Ethanol Does Not Delay Muscle Recovery but Decreases Testosterone/Cortisol Ratio

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ABSTRACT

HAUGVAD, A., L. HAUGVAD, H. HAMARSLAND, and G. PAULSEN. Ethanol Does Not Delay Muscle Recovery but Decreases Testosterone/Cortisol Ratio. *Med. Sci. Sports Exerc.*, Vol. 46, No. 11, pp. 2175–2183, 2014. **Purpose**: This study investigated the effects of ethanol consumption on recovery from traditional resistance exercise in recreationally trained individuals. **Methods**: Nine recreationally trained volunteers (eight males and one female, 26 ± 4 yr, 81 ± 4 kg) conducted four resistance exercise sessions and consumed a low (0.6 (females) and 0.7 (males) g·kg^{-1} body mass) or a high dose (1.2 or 1.4 g·kg^{-1} body mass) of ethanol 1–2.5 h after exercise on two occasions. The first session was for familiarization with the tests and exercises and was performed without ethanol consumption. As a control trial, alcohol-free drinks were consumed after the exercise session. The sequence of trials, with low and high ethanol doses and alcohol-free drinks (control), was randomized. Maximal voluntary contractions (MVC) (knee extension), electrically stimulated contractions (knee extension), squat jumps, and hand grip strength were assessed 10–15 min and 12 and 24 h after the ethanol/placebo drinks. In addition to a baseline sample, blood was collected 1, 12, and 24 h after the ethanol/placebo drinks. The exercise session comprised 4 × 8 repetition maximum of squats, leg presses, and knee extensions. **Results**: MVC were reduced by 13%–15% immediately after the exercise sessions ($P < 0.01$). MVC, electrically stimulated force, and squat jump performance were recovered 24 h after ethanol drinks. MVC was not fully recovered at 24 h in the control trial. Compared with those in the control, cortisol increased and the free testosterone/cortisol ratio were reduced after the high ethanol dose ($P < 0.01$). **Conclusions**: Neither a low nor a high dose of ethanol adversely affected recovery of muscle function after resistance exercise in recreationally strength-trained individuals. However, the increased cortisol levels and reduced testosterone/cortisol ratio after the high ethanol dose could translate into long-term negative effects. **Key Words:** ALCOHOL, STRENGTH TRAINING, MUSCLE PERFORMANCE, ELECTRICALLY STIMULATED CONTRACTIONS, CREATINE KINASE, LEUKOCYTES

According to statistics from the Norwegian Institute for Alcohol and Drug Research, ethanol consumption increased by 20% from 1993 to 2000. From 2000 to 2010, it further increased by approximately 1 L·yr^{-1} per inhabitant (www.sirus.no). Alcohol consumption among professional athletes shows no exception to these trends. In fact, reports suggest an even larger and more frequent intake in sportsmen as compared with that in the general population (21,24).

Although alcohol consumption after exercise is quite common, surprisingly few studies have investigated the effects of alcohol on recovery from and adaptation to exercise. However, recent experiments have shown that alcohol may hinder muscle recovery (1,2,4). Barnes et al. (1) found that 1 g ethanol·kg^{-1} body mass prolonged recovery of muscle function after 300 eccentric contractions. In a similar study, the same investigator reported that a lower dose of 0.5 g ethanol·kg^{-1} body mass did not exert such effects (3). Investigating recovery after rugby matches, Barnes et al. (5) and Murphy et al. (23) both found evidence for negative effects on muscle power when 1 g ethanol·kg^{-1} body mass was consumed in the hours after a match.

The mechanisms behind the adverse effects of ethanol on muscle recovery from exercise are uncertain (37) but probably relate to interference with circulatory levels of cortisol, growth hormone, and androgens (14,34,36). In addition, metabolic processes (15,37), hypertrophic cellular signaling (e.g., reduced mechanistic target of rapamycin (mTOR) activation), and protein synthesis may be considered (18,19). Adverse effects on protein synthesis have, however, not been verified in experiments where ethanol administration was preceded by exercise.

Importantly, in the studies of Barnes et al. (1,2,4), intensive eccentric exercise was applied. Eccentric exercise is known to induce far more muscle damage than that induced by traditional resistance exercise (27). Similarly, rugby is a physically rough, intensive sport (5,23), regularly requiring body contact that causes muscle contusions (16). Thus, it is implied that the observed effects of ethanol in these studies may be associated with recovery from muscle damage that

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requires remodeling and regeneration. The aforementioned studies used nontraditional exercise models in which post-exercise recovery is likely to be intensive. However, the effects of ethanol consumption on recovery markers after more traditional exercise protocols remain unexplored.

