Eur J Appl Physiol (2010) 108:1009–1014
DOI 10.1007/s00421-009-1311-3

ORIGINAL ARTICLE

Post-exercise alcohol ingestion exacerbates eccentric-exercise induced losses in performance

Matthew J. Barnes · Toby Mündel · Stephen R. Stannard

Accepted: 23 November 2009 / Published online: 11 December 2009

Abstract The effect of acute alcohol intake on muscular performance in both the exercising and non-exercising legs in the days following strenuous eccentric exercise was investigated to ascertain whether an interaction between post-exercise alcohol use and muscle damage causes an increase in damage-related weakness. Ten healthy males performed 300 maximal eccentric contractions of the quadriceps muscles of one leg on an isokinetic dynamometer. They then consumed either a beverage containing 1 g of ethanol per kg bodyweight ethanol (as vodka and orange juice; ALC) or a non-alcoholic beverage (OJ). At least 2 weeks later they performed an equivalent bout of eccentric exercise on the contralateral leg after which they consumed the other beverage. Measurement of peak and average peak isokinetic (concentric and eccentric) and isometric torque produced by the quadriceps of both exercising and non-exercising legs was made before and 36 and 60 h post-exercise. Greatest decreases in exercising leg performance were observed at 36 h with losses of 28.7, 31.9 and 25.9% occurring for OJ average peak isometric, concentric, and eccentric torques, respectively. However, average peak torque loss was significantly greater in ALC with the same performance measures decreasing by 40.9, 42.8 and 44.8% (all $p < 0.05$). Performance of the non-exercising leg did not change significantly under either treatment. Therefore, consumption of moderate amounts of alcohol after damaging exercise magnifies the loss of force associated with strenuous eccentric exercise. This weakness appears to be due to an interaction between muscle damage and alcohol rather than the systemic effects of acute alcohol consumption.

Keywords Ethanol · Muscle strength · Soft tissue injuries

Communicated by William Kraemer.

M. J. Barnes (✉) · T. Mündel · S. R. Stannard
Institute of Food, Nutrition, and Human Health,
Massey University, Private Bag 11-222,
Palmerston North, New Zealand
e-mail: M.Barnes@massey.ac.nz

Introduction

The mechanisms and consequences of exercise-induced muscle damage (EIMD) have received considerable scientific attention over the past 20 years. Strenuous eccentric muscle action is now known to cause micro-structural damage resulting in delayed onset muscle soreness (DOMS), inflammation and more importantly, impaired muscle function which typically lasts for a number of days, depending on the severity of the damage (Cleak and Eston, 1992; Proske and Morgan, 2001). Over this same period EIMD has been employed as a model of soft tissue injury where a number of modalities aimed at improving the rate of recovery (e.g. cold water immersion therapy (Eston and Peters 1999), non-steroidal anti-inflammatory medication (Gulick et al. 1996), massage (Jönhagen et al. 2004) and compression therapy (Kraemer et al. 2001)) have been tested. This research has provided mixed and often inconclusive results. Surprisingly, compared to these and other recovery modalities, less attention has been afforded to post-exercise behaviours that may simultaneously impair the recovery process after EIMD.

One such behaviour is post-exercise alcohol use. While the consumption of large amounts of alcohol by sportspeople, often after competition or training, is common place (Nelson and Wechsler 2001; Snow and Munro 2006; O'Brien et al. 2007) little is known about how this behaviour effects recovery and subsequent performance in

the days after exercise. We have recently shown that moderate amounts of alcohol (1 g per kg of body weight) consumed immediately after strenuous eccentric exercise exacerbates the weakness normally seen in the days following such exercise (Barnes et al. 2009). This finding suggests that athletes competing in sports involving strenuous eccentric muscular work or those suffering from soft tissue injury should avoid alcohol consumption, at least immediately after exercise or injury, if rapid recovery is important. However, the design of this previous study did not allow us to ascertain whether this alcohol-induced weakness was due to delayed recovery from strenuous eccentric exercise or whether alcohol alters the ability to recruit and create tension in any muscle group in the 60-h period after drinking.

