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Effects of Alcohol Consumption on Muscle Soreness and Inflammation During Recovery From Strenuous Exercise

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Abstract

Although parties or get-togethers with alcoholic beverages after sporting competitions are popular, studies on the effects of alcohol ingestion after strenuous exercise on muscle damage and inflammation in non-drinkers' are few and ambiguous. Therefore, the aim of this study was to investigate the effects of alcohol ingestion during recovery from an acute bout of exercise on muscle soreness and inflammatory markers in regular exercisers who do not regularly consume alcohol. Male participants (n = 15) completed two bouts of exercise on a rowing ergometer for 2000 m in a randomized fashion. All participants ingested 5 mL of alcoholic (AL) or placebo (PL) beverage per kg of body weight within 10 min post-exercise. Blood samples for blood alcohol, creatine kinase (CK), C-reactive protein (CRP), and interleukin (IL)-6 concentrations were collected pre-exercise (T0), and at 1 (T1), 3 (T2), and 24 h (T3) post-ingestion. Self-reported muscle soreness was assessed at the same time points. Lactate levels were measured before exercise and within 1 h post-exercise. Muscle soreness was significantly lower in the AL than the PL trials at T3 ($p < 0.05$). Although CK, IL-6 and CRP levels were significantly higher during recovery than before exercising, there was no significant difference between the AL and PL trials. In addition, no significant difference in lactate concentrations between the two trials was evident in the 1 h after exercise. For regular exercisers, the alcoholic beverage ingested did not increase CK, IL-6, or CRP compared to their placebo trial, despite attenuated muscle soreness. Comparisons between drinkers and non-drinkers of high fitness ingesting permissible alcohol doses should be performed in the future.

Keywords: alcohol, inflammation, strenuous exercise, muscle damage

1. Introduction

Although the ingestion of alcoholic beverages may enhance post-consumption sports performance by reducing the sensation of muscle soreness or pain post-exercise (Williams, 1991), others have found that, instead, alcohol ingestion after exercise decreases vertical jump height, magnifies losses in dynamic and static strength, and decreases endurance performance (Barnes, Mündel, & Stannard, 2012; Lecoultre & Schutz, 2009). Moreover, consuming too much alcohol can lead to dysfunction in energy metabolism, an increased load on the cardiovascular system and muscle protein synthesis impairment after exercise (Heikkonen et al., 1998; Parr et al., 2014). Therefore, athletes are strictly advised to pay attention to or abstain from consuming alcohol during training or competition, although the American College of Sports Medicine (ACSM) has issued guidelines for certain alcohol dosages (American College of Sports Medicine, 1988).

However, a few studies have reported that alcoholic beverages are widely consumed during recovery periods after training or competitions, particularly during parties or get-togethers (Burke & Read, 1988; Dietez, Fitzgerald, & Jenkinson, 2008). Because strenuous exercise or training can lead to increases in indicators of muscle damage or inflammation (Mendham, Donges, Liberts, & Duffiel, 2011; Scharhag et al., 2005) and the results regarding the effect of alcohol ingestion on inflammatory markers during the recovery appear inconsistent (Barnes, Mündel, & Stannard, 2012; Murphy, Snape, Minett, Sekin, & Duffield, 2013), those that consume alcohol after exercise should be cautious. In the above-mentioned studies (Barnes et al., 2012; Murphy et al., 2013), all participants were resistance-training athletes or rugby players with a habit of regularly consuming alcoholic beverages. However, studies regarding the effect of alcoholic beverage ingestion after strenuous exercise on irregular social drinkers or non-drinkers are few. To compound the paucity of data on those that are not regular drinkers of alcohol, studies on the effect of acute alcohol ingestion during recovery on the markers of muscle damage or inflammation in irregular social drinkers or non-drinkers

that regularly exercise or have high fitness are less systematic (Lecoultre & Schutz, 2009; Raum, Gebhardt, Buchner, Schiltenwolf, & Brenner, 2007) compared with the existing literature on regular drinkers. Indeed, there was no measurement of inflammatory markers in Lecoultre et al.'s study (2009) and no exercise intervention in Raum et al.'s study (2007). Thus, the purpose of the current study was to investigate the effects of acute alcohol consumption during post-exercise recovery on muscle soreness, CK, CRP, and IL-6 in regular exercisers with no habit of alcohol consumption. This study aimed to examine whether there was any difference in those parameters due to alcohol consumption after an acute bout of exercise, as the variables are already known to increase after strenuous exercise (Mendham et al., 2011; Scharhag et al., 2005). The hypothesis was that muscle soreness might be lessened during recovery due to acute alcohol consumption, although in past research CK, IL-6, and CRP did not show significant differences between the alcohol and non-alcohol trials during the recovery.