The present study is the first to investigate the effects of ethanol on the recovery of muscle function after a session of traditional resistance exercise. Furthermore, a possible dose–response effect of ethanol consumption on recovery after exercise was explored. Hence, it was hypothesized that ethanol consumption in the hours after exercise would delay the recovery of muscle function and that a high dose (1.2 (female) and 1.4 (male) $g \cdot kg^{-1}$ body mass) of ethanol would exert larger effects than a low dose (0.6 (female) and 0.7 (male) $g \cdot kg^{-1}$ body mass).

METHODS

Participants

Twelve healthy nonsmoking individuals volunteered to participate in this study. All participants were recreationally trained, which means that they had conducted resistance exercise more than two times per week during the 12 months before the experiment; they had 10 ± 6 yr of experience with resistance exercise. Because of injury and causes not related to the experiment, nine completed the study: eight males and one female (26 ± 4 yr of age, 81 ± 4 kg body mass, 179 ± 5 cm in height). All participants were accustomed to drinking but consumed alcohol no more than 3 $d \cdot wk^{-1}$ and fewer than 10 drinks/units per week (1 unit equals 13 g of ethanol).

All volunteers were fully informed of the experimental procedures before the start of the study, and they all signed a written consent. The study complied with the Declaration of Helsinki, and the regional ethical committee in Norway identified no ethical concerns.

Experimental Design

After a session to provide familiarization with all tests and the exercise session, the participants completed three trials in a randomized order (Fig. 1): one trial with a low ethanol dose (LALC), one trial with a high ethanol (HALC) dose, and one trial with placebo drinks (CONT). The drinks in each trial were consumed 60–150 min after the resistance exercise session. The investigators who conducted the muscle function tests and the participants were all blind to the content of the drinks. However, because of different ethanol concentrations, the drinks did have a slightly different taste.

On each trial day, muscle function tests were performed before the exercise bout (15–30 min), immediately after the bout (10–15 min), and at 12 and 24 h after drinks. A minimum of 2 d and a maximum of 6 d separated the intervention days (i.e., a minimum of 3 d between exercise sessions). Venous blood samples were drawn before exercise (only before the familiarization trial) and 1, 12, and 24 h after drinks (Fig. 1). Exercise sessions were always conducted between 4:00 and 6:00 p.m.

Food and fluid intake were standardized on trial days and individualized for each participant. Intoxication after drinks was monitored every 15 min by repeated breath alcohol tests (AL 7000 alcohol detector; South Korea). After confirming that the ethanol levels were declining (after 2–3 h), the participants were driven home by one of the investigators. The participants were instructed to go straight to bed to get 7–8 h of sleep.

Exercise Session

Squats, leg presses, and bilateral knee extensions were performed in four sets with a load of eight-repetition maximum. This load was determined during the familiarization session and was based on the participants' experience. Because recruited participants were asked to "explore" their eight-repetition maximum loads in these exercises before the familiarization session, appropriate loads were found without difficulty. The loads were continuously adjusted during each exercise session. The contractions were performed slowly and in a well-controlled manner. Each set was followed by 2 min of rest. Each exercise session lasted for approximately 27 min.

Drinks

Sixty minutes after exercise, the participants started to consume either a low or a high dose of ethanol or a placebo

