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REVIEW ARTICLE

Effects of Alcohol Consumption Following Resistance Training: A Narrative Review of Physical Performance and Metabolic Recovery

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Abstract

Background: **Resistance training is a popular exercise modality that aims to increase muscle strength, power, and endurance. Meanwhile, alcohol is a widely used recreational beverage that has been found to negatively affect physical performance and recovery. Thus, understanding the effects of alcohol consumption following resistance training is crucial for athletes and fitness enthusiasts.**

Method: **A literature search from 2010 to 2022 was conducted on the PubMed database to identify relevant studies investigating the effects of alcohol consumption after resistance training. The following search terms were used: "alcohol," "resistance training," "physical performance," "recovery," and "metabolism." Only studies published with human participants were included. A total of 12 studies were included in this review. The results were divided into several topics: strength, power, delay-onset muscle soreness (DOMS), immune function, serum creatinine kinase (CK), anabolic markers, and testosterone and cortisol levels.**

Results: **No significant effects on power, DOMS, or CK were found. Diverse results were found regarding immune function and testosterone/cortisol levels, suggesting that the effects of alcohol consumption on resistance training may be individual-specific. For strength and anabolic markers, dose-dependent reductions were observed in several studies, particularly in male participants, indicating a potential sex-related effect.**

Conclusion: **Alcohol consumption may impair strength and anabolic markers in a dose-dependent manner, particularly in male participants. The effects on immune function and testosterone/cortisol levels were individual-specific, with varying results among studies. These findings may have practical implications for athletes and fitness enthusiasts, emphasizing the importance of alcohol moderation to optimize physical performance and recovery.**

Keywords: Resistance exercise, Alcohol ingestion, Athlete's performance

1. Introduction

Resistance training (RT) is a type of physical activity that can increase muscle strength, power, size, and endurance, making it a popular choice in the last few decades.[1] While non-athletes mostly perform RT to improve their physical appearance, athletes view it as a way to improve their performances in various sports,[2] making RT a crucial factor for sports people at all levels.

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RT is a powerful stimulus that changes homeostatic status, causing adaptive reactions in metabolism, which repairs the structural damage caused by previous training sessions.[3] Therefore, proper recovery is essential and can lead to numerous benefits in training and upcoming competitions.[4]

Alcohol (ALC) consumption is a well-known unnecessary stressor during recovery after RT,[5,6] with an estimated average of 4.3 L of pure alcohol consumed yearly per person worldwide.[7] It is known that alcohol affects various organs and tissues in the body, causing injury and dysfunction.[8] In relation to post-exercise recovery, studies have shown that alcohol consumption shortly after exercise can decrease muscle protein synthesis, and increase the expression of enzymes associated with muscle atrophy,[5,9] while the real effects of alcohol consumption after resistance training still remain unclear.

Based on the above circumstances, this review aims to understand the effects of alcohol consumption on physical performance and metabolic recovery after resistance training.

2. Methods

A search was performed using the PubMed search engines to investigate the acute effects alcohol after resistance training may have on physical performance and recovery factors.

The key search terms 'alcohol consumption' OR 'alcohol ingestion' OR 'ethanol consumption' OR 'ethanol ingestion' were used in combination with the following: 'resistance training', 'resistance exercise', 'eccentric training', 'eccentric exercise'.

Articles considered for inclusion in this review had to address all the criteria.

1. The inclusion criteria only encompassed studies that specifically investigated the impacts of alcohol intake "post" physical training or exercise.
2. The research must have a publication date after January 1st, 2010, in order to ensure the incorporation of current research.
3. This review only includes studies that have been conducted on human subjects, in order to ensure the applicability of the findings (Tables 1 and 2).

3. Strength performance

Seven retrieved studies analyzed the muscle strength performance. While 4 studies revealed no statistical significance, three studies showed a decrease in peak or average strength after resistance training followed by alcohol consumption, and there may be a sex-related effect.

