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Alcohol Metabolism and Natural Nutritional Interventions: A Comprehensive Review

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Introduction

Alcohol use disorder (AUD) represents a major global public health challenge, contributing to the loss of millions of lives worldwide. Despite its widespread prevalence, a substantial proportion of individuals affected by AUD do not receive appropriate or timely intervention. This significant treatment gap highlights the urgent need for expanded awareness, improved understanding, and the development of effective intervention strategies to address this critical health issue. Chronic alcohol consumption disrupts metabolic processes and adversely affects multiple organ systems, including the liver, brain, cardiovascular system, and gastrointestinal tract. Because alcohol is rapidly metabolized and widely distributed throughout the body, a comprehensive understanding of its metabolic pathways and of supportive interventions that may facilitate physiological recovery is essential. Furthermore, the complex interplay between alcohol metabolism, nutritional deficiencies, and natural or adjunctive therapeutic approaches presents valuable opportunities to address the multifactorial physiological challenges experienced by individuals with alcohol use disorder.

Alcohol Metabolism: Pathways of Enzymatic Processes and Physiological Effects

Alcohol metabolism occurs primarily in the liver through a series of enzymatic reactions. Ethanol is first oxidized to acetaldehyde by alcohol dehydrogenase (ADH), and acetaldehyde is subsequently converted to acetic acid by acetaldehyde dehydrogenase (ALDH), facilitating its further metabolism and elimination (Hyun et al., 2021). The efficiency of these metabolic pathways varies considerably among individuals and is influenced by genetic polymorphisms, biological factors, sex, nutritional status, and dietary patterns. Emerging evidence indicates that alcohol metabolism extends beyond direct hepatic enzymatic conversion and involves complex gut–brain interactions that significantly influence systemic physiology, immune function, and metabolic regulation (Lehner et al., 2024).

High alcohol consumption aids in the development of the dysfunction of metabolism, and the dysfunction becomes achievable through the infiltration of reactive oxygen species (ROS), lipid peroxidation, loss of nicotinamide adenine dinucleotide (NAD+), and subsequent dysfunction of the mitochondrion (Lee et al., 2025). All these lead to metabolic imbalances in various body systems especially the liver and heart, pancreas and the brain. The pathological effects of chronic alcohol intake on cells are far-reaching, and the effects of intoxication are acute (Birková et al., 2021). Integration of alcohol metabolism and other metabolic health has been noted to be increasingly accepted, especially in steatotic liver disease (MASLD), which is associated with metabolic dysfunction. According to Acierno

et al. (2025), nutritional and pathophysiological backgrounds need to be considered when studying the patterns of alcohol use and its effects on liver metabolism, since the disruption of the metabolism caused by alcohol-induced liver damage is an important part of the metabolic syndrome and its consequences. Also, the effects of alcohol are seen in terms of insulin resistance and metabolic dysfunction. The results obtained show that the relation between alcohol intake patterns and diverse scales of insulin resistance risks is significant (Obrador de Hevia et al., 2025).

Nutrient Depletion and Metabolic Disruption

Chronic alcohol consumption profoundly disrupts nutrient intake, absorption, storage, and utilization through a network of interdependent mechanisms. Poor dietary quality, gastrointestinal dysfunction, impaired hepatic metabolism, and increased oxidative stress collectively contribute to widespread micronutrient deficiencies in individuals with alcohol use disorder (Baj et al., 2020). These deficiencies perpetuate a self-reinforcing cycle, in which inadequate nutrient availability further compromises the body's ability to metabolize alcohol efficiently and to repair alcohol-induced tissue damage. Among the most clinically significant deficiencies is thiamine (vitamin B1), a critical cofactor in glucose metabolism and cellular energy production. Emerging evidence indicates that thiamine deficiency and neuroinflammation are central contributors to the progression of alcohol use disorder and its neurological sequelae, yet these factors remain underrecognized in routine clinical practice despite their profound impact on neurological outcomes (Kalapatapu et al., 2025). The longstanding association between thiamine deficiency and alcohol-related neurological disorders has prompted renewed interest in fortification and supplementation strategies. As discussed by Jedamzik and Juckel (2025), revisiting historical lessons may help inform more effective preventive approaches against Wernicke encephalopathy and Korsakoff syndrome.

In addition to vitamin deficiencies, chronic alcohol use is strongly associated with widespread mineral imbalances. Numerous studies have documented deficiencies in magnesium, calcium, potassium, sodium, phosphorus, selenium, zinc, and chromium among individuals with alcohol use disorder (Baj et al., 2020). A meta-analysis by Vanoni et al. (2021) demonstrated significantly reduced total and ionized circulating magnesium levels in this population, with important implications for neuromuscular function, cellular energy metabolism, and the severity of alcohol withdrawal symptoms. Collectively, these trace element imbalances exacerbate oxidative stress and impair endogenous antioxidant defense systems, contributing to ongoing cellular injury and systemic dysfunction.