Previous research (Poulsen et al. 2007) suggests that the acute consumption of clinically relevant levels of alcohol does not affect muscular performance in the days following a drinking session. Further, indices of EIMD are unaffected when alcohol is acutely consumed prior to a bout of damaging exercise (Clarkson and Reichsman 1990). The results of these studies suggest that our previous findings are due to the alcohol affecting the recovery processes in already damaged muscle, not systemically weakening all skeletal muscles.

The purpose of this study, therefore, was to investigate whether alcohol interacts with damaged skeletal muscle to magnify the typical weakness associated with EIMD or whether our previous findings are due to the systemic effects of acute alcohol consumption, independent of EIMD.

Methods

Overview

The current study utilised a modified version of the protocol described by Barnes et al. (2009). Adapted from the work of MacIntyre et al. (1996), this exercise protocol has previously been shown to bring about significant levels of muscle damage as characterised by decreased muscular performance, DOMS and elevations in circulating creatine kinase concentrations (MacIntyre et al. 1996; Barnes et al. 2009). Briefly, subjects performed 300 maximal eccentric contractions of the quadriceps muscles of one leg on an isokinetic dynamometer. They then consumed either an alcoholic beverage or a non-alcoholic control beverage. At least 2 weeks later they performed an equivalent bout of eccentric exercise on the contralateral leg after which they consumed the other beverage. Muscular performance of the damaged (exercising) and non-damaged (non-exercising) legs was measured prior to the damaging exercise bout and at 36 and 60 h post-exercise.

Subjects

Twelve males volunteered to participate in this study. However, due to an obvious learning effect the results of two subjects were excluded from statistical analysis, as their strength was higher than pre-exercise values in the days after the damaging protocol. Analysis was therefore carried out on the results of 10 subjects (age 23.5 ± 5.1 years, body mass 76.9 ± 12.9 kg). All subjects were healthy and had at least 2 years of resistance training experience at a recreational level (minimum twice per week). The protocol was approved by the Massey University Human Ethics Committee and written consent was obtained from each subject.

Familiarisation of the protocol was carried out at least 1 week before the first trial. Subjects were instructed to abstain from alcohol consumption and any form of exercise from 48 h before and until 60 h after the exercise bout. Subjects were also instructed to abstain from practices that could potentially improve or delay their recovery during the 60 h post-exercise period. Subject's diets were replicated between trials by way of a food diary that was filled out from the morning of the first trial until the last measurements were taken at 60 h post-exercise. Utilising a single cross-over design, treatment and leg were randomly allocated in a counter-balanced fashion. This was done to account for ordering and (leg) dominance effects.

Muscular performance

Four hours after consuming a standardised, solid meal (3765 kJ; CHO 133 g, Fat 28 g, Protein 25 g) subjects returned to the laboratory in the evening and warmed up on a cycle ergometer (Monark, Varberg, Sweden) for five minutes at 100 W. They were then seated on a Biodex® isokinetic dynamometer (Biodex Medical Systems, New York, USA) and straps were fixed across the chest, hips and active leg to isolate movement to the quadriceps. Knee joint range of motion was set and recorded for use in subsequent follow-up tests. Subjects then performed separate sets of five maximal isometric, concentric and eccentric contractions of the quadriceps muscles of both the non-exercising and the exercising legs. Each set was separated by 2 min of passive recovery. Isometric tension was measured at a knee angle of 75° (1.31 rad). Concentric and eccentric torques were measured at an angular velocity of $30°s^{-1}$ ($0.52\ rad\ s^{-1}$). Absolute peak torque and average peak torque over five contractions were recorded. Pre-exercise muscular performance measures were made in the early evening with subsequent follow-up measurements made 36 and 60 h post-exercise. The time of day at which subjects completed these measurements was standardised between trials. Although time of day was different between

measurements the lack of change in the performance of the non-exercising leg illustrates that changes in circadian rhythms did not affect force development capabilities in the current study.

