hypertensive risk in individuals with an altered autonomic function [20-26].

### **Mental Health and Alcohol Intoxication**

Alcohol consumption and intoxication are major cultural practices in most societies and divorced individuals from any age and of any country. The appeal to alcohol is economically on a global scale. The alcohol-enticing effects of the various forms of media are only a shadow of environmental forces that have acted upon and within human cultures for centuries if not millennia [27-30]. This scenario might partly mirror the fact that alcoholic exposure has been part of the human diet since prehistoric times. Other theories speculated on the function of ethyl alcohol in relation to its sweetness. In addition to ethnobotanical approaches, there are two evolutionary theories for alcohol consumption. The first emphasizes the production of ethanol in ripe fruits, as a consequence of the yeast phenotypic plasticity. The second theory paradigmatically states that ethanol has a moderating social effect among sociable animals, by removing their more aggressive dispositions in favour of co-operation within the specific animal groups [31-35].

### **Depression and Anxiety**

However, these negative emotions can be activated not only by the external negative stressor but also by internal or personal factors. Alcohol is one of the most common self-prescribed anxiolytics, and alcohol acts quickly, and some of its reinforcing effects are quite effective. Drinking helps to deal with the world of social relationships, economic and personal fears, and oppression. Drinkers who are alcohol dependent manifest symptoms of anxiety and often of the depression that would be relieved by the use of anxiolytic or antidepressant drugs. Although self-medication may be realistic elements, it does at least allow an escape from other elements to provide effective short-term relief. When intoxicating doses of alcohol are taken, some of this relief is immediate, and the drinker may truly feel "no pain". Alcohol intoxication usually results in a release of "tensions", anxiety, and various forms of depression. Especially anxiety and depression are common stress-induced states. In the mood alteration which they produce, stress, depression, and anxiety are typical negative appraisals of the situation and may be regarded as emotional elements in the coping process [8]. Depression can, therefore, result from a "helpless" situation or a perceived "hopeless" inability to escape from an adverse event. State anxiety is a negative emotion that initiates the action system and prepares for action. When the stressor conditions involve punishment and uncertainty, activation of the inhibitory system results in a "freezing" behavior or other anxiety disorders [11].

### **Risk of Suicide**

The large number of closely spaced intoxication suicide deaths which occur in Europe and the fact that the BAC level associated with suicide completions is also associated with fatal intoxication accidents is evidence that the lethal risk does not require the addition of any additional motive; the lethal risk of those with a large BAC is directly increased by the pharmacological effect of alcohol and most of the psychology of the situation is believed to be related to how these intoxicated conduct themselves [9]. First, the ethanol increases impulsivity so that intoxicants are particularly impatient for smooth, reliable action in uncorking front doors or windows to provide the more immediately available exit and also selects a corner site within a distance they may travel and delivers the downing inclination. Second, those with a large BAC begin to drop when the BAC level moves down from its highest level. A prospect of an escape route is solely associated with sitting in this erfame position and on the other side, they spend easy rise thinking through the inevitable long-drawn-out awkwardness of any conceivable escape stratagem. A recent study has shown that the lobby associated with risk from all sample intoxication deaths is seventeen times more substance that they contain beyond the individual's own constructs from Maria through jeans. In regard to suicide, the authors failed to demonstrate that people take their lives more impromptu when under the influence of alcohol [11]. The body of suicidology literature which has appeared over the past 3 to 4 decades has consistently demonstrated that the likelihood of suicidal behavior increasing with the level of the BAC till driving while drunk (which is around 160 mg or percent BAC) and then failing to increase any further. This is illustrated by a Norwegian study which showed a (0.87) female and 1.08 (male) times higher the risk of suicide for every 10 mg increase in BAC. It is



important to recognize that this conclusion about level emerges not from a single line of evidence but from a variety of independent sources: a) the consideration of the BAC levels present in blood samples of both accident-prone and non-accident-prone groups, b) the demonstration that a majority of completed suicides are associated with a BAC of greater than zero, c) the evidence of even stronger association of BAC amongst autopsied suicide derides and d) strong elevation in risk of accidental causation at levels substantially lower than toxic than the homicides tics effective level [13].