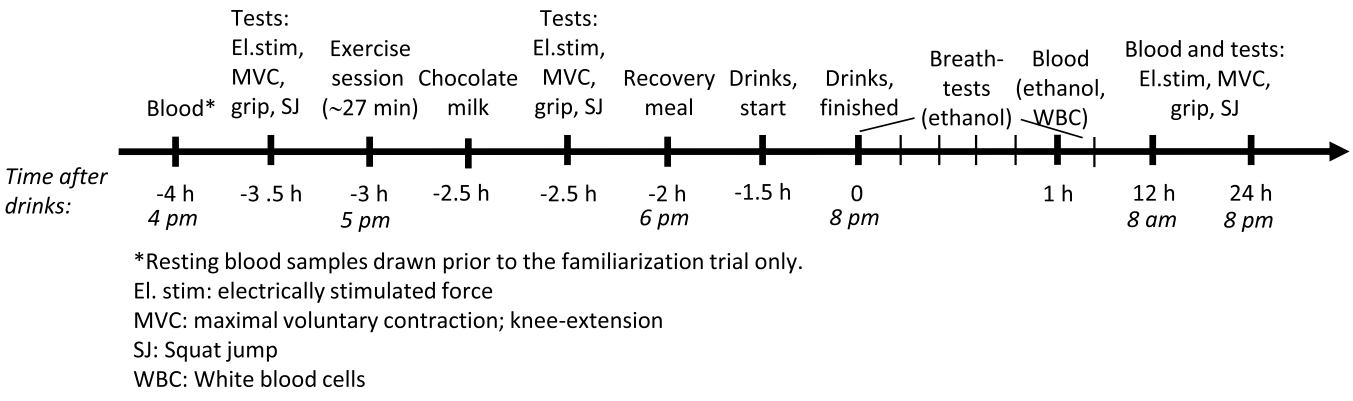

FIGURE 1—Timeline for the trials.

(low dose, 0.6 (females) and 0.7 (males) g ethanol·kg^{-1} body mass; high dose, 1.2 (females) and 1.4 (males) g ethanol·kg^{-1} body mass). Ethanol (40% ethanol/volume, Absolut vodka) was diluted with 200-mL sugar-free lemonade (raspberry flavor) and water to a total of 1.5 L. The control beverage consisted of 300-mL sugar-free lemonade (raspberry flavor) and water (total of 1.5 L). The lemonade was added to minimize the taste of the ethanol. The beverage was consumed in about 90 min.

Nutrition

Immediately postexercise, the participants consumed 0.5 L of chocolate milk for rehydration and nutrition (16.5 g of protein, 7.5 g of fat, and 45 g of CHO). Approximately 30 min after exercise, the participants consumed a standard meal of oatmeal, milk, raisins, a banana, and strawberry jam—*ad libitum*—during the familiarization trial. On the basis of the familiarization trial, the individually adapted nutrient and fluid intakes were replicated during the following three trials. For the unstandardized meals (e.g., breakfast), participants were instructed to follow and record their habitual diet from the familiarization trial days and replicate these meals during the following trials.

Performance Tests

The exercise session targeted the knee and hip extensor muscles, specifically. Therefore, the recovery of these muscles was monitored. The contractile properties were tested in three different ways: 1) electrically stimulated isometric knee extensions, 2) maximal voluntary isometric knee extension, and 3) squat jumps (muscle power). The tests were always performed in this order. In addition, the participants performed a hand grip strength test (nonexercised muscles).

The performance tests were conducted immediately preexercise, postexercise, and 12 and 24 h after beverage consumption (which followed the exercise bout). Before testing, participants performed a 3-min warm-up on a cycle ergometer (100 W) except before the tests conducted immediately after exercise.

Electrically stimulated isometric contractions and maximal voluntary isometric contractions. Electrically stimulated isometric contractions (El.stim) force and maximal voluntary isometric contractions (MVC) were both measured with a force transducer (HBM U2AC2; Darmstadt, Germany) in a modified Gym2000 knee extensor apparatus. The right knee was fixed at 90°, with the ankle bar placed at a level just superior to the medial and lateral malleoli. The left leg was not tested. During tests, the participants were strapped to the chair with seat belts over both shoulders and hips.

The skin over the m. vastus medialis was shaved and cleaned with isopropanol. Electrodes (5 × 10 cm, Polartrode; Medistim, Oslo, Norway) were placed longitudinally on the m. vastus medialis. The sites were marked with a waterproof pen so that the same sites were used during all tests. With fixed voltage of 120 V and 500-μs square wave pulses, the muscle was stimulated twice for 300 ms with a 20-Hz current and twice with a 50-Hz current (S11 Stimulator; Grass Instruments, Massachusetts). The mean values from the two stimulations at each frequency were used to calculate the 20/50 Hz force ratio. The test procedures are as presented in previous studies from our laboratory (31). After the El.stim, three 5-s MVC were performed, separated by 1 min of rest. The peak values were included in the data analysis.