Exercise protocol

Subjects remained on the Biodex and completed 3 sets of 100 maximal eccentric contractions, over a 60° (1.05 rad) range of motion at an angular velocity of $30°s^{-1}$, using the quadriceps muscles of one leg. Each set was separated by 5 min of passive recovery. Subjects were verbally encouraged to resist the downward action of the dynamometer arm as hard as possible and had access to visual feedback of their torque throughout the protocol to ensure continuous maximal effort. Total work completed during the eccentric exercise bouts was not significantly different between trial one (34.9 ± 11.5 kJ) and trial two (36.7 ± 11.1 kJ) ($p = 0.14$) or between control (OJ) (35.2 ± 11.5 kJ) and alcohol (ALC) trials (36.5 ± 11.5 kJ) ($p = 0.29$).

Treatment

Thirty minutes after completion of the exercise bout, and having consumed a standardised meal immediately after exercise (1532 kJ; CHO 50.4 g, fat 9.1 g, protein 7 g), subjects began drinking a beverage containing either 1 g of alcohol per kg of body weight as vodka (37.5% alcohol/volume; Smirnoff, Australia) in orange juice (Frucor Beverages, New Zealand) (ALC) or a control beverage of orange juice alone (OJ). Equivalent to 8 ± 2.8 standard drinks, the mean volume of vodka consumed per subject was 211.9 ± 51.4 ml. In order to balance total energy value (2794.5 ± 476.1 kJ) and fluid volume (1638 ± 268 ml) between trials, subjects consumed a greater volume of orange juice in the OJ trial while in the ALC trial they consumed an additional volume of water (751 ± 128 ml) along with the alcoholic beverage. An equal volume of beverage was consumed every 15 min over a total time of 90 min. Once the required amount of beverage was consumed participants were driven home and instructed to go directly to bed.

For the second trial, the contralateral leg was exercised and the other beverage was consumed using the same protocol, as outlined above.

Statistical analysis

Data were analysed using the Statistical Program for Social Sciences (SPSS) for Windows (version 15.0, SPSS Inc., Chicago, IL). A general linear-model three-way repeated-measures ANOVA (treatment × time × leg) was used to compare conditions over time for each performance measure. This analysis provided main effects of time, treatment and leg; thus treatment × time, treatment × leg, time × leg and treatment × time × leg interactions were also investigated. If conditions differed significantly, post hoc pairwise comparisons using Bonferroni adjustment were performed to identify the differences between time points within each treatment and leg. As no significant change was seen in muscular performance of the non-exercising leg (see Results section for details) and to allow comparison between the results of the current study and our previous findings (Barnes et al. 2009), exercising leg data was analysed separately with two-way repeated-measures ANOVA providing additional treatment × time interactions. As different legs were used for each trial, resulting in significantly different pre-exercise values between treatments, data was analysed as absolute change in torque relative to pre-exercise values. Reported values are means ± standard deviation (SD). Statistical significance was set at the 95% level of confidence ($P < 0.05$).

Results

Completion of 300 eccentric muscular contractions of the quadriceps resulted in significant decreases in isometric, concentric and eccentric peak and average peak torque over time in the exercising leg only (all $P < 0.001$, Table 1). No significant change in non-exercising leg performance was observed at any time point under either treatment (all $P > 0.2$). Significant Treatment × Time × Leg interactions were found for isometric ($P = 0.036$) and eccentric ($P = 0.02$) peak torques as well as for isometric ($P = 0.032$), concentric ($P = 0.032$) and eccentric ($P = 0.023$) average peak torques.

Analysis of the exercising leg data, independent of the leg variable, found significant changes over time for all performance measures (all $P < 0.001$). Significant treatment effects (all $P < 0.02$) and treatment × time interactions (all $P < 0.05$) were seen for all performance measures except peak concentric torque ($P = 0.16$ and 0.42, respectively). Greatest decreases in performance were seen with ALC at 36 h while no significant change in performance was observed between 36 and 60 h under either treatment.

Repeated-measures ANOVA of trial one versus trial two found no significant order effect for any of the muscular performance measures (all $P > 0.15$).