2. Methods

Participants were recreationally active individuals who regularly engaged in exercisers. The average age, body weight, BMI, and VO_2max of all participants were 24.7 ± 7.3 yrs, 76.2 ± 3.1 kg, 22.6 ± 1.4 kg/m^2, and 53.7 ± 3.2 mL/min/kg, respectively. The alcohol ingestion of all participants was assessed by a daily drinking questionnaire (DDQ), which measured the number of standard drinks ingested by individuals each day in a typical week during the past month (Collins, Parks, & Marlatt, 1985). Eligible participants were included if they matched the following conditions: no or little alcohol consumption (< 350 mL of beer, < 100 mL of red wine, or < 40 mL of whisky) in a month and having no allergies to alcohol (participant data was not included if they were allergic to, or had any adverse reaction to alcohol ingestion during the study). In addition, participants underwent a general medical check-up to ensure that they were free of diseases such as hypertension, diabetes, and hyperlipidemia. We calculated the statistical power of the numbers of subjects based on the changes in CRP values between baseline and at 24 h post-exercise, as in the study by Plaisance et al. (2007). Power analysis software G*Power 3.0 (Franz Faul, Kiel University, Germany) indicated that a total of 14 participants would provide 90% power at the 0.05 level of significance to detect a difference of 5% in CRP given a crossover design. All participants provided written informed consent after they understood the goals, procedures, and potential risks of this research. This study was approved by the institutional review board of Kaohsiung Medical University.

2.1 Overview of the experiment

Prior to determining maximal oxygen capacity, and at each exercise session, it was important to confirm that participants were free of trauma, upper respiratory tract infections (URTIs), and the common cold (e.g., having symptoms such as a cough, runny nose, or nasal congestion), as these conditions could influence the parameters being measured. After 10 h of fasting, participants reported to the laboratory where a blood sample (T0) was collected. Participants were then provided a standard breakfast (a sandwich and soy milk with a total caloric content of 335 kcal: 55% carbohydrates, 20% fat, and 25% protein). One hour after the meal, participants warmed up at a self-determined pace at <150 W for 2-3 min before the main test: a 2000-m session on the rowing ergometer performed as quickly as possible. This strenuous exercise model was similar to the experimental design in Skarpanska-Stejnborn et al.'s study (2011), which examined the effect of plant extract supplementation on inflammation and oxidative stress after a 2000-m rowing-ergometer test.

Each participant consumed either a single alcoholic beverage (Taiwan Beer, 4.5% alcohol content, Taiwan, AL) or a placebo beverage (plain water, PL) at a dose of 5 mL/kg body weight within 10 min of completion of this trial, which was modified from the study by Tousoulis et al. (2008). The alcoholic and placebo beverages were provided in an opaque container after their volume had been determined and participants wore a nose clip when they drank the beverages. The goal of these procedures was to reduce the differences between the two beverages, with respect to their colorings and smells between the two beverages. The two experimental trials were performed in a randomized order generated by a computerized random number generator and separated by at least 7 days. Blood samples were collected at 1 (T1) and 3 h (T2) post-ingestion and the next day after overnight fasting (T3). An overnight urine sample was also collected in addition to a blood sample at T3 to determine hydration status. All participants showed euhydration, corresponding to a urine specific gravity of <1.020 g/mL.

2.2 $\dot{V}O_2$max measurement

Maximal aerobic capacity ($\dot{V}O_2$max) was determined after one night of fasting. Each participant sat quietly on a wind resistance-braked rowing ergometer (Concept II, Morrisville, VT, USA) for 1 min before rowing at 100 W. The load was increased by 30 W every 2 min until maximal voluntary exhaustion was reached. Gas concentrations were measured and analyzed with a SensorMed VU MAX29 (Yorba Linda, CA, USA). The value of VO_2max was accepted if at least two of the following criteria were met: 1) a respiratory quotient (RQ) >1.1 with a plateau VO_2 reading despite an increase in exercise intensity, 2) a heart rate within 10 bpm of the age-predicted maximum (Vantage XL Polar System, Port Washington, NY), and 3) a rating of perceived exertion (RPE) of greater than 18 on the Borg scale (Borg, 1982). All instruments were calibrated in accordance with the manufacturers' manuals.