Grip strength. After the El.stim and MVC, maximal grip strength (nonexercised muscle) was measured using a hydraulic hand dynamometer (#43050; Chattanooga Group, Inc., Hixson, TN). The dynamometer was individually adjusted so that it reached from the hypothenar to the middle phalanges. The participants used their dominant hand and were given two attempts. The peak values are included in the data analysis. The test of a nonexercised muscle was adapted from Overgaard et al. (25) and was assumed to monitor changes in central activation levels, independent of processes in the exercised muscles.

Squat jump. Squat jumps were performed on a force platform (FP4; Huruk Co., Tampere, Finland, and SG-9; Advanced Mechanical Technologies, Newton, MA). Jumps were performed with no countermovement from a knee angle of 90°, hands fixed to the hip. Jump height was calculated from the impulse during takeoff. The highest jumps were included in the data analysis.

Blood Sampling and Biochemical Analysis

Venous blood samples were drawn from an antecubital vein into two EDTA tubes (4.5 mL) and one serum tube (9 mL). Differential counts and hemoglobin (Hb) were analyzed in whole blood (Sysmex K-1000; TOA Medical Electronics Co., Ltd., Kobe, Japan). Serum and plasma were analyzed at Fürst Medical Laboratory (Oslo, Norway) using a Siemens Advita Centaur XP system for cortisol, testosterone, and sex hormone-binding globulin (SHBG). A Siemens Advita 2400 system was used to analyze creatine kinase (CK) and ethanol. All analytic coefficients of variation were <10%. Free testosterone was calculated as testosterone/SHBG multiplied by a factor of 10 (38).

Statistics

We used a within-subject design with three trials (LALC, HALC, and CONT), and in each trial, two time points were assessed (12 and 24 h after drinks). The data were analyzed with a two-way repeated-measures ANOVA (treatment and time) and the Holm–Sidak multiple comparisons test. When the two time points (12 and 24 h after drinks) were combined (averaged), we applied a one-way repeated-measures ANOVA (treatment) and the Holm–Sidak multiple comparisons test. Muscle function variables were expressed as the percentage change from baseline (before exercise). Blood variables (hormones, etc.) were tested as absolute values and

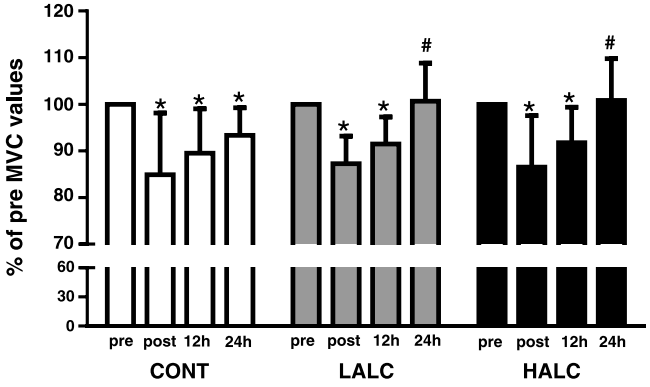

FIGURE 2—Recovery of MVC of the knee extensors. Tests were conducted before (preexercise) and after exercise (10–15 min postexercise) and 12 and 24 h after drinks: CONT, LALC, and HALC (n = 9) (mean ± SD). *Significantly different from preexercise (100%). #Significantly different from CONT.

relative values. The relative values were calculated as percentage difference from the placebo trial. For values combined across trials, to test differences between morning and evening measurements, a paired Student's t-test was applied. Outliers were identified using the Grubbs test. The α level of significance was set to 0.05. Values are given as mean ± SD. Statistics were calculated and figures were generated in GraphPad Prism (version 6.00 for Windows GraphPad software; GraphPad, La Jolla, CA).

RESULTS

During the resistance exercise session, the participants lifted a total of 10,165 ± 2170, 10,168 ± 1832, and 10,227 ± 1991 kg in the CONT, LALC, and HALC trials, respectively. There were no statistical differences between trials ($P > 0.97$).

The highest measured blood concentration values of ethanol, estimated by breath measurements (alcometer), were 0.8% ± 0.1% and 1.6% ± 0.1% after LALC and HALC, respectively (both, $P < 0.01$). Assessed directly in plasma 1 h after drinks, the promille values were 0.5% ± 0.1% and 1.4% ± 0.1% after LALC and HALC, respectively (both, $P < 0.01$). No ethanol was detected in participants' blood in the CONT trial.