Discussion

The aim of the present study was to investigate whether the systemic effects of alcohol bring about muscular weakness

Table 1 Absolute changes in torque (N-m) following strenuous eccentric exercise

	Exercising leg			Non-exercising leg		
	Pre	36 h	60 h	Pre	36 h	60 h
Peak ISO						
OJ	275.8 ± 48.9	−78.8 ± 38.7*	−72.9 ± 48.7*	286.0 ± 51.2	−3.4 ± 16.0	−0.1 ± 19.6
ALC	295.6 ± 49.8†	−113.7 ± 45.3*†	−108.9 ± 84.4*	289.6 ± 62.6	−10.0 ± 21.8	−3.0 ± 42.7
Peak CON						
OJ	227.8 ± 50.3	−71.5 ± 32.4*	−57.9 ± 22.8*	255.9 ± 69.4	−11.5 ± 26.0	−12.1 ± 23.2
ALC	240.9 ± 50.4	−88.3 ± 44.4*	−78.2 ± 64.6*	238.3 ± 45.3	−11.4 ± 24.2	−3.5 ± 37.7
Peak ECC						
OJ	284.9 ± 77.3	−77.1 ± 59.3*	−63.5 ± 51.9*	311.0 ± 81.7	−0.3 ± 32.2	4.9 ± 37.8
ALC	345.6 ± 81.7‡	−150.2 ± 58.8*‡	−133.2 ± 88.2*†	335.6 ± 79.0	−22.6 ± 47.7	−33.0 ± 73.1
Average ISO						
OJ	256.9 ± 39.0	−73.8 ± 41.5*	−72.0 ± 55.7*	271.3 ± 40.1	−7.8 ± 18.7	−1.5 ± 22.1
ALC	281.1 ± 37.7†	−115.1 ± 50.3*†	−111.7 ± 86.8*†	271.3 ± 54.1	−8.9 ± 15.3	−3.7 ± 43.7
Average CON						
OJ	205.1 ± 31.2	−65.6 ± 32.7*	−55.3 ± 32.7*	230.7 ± 50.2	−10.0 ± 19.7	−9.9 ± 32.8
ALC	226.8 ± 37.9†	−97.0 ± 47.7*†	−91.0 ± 64.0*†	222.7 ± 36.2	−11.6 ± 16.8	−9.4 ± 40.5
Average ECC						
OJ	266.9 ± 76.9	−69.1 ± 93.6$^{\#}$	−63.6 ± 47.4*	291.3 ± 84.3	2.6 ± 33.7	6.4 ± 41.5
ALC	320.4 ± 70.1†	−143.5 ± 68.6*†	−127.8 ± 91.6*†	306.3 ± 75.9	−8.6 ± 37.1	−23.8 ± 60.1

ISO isometric, *CON* concentric, *ECC* eccentric torque (N-m), *OJ* control, *ALC* alcohol treatment

Data presented as mean ± SD

Significant difference from pre-exercise value: $^{*}P < 0.01$, $^{\#}P < 0.05$

Significant difference between trials: $^{\ddagger}P < 0.01$, $^{\dagger}P < 0.05$

No significant difference between 36 and 60 h values under either treatment

in the days following alcohol consumption after strenuous eccentric exercise or whether alcohol interacts with EIMD to exacerbate the loss of muscular performance, as previously observed (Barnes et al. 2009).

Completion of 300 maximal eccentric contractions of the quadriceps resulted in significant decreases in all performance measures in the exercising leg only (Table 1). In accordance with the results of Poulsen et al. (2007), our data confirm that a moderate dose of alcohol has no affect on muscular performance in the days following a drinking episode provided the muscle has not been damaged as a result of strenuous eccentric work. The results of our previous study (Barnes et al. 2009) are thus due to an interaction between post-exercise alcohol consumption, the damaged muscle and/or the recovery processes initiated by EIMD.