Natural interventions: Parallel to Nutritional interventions Evidence

N-Acetylcysteine (NAC)

N-acetylcysteine use constitutes one of the currently promising natural interventions that facilitate alcohol metabolism and the recovery process. Otherwise, NAC helps in cellular detoxification and anti-oxidative stress, being a precursor of glutathione, the most antioxidant of nature, within the body (Quintanilla et al., 2020). Studies have shown that NAC, especially when used alongside acetylsalicylic acid, can have a major reduction in ethanol consumption via complementary levels such as oxidative stress decreasing processes and anti-inflammatory pathways. Quintanilla et al. (2020) established that the AC therapy, together with NAC, was effective at decreasing the number of ethanol drinks by 65-70 percent, which is an indication of strong synergies.

The potential therapeutic use of NAC does not only limit itself to alcohol use reduction, but also to the maintenance of neurological and liver health. NAC fills NAD+ regenerative circles by restoring stores of glutathione, which provides multiple pathophysiological pathways that result in alcohol-induced harm. Its mechanism of action in regulating the glutamate system could also help in suppressing cravings and maintaining abstinence.

Omega-3 Polyunsaturated Fatty Acids

The other evidence-based natural intervention that has numerous modes of action is omega-3 fatty acids. Cardona et al. (2025) conducted a systematic review on the effectiveness of omega-3 polyunsaturated fatty acids in alcohol use and adverse effects and discovered that the interventions are able to alleviate behavioral, biochemical, and inflammatory processes of alcohol consumption. The anti-inflammatory effect of omega-3s is most pertinent because it is the inflammation that plays a role in causing this tissue damage

when one consumes alcohol.

Zhang et al. (2025) also offered some mechanistic details about the protection of alcoholic liver disease by omega-3, showing that the protective factor in fish oil is the primary element, which is the docosahexaenoic acid (DHA), and the protective mechanism is by increasing retinol saturase (Retsat). The presented study uncovers certain molecular mechanisms according to which omega-3 supplementation could relieve liver damage caused by chronic alcoholism, providing precise treatment opportunities. The cognitive function and mood are also supported by the neuroprotective properties of omega-3s in the process of recovery.

B-Complex Vitamins and Minerals

Beyond thiamine, the broader B-complex vitamin group plays a critical role in energy metabolism, neurotransmitter synthesis, and red blood cell production processes that are frequently impaired in individuals with alcohol use disorder (AUD). Deficiencies in vitamins B6, B12, and folate are commonly observed and contribute to cognitive dysfunction, mood disturbances, and neurological impairments that occur independently of thiamine deficiency (Kalapatapu et al., 2025). These deficiencies further exacerbate disruptions in methylation pathways, homocysteine metabolism, and neural repair mechanisms, underscoring the importance of comprehensive B-complex repletion in supporting neurological function and metabolic recovery in AUD.

Antioxidants and Liver Support

In alcohol-induced damage, oxidative stress is central to the occurrence of the damage; hence, antioxidant supplementation would be a rational choice of treatment. Contreras-Zentella et al. (2025) investigated oxidative stress in liver metabolic dysfunction and diseases, with special attention paid to hepatogenic diabetes and the impact of alcohol consumption. Their study focuses on the role that chronic oxidative stress can continue to disrupt metabolism, as well as indicates the promise of antioxidant interventions to decouple this disease process. The vitamins C and E and alpha-lipoic acid offer direct antioxidant protection, countering the effects of the free radicals, and facilitating the process of tissue repair. Such nutrients, in combination with such compounds as NAC, are synergists that comprise an all-around antioxidant defense in the process of restoring alcohol damage.

Metabolic Dysfunction and Reproductive Health

In addition to its well-documented effects on major organ systems, chronic alcohol consumption exerts significant metabolic consequences that adversely affect reproductive health an aspect of alcohol-related pathology that is frequently overlooked. Alcohol-induced metabolic dysfunction alters endocrine signaling, disrupts mitochondrial energy production, and increases oxidative stress, all of which play critical roles in reproductive physiology. Genchi et al. (2024) examined alcohol within the context of metabolic pathomedicine and male infertility, demonstrating that chronic alcohol exposure induces dysmetabolic changes that impair hormonal homeostasis, compromise spermatogenesis, and reduce sperm quality, viability, and reproductive potential. These metabolic disturbances are closely linked to disruptions in testosterone synthesis, altered gonadotropin signaling, and increased oxidative damage within reproductive tissues.