MVC measured before each exercise session showed stable values, i.e., 608 ± 124, 598 ± 128 and 592 ± 134 N in the CONT, LALC, and HALC trials, respectively ($P > 0.44$ between trials). The exercise sessions induced an immediate, consistent decrease of 13%–15% in MVC; MVC was still suppressed 12 h after drinks in all trials ($P < 0.05$) (Fig. 2). MVC normalized within 24 h after both LALC and HALC but not after CONT ($P < 0.05$) (Fig. 2). The recovery from immediately after exercise (before drinks) to 12 and 24 h was, however, not different between trials.

Electrically stimulated force generated by a 50-Hz current and the 20/50 Hz ratio measured before each exercise session showed stable values. The 50-Hz forces were 54 ± 20, 51 ± 18, and 49 ± 20 N in the CONT, LALC, and HALC trials, respectively ($P > 0.44$ between trials), and the 20/50 Hz ratios were 0.78 ± 0.06, 0.79 ± 0.06, and 0.77 ± 0.05, respectively ($P > 0.44$ between trials). The 50-Hz force and the 20/50 Hz ratio decreased immediately after exercise in all trials ($P < 0.01$; Table 1). Twelve hours after exercise, the 50-Hz force had normalized in all trials, whereas the 20/50 Hz ratio had normalized after CONT and HALC. After LALC, the 20/50 Hz was still reduced 12 h after drinks ($P < 0.05$) and different from that after CONT ($P < 0.05$). However, the reduction immediately after exercise in the LALC trial (before drinks) was also larger than that after the CONT trial ($P < 0.05$). This means that the recovery of the 20/50 Hz ratio from the time point immediately after exercise to 12 h after drinks was actually not different between trials ($P = 0.35$).

Squat jump heights measured before each exercise session showed stable values, i.e., 39.5 ± 5.7, 39.4 ± 4.4, and 38.5 ± 4.8 cm, in the CONT, LALC, and HALC trials, respectively ($P > 0.43$ between trials). Squat jump performance was reduced in all trials immediately after exercise and 12 h after drinks ($P < 0.05$) (Table 1). No trial differences were found, except that jump performance was reduced more at 12 h after drinks in the LALC trial than that after drinks in the CONT trial (Table 1). Because jump performance also tended to be lower immediately after exercise ($P = 0.07$, before ethanol consumption), recovery in the period from immediately after exercise to 12 h after drinks was not different from that in CONT ($P = 0.27$).

Grip strength did not differ between trials, but grip strength across trials was consistently lower after 12 h than that after 24 h after drinks (mean of all trials, $P < 0.01$) (Table 1).

Cortisol levels did not differ significantly between trials at any specific time point. However, when the 12- and 24-h cortisol values were combined (averaged), the cortisol levels were elevated after HALC compared with those after CONT

TABLE 1. Muscle function immediately after exercise (−2.5 h) and 12 and 24 h after drinks.

	CONT			LALC			HALC		
Hours after Drinks	−2.5 (Postexercise)	12	24	−2.5 (Postexercise)	12	24	−2.5 (Postexercise)	12	24
50 Hz (%)	86 ± 8*	91 ± 13	98 ± 6	85 ± 17*	92 ± 15	97 ± 14	82 ± 7*	94 ± 10	98 ± 7
20/50 Hz (%)	79 ± 6*	97 ± 4	98 ± 7	71 ± 16*,**	89 ± 10*,**	92 ± 9	76 ± 9*	95 ± 6	98 ± 4
Squat jump (%)	93 ± 3*	96 ± 5*	97 ± 4	90 ± 5*	91 ± 6*,**	99 ± 6	92 ± 4*	92 ± 5*	97 ± 5
Grip test (%)	100 ± 4	95 ± 6*	99 ± 6	102 ± 8	96 ± 8*	102 ± 1	102 ± 6	96 ± 7*	99 ± 8

Values are expressed in percentage of preexercise levels for the three trials: CONT, LALC, and HALC (n = 9) (mean ± SD).
*Significantly different from preexercise (100%).
**Significantly different from CONT.

(P = 0.03) (Fig. 3). Across trials, the cortisol levels were consistently higher at 12 h than those at 24 h after drinks (mean of all trials, $P < 0.01$) (see Guignard et al. (13) for circadian rhythm of cortisol and testosterone).