Barnes et al., 2010a,[10] Barnes et al., 2010b,[11] Barnes et al., 2011,[12] and Barnes et al., 2012[13] experimented with similar protocols, ten to twenty-three male participants were asked to perform a session of a total of 300 repetitions of single leg quadriceps femoris extension, and beverages containing 1 g(g)[10,11,13] or 0.5 g[12] per kilogram (kg) body weight was administered. Peak and average torque[10−12] or maximal voluntary isometric contraction[13] (MVIC) were recorded. After two weeks, the identical session was employed, alcohol-free beverages were given this time, and the same measurements were performed.

All of the studies above are conducted on male participants, and showed a significant decrease in muscle performance after training despite conditions, Barnes et al., 2010a[10] and Barnes et al., 2010b[11] both revealed a significantly greater loss in peak or average strength with moderate alcohol consumption (1 g/kg body weight ethanol), and Barnes et al., 2012[13] also showed a significant decrement in MVIC, compared with placebo beverage consumption. However, Barnes et al., 2011[12] showed no difference between mild alcohol consumption (0.5 g/kg body weight) and alcohol-free beverage ingestion. The difference between those four similar studies may imply that the effect of peak strength loss caused by alcohol consumption may be a dose-dependent effect, at least in males.

Levitt et al.[14] and McLeay et al.[15] performed their experiments on females, with similar experiment protocols compared to Barnes et al.[10−13] researches. Thirteen and eight female participants were recruited, respectively, and performed two identical bouts of exercise (A total of 300 repetitions of single leg quadriceps femoris extension), followed by alcohol (with the dosage of 1.09g/kg fat-free body weight[14] or 0.88g/kg body weight[15]) or placebo ingestion, maximal torque production was measured

Table 1. A brief summary of the included studies.

Author	Participants (Age)	ALC dosage	Resistance exercise	Measurements	Results
Barnes[10]	11 males (23.9 ± 4.7 years)	1 g/kg BW	1 x 300 eccentric contractions of the quadriceps muscles	Peak and averaged torque, Soreness, and CK at 36 and 60 h post-exercise	Significantly decreased peak strength in the ALC group. No effects on plasma CK and ratings of muscle soreness between conditions
Barnes[11]	10 males (23.5 ± 5.1 years)	1 g/kg BW	1 x 300 eccentric contractions of the quadriceps muscles	Peak and averaged torque at 36 and 60 h post-exercise	Significantly decreased average and peak torque for the ALC group except for peak concentric torque.
Barnes[12]	10 males (20.8 ± 1.6 years)	0.5 g/kg BW	1 x 300 eccentric contractions of the quadriceps muscles	Peak and averaged torque at 36 and 60 h post-exercise	No effects on peak or averaged torque between conditions
Barnes[13]	23 males (24.1 ± 5.6 years)	1 g/kg BW	3 x 100 eccentric contractions of the quadriceps muscles	MVIC, iEMG, VA, LFF, and CK at 36 and 60 h post-exercise	Significantly decreased VA and MVIC in the ALC group, with no effects on iEMG, LEF, and serum CK levels between conditions
Vingren[45]	8 males (25.3 ± 3.2 years)	1.09 g/kg fat-free body mass	6 x 10 Smith squats	TT, FT, cortisol, SHBG, and estradiol at 20−40, 60−120, and 140−300 min post-exercise	Significantly higher serum TT, FT, and free androgen in the ALC group at 140−300 min post-exercise, no effects on SHBG, cortisol, or estradiol between conditions
Parr[6]	8 males (21.4 ± 4.8 years)	1.5 g/kg BW	8x5 leg extension (80 % 1-RM), 30 min (63 % PPO), and high-intensity interval (10 × 30 s, 110 % PPO) cycling	Muscle Biopsy, Blood glucose, and plasma AA concentration at 2, 4, 6, and 8 h post-exercise	Significantly lower phosphorylation of mTOR in the ALC group 2 h post-exercise. Blood glucose varied between conditions, and protein intake groups had higher plasma AA levels
Haugvad[16]	8 males and 1 female (26 ± 4 years)	Low: 0.7 (0.6) & High: 1.2 (1.4) g/kg BW for male (female)	4 x 8 Squats, leg presses, and bilateral knee extensions	MVC, Power, Cortisol, Testosterone, SHBG, CK, and leukocyte at 12 and 24 h post-exercise	Significantly increased cortisol and reduced testosterone/cortisol ratio in the ALC group, but no effects on muscle function, serum CK or leukocyte count between conditions
Levitt[22]	10 males (25 ± 3 years) and 8 females (23 ± 2 years)	1.09 g/kg fat-free body mass	6 x 10 back squat (80 % 1-RM)	IFNγ, TNF-α, IL-1β, IL-6, IL-8, and IL-10 at 3 and 5 h	IL-6 production decreased in the ALC group at 3 h, IL-8 at 5 h post-exercise, no effects on IFNγ, TNF-α, IL-1β/6/10 after 5 h between conditions
Levitt[14]	13 females (21−34 years)	1.09 g/kg fat-free body mass	3 x 100 eccentric contractions of the quadriceps muscles	TNF-α, IL-1β/6/8/10, muscle soreness, CK, and peak torque at 5, 24 and 48 h post-exercise	No effects on torque production, CK or inflammatory capacity between conditions
McLeay[15]	8 females (23 ± 2 years)	0.88 g/kg BW	3 x 100 eccentric contractions of the quadriceps muscles	Peak torque, CK, and soreness at 36 and 60 h post-exercise	No effects on peak torque, CK and soreness between conditions
Duplanty[26]	10 males (21−29 years) and 9 females (21−26 years)	1.09 g/kg fat-free body mass	6 sets of heavy Smith machine squats (50−100 % 1-RM)	Muscle biopsy at 3 and 5 h post-exercise	Significantly attenuated phosphorylation of the mTORC1 signaling pathway post exercise in the ALC group
Levitt[17]	10 males (21−28 years)	1.09 g/kg fat-free body mass	4 x 10 eccentric squat (110 % concentric 1-RM)	Soreness, VJ Peak power, and peak force at 24 and 48 h post-exercise	No effects on soreness, VJ Peak power, and peak force between conditions