2.3 Dietary and physical activity records

Aside from the 10 min post-exercise period, participants were reminded not to ingest any alcoholic beverages from 24 h before the study until after collection of the final blood sample. In order to standardize the nutritional status of the sample group, a dietician instructed participants to record their food intake, based on their categories and portions, with photos, over a two-day period. Food calories and compositions were analyzed using software (DietOrganizer 2.2; MulberrySoft, USA). In addition, two-day physical activity logs for all participants were recorded for each protocol. For the second session, subjects were asked to follow the physical activity logs from the previous session and to avoid physical activity beyond a moderate intensity.

2.4 Measurement of muscle soreness

Muscle soreness in the arms (biceps and triceps) and legs (quadriceps) was determined using a self-reported muscle-pain intensity scale (Cook, O'Connor, Eubanks, Smith, & Lee, 1997). This scale ranged from 0 (no pain at all) to 10 (extremely intense pain, almost unbearable) and the same researcher was responsible for the palpation of all muscle groups. The points of the upper and lower muscle groups were averaged for statistical analyses. Muscle soreness was assessed at the same time points as venous blood collection.

2.5 Lactate measurement

Fingertip blood samples were collected for lactic acid analysis prior to and immediately after exercise, and at 15, 30, 45 and 60 min post-exercise. Before taking the fingertip capillary blood sample, participants were asked to immerse one hand in warm water at ~40°C for 30 s. The hand was dried and one finger pierced with a sterile lancet (1.5 mm). The first drop of blood was sampled and ~10 μL was used to determine lactate concentrations with the Lactate Pro analyzer (Arkray, Japan).

2.6 Blood analysis

Venous blood samples were collected at four time points: prior to exercise (T0) and at 1 (T1), 3 (T2), and 24 h (T3) post-alcohol ingestion to determine the alcohol, CK (Dinabott, Tokyo, Japan), CRP (Immulite 2000, Diagnostic Products, Los Angeles, CA, USA), and IL-6 (R&D Systems, Minneapolis, MN, USA) concentrations. For each participant, a 7 mL blood sample was collected at each time point. Blood was allowed to clot at room temperature and then centrifuged at 3000 rpm for 10 min. Blood alcohol levels were analyzed using an enzymatic colorimetric assay. CK concentrations were determined spectrophotometrically (VP-Super, Dinabott, Tokyo, Japan), and CRP and IL-6 levels were analyzed using enzyme-linked immunosorbent assays (ELISAs). All serum variables were measured in duplicate. The intra-assay coefficients of variation for CK, CRP, and IL-6 were 3.1%, 3.9%, and 5.2%, respectively.

2.7 Data analysis

All values are expressed as the mean ± standard deviation (SD). The normality of the data was tested using the Kolmogorov-Smirnov test. If the data failed to meet the assumptions of normal distribution, the data were log-transformed. All parameters were analyzed using a two-factor (trial × time) repeated-measures analysis of variance (ANOVA). Tukey's *post-hoc* test was applied in the event of a significant *F* ratio. The Greenhouse-Geisser epsilon correction was used to adjust the significance level of the test statistics for violations of assumed sphericity. In addition, a paired Student's *t*-test was used to compare pre- and post-exercise values for a single exercise session at a given trial. Relationships among variables in each trial were analyzed using Pearson's product-moment correlation coefficients. The SPSS statistical package (version. 15.0, Chicago, IL, USA) was used for all statistical procedures. For all statistical comparisons, the threshold was set to $\alpha < 0.05$.

3. Results

All participants were categorized as consuming little or no little alcohol according to the results of the DDQ. For the 2000-m ergometer exercise test, the AL and PL trials yielded completion times of 8.0 ± 0.9 and 7.9 ± 1.0 min, respectively, with no significant difference observed between trials; RPE differences were not significant (17.4 ± 1.5 vs. 17.6 ± 1.6, $p > 0.05$), either. Before exercise, there were no significant differences for muscle soreness sore or lactate, CK, CRP, or IL-6 levels between the two trials. All participants were able to finish the alcoholic or placebo beverage (319 ± 23.5 mL) within 10 min without any side effects. After inference using the calculations of Dill and Costill (1974), no significant differences were evident in blood plasma concentrations at the three post-ingestion time points compared with T0. The blood alcohol levels in the AL trial were 13.7 ± 2.4 and 8.9 ± 1.7 mg/dL at T1 and T2, respectively, and were undetectable in the blood at T3. Blood alcohol was undetected at T1, T2, or T3 in the PL trial.