### **Prevention and Treatment Strategies**

The World Health Organization (WHO) has suggested possible strategies to reduce alcohol-related injuries. These strategies include the obvious restriction of alcohol sales at places where there is a high rate of alcohol abuse. This has been implemented at gasoline outlets, for example, in many locales in the United States [11-16]. It has been demonstrated that reducing alcohol price may be effective in reducing violence and injuries. With a high price for alcohol products, poor and young males may decide to drink less. Screening and brief intervention for alcohol and other drug use have been shown to be very effective, inexpensive, and do not take much time. Therefore, there can be no reason why all physicians and healthcare workers would not screen and intervene when face-to-face with any individual or in the emergency room, urgent care center, and clinics for any drug or alcohol use. There are different ways to enact stricter enforcement of the laws associated with alcohol offenses, such as zero tolerance for drunk drivers [12]. High visibility police patrols, mobile breath testing, or sobriety checkpoints may have a short-term effect by creating public awareness of the law. Additionally, regulations for the density of alcohol outlets might also affect violence and related injuries. Prevention and treatment strategies for heavy alcohol intoxication are most practically addressed immediately prior to or at an event where heavy alcohol consumption is anticipated. It would not be practical to screen every patient in a trauma room or in a clinic office for recent drug and alcohol consumption. Also, it would be unlikely that individuals would seek treatment for potential intoxication. However, fear of toxic side effects, treatment of hangover symptoms, or for the treatment of negative blood alcohol levels with trauma from falls or fights may prompt such visits or calls. Finding a person asleep who has consumed a large amount of alcohol is a common clinical problem. The goal of any upcoming treatment or prevention strategy for heavy alcohol consumption and intoxication is to promote and enhance lower BAC levels [8-9].

### **Public Health Interventions**

All of these quite different, but potentially effective, types of public health countermeasures are grounded in evidence of adverse effects of risky drinking and the benefits of reducing, eliminating, and substituting excessive drinking. As there is no single root cause of risky alcohol consumption and the problems it generates, it is unlikely that there is only a single, simple measure to effectively diminish high alcohol intoxication and its related adverse events [12-18]. Therefore, about half of the potential environmental policies and only vaguely defined pharmacological preventions are under-investigated and under-used. This important problem demands more research and experiments because a multiplicity of possible solutions gives hope of more effective responses than relying on a few impractical and incomplete strategies. The above reported public health problems related to excessive levels of alcohol intoxication have several different causes and reasons, including society and individuals. Hence, they need a wide range of possible interventions and probably a combined effort to be successfully and substantially addressed. Furthermore, it is important to start primary prevention interventions because many of the most compelling and effective interventions are those that address proven risks before problems are encountered [19-20]. Therefore, useful interventions range from mass media campaigns to alcohol beverage pricing and food policies, to broad-based responsible-service policies and training, to physician advice and brief intervention programs. Additionally, the possibility of using modern technology to help detect and avoid risky occasions of heavy drinking and its negative effects is worth considering [21-25].

### **Therapeutic Approaches**

The management of the intoxicated patient aims at preventing secondary damage and the return to normal daily activities. The adoption of an approach that meets patients' needs and the use of the correct healthcare interventions can reduce waiting time at first aid posts, shorten hospital stays, and increase customer satisfaction. There should be easy access to psychological triage, medical, mental health, and social workers [21-35]. The acute phase of methanol, ethylene glycol, or isopropanol poisoning can be managed correctly and efficiently, so as to quickly reverse the symptoms and signs of intoxication and prevent death or severe long-term consequences. Alcohol has a complex interaction with therapeutic drugs and can trigger other diseases or blunt their signs. Proper management depends on the exact history and reliable diagnostic tools (such as specific blood tests that can confirm the diagnosis of alcohol abuse). In managing alcohol intoxication, do not forget basic principles like resuscitation, elimination, and

detoxification [37-38]. Medical personnel should be able to manage patients effectively at a general level and be alert to the circumstances in which patients should be referred to higher-level care. Public health measures are aimed at reducing alcohol consumption in general and involve the epidemic triad of host, agent, and environment. For instance, emotional or professional support for the family helps to prevent overconsumption of alcohol, and holistic therapeutic procedures should always be considered for the appropriate management of addiction. Therapeutic approaches include 3 main areas - prevention, early intervention, and acute intoxication management [12-15].

### CONCLUSION

Alcohol intoxication remains a pervasive issue with far-reaching implications for individual and public health. The multifaceted nature of alcohol intoxication, encompassing biological, psychological, and social dimensions, necessitates a comprehensive understanding of its mechanisms and effects. Acute intoxication impairs motor skills and cognitive function, while chronic consumption leads to severe health conditions such as liver disease, cardiovascular problems, and mental health disorders. Effective prevention and treatment strategies must integrate public health policies, educational initiatives, and clinical interventions. Future research should focus on refining these strategies and exploring innovative approaches to reduce alcohol-related harm. Collaborative efforts across healthcare providers, policymakers, and communities are essential to address the global burden of alcohol intoxication.

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