Table 2. A brief summary of short, medium and long-term effects.

Author	Short-term effects (<6 h post-exercise)	Medium-term effects (6−24 h post-exercise)	Long-term effects (>24 h post-exercise)
Barnes[10]	NA	NA	Decreased peak strength in the ALC group
Barnes[11]	NA	NA	Decreased torque for the ALC group
Barnes[12]	NA	NA	No effects on peak or averaged torque
Barnes[13]	NA	NA	Decreased VA and MVIC in the ALC group
Vingren[45]	Elevated serum TT, FT, and free androgen in the ALC group	NA	NA
Parr[6]	Decreased phosphorylation of mTOR in the ALC group	NA	NA
Haugvad[16]	NA	Increased cortisol and reduced testosterone/ cortisol ratio in the ALC group	NA
Levitt[22]	Decreased IL-6 and IL-8 production in the ALC group	NA	NA
Levitt[14]	No effects on torque production, CK or inflammatory capacity	No effects on torque production, CK or inflammatory capacity	No effects on torque production, CK or inflammatory capacity
McLeay[15]	NA	NA	No effects on peak torque, CK and soreness between conditions
Duplanty[26]	Attenuated phosphorylation of the mTORC1 signaling in the ALC group	NA	NA
Levitt[17]	NA	No effects on soreness, VJ peak power, and peak force between conditions	No effects on soreness, VJ peak power, and peak force between conditions

Abbreviations: AA: Amino acid; ALC: Alcohol consumption; BW: Body weight; CK: Creatinine kinase; FT: Free testosterone; IFNr: Interferon gamma; IL: Interleukin; iEMG: Integrated Electromyography; LEF: Low-frequency fatigue; mTOR: Mammalian target of rapamycin; mTORC1: Mammalian target of rapamycin complex 1; MVIC: Maximum voluntary isometric contraction; PPO: Peak power output; RPE: Rating of perceived exertion; SHBG: Sex hormone binding globulin; TNF-α: Tumor necrosis factor alpha; TT: Total testosterone; VA: Voluntary activation; VJ: Vertical jump.

for each leg before and after the exercise bouts. Both of the studies showed significant decreases in maximal torque regardless of drink condition, but there was no main effect of treatment (alcohol) compared to placebo. Compared with the studies conducted by Branes et al.,[10−13] implying that the effect of peak strength loss caused by alcohol ingestion may have a gender difference.