Confirming our previous observations, in the current study significant differences in post-exercise muscle performance between treatments were seen after 36 h. At this time point, isometric and eccentric peak torques were 39 and 44% lower than pre-exercise measures, respectively, with ALC compared to losses of 29 and 27% for the same measures with OJ. Perhaps more important than a single all out effort, the ability to generate force repeatedly was greatly reduced, with losses in average peak torque of 41% (isometric), 43% (concentric) and 45% (eccentric) with ALC compared to 29, 32 and 26% with OJ, respectively. Together with the results of our previous work (Barnes et al. 2009), the current study reinforces the observation that the consumption of moderate amounts of alcohol after damaging exercise magnifies the loss in force production capability typically associated with EIMD.

To date considerable effort has been made to identify modalities that improve the rate of performance recovery after strenuous eccentric exercise. The majority of this research, however, has failed to conclusively show that losses in performance can be minimised if a particular modality is used during the post-exercise period (Cleak and Eston 1992; Barnett 2006; Wilcock et al. 2006). An alternative, as suggested by Reilly and Ekblom (2005), is to adhere to proper nutritional strategies including moderation when drinking alcohol. Indeed, given our current and previous findings, moderation of alcohol after strenuous, damaging exercise is a sound advice if a timely return to optimal performance is desired. However, whether moderation alone is sufficient to avoid the negative effects of

alcohol on recovery after damaging exercise is currently unclear as only one dose (1 g of alcohol per kg of body weight) has been investigated. In fact, the dose used in the current study is considerably lower than levels of alcohol consumption frequently reported by sportspeople (Snow and Munro 2006; O'Brien et al. 2005; O'Brien et al. 2007) suggesting that alcohol use would have to be restricted to an even greater extent if results such as those observed in the present study are to be avoided. Further research is warranted to investigate the dose effects of alcohol use in the post-exercise period.

Although the mechanisms behind our findings are not yet understood, previous research into acute alcohol use suggests that a number of similarities may exist between the separate effects of EIMD and acute alcohol consumption on skeletal muscle. Alterations in excitation–contraction (E–C) coupling and central nervous system (CNS) function have been proposed as contributors to the force loss associated with EIMD (Deschenes et al. 2000; Carson et al. 2002; Prasartwuth et al. 2005; Racinais et al. 2008; Dartnall et al. 2009). Similarly, acute alcohol exposure has been shown to negatively affect sarcoplasmic Ca^{2+} transport, thus altering E–C coupling (Cofán et al. 2000); while alcohol acts on the CNS to impact axonal conductance and neurotransmission leading to dose-dependent impairment of psychomotor and cognitive skills. These actions contribute to the popularity of alcohol as a recreational drug (Valenzuela 1997; Reilly 2003). Finally, a well-coordinated immune response is initiated by EIMD to facilitate repair and recovery of damaged tissue (Tidball 2005). As acute alcohol use has been shown to adversely affect recovery from trauma/injury by altering the normal inflammatory response (Szabo and Mandrekar 2009), our results may be due to alcohol-related impairment of the normal recovery processes. Whether any or all of these factors combine to bring about the results observed in the current study is worth further investigation.

Conclusion

When consumed after strenuous eccentric exercise, a moderate dose of alcohol magnifies the temporary loss of muscular performance associated with EIMD but does not affect performance of the unexercised muscle. Alcohol thus appears to exert its ergolytic effect by impairing the normal recovery processes which occur following exercise-induced microstructural damage, not by a systemic effect on skeletal muscle innervation. This study provides evidence that the management of alcohol use after strenuous eccentric exercise is as important, if not more important, than the use of popular recovery modalities if optimal recovery of performance is desired. The mechanisms behind our findings are yet to be fully elucidated.

Conflict of interest statement None.