Figure 1 displays the changes in lactate observed during the 1 h recovery post-exercise. A time-dependent effect was found and lactate levels were significantly higher immediately post-exercise and at 15, 30, 45, and 60 min post-exercise compared to their pre-exercise values ($p < 0.05$). However, no trial or interaction effect was evident between the AL and PL trials.

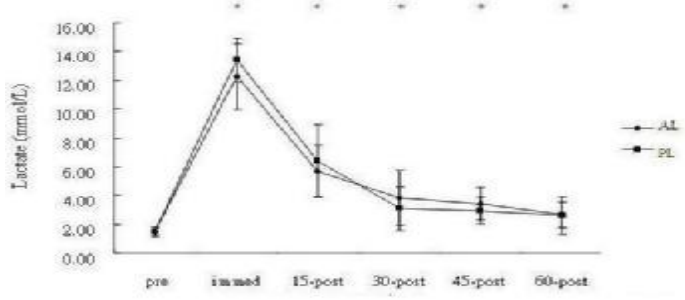

Figure 1 Changes in lactate levels from pre-exercise to 60 min post-exercise for the alcohol (AL) and placebo (PL) trials
* significantly different from the pre-exercise value

For muscle soreness, there was a significant interaction effect between the two trials, and a main effect of time was evident after the ergometer exercise. Although the participants' muscle soreness score was significantly higher at T1, T2 and T3 compared to their score at T0 in both of the two trials, the AL trial had significantly lower muscle soreness than the PL trial at T3 (AL: 6.1 ± 0.7, PL: 7.1 ± 1.1, $p < 0.05$, Fig. 2).

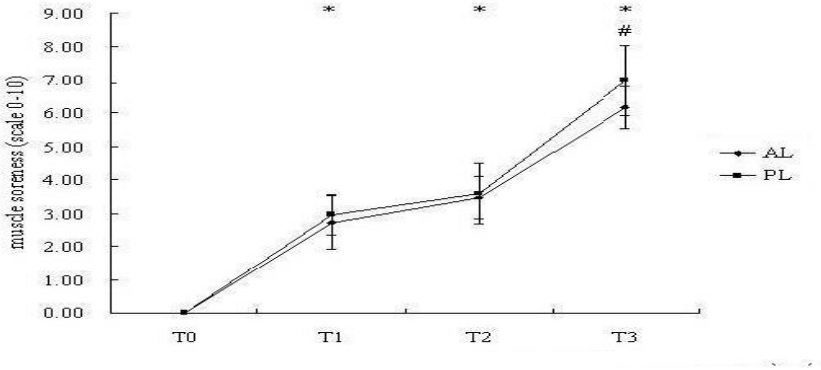

Figure 2 Scores of muscle soreness at T0, T1, T2 and T3 for the alcohol (AL) and placebo (PL) trials
* significantly different from the pre-exercise value
# significantly different from the value of the PL trial at T3
T0: prior to exercise; T1, T2, and T3: 1, 3, and 24 h post-ingestion, respectively

Table 1 shows the CK and IL-6 levels from pre-exercise to 24 h post-exercise in the AL and PL trials. CK concentrations after strenuous exercise showed a main effect of time after a 2000-m ergometer exercise test exercise. Although CK levels were significantly higher at T1, T2, and T3 than at T0 in both trials (all $p < 0.05$), neither a trial or interaction effect were found between the two trials. On the other hand, IL-6 concentrations peaked at T2 after strenuous exercise and were significantly higher at T1, T2, and T3 compared with the pre-exercise value in both trials (all $p < 0.05$); however, neither a main effect of trial or an interaction effect were found between the two trials.