Haugvad et al.[16] conducted the study by recruiting 9 recreationally-trained participants (8 males and 1 female) for 4 resistance training sessions (the first was familiarization) and consumed a low (0.6 g/kg body mass for females and 0.7 g/kg body mass for males) or a high dose (1.2 or 1.4 g/kg body mass) of ethanol or alcohol-free drink 1−2.5 h after exercise. The exercise session includes 4 sets of 8 repetitions maximum of squats, leg presses, and knee extensions. MVC (knee extension) was recorded and showed immediately reduced strength after the exercise sessions despite drinking conditions. There were no consistent differences in the recovery of muscle function between conditions, and full recovery of MVC was found 24 h after ethanol consumption. The different results between this study and Barnes et al., 2010a,[10] Barnes et al., 2010b,[11] and Barnes et al., 2012[13] may be due to different resistance training programs, with Barnes et al. using 300 repetitions of contraction to produce a great amount of muscle damage, and Haugvad et al.[16] being the more realistic with the training program. We may conclude that the effect of alcohol on

delayed muscle recovery exists training may exist, but enough dose of ethanol and muscle damage are required to reveal the difference.

4. Power performance

Two studies measured power after resistance training followed by alcohol consumption, and no statistical significance results were shown. Haugvad et al.[16] analyzed the simple vertical squat jump performed on a force platform, and the jump height was recorded. Levitt et al., 2018[17] also included the vertical jump test, but with a countermovement beforehand. Both studies revealed a time effect for a reduction in vertical jump power after resistance training, but no differences between groups (alcohol consumption, alcohol-free consumption) were presented. The findings imply that alcohol ingestion may not worsen the performance of lower body muscles.

5. Delay-onset muscle soreness

Delayed Onset Muscle Soreness (DOMS) is a discomfort within the skeletal muscle following unaccustomed physical activity. DOMS is usually associated with unfamiliar, high-force muscular work and is precipitated by eccentric actions. The intensity of discomfort increases within the first 24 h following cessation of exercise, peaks between 24 and 72 h, subsidies, and eventually disappears by 5−7 days post-exercise. DOMS can affect athletic performance by causing a reduction in joint range of motion, shock attenuation, and peak torque.[18]

Four retrieved studies[10,14,15,17] included DOMS measurements, and none of them showed a significant increase in muscle soreness after resistance training followed by alcohol ingestion. The rating method included self-evaluating questionnaire,[17] applying pressure on the tested muscle[14] or conducting concentric targeted muscular contraction.[10,15] All of the studies showed an increase in muscle soreness after the session of resistance training, however, no significant differences were found between the alcohol-consumption group and the non-alcohol-consumption group. The results indicate that resistance training may be a factor that induces DOMS, but alcohol consumption may not be a strong contributing factor in muscle soreness.

6. Immune function

Both the innate and adaptive immune systems are activated after muscle injury. Immune cells are recruited orderly to the lesion after trauma, and are responsible for debris clearance and microenvironment modification by secreting various types of cytokines, growth factors and enzymes.[19] Pro-inflammatory cytokines, including TNF-α, IFNγ, IL-1β, IL-8 are responsible for promoting inflammation and macrophage activation, while anti-inflammatory cytokines like IL-10 attenuate inflammation.[20] Research has shown that interleukin 6 (IL-6) is both proinflammatory and anti-inflammatory, depending on the context.[21]