Neither testosterone nor SHBG was altered significantly by ethanol consumption (Table 2). The serum testosterone levels across trials were consistently higher at 12 h than those at 24 h after drinks (mean of all trials, $P < 0.01$).

Calculated free testosterone was not different between trials. However, when combining (averaging) the levels at 12 and 24 h after drinks, the levels tended to be reduced after HALC compared with those after both the CONT and LALC (both P = 0.06) (Fig. 3).

Compared with that in CONT, the calculated free testosterone/cortisol ratio (T/C ratio) was reduced during the 24-h period after HALC ($P < 0.01$) but not after LALC (Fig. 3).

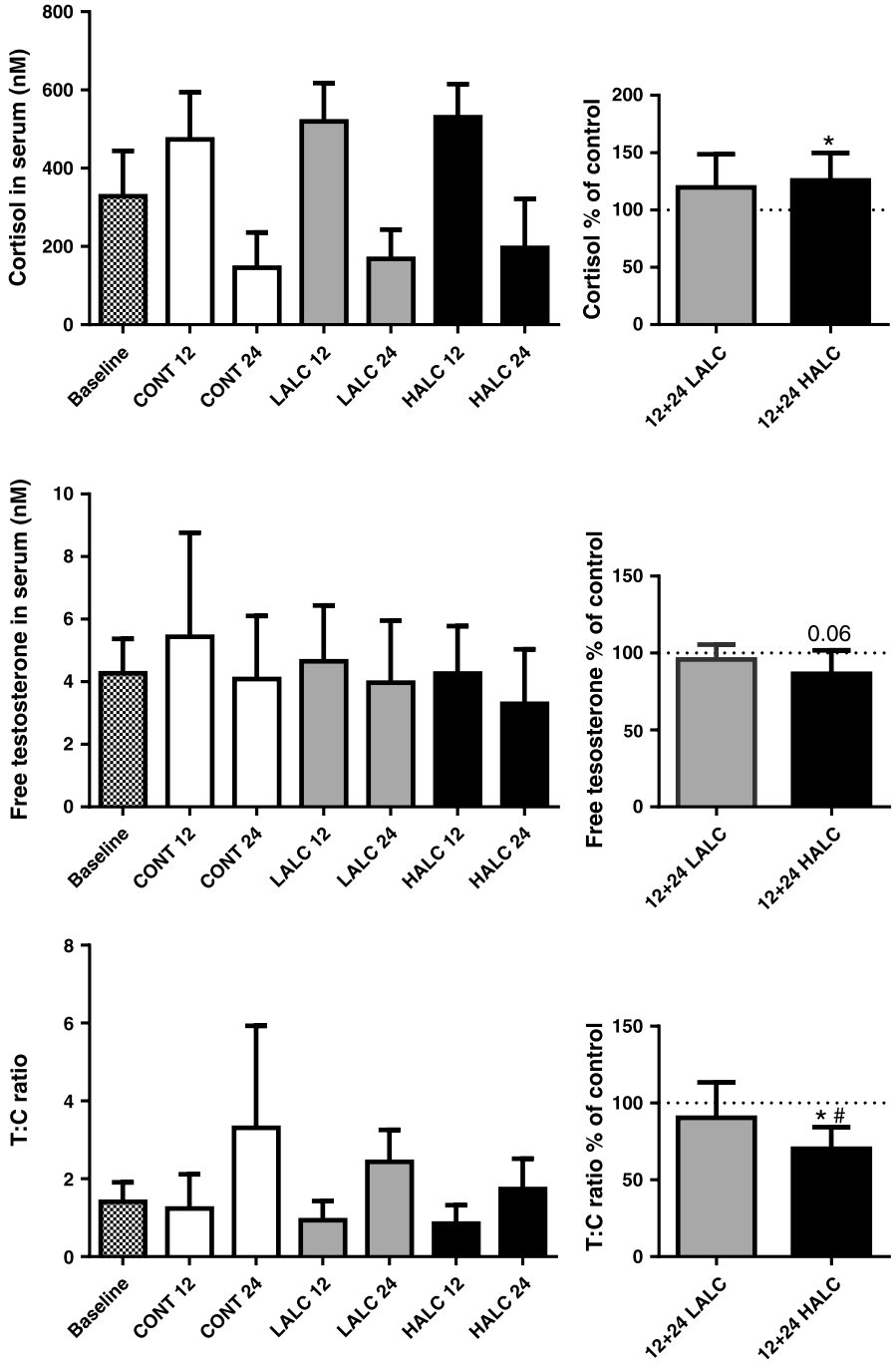

FIGURE 3—Blood concentrations of cortisol (n = 9) (mean ± SD), calculated free testosterone and the T/C ratio (n = 8) 12 and 24 h after drinks: CONT, LALC, and HALC. Note that the female participant was omitted from testosterone data. The baseline (preexercise) levels are measured at approximately 4:00 p.m. The right columns of the graphs show the individual averaged values for the 12 and 24 h measurements as percentage of CONT. *Significantly different from CONT, $P < 0.01$. #Significantly different from LALC, $P < 0.01$.

TABLE 2. Serum concentrations of testosterone and SHBG for the three trials: CONT, LALC, and HALC (n = 9) (mean ± SD).

	Baseline	CONT		LALC		HALC	
Hours after Drinks	−(Approximately 4:00 p.m.)	12 (Approximately 8:00 a.m.)	24 (Approximately 8:00 p.m.)	12 (Approximately 8:00 a.m.)	24 (Approximately 8:00 p.m.)	12 (Approximately 8:00 a.m.)	24 (Approximately 8:00 p.m.)
Testosterone (nM)	11.7 ± 4.9	12.7 ± 5.3	10.3 ± 5.7	12.0 ± 5.1	9.9 ± 4.9	11.3 ± 5.3	8.3 ± 4.1
	(13.0 ± 2.9)[a]	(14.2 ± 2.9)[a]	(11.6 ± 4.7)[a]	(13.4 ± 3.2)[a]	(11.0 ± 3.9)[a]	(12.7 ± 3.7)[a]	(9.3 ± 3.3)[a]
SHBG (nM)	35.2 ± 14.3	33.5 ± 15.9	35.0 ± 15.9	34.0 ± 15.2	34.8 ± 15.1	34.6 ± 15.1	34.6 ± 14.3

[a]Testosterone levels excluding the female participant.

Moreover, the T/C ratio after HALC was reduced more than that after LALC.