References

Barnes MJ, Mündel T, Stannard SR (2009) Acute alcohol consumption aggravates the decline in muscle performance following strenuous eccentric exercise. J Sci Med Sport. doi:10.1016/j.2008.12.627

Barnett A (2006) Using recovery modalities between training sessions in elite athletes: does it help? Sports Med 36(9):781–796

Carson RG, Riek S, Shahbazpour N (2002) Central and peripheral mediation of human force sensation following eccentric or concentric contractions. J Physiol 539(3):913–925

Clarkson PM, Reichsman F (1990) The effect of ethanol on exercise-induced muscle damage. J Stud Alcohol 51(1):19–23

Cleak MJ, Eston RG (1992) Muscle soreness, swelling, stiffness and strength loss after intense eccentric exercise. Br J Sp Med 537(2):267–272

Cofán M, Nicholás JM, Fernández-Solá J et al (2000) Acute ethanol treatment decreases intracellular calcium-ion transients in mouse single skeletal muscle fibres in vitro. Alcohol Alcohol 35(2):134–138

Dartnall TJ, Rogasch NC, Nordstrom MA et al. (2009) Eccentric muscle damage has variable effects on motor unit recruitment thresholds and discharge patterns in elbow flexor muscles. J Neurophys. doi:10.1152/jn.91285.2008

Deschenes MR, Brewer RE, Bush JA et al (2000) Neuromuscular disturbance outlasts other symptoms of exercise-induced muscle damage. J Neurol Sci 174:92–99

Eston R, Peters D (1999) Effects of cold water immersion on symptoms of exercise-induced muscle damage. J Sports Sci 17:231–238

Gulick DT, Kimura IF, Sitler M et al (1996) Various treatment techniques on signs and symptoms of delayed onset muscle soreness. J Athl Train 31(2):145–152

Jönhagen S, Ackermann P, Ericksson T et al (2004) Sports massage after eccentric exercise. Am J Sports Med 32(6):1499–1503

Kraemer WJ, Bush JA, Wickham RB et al (2001) Influence of compression therapy on symptoms following soft tissue injury from maximal eccentric exercise. J Orth Sports Phys Ther 31(6):282–290

MacIntyre DL, Reid WD, Lyster DM et al (1996) Presence of WBC, decreased strength, and delayed soreness in muscle after eccentric exercise. J Appl Physiol 80:1006–1013

Nelson TF, Wechsler H (2001) Alcohol and college athletes. Med Sci Sports Exercise 33(1):43–47

O'Brien KS, Blackie JM, Hunter JA (2005) Hazardous drinking in elite New Zealand sportspeople. Alcohol Alcohol 40(3):239–241

O'Brien KS, Ali A, Cotter JD et al (2007) Hazardous drinking in New Zealand sportspeople: level of sporting participation and drinking motives. Alcohol Alcohol 42:376–382

Poulsen MB, Jakobsen J, Aagard NK et al (2007) Motor performance during and following acute alcohol intoxication in healthy non-alcoholic subjects. Eur J Appl Physiol 101:513–523

Prasartwuth O, Taylor JL, Gandevia SC (2005) Maximal force, voluntary activation and muscle soreness after eccentric damage to human elbow flexor muscles. J Physiol 567:337–348

Proske U, Morgan DL (2001) Muscle damage from eccentric exercise: mechanism, mechanical signs, adaptation and clinical applications. J Physiol 537(2):333–345

Racinais S, Bringard A, Puchaux K et al (2008) Modulation in voluntary neural drive in relation to muscle soreness. Eur J Appl Physiol 102:439–446

Reilly T (2003) Alcohol, anti-anxiety drugs and sport. In: Mottram DR (ed) Drugs in sport. Routledge, London, pp 258–287

Reilly T, Ekblom B (2005) The use of recovery methods post-exercise. J Sports Sci 23(6):619–627

Snow P, Munro G (2006) Alcohol (mis)use in metropolitan amateur football clubs. ACHPER Healthy Lifestyles J 53:7–11

Szabo G, Mandrekar P (2009) A recent perspective on alcohol, immunity and host defence. Alcohol Clin Exp Res 33(2):220–232

Tidball JG (2005) Inflammatory processes in muscle injury and repair. Am J Physiol 288:R345–R353

Valenzuela CF (1997) Alcohol and neurotransmitter interactions. Alcohol Health Res World 21(2):144–148

Wilcock IM, Cronin JB, Hing WA (2006) Physiological response to water immersion: a method of sport recovery? Sports Med 36(9):747–765