Two retrieved studies measured the concentration of circulating LPS-stimulated cytokines after resistance training with or without alcohol consumption and showed different results. Levitt et al., 2016[22] asked 18 recreationally trained males and females to finish 2 sessions of squats, and alcohol-containing drinks (with 1.09g ethanol/kg fat-free body mass) or non-alcohol beverages were administered after the exercise. Blood samples revealed increased IFNγ, TNF-α, and IL-1β 5 h post-exercise, and decreased IL-10 3 h and 5 h after exercise, regardless of drinking conditions. However, there were reduced productions of IL-6 at 3 h and 5 h in the ALC group after the exercise compared with placebo. Levitt et al., 2017[14] recruited 13 recreationally resistance-trained females and requested them to finish a session of 3 x 100 maximal single-leg eccentric leg extensions, followed by alcohol-containing drinks (with 1.09g ethanol/kg fat-free body mass) or non-alcohol beverages. The blood samples showed a significant main effect of time for LPS-stimulated concentrations of IL-10, IL-8, and TNF-α. Hence, IL-6 and IL-1β remained unchanged over time. No main effect was found for any of the cytokines investigated.

Both studies showed altered immune function after resistance exercise, although there was a difference in IL-6 level between conditions in Levitt's research,[14] alcohol seems to be a less contributing factor for immune modulation.

7. Creatinine kinase

Resistance exercise can result in localized damage to muscle tissue. Usually, the damage is accompanied by the release of enzymes such as creatinine kinase (CK) and lactate dehydrogenase, myoglobin, and other proteins into the blood.[23] Serum CK has been proposed as one of the best indirect indicators of muscle damage due to its ease of identification and the relatively low cost to quantify.[24]

Five Retrieved studies[10,13–16] included venous Creatinine Kinase (CK) measurement, and none of them revealed an alcohol-induced effect on serum CK concentration. All of the research showed increased CK after each resistance training session, whether with or without alcohol consumption. However, no significant differences were shown between the alcohol consumption group and the non-alcohol consumption group in any of the studies. The finding may imply that although resistance training may induce a certain amount of muscle damage, alcohol consumption may not be a modulating factor for venous CK concentration after resistance training.

8. Anabolic markers

Protein synthesis provides the basis for adaptations (for example, muscle growth, and recovery) to resistance training. Regulation of protein synthesis and subsequent muscle growth is complex and involves numerous factors. The mammalian target of rapamycin (mTOR), a protein Ser-Thr kinase that resides within a multiprotein complex called mTOR complex 1 (mTORC1), may be an essential pathway for regulating skeletal muscle protein synthesis. Bodine et al.[25] demonstrated the importance of mTORC1 signaling in resistance training by blocking mTOR using rapamycin, and reduced contraction-induced muscle growth in rats was found.

Two included studies measured the phosphorylation level of mTORC1, and both showed elevated phosphorylation of the mTORC1 signaling, but this effect was significantly attenuated by alcohol consumption in males. Parr et al.[6] asked 8 physically active males to complete resistance training (8 x 5 reps leg extension with 80 % 1 RM) followed by continuous (30min, 63 % PPO) and high intensity (10 × 30sec, 110 % PPO) cycling. Whey protein (PRO), alcohol co-ingest with protein (ALC-PRO), or alcohol co-ingest with carbohydrates (ALC-CHO) were administered randomly. Muscle biopsy was taken, and showed increased mTOR phosphorylation for all treatments (PRO, ALC-PRO, and ALC-CHO) compared with the pre-exercise condition, However, mTOR phosphorylation was significantly lower for the ALC group compared with the PRO group.

Duplanty et al.[26] recruited 19 resistance-trained males and females to complete 2 trials of resistance training (a set of 8–10 reps at 50 % 1 RM, followed by a set of 2–5 reps at 85 % and 4–5 sets of 1 rep at 100 % 1RM), followed by ingestion of either alcohol or placebo. Muscle tissue was obtained from vastus lateralis by biopsies. For the placebo, mTORc1 phosphorylation was significantly higher at 3 h after resistance training than pre-exercise. For male participants, mTOR phosphorylation was significantly lower for alcohol than for placebo, and these female participants didn't have the same effects. Those two studies indicated that alcohol may attenuate the anabolic process stimulated by resistance training, decreasing the benefit of muscle protein synthesis and hypertrophy, at least for males.