Table 1 Creatine kinase (CK) and interleukin (IL)-6 concentrations at T0, T1, T2, and T3 for alcohol (AL) and placebo (PL) trials

	T0	T1	T2	T3
CK (U/L)				
AL	175.9 ± 8.2	872.0 ± 82.1*	1022.7 ± 60.9*	1664.7 ± 112.8*
PL	169.7 ± 9.0	831.7 ± 86.5*	995.3 ± 69.6*	1739.7 ± 111.5*
IL-6 (pg/mL)				
AL	1.1 ± 0.2	5.0 ± 0.3*	7.3 ± 0.5*	3.1 ± 0.4*
PL	1.1 ± 0.2	4.9 ± 0.3*	6.8 ± 0.7*	2.8 ± 0.5*

* Significantly different from T0.

T0: prior to exercise; T1, T2, and T3: 1, 3, and 24 h post-ingestion, respectively.

CRP levels displayed no interaction post-exercise, either. CRP levels gradually increased from T1 to T3 and were significantly higher at T2 and T3 than at T0 and T1 in both trials ($p < 0.05$) (Fig. 3). Although the highest levels of CRP were found at T3 in both trials and there was a tendency for it to be higher in the AL than in the PL trials at T3 (AL: 0.62 ± 0.08, PL: 0.55 ± 0.10 mg/L, $p = 0.06$), no significantly difference was observed.

Correlations

Associations among the serum variables in each trial were analyzed using Pearson's product-moment correlation coefficients. There were no significant associations among the parameters in the AL or PL sessions at any time point, with the exception of CRP and IL-6 levels, which showed a significant correlation at T3 in the AL trail ($r = 0.55$, $p < 0.05$).

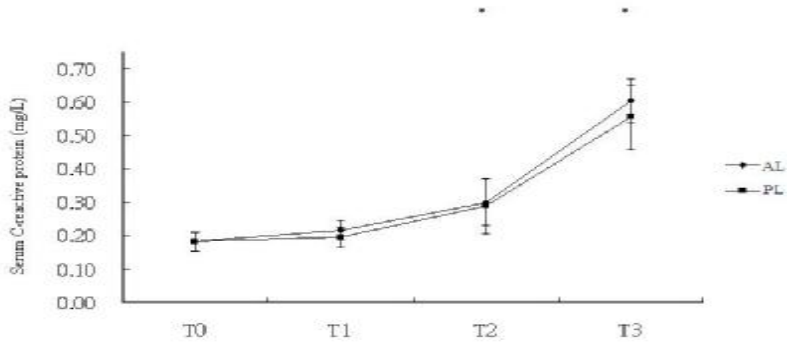

Figure 3 C-reactive protein (CRP) levels at T0, T1, T2 and T3 for the alcohol (AL) and placebo (PL) trials

* significantly different from the pre-exercise value

T0: prior to exercise; T1, T2, and T3: 1, 3, and 24 h post-ingestion, respectively

4. Discussion

Few studies have reported the effect of alcoholic beverage ingestion during recovery on non-drinkers with regular exercise or highly fitness. Our current study demonstrated that alcohol ingestion after exercise did not result in significant differences in CK, IL-6, or CRP up to 24 h post-exercise, or in lactate response up to 1 h post-exercise. However, acute alcohol ingestion may attenuate muscle soreness at 24 h post-ingestion (T3).

The lack of significant difference in RPE or the completion time of the 2000-m ergometer session between the two trials indicated that the physiological stress from the exercise was similar. On the other hand, although the lactate responses were significantly higher during the one-hour recovery after acute exercise than before exercise in both the AL and PL trials, no significant differences between the two trials were found at any time point. These results are in line with those of Lecoultre and Schutz (2009). We also showed that the alcohol dose used in this study did not induce a significant difference in lactate metabolism or clearance compared with the PL trial.

In regards to muscle soreness, although William et al. have reported that alcohol ingestion could improve subsequent sports performance by reducing pain and anxiety (Williams, 1991), results in several other studies (Barnes et al., 2012; Murphy et al., 2013) differ. Our results are similar to several studies that have suggested that alcohol use could reduce pain sensitivity and muscle soreness (Murphy et al., 2013; Woodrow & Eltherington, 1988): the muscle soreness scores for the AL trial were significantly lower than those for the PL trial at T3. However, this result differed from a study by Barnes et al. (2012), which reported no significant difference in muscle soreness score regardless of alcohol ingestion. Possible reasons for this discrepancy include the characteristics of the participants: in their study, participants were known to not be naive to alcohol, but no any further information was reported, such as their drinking habits or typical alcoholic consumption in one month. Several researchers have also suggested that individual drinking history, preference for alcoholic over non-alcoholic beverages, and a family history of alcoholism should be considered in addition to drinking habits when assessing the effect of ethanol on pain responses or analgesia (Perrino et al., 2008). We hypothesized that participants in the current study were likely to exhibit decreased sensitivity or elevated tolerance to muscle soreness due to the analgesic effects of alcohol ingestion. It is notable that the rugby players who were binge drinkers in Murphy et al.'s study (2013) differ from the non-drinkers in our study, yet alcohol ingestion during recovery ameliorated muscle soreness in both population. There are unfortunately limited data available with respect to abstainers and non-drinkers, or comparisons between non-drinkers and habitual drinkers within the athlete cohort. The lack, coupled with the findings of the present study and how it compares with the literature, strongly suggest that rigorous screening of participants' characteristics based on their alcohol habits and history should be considered in future studies.

