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Alcohol Use in Sport: Effects on Performance, Recovery, and Health Outcomes in Recreational and Competitive Athletes — A Narrative Review

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Abstract

Background. Alcohol consumption is disproportionately prevalent among athletes, particularly in team sports, where post-competition drinking coincides with the critical recovery window. A comparative analysis of alcohol's effects in recreational versus competitive athletes remains absent from the literature.
Aim. To evaluate the effects of alcohol on sport performance, post-exercise recovery, and long-term health, with a comparative analysis across athletic levels.
Material and methods. A narrative review of 24 peer-reviewed articles identified via PubMed and Scopus (primary range: 2020–2025; seminal works included).
Results. Athletes exhibit higher rates of hazardous episodic drinking than non-athletic peers. Acutely, alcohol impairs neuromuscular coordination, endurance, and post-exercise muscle protein synthesis (mTORC1 suppression; reductions up to 37%). Sleep architecture is dose-dependently disrupted; even low doses (≤0.50 g/kg) delay REM onset by 18 minutes. Immune-mediated muscle repair is blunted through cytokine dysregulation. Chronically, alcohol disrupts the hypothalamic-pituitary-gonadal axis and is associated with dose-

dependent cardiovascular mortality risk. Competitive athletes are disproportionately vulnerable due to higher training loads and narrower performance margins.
Conclusions. Alcohol exerts multisystemic negative effects on athletic performance and recovery. Sport-specific harm reduction strategies are preferable to generic abstinence messaging.

Key words: alcohol; ethanol; athletic performance; sport recovery; muscle protein synthesis; competitive athletes; recreational athletes; health

1. Introduction

Alcohol (ethanol) is one of the most widely consumed psychoactive substances worldwide, and its habitual use is associated with dose-dependent increases in all-cause mortality, cardiovascular disease, and multiple organ pathologies, with no established safe level of intake [1, 2]. Within the athletic context, the relationship between alcohol use and sport participation is particularly complex. Contrary to the expectation that athletes, by virtue of their health-conscious lifestyle, would consume less alcohol than the general population, the available evidence consistently suggests the opposite: athletes, particularly those involved in team sports at the collegiate and sub-elite levels, not only consume alcohol more frequently but also engage in hazardous episodic drinking at rates substantially exceeding those observed in non-athletic peers [3, 4, 5].

The cultural embeddedness of alcohol within sport is well-documented. Post-competition drinking is a widely practised ritual in team sports across multiple countries, serving social functions related to group cohesion, celebration, and stress relief [3]. Barnes (2014) reported that hazardous alcohol consumption — defined as regular single-session intake four to nine times the recommended safe amount — has been documented among rugby league, rugby union, and Australian Rules football players [3]. Similarly, a large-scale analysis of collegiate athletes in the United States found that, after adjusting for sociodemographic and mental health covariates, student-athletes were significantly more likely than non-athletes to be past and occasional users of alcohol [4]. This pattern appears across both sexes and multiple levels of competition, though males and team-sport participants consistently report the highest prevalence of binge drinking [3, 4, 5].

The physiological consequences of alcohol ingestion are multisystemic and of direct relevance to sport. Ethanol exerts acute depressant effects on the central nervous system, impairing reaction time, neuromuscular coordination, balance, and fine motor control — all critical components of athletic performance across a wide range of disciplines [3, 7]. At the metabolic level, alcohol suppresses muscle protein synthesis (MPS) through inhibition of the mechanistic target of rapamycin complex 1 (mTORC1) signalling pathway [9, 10]. In the post-exercise recovery window, these anabolic disruptions are particularly consequential, as both endurance and resistance training place extraordinary demands on the regenerative capacity of skeletal muscle. Furthermore, even moderate pre-sleep alcohol consumption disrupts sleep architecture — specifically delaying and reducing rapid eye movement (REM) sleep in a dose-dependent fashion — thereby compromising the hormonal and anabolic

processes that are preferentially active during nocturnal recovery [6, 11]. Chronic alcohol use introduces additional long-term health risks, including cardiovascular dysfunction, endocrine disruption affecting the hypothalamic-pituitary-gonadal axis, and immunosuppression, all of which have specific implications for the athletic population [9, 11, 12].

Despite the breadth of existing research on alcohol and physiology, a critical gap remains in the literature: the comparative analysis of alcohol's effects across different levels of athletic engagement. Recreational athletes — individuals who exercise regularly for health, enjoyment, or social participation without systematic competitive goals — and competitive athletes — those who train with structured periodisation and compete at organised levels — differ substantially in training volume, physiological adaptation, recovery demands, and the functional consequences of performance impairment. Accordingly, the magnitude and clinical significance of alcohol-induced disruption may differ meaningfully between these groups. Most existing narrative and systematic reviews have addressed either the general population or elite athletes in isolation, without explicitly framing this comparison [3, 5, 7]. The present review addresses this gap by adopting a comparative framework as its central organising principle.

Research Objective. The aim of the present narrative review is to synthesise current evidence on the effects of alcohol use on sport performance, post-exercise recovery, and long-term health outcomes, with a specific focus on the comparative analysis of recreational and competitive athletes.

Research Problems. (1) What are the acute and chronic effects of alcohol on physiological parameters directly relevant to sport performance, including neuromuscular function, aerobic capacity, and metabolic recovery? (2) How do the magnitude and clinical significance of these effects differ between recreational and competitive athletes? (3) What dose–response relationships and sex-related differences can be identified in the available evidence?

Research Hypotheses. H1: Alcohol consumption impairs key indices of athletic performance — including strength, endurance, and reaction time — in a dose-dependent manner across both recreational and competitive athletes. H2: Competitive athletes experience proportionally greater functional impairment from post-exercise alcohol consumption due to higher baseline training loads, narrower performance margins, and greater sensitivity of the anabolic recovery window. H3: The long-term health consequences of habitual alcohol use are modified by physical fitness, such that higher cardiorespiratory fitness attenuates, but does not eliminate, alcohol-associated mortality risk.

2. Research materials and methods

2.1. Search strategy

A narrative review of peer-reviewed literature was conducted between April and May 2025 using PubMed and Scopus electronic databases. The search strategy was structured around three conceptual domains: (1) the substance (alcohol/ethanol), (2) the population (athletes/sport participants), and (3) the outcome (performance, recovery, health). The primary search string applied was: ("alcohol" OR "ethanol") AND ("athlete" OR "sport*" OR

"exercise") AND ("performance" OR "recovery" OR "health" OR "muscle protein synthesis" OR "testosterone" OR "sleep" OR "cardiovascular" OR "hydration" OR "immunity"). Additional domain-specific searches were performed for individual outcome categories, including: ("alcohol" AND "mTOR"), ("alcohol" AND "sleep architecture"), ("alcohol" AND "cardiovascular disease"), and ("alcohol" AND "endocrine" AND "exercise"). Supplementary manual searches were conducted within the reference lists of included articles. The primary time frame was January 2020 to May 2025; however, seminal works published prior to 2020 were included as foundational references where no more recent evidence of equivalent methodological quality was available. A total of 24 articles were included in the final synthesis.

2.2. Eligibility criteria

Inclusion criteria: (1) original research articles, systematic reviews, meta-analyses, or narrative reviews published in peer-reviewed journals; (2) published in English; (3) involving adult human participants (aged ≥18 years) or, where relevant to establishing population-level context, adolescent samples (aged ≥13 years); (4) addressing the relationship between alcohol consumption and at least one of the following outcomes: physical performance, post-exercise recovery, sleep quality, hormonal or immune function, cardiovascular health, hydration status, body composition, mental health, or injury risk; (5) reporting data specifically on athletic or physically active populations, or providing mechanistic evidence directly applicable to such populations.

Exclusion criteria: (1) studies conducted exclusively in animal models; (2) studies focused solely on clinical alcohol use disorder or alcohol dependence syndrome without relevance to athletic or physically active populations; (3) non-peer-reviewed sources, including grey literature, conference abstracts, and media reports; (4) articles for which full text was unavailable; (5) duplicate publications reporting the same dataset.

2.3. Data collection and analysis

2.3.1. AI

AI was utilized for two specific purposes in this research. Assistance in identifying relevant search terms and organizing literature by thematic categories. Assistance in refining the academic English language of the manuscript, ensuring clarity, consistency, and adherence to scientific writing standards. It is important to emphasize that all AI tools were used strictly as assistive instruments under human supervision. The final interpretation of results, synthesis of evidence, and conclusions were determined by the human authors. The AI tools served primarily to enhance efficiency in literature organization and linguistic refinement, rather than replacing human judgment in the analytical process.

3. Research results

3.1. Prevalence of alcohol use among athletes

Alcohol consumption is disproportionately prevalent among athletic populations compared to the general public. Despite the intuitive expectation that health-oriented individuals would consume less alcohol, the evidence consistently demonstrates the opposite pattern. Barnes (2014) reported that at least half of surveyed sportspeople engaged in regular hazardous binge-drinking behaviour, compared to approximately 16.5% of the general population [3]. This paradox is particularly pronounced in team sports, where alcohol use is deeply embedded in post-competition culture as a vehicle for social bonding, celebration, and stress relief [3].

At the collegiate level, Charest et al. (2021), analysing data from 110,750 students across 2011–2014, found that collegiate athletes were significantly more likely than non-athletes to be past users of alcohol (OR = 1.317) and occasional users (OR = 1.173) after full adjustment for sociodemographic and mental health covariates [4]. Importantly, frequent alcohol use was not elevated among athletes, suggesting that the predominant pattern is episodic rather than chronic heavy use — consistent with the binge-drinking profile characteristic of post-competition rituals. Athletes were also less likely than non-athletes to use cigarettes, cannabis, cocaine, and sedatives, underscoring the specificity of alcohol as the substance of choice within the athletic milieu [4].

The relationship between sport participation and alcohol use is moderated by several variables, including sex, sport discipline, and level of competition. Males consistently exhibit higher rates of hazardous drinking than females across all athlete categories [3, 4, 14]. With respect to sport type, team and contact sports are associated with the greatest alcohol use; a scoping review by Walczak et al. (2023) reported that participation in high-contact sports conferred an odds ratio of 1.860 for being drunk in the past 30 days among adolescent athletes, compared to 1.477 for sport participation in general [14]. Ball sports and football disciplines were most consistently associated with elevated alcohol consumption across both sexes and age groups [14].

An important distinction between recreational and competitive athletes concerns the level of professionalisation and its moderating effect on alcohol use. Barnes (2014) noted that drinking as a post-match reward is most prevalent among sub-elite athletes, while elite athletes, subject to tighter schedules, dietary monitoring, and anti-doping obligations, appear to consume less alcohol — though not necessarily at safer levels [3]. This distinction is supported by data from Baskerville et al. (2024), who observed that immune-related health risks associated with alcohol misuse may be differentially distributed across competitive levels, with recreational athletes representing a particularly under-examined at-risk group [12]. Among adolescent athletes, Schmidt et al. (2024) reported that 70% consumed alcohol, 30% exhibited heavy episodic drinking (HED), and 10% reported high-frequency HED — rates exceeding those in the general adolescent population in the same country [15]. Greater engagement and personal fulfilment derived from sport participation were identified as protective factors against HED, underscoring the importance of sport experience quality in prevention efforts [15].

3.2. Acute effects of alcohol on physical performance

Ethanol exerts a complex pattern of acute effects on physiological systems relevant to athletic performance. As a central nervous system (CNS) depressant, alcohol impairs neuromuscular coordination, reaction time, balance, and fine motor control in a dose-dependent manner [3, 7]. These effects are mediated primarily through facilitation of GABA-A receptor activity and inhibition of glutamate transmission, resulting in generalised CNS depression that manifests functionally as slowed decision-making, impaired spatial processing, and reduced postural stability — all of which are critically relevant to sport performance across a wide range of disciplines [3, 7, 16].

With respect to aerobic endurance capacity, the evidence consistently demonstrates a dose-dependent impairment. Vella and Cameron-Smith (2010) identified an ethanol threshold of approximately 20 mmol/L, beyond which significant aerobic performance decrements become evident, with this relationship operating in a dose-dependent manner [5]. At practically relevant doses consumed by athletes — such as 0.5 g/kg lean body weight — reductions in total work performed during cycling time trials have been documented [7]. Shirreffs and Maughan (2006) further noted that the effects of alcohol on aerobic performance are mediated through multiple mechanisms, including impaired hepatic gluconeogenesis, peripheral vasodilation impairing thermoregulation, and disruption of carbohydrate metabolism during sustained exercise [7]. Pesta et al. (2013) confirmed this pattern in their comprehensive pharmacological review, noting that ethanol acutely reduces maximal oxygen uptake and impairs substrate utilisation during aerobic exercise [16].

The evidence regarding anaerobic performance — including maximal strength and power output — is less consistent. Barnes (2014) reviewed evidence showing that isometric and isokinetic strength are largely unaffected by acute alcohol ingestion at doses up to 1.59 g/kg in healthy adults, a finding partially explained by the preservation of peripheral muscle contractility under moderate blood alcohol concentrations [3]. However, sprint performance, which requires a high degree of neuromuscular coordination and motor control, has been shown to be impaired in a dose-dependent manner even at breath alcohol concentrations as low as 0.01 mg/ml, with greater impairment at 0.10 mg/ml across distances of 200 m to 1500 m [3, 16]. This distinction between gross strength and performance tasks with high technical demand is of practical importance: alcohol may not impair a single maximal effort but will likely compromise sport-specific performance that integrates strength, speed, timing, and coordination.

3.3. Alcohol and post-exercise recovery

An important contextual consideration is that most athletes consume alcohol after, rather than before, competition. Murphy et al. (2013) investigated the post-match effects of alcohol ingestion in competitive Rugby League players using a randomised crossover design, in which participants consumed 1 g of ethanol per kilogram body mass four hours after a competitive match, reporting that this protocol resulted in measurably impaired recovery of neuromuscular performance and elevated perceptions of fatigue compared to a non-alcoholic

control [17]. This post-exercise drinking pattern — ubiquitous in team sports — means that the acute performance consequences of alcohol manifest not as direct impairment of the current competition, but as impairment of recovery capacity for subsequent training and competition, which is arguably a more consequential effect for the competitive athlete. A further dimension of this post-exercise vulnerability is hydration: Desbrow et al. (2013) demonstrated that full-strength beer (4.8% ABV) consumed following exercise-induced dehydration produced significantly worse net fluid balance than low-alcohol beer (2.3% ABV) with added sodium, with substantially greater urine output observed in the full-strength condition [23]. These findings indicate that the alcohol content of beer directly exacerbates post-exercise fluid losses, compounding the physiological deficits incurred during competition.

The post-exercise recovery period is particularly vulnerable to the deleterious effects of alcohol. Three primary recovery domains are discussed below: (1) muscle protein synthesis, (2) sleep quality, and (3) immune-mediated muscle repair.

3.3.1. Muscle protein synthesis (MPS)

Alcohol consumption following resistance exercise suppresses the anabolic signalling cascade, specifically the mTORC1 pathway, which is central to post-exercise MPS. The landmark study by Parr et al. (2014) provided the first direct human evidence of this effect, demonstrating that ingestion of 1.5 g/kg body mass of ethanol after concurrent resistance, continuous, and high-intensity interval exercise reduced myofibrillar fractional synthetic rates (FSR) by 24% compared to protein ingestion alone, and by 37% when alcohol was consumed without protein — even though blood amino acid profiles were similar between conditions [19]. The mechanistic basis of this suppression involves attenuation of mTORSer2448 phosphorylation and downstream p70S6K signalling, mediated in part through upregulation of the negative mTOR regulator REDD1 and increased mTOR-raptor binding [10, 19]. A comprehensive evidence-based narrative review by Levitt et al. (2022) confirmed that acute ethanol consistently suppresses mTORC1 pathway signalling, with effects moderated by sex, nutritional co-ingestion, training status, dose, and timing of ethanol intake [10]. Importantly, the evidence indicates that anabolic stimuli from protein ingestion can only partially rescue this inhibition, and the mTOR suppression is not fully counteracted even under conditions of optimal nutritional provision [10, 19]. From the perspective of the competitive athlete, for whom each training session represents a critical stimulus for adaptation, this partial rescue is insufficient to prevent meaningful impairment of the recovery process.

3.3.2. Sleep and recovery

Alcohol consumption consistently and dose-dependently disrupts sleep architecture in a manner directly relevant to athletic recovery. While it may shorten sleep onset latency at high doses (≥0.85 g/kg), this effect does not translate into improved sleep quality. The most comprehensive quantification of these effects comes from the systematic review and meta-analysis by Gardiner et al. (2025), which synthesised 27 controlled crossover studies and found that even a low dose of alcohol (≤0.50 g/kg, approximately three standard drinks)

delays REM sleep onset latency by a mean of 18.0 minutes and reduces total REM sleep duration by 11.3 minutes, with progressively greater disruptions at higher doses [6]. Shortening of sleep onset latency and latency to deep sleep (N3) was only observed at high doses (≥0.85 g/kg), meaning any perceived benefit to sleep initiation is achieved at the cost of substantially worsened REM disruption [6]. These findings were corroborated by the prospective smartwatch-based study by Strüven et al. (2025), which demonstrated that moderate alcohol consumption (40 g/day for women, 60 g/day for men over three consecutive evenings) significantly elevated nocturnal resting heart rate from 63.6 ± 9.2 bpm at baseline to 66.6 ± 9.0 bpm during exposure ($p < 0.001$), with rapid normalisation after cessation, while no significant changes in objective sleep architecture were observed [20]. The elevation in nocturnal heart rate reflects autonomic dysregulation — increased sympathetic activity with reduced vagal tone — that likely impairs the physiological recovery quality during sleep [20]. Supporting evidence from a large cross-sectional study of 933 student-athletes demonstrated using structural equation modelling that poor sleep quality — itself negatively predicted by unhealthy lifestyle behaviours including alcohol consumption — was significantly associated with a higher number of injuries, while physical fitness did not show a direct protective effect against injury occurrence [24]. For competitive athletes, who frequently operate at the limits of recovery capacity and for whom sleep architecture is a critical determinant of adaptation, the disruption of even one or two REM cycles through post-competition drinking represents a meaningful performance liability.

3.3.3. Immune response to muscle injury

A further dimension of post-exercise recovery is the immune-mediated repair of exercise-induced muscle damage. Under normal circumstances, the innate immune system initiates a coordinated inflammatory response to muscle trauma, directing phagocytosis, satellite cell activation, and myofibrillar regeneration [3]. Barnes (2014) summarised evidence that acute alcohol consumption disrupts this process by shifting the cytokine balance toward an anti-inflammatory environment: specifically, alcohol downregulates tumour necrosis factor-alpha (TNF-α) production, inhibits interleukin-1β expression, and increases anti-inflammatory mediators including TGF-β, IL-10, and prostaglandin E2 [3]. While a transient reduction in inflammation may seem beneficial, this disruption impairs the coordinated immune sequence required for effective tissue repair, potentially delaying full muscle recovery between training sessions or competitions. For athletes engaged in high-frequency training or multi-day competition schedules, this alcohol-induced blunting of the repair response may contribute to cumulative under-recovery and increased injury susceptibility [3].

3.4. Long-term health consequences

3.4.1. Cardiovascular system

The relationship between alcohol consumption and cardiovascular health has historically been characterised by the J-curve hypothesis, which proposed cardioprotective effects at low-to-moderate intake levels. However, this hypothesis has been substantially challenged by methodologically rigorous evidence using Mendelian randomisation (MR) — a technique that uses genetic variants as proxies for alcohol exposure, thereby circumventing confounding by

healthy lifestyle factors that disproportionately characterise moderate drinkers. Biddinger et al. (2022), using MR analyses in 371,463 UK Biobank participants, demonstrated that a 1-SD increase in genetically predicted alcohol consumption was associated with significantly elevated odds of hypertension (OR 1.28, 95% CI 1.18–1.39), coronary artery disease (OR 1.38, 95% CI 1.10–1.74), myocardial infarction (OR 1.37, 95% CI 1.05–1.78), stroke (OR 1.26, 95% CI 1.04–1.54), heart failure (OR 1.39, 95% CI 1.08–1.78), and atrial fibrillation (OR 1.24, 95% CI 1.08–1.44) [2]. Critically, the apparent cardioprotection of light drinking in observational studies was substantially attenuated after adjustment for six lifestyle confounders, strongly suggesting that the J-curve reflects confounding rather than a genuine biological benefit [2]. Zhao et al. (2023), in a systematic review and meta-analysis of 107 cohort studies, confirmed that no safe level of alcohol consumption for all-cause mortality could be established, and that risk estimates previously suggesting a protective effect largely disappeared after methodological improvements controlling for abstainer bias [1]. The prospective HUNT Study (Nauman et al., 2026), which examined the joint effects of changes in alcohol intake and cardiorespiratory fitness (CRF) over 10 years, found that changes in alcohol intake were positively associated with mortality risk, but that maintaining or improving CRF was a stronger predictor of all-cause mortality than changes in alcohol intake — providing important nuance for the physically active population, suggesting that fitness attenuates but does not eliminate alcohol-associated mortality risk [21].

3.4.2. Hormonal system

Chronic alcohol consumption exerts dose-dependent and bidirectional effects on the endocrine system with direct implications for athletic performance and recovery. Rachdaoui and Sarkar (2013) provided a comprehensive review of alcohol-endocrine interactions, documenting that chronic heavy alcohol use disrupts the hypothalamic-pituitary-gonadal (HPG) axis, leading to suppression of luteinising hormone (LH) and follicle-stimulating hormone (FSH) secretion, reduced gonadal testosterone synthesis, and elevated cortisol through dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis [11]. Elevated cortisol in the context of suppressed testosterone is a highly unfavourable hormonal milieu for the athlete, as it promotes net protein catabolism and impairs anabolic adaptation to training. Haugvad et al. (2014), investigating post-exercise alcohol consumption in resistance-trained subjects, demonstrated that while ethanol did not significantly delay markers of muscle damage recovery, the testosterone-to-cortisol ratio was significantly reduced in the alcohol condition — an important indicator of the anabolic-catabolic balance that governs training adaptation [22]. These hormonal consequences are compounded by alcohol-induced disruption of sleep architecture, as described in section 3.3.2, and by direct suppression of Leydig cell testosterone production in the testes at higher doses [11]. Collectively, these endocrine disruptions attenuate the hormonal environment required for muscle hypertrophy, strength development, and aerobic adaptation, thereby limiting an athlete's capacity to realise training-induced gains and sustain long-term athletic development.

3.4.3. Musculoskeletal system

Chronic alcohol use impairs skeletal muscle regenerative capacity at the cellular level. Khair et al. (2022) demonstrated that alcohol decreases the differentiation potential of muscle stem cells (myoblasts) into mature myotubes, disrupts myoblast bioenergetics by reducing glycolytic capacity, and increases expression of class IIA histone deacetylases that impair myogenic gene expression [9]. These effects mean that habitual alcohol use may progressively compromise the capacity for structural muscle adaptation, limiting long-term gains in muscle mass and strength independently of acute training stimuli. From a bone health perspective, alcohol disrupts calcium metabolism and osteoblast function, reducing bone mineral density and increasing fracture risk — consequences of particular relevance to contact sport athletes and those in high-impact disciplines [9].

3.4.4. Immune function

Alcohol exerts dose- and chronicity-dependent effects on immune function that are of particular relevance to the athletic population. Khair et al. (2022) reviewed evidence from the 2021 AIRIG symposium demonstrating that alcohol misuse reduces natural killer (NK) cell numbers and cytolytic activity, impairs alveolar macrophage phagocytic function through mitochondrial bioenergetic disruption, increases susceptibility to bacterial pneumonia, and promotes neuroinflammation through amplification of IL-6 signalling pathways in the prefrontal cortex [9]. This latter finding warrants specific clarification in the context of the present review: as described in section 3.3.3, acute alcohol exposure inhibits IL-6 expression in peripheral skeletal muscle, thereby blunting the local pro-inflammatory response to exercise-induced damage. Chronic alcohol exposure, by contrast, amplifies IL-6 signalling in the central nervous system via GP130/STAT3 pathway upregulation, contributing to neuroinflammation and neuronal damage [9]. These opposing effects reflect the tissue-specificity and exposure-duration-dependency of alcohol's immunomodulatory properties. These immunosuppressive effects are exacerbated by the immunological stress of intense training. The post-exercise period of reduced immunity has been described as an "open window" providing opportunity for pathogen invasion, though Baskerville et al. (2024) note that this model is contested — the transient reductions in circulating lymphocyte numbers may reflect redistribution to peripheral tissues rather than true immunosuppression [12]. There is nonetheless consensus that immunodepression occurs following extreme and prolonged exercise, and Baskerville et al. (2024) concluded that the combination of heavy alcohol consumption and high-volume training creates a particularly high-risk profile for upper respiratory tract infections [12]. In this context, post-competition binge drinking among competitive athletes represents a compounded immunological insult: occurring precisely when the athlete is most immunologically vulnerable, it further depresses an already challenged immune system and may meaningfully extend the period of heightened infection susceptibility.

3.4.5. Body composition

Alcohol is a calorically dense macronutrient substitute, providing approximately 7 kcal per gram — second only to dietary fat [7]. Its consumption contributes to total energy intake without providing nutritional value, and its metabolism generates acetate, which suppresses lipid oxidation and promotes de novo lipogenesis [16]. The net effect is a shift in body composition toward increased fat deposition and reduced lean mass, particularly relevant in athletes for whom body composition is a key performance determinant [16]. The BEER-HIIT study (Molina-Hidalgo et al., 2020) provided reassuring evidence that moderate, regular ethanol or beer consumption (approximately 1.7–2.6 drinks/day for men) during 10 weeks of HIIT did not negatively impact lean mass gains or fat loss compared to alcohol-free controls [18], suggesting that moderate chronic intake may not meaningfully compromise body composition when combined with regular structured exercise. However, these findings cannot be extrapolated to heavier drinking patterns or to competitive athletes with more stringent body composition requirements. The mental health dimension of alcohol use — including alcohol as a coping mechanism for competitive stress, burnout, or career-related anxiety — is a further consideration in the athletic context. Oevreboe et al. (2023) reported that mental distress is highly prevalent in elite Norwegian athletes, with significant proportions meeting criteria for clinical anxiety and depression [13], conditions that are established risk factors for hazardous alcohol use and that may perpetuate a cycle of drinking as self-medication.

3.5. Comparative analysis: recreational versus competitive athletes

The effects of alcohol on performance and health are not uniform across athletic populations, and a comparative framework reveals meaningful differences in the magnitude, context, and clinical significance of these effects between recreational and competitive athletes. These differences operate across three principal dimensions: training demands and recovery windows, performance margins, and social-cultural context of alcohol use.

With respect to training demands and recovery windows, competitive athletes typically operate at higher training volumes, greater intensity distribution, and more frequent competition cycles than recreational counterparts. This creates a narrower recovery window within which alcohol-induced impairments of MPS, hormonal balance, and sleep architecture are disproportionately consequential. A recreational athlete who trains three times per week has substantially greater buffer capacity for sub-optimal recovery than a competitive athlete who trains twice daily with weekly competition obligations. In this context, even a modest 24% reduction in MPS (as reported by Parr et al. [19]) or a 37% reduction in the absence of co-ingested protein may represent the difference between positive net protein accretion and net catabolism over a training week. Furthermore, the hormonal consequences of post-competition drinking — reduced testosterone-to-cortisol ratio [22], impaired growth hormone secretion during sleep [6] — will cumulatively impair adaptation over a competitive season in a manner less likely to be offset in the recreational athlete [11].

With respect to performance margins, competitive athletes operate within narrower bands of performance where small absolute impairments translate into competitive disadvantage. The

neuromuscular and cognitive impairments associated with even moderate alcohol consumption — slowed reaction time, impaired fine motor coordination, reduced balance — may be functionally irrelevant for a recreational athlete jogging for fitness, but critically relevant for a competitive athlete whose performance depends on millisecond reaction times, precise technical execution, and optimal tactical decision-making. Barnes (2014) emphasised this distinction, noting that the absence of a statistically significant effect of alcohol on maximal isometric strength does not imply an absence of practically meaningful performance impairment in sport-specific tasks that integrate multiple capacities [3].

With respect to social-cultural context, the paradox of high alcohol prevalence in competitive sport — documented across rugby [17], Australian rules football [3], collegiate athletics [4], and adolescent sport [15] — reflects the deeply embedded cultural role of post-competition drinking in team sport identity and social cohesion. Barnes (2014) observed that drinking as post-match reward is most prevalent among sub-elite competitive athletes, while the evidence suggests that elite athletes at the highest competitive levels may exhibit more restraint, though not necessarily at physiologically safe levels [3]. Recreational athletes, while also embedded in drinking cultures, typically face lower social pressure around post-competition drinking and carry lower stakes for recovery quality, potentially making them more amenable to behavioural modification. The immune vulnerability window is an important dimension specific to competitive athletes: Baskerville et al. (2024) noted that high-volume training creates a transient immunosuppressed state that is substantially worsened by alcohol consumption [12], making the post-competition period — precisely when binge drinking is most culturally sanctioned — the moment of greatest immunological risk.

4. Discussion

This narrative review synthesises evidence across multiple physiological domains to provide a comprehensive picture of how alcohol consumption affects sport performance, post-exercise recovery, and long-term health in recreational and competitive athletes. The overarching conclusion is that alcohol is broadly detrimental to the athlete at both acute and chronic timescales, with effects operating through CNS depression, metabolic disruption, anabolic signalling inhibition, autonomic dysregulation during sleep, hormonal suppression, immune compromise, and direct organ toxicity. The magnitude and clinical significance of these effects are dose-dependent and moderated by training status, sex, nutritional context, and the timing of ingestion relative to exercise.

A key contribution of this review is the adoption of a comparative framework distinguishing recreational from competitive athletes — a distinction that has been underserved in the existing literature. The evidence converges on the conclusion that competitive athletes are disproportionately vulnerable to alcohol-induced impairments of recovery and adaptation, due to higher training loads, narrower performance margins, and the specific timing of post-competition drinking during the recovery window. This comparative perspective has direct implications for sports medicine practice: alcohol management strategies and educational interventions should be calibrated to the athlete's competitive level, training load, and specific

recovery requirements, rather than applying uniform public health messaging designed for the general population.

Several important nuances emerge from the evidence. First, the effects of alcohol on strength and power appear less consistent than those on endurance, neuromuscular coordination, and recovery — suggesting that sport disciplines with high technical and endurance demands may be more sensitive to alcohol-related impairment than purely strength-based activities. Second, moderate chronic alcohol consumption (approximately 1–2 standard drinks per day), as investigated in the BEER-HIIT study [18], does not appear to negate the cardiovascular and muscular adaptations to structured exercise training — a finding with practical relevance for recreational athletes for whom complete abstinence is neither expected nor required. Third, the cardiovascular risk literature has undergone substantial revision: the putative J-curve of cardioprotection has been refuted by Mendelian randomisation evidence [1, 2], and the implication that no level of alcohol consumption is universally safe for long-term cardiovascular health applies to athletes as it does to the general population. Fourth, emerging data from Nauman et al. (2026) suggest that high cardiorespiratory fitness attenuates alcohol-associated mortality risk [21], providing a biologically plausible mechanism through which athletes may partially offset alcohol's long-term harms through sustained physical conditioning — though this finding should not be interpreted as a licence for hazardous drinking.

Several limitations of the existing evidence base should be acknowledged. A substantial proportion of the available data on alcohol and athletic performance relies on self-reported alcohol intake, which is known to systematically underestimate actual consumption. Ethical constraints prevent the conduct of randomised controlled trials in which participants are assigned to heavy drinking conditions, limiting the mechanistic precision of human evidence. Most studies have been conducted in young, male, collegiate populations, limiting generalisability to female athletes, masters athletes, and non-Western populations. The comparative analysis of recreational versus competitive athletes is hampered by the absence of a universally accepted operational definition for these categories and by the limited number of studies that explicitly stratify analyses by competitive level. Finally, the rapidly evolving landscape of evidence — particularly regarding cardiovascular risk and sleep physiology — means that conclusions should be interpreted in light of ongoing research that may refine current understanding. Future research should prioritise prospective longitudinal designs stratifying by competitive level, sex-specific analyses of hormonal and recovery outcomes, investigation of the interaction between alcohol consumption and sport-specific training modalities, and the development of evidence-based alcohol harm reduction frameworks specifically designed for athletic populations.

5. Conclusions

Alcohol consumption exerts multisystemic negative effects on athletic performance, post-exercise recovery, and long-term health outcomes across both recreational and competitive athletes. Acutely, ethanol impairs CNS function, endurance performance, and post-exercise anabolic signalling — particularly through suppression of mTORC1-mediated muscle protein

synthesis — and disrupts sleep architecture in a dose-dependent manner that further compromises recovery. Chronically, habitual alcohol use is associated with adverse cardiovascular outcomes, hormonal dysregulation, impaired skeletal muscle repair and adaptation, immunosuppression, and unfavourable body composition changes.

Competitive athletes are disproportionately vulnerable to these effects compared to recreational athletes, owing to higher training loads, narrower performance margins, and the specific context of post-competition drinking occurring during the critical anabolic recovery window. The embedded cultural role of alcohol in team sport identity represents a significant barrier to harm reduction, requiring evidence-based educational and cultural interventions tailored to the athletic environment.

The evidence reviewed supports several specific conclusions regarding the mechanisms through which alcohol impairs recovery. Post-exercise anabolic signalling is disrupted through suppression of mTORC1-mediated muscle protein synthesis, with reductions of up to 37% documented in the absence of co-ingested protein [19]. Sleep architecture is dose-dependently disrupted at any level of alcohol intake, with even low doses (≤0.50 g/kg) delaying REM sleep onset by a mean of 18 minutes and reducing REM duration by 11 minutes [6]. The normal immune-mediated response to exercise-induced muscle damage is blunted through alcohol-induced cytokine dysregulation, potentially delaying full tissue repair between training sessions [3]. The diuretic effect of full-strength beer further compounds post-exercise fluid deficits, with significantly greater urine output observed compared to low-alcohol alternatives [23]. Chronically, alcohol disrupts the hypothalamic-pituitary-gonadal axis, reduces the testosterone-to-cortisol ratio, impairs myoblast differentiation, and is associated with dose-dependent increases in cardiovascular mortality risk that are not abrogated by physical fitness alone [1, 2].

The comparative analysis of recreational and competitive athletes reveals that the consequences of alcohol use are not uniform across athletic populations. Competitive athletes face disproportionate risk due to narrower recovery margins, higher training frequencies, and the specific timing of post-competition drinking during the anabolic window. However, evidence from the BEER-HIIT study suggests that moderate, regular alcohol consumption does not significantly attenuate fitness adaptations in recreationally active individuals [18], and high cardiorespiratory fitness appears to attenuate, but not eliminate, alcohol-associated mortality risk [21] — findings that underscore the dose- and context-dependency of alcohol's effects. The paradox of elevated alcohol prevalence in sport reflects the deep cultural embeddedness of post-competition drinking and represents a meaningful public health challenge requiring sport-specific harm reduction strategies rather than generic abstinence messaging. Clinicians, sports medicine practitioners, coaches, and performance nutritionists should communicate evidence-based information about the specific physiological consequences of post-exercise alcohol consumption, tailored to the athlete's competitive level and recovery demands. When alcohol consumption occurs, strategies to minimise harm include delaying consumption until after initial recovery nutrition has been completed, limiting total intake, ensuring adequate hydration, and avoiding alcohol in the immediate pre-sleep period.

These findings have direct implications for sports medicine practitioners, coaches, and athletes, and underscore the importance of evidence-based alcohol education within sporting environments.

Disclosure

Supplementary Materials

Not applicable.

Author Contributions

Conceptualization: Zięty M., Woźniak D.; Methodology: Zięty M., Wojtyniak W.; Literature Search: Wojtyniak W., Jóźwicka M., Makowski P., Banyś K.; Writing – Original Draft: Zięty M., Woźniak D., Tomasik J.; Writing – Review & Editing: all authors; Supervision: Zięty M. All authors have read and agreed to the published version of the manuscript.

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Conflicts of Interest

The a

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