9. Testosterone and cortisol

Testosterone plays a crucial role in the production of muscle protein,[27] and it is essential for adapting to resistance training.[28,29] When heavy resistance exercise is performed, there is a temporary increase in the levels of both total testosterone (TT) and free testosterone (FT) in serum.[30,31] The correlation between testosterone levels and alcohol consumption may differ, as studies have indicated rises,[32–34] no connection[35–37] or reduced levels of testosterone[38,39] with alcohol consumption.

Cortisol is a stress hormone which plays a vital role in muscle catabolism,[40] and has important functions on lipids, proteins, and glucose regulation. Studies conducted on adults have shown that the level of cortisol in the body increases in relation to the intensity and duration of the activity.[41,42] And

most of the research also showed a positive relationship between alcohol consumption and cortisol levels.[43,44]

Two studies measured the effects of post-resistance training alcohol consumption on testosterone and cortisol level, and different results were found. Vingren et al.[45] showed significantly elevated serum TT, FT, Free androgen index and FT/cortisol ratio with alcohol ingestion compared with placebo during 140−300 min after the exercise, with no significant difference in cortisol levels were found. While in Haugvad et al.[16] study, compared with placebo, the FT/cortisol ratio was reduced after 12 h post-exercise followed by high dose ethanol ingestion, and the cortisol level also increased for the ethanol group.

There are two possible reasons for the differences between studies, the first is the time of measurement, Vingren's study measured serum sex hormone from the consumption post-exercise to 5 h afterward, while Haugvad's research chose to focus on the effects after 12 and 24 h post-exercise. So the results may be different interpretations of the same course. The second reason may be the concentration of ethanol, while Vingren's research used 1.09 g/kg (fat-free body mass) as the single concentration for all participants, Haugvad chose to administer low (0.6&0.7 g/kg body mass) and High (1.2&1.4 g/kg body mass) for male & female. The low dose alcohol consumption group had the similar trait of cortisol and testosterone level, but none of the results reached significance compared with the placebo. This may indicate that the results of Haugvad's study are dose-dependent, and may be the reason for the differences between the two studies.

10. Temporal effects

Five included studies discussed the short-term effects (<6 h post-exercise), both Parr et al.[6] and Duplanty et al.[26] revealed reduced mTOR signaling in the ALC group, and Vingren et al.[45] showed elevated serum testosterone and free androgen in the ALC group. While Levitt et al., 2016[22] reported decreased IL-6 and IL-8 production in the ALC group, Levitt et al., 2017[14] showed no differences in inflammatory capacity, torque or CK between conditions.

For the medium-term effects (6−24 h post-exercise), Levitt et al., 2017[14] revealed no alteration in torque, CK or inflammatory capacity, and Levitt et al., 2018[17] also reported no differences in soreness, peak power and force between conditions. While Haugvad et al.[16] showed increased cortisol and reduced testosterone/cortisol ratio in the ALC group.

Seven included researches demonstrated the long-term effects (>24 h post-exercise) of alcohol consumption following resistance training, while 3 studies revealed decreased strength or power,[10,11,13] the remaining articles reported no effects on torque production.[12,14,15,17] No differences in CK[14,15] or soreness[15,17] between conditions are shown.

In summary, while acute alcohol consumption can impact mTOR signaling and hormone levels, the medium and long-term effects on strength, power and other performance-related factors appear to vary, suggesting the effects may be complex and influenced by various factors.

11. Conclusion

In this review of 12 studies, alcohol consumption after resistance exercise had no effects on power and delay-onset muscle soreness, but may decrease strength performance in a dose-dependent manner, especially for males. Studies showed diverse results regarding immune function and metabolic hormones, with alcohol ingestion after resistance exercise possibly attenuating anabolic marker mTOR in males. Further studies with larger cohorts are necessary to better understand the effect of sex and the underlying mechanisms of alcohol and resistance training.

Conflicts of interest

Dr. Chueh-Hung Wu, an editorial board member at *Rehabilitation Practice and Science*, had no roles in the peer review process of or decision to publish this article. The other author declared no conflicts of interest in writing this paper.

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