No significant changes in the circulating levels of CK were found between trials (Table 3). The pre-CK levels were not statistically different from the postexercise data (Table 3). Moreover, no differences between trials were detected regarding the total number of white blood cells (WBC), neutrophils, lymphocytes, or Hb (Table 3) and hematocrit. The hematocrit levels were generally slightly higher in the morning than those in the evening (mean of all trials, 41.6 ± 1.9 vs 40.4 ± 2.7; P = 0.01). Compared with the resting levels (baseline) (Table 3), the exercise session caused a subclinical leukocytosis 1 h after exercise (mean of all trials, 24% ± 30%; $P < 0.01$). Note that the blood data were not corrected for plasma shifts because there was no observed hemoconcentration.

DISCUSSION

This study investigated the effects of ethanol consumption on the recovery of muscle function after a traditional strength training session. The major finding was that ethanol consumption 60–150 min after exercise did not hamper recovery of muscle strength and power. However, the high ethanol dose increased the cortisol levels and lowered the T/C ratio during the 24 h after drinks.

Contrary to our hypothesis, no consistent differences in the recovery of muscle function between trials were found either for voluntary or electrically stimulated contractions, nor were nonexercised muscles affected. Intriguingly, an opposite effect was observed, as full recovery of MVC at 24 h was found after ethanol consumption (low and high doses) but not after the control trial. Noteworthy, the rate of recovery from immediately after exercise (before drinks) to 12 and 24 h did not differ between trials—indicating that the slightly larger reductions in MVC during the exercise explain the difference between trials. It is difficult to envision ethanol consumption facilitating muscle recovery, but further research should investigate this finding.

The low ethanol dose seemed to cause a delayed recovery of the 20/50 Hz ratio and squat jump performance at 12 h after drinks. This difference seemed, however, to be a consequence of unexplained larger reductions immediately after exercise (before ethanol consumption). In fact, the rate of recovery from the levels immediately after exercise to 12 and 24 h after drinks was not different between trials. Moreover, because we did not see this effect after the high dose, it is highly unlikely that this was due to ethanol intoxication. We should, on the other hand, keep in mind that other investigators have reported recovery of jumping performance (muscle power) to be more sensitive to ethanol consumption after exercise than after MVC (5,23).

Depression of the 20/50 Hz ratio and low-frequency fatigue indicates impairment of the excitation–contraction coupling (intracellular Ca^{2+} release) (10), and values are typically reduced after resistance exercise (31). Barnes et al. (4) reported, in agreement with our observation, that ethanol consumption did not affect low-frequency fatigue after 300 eccentric contractions. Barnes et al. (4) concluded, however, that ethanol consumption (1 $g \cdot kg^{-1}$ body mass) led to a decreased neural drive.

In addition to the voluntary (MVC) and electrically (50 Hz) stimulated contractions, we included assessments of maximal force in nonexercised muscles (grip strength) to distinguish between peripheral (muscular) and central (neural) mechanisms behind the muscle force deficits (25). Collectively, our results do not indicate that ethanol reduced neural drive to the exercised muscles, in contrast to Barnes et al. (4). Grip force levels were unaffected by ethanol consumption, which lends support to the observations of Barnes et al. (2,4), who found no ethanol effect in nonexercised muscles. Because muscle strength and performance seem dependent on circadian rhythms (20), we suggest that the reduced grip

TABLE 3. Circulating levels of CK, Hb, total number of WBC, lymphocytes, and neutrophils for the three trials: CONT, LALC and HALC.

	Baseline	CONT		LALC		HALC	
Hours after Drinks	−(Approximately 4:00 p.m.)	12 (Approximately 8:00 a.m.)	24 (Approximately 8:00 p.m.)	12 (Approximately 8:00 a.m.)	24 (Approximately 8:00 p.m.)	12 (Approximately 8:00 a.m.)	24 (Approximately 8:00 p.m.)
CK (IU)	328 ± 232	435 ± 189[a]	491 ± 259[a]	463 ± 317	487 ± 289	460 ± 363	486 ± 290
Hb ($g \cdot dL^{-1}$)	15.4 ± 1.0	15.3 ± 0.8	15.1 ± 0.9	15.4 ± 0.6	15.3 ± 0.6	15.4 ± 0.7	15.3 ± 0.5
WBC (10^3 μL^{-1})	6.8 ± 1.4	5.9 ± 1.4	7.7 ± 2.2	5.7 ± 1.6	7.3 ± 2.3	5.8 ± 1.0	7.1 ± 1.9
Lymphocytes, % of WBC	35 ± 5	40 ± 4	34 ± 5	40 ± 8	35 ± 7	40 ± 9	36 ± 9
Neutrophils, % of WBC	56 ± 5	48 ± 5	55 ± 5	49 ± 8	54 ± 9	48 ± 9	54 ± 9

Values are expressed as mean ± SD; n = 9.
[a]An outlier was excluded.

strength levels at 12 h after drinks (including the placebo) were a consequence of this assessment being conducted in the morning.

Considering the combination of muscle function tests applied, our observations suggest that neither the local events in the exercised muscles nor the voluntarily ability to activate the muscles was detectably affected by alcohol intoxication. This corresponds to some studies (3,8,28) but differs from others that have reported delayed recovery from exercise when ethanol was consumed in the restitution period (1,2,5,23).

The studies that observed no effects administered a low dose (0.5 $g \cdot kg^{-1}$ body mass) (3), or applied a study design that somewhat differed from the present study. Poulsen et al. (28) used a "gentle" exercise protocol (30 maximal isokinetic concentric contractions), and Clarkson and Reichsman (8) administrated the ethanol before eccentric exercise. These studies detected no reductions in muscle motor performance under or after ethanol intoxication even with rather high doses, i.e., 1 $g \cdot kg^{-1}$ body mass (8) and 1.4 $g \cdot kg^{-1}$ body mass (28).

Barnes et al. (1,2) observed rather clear adverse effects of ethanol intoxication on the recovery from intensive eccentric exercise. The difference between muscle-damaging eccentric exercise and traditional resistance exercise could explain the discrepancy between the studies of Barnes et al. (1,2) and the present study. Indeed, on the basis of the suggestions of Paulsen et al. (27), the decrements and recovery times of muscle function in the present study suggest merely mild muscle damage (<20% reduction in muscle function and/or recovery within 48 h), whereas the damage inflicted in the studies of Barnes et al. (1,2) seemed moderate or large (large damage, >50% reduction in muscle function and/or recovery longer than 1 wk). Importantly, our recreationally trained participants