In addition to assessing muscle soreness, we evaluated muscle damage using CK concentrations. In this study, CK levels increased after strenuous exercise and reached a peak at 24 h post-exercise in both trials, signaling that muscle damage occurred in each trial. However, CK levels did not differ between the trials. In addition, IL-6 levels displayed no significant differences between the trials in this study, although both trials showed an elevation in IL-6 levels after acute exercise. The latter result echoes similar findings in the literature (Arent, Senso, Golem, & McKeever, 2010); however, the former is a novel finding. Although one study (Tousoulis et al., 2008) has reported no significant differences in IL-6 levels after acute alcohol ingestion compared with placebo (water) trials, and its blood sampling times were comparable to our study (at baseline without alcohol ingestion and at 4 h post-ingestion vs. pre- and 1, 3, and 24 h post-ingestion), what makes our study clearly novel is the inclusion of an exercise intervention. Our results

indicate that IL-6 levels did not significantly differ due to alcohol ingestion during recovery from an acute bout of exercise compared with the same exercise session without alcohol consumption. Therefore, the dose of alcohol (0.23 g/kg body weight) after strenuous exercise ingested in this study might not increase CK or IL-6 levels.

The CRP responses in the two trials were in agreement with previous studies, which indicated that strenuous exercise could lead to an increase in CRP levels (Mendham et al., 2011; Scharhag et al., 2005). However, our results contrast with those Raum et al.'s study, which found no significant change in CRP from pre-alcohol ingestion to the period post-alcohol ingestion (Raum et al., 2007). The fact that no acute strenuous exercise was performed in Raum et al.'s study can account for this difference. The other goal of our study was to explore whether CRP levels differed due to alcohol consumption during the recovery after strenuous exercise in non-drinkers with regular exercise. The results and trends indicating increased CK and CRP without significant differences between the alcohol and non-alcohol trials resembled those from Murphy et al. (2013). Based on these findings, we conclude that the present dose of alcohol during recovery may have a small, but negligible, effect on the markers of muscle damage and systemic inflammation in regular exercisers who are non-drinkers.

We further examined whether CRP was related to other markers of muscle damage. IL-6 and other factors, such as nuclear factor (NF)-κB and fibrinogen, have been analyzed for their association with CRP changes in studies on long-term alcohol ingestion (Blanco-Colio et al., 2000) and acute bouts of exercise (Scharhag et al., 2005; Shojaei, Farajov, & Jafari, 2011). In spite of a significant relationship between IL-6 and CRP at T3 in the AL trial, our result did not support the notion of alcohol-causation, as CK and IL-6 during the recovery did not significantly differ between the two trials. However, the relationship between IL-6 and other markers of muscle damage, such as myoglobin, is a rational direction in which to address the speculation of alcohol effects on CRP metabolism.

4.1 Limitations

The findings, under alcohol dose condition in the current study, are in line with the conclusion in a review article by Bare (2014), who suggested that a dose of approximately 0.5 g/kg body weight (0.23 g/kg body weight in the present study) post exercise was unlikely to have negative influence on the markers we examined here. However, one limitation should be mentioned. The volume of alcohol in the current study was lower than what is used in the existing literature, and so caution should be taken when applying our findings to related studies.

5. Conclusion

In this study, muscle soreness after strenuous exercise may be attenuated by ingestion of alcoholic beverages. Further studies are warranted based on the allowable amount of alcohol described by the ACSM guidelines or greater alcohol consumption depending on participant characteristics, especially for comparisons of muscle damage and inflammation between non-drinkers (abstainers) and habitual drinkers.

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