Importantly, this research expands the conventional understanding of alcohol's impact beyond hepatic, neurological, and cardiovascular systems to include reproductive function as a metabolically sensitive target. The findings suggest that interventions aimed at restoring metabolic balance particularly those addressing oxidative stress, mitochondrial dysfunction, and micronutrient depletion may offer meaningful support for reproductive health in individuals with a history of chronic alcohol use. Nutritional and metabolic therapies that enhance cellular energy production and hormonal regulation may therefore play a complementary role in mitigating alcohol-related reproductive dysfunction and supporting broader physiological recovery.

Conclusion

The sources indicate that alcohol metabolism is a complex physiological process that goes far beyond the basic enzyme conversion process in the liver. Chronic alcoholism causes an overall condition of dysmetabolism, loss of minerals, and oxidative stress, as well as

inflammation, at the systemic level. The natural nutritional interventions are the evidence-based interventions that can be employed to assist people with AUD, i.e., the N-acetylcysteine, omega-3 fatty acids, vitamin B-complex, magnesium, zinc, and antioxidants. These interventions have complementary effects on the improvement of nutrient deficiencies, lowering oxidative stress, liver functionality, and targeting inflammation. Natural approaches make valuable prevention and recovery tools when they are encompassed in the holistic treatment approaches that are maintained with proper medical guidance. The ongoing studies on the topic should address the personalized nutritional interventions depending on personal genetic factors, the most appropriate dosing, and maintaining the effects of such accessible and safe interventions in the long-term to reach their full therapeutic potential.

References

1. Acierno C., et al. "Alcohol consumption and liver metabolism in the era of MASLD: Integrating nutritional and pathophysiological insights". Nutrients 17.13 (2025): 2229.
2. Baj J., et al. "Magnesium, calcium, potassium, sodium, phosphorus, selenium, zinc, and chromium levels in alcohol use disorder: A review". Journal of Clinical Medicine 9.6 (2020): 1901.
3. Birková A., et al. "Current view on the mechanisms of alcohol-mediated toxicity". International Journal of Molecular Sciences 22.18 (2021): 9686.
4. Cardona D., et al. "Impact of omega-3 polyunsaturated fatty acids on alcohol use and negative consequences: A systematic review". Nutrition Reviews 83.7 (2025) 1214–1226.
5. Contreras-Zentella ML, Hernández-Espinosa LC and Hernández-Muñoz R. "Oxidative stress in liver metabolic dysfunction and diseases, with a focus on hepatogenic diabetes: Effect of alcohol consumption". Antioxidants 14.12 (2025): 1494.
6. Genchi VA., et al. "Understanding the role of alcohol in metabolic dysfunction and male infertility". Metabolites 14.11 (2024): 626.
7. Hyun J., et al. "Pathophysiological aspects of alcohol metabolism in the liver". International Journal of Molecular Sciences 22.11 (2021): 5717.
8. Jedamzik T and Juckel G. "Fortifying alcoholic beverages with thiamine: Lessons from history and future opportunities". Drug and Alcohol Dependence Reports 16 (2025): 100358.
9. Kalapatapu N., et al. "Thiamine deficiency and neuroinflammation are important contributors to alcohol use disorder". Pathophysiology 32.3 (2025): 34.
10. Lee J, Lee J-Y and Kang H. "Excessive alcohol consumption: A driver of metabolic dysfunction and inflammation". Frontiers in Toxicology 7 (2025): 1670769.
11. Lehner T, Gao B and Mackowiak B. "Alcohol metabolism in alcohol use disorder: A potential therapeutic target". Alcohol and alcoholism 59.1 (2024): agad077.
12. Obrador de Hevia J., et al. "Association between alcohol consumption, other healthy habits, and sociodemographic variables and the values of different insulin resistance risk scales in 139,634 Spanish workers". Healthcare 13.8 (2025): 921.
13. Quintanilla ME., et al. "N-acetylcysteine and acetylsalicylic acid inhibit alcohol consumption by different mechanisms: Combined protection". Frontiers in Behavioral Neuroscience 14 (2020): 122.
14. Vanoni FO., et al. "Magnesium metabolism in chronic alcohol-use disorder: Meta-analysis and systematic review". Nutrients 13.6 (2021): 1959.
15. Zhang Y., et al. "n-3 polyunsaturated fatty acids-enriched fish oil attenuates chronic alcohol-induced liver injury via a mechanism involving the upregulation of Retsat". Journal of Nutritional Biochemistry (2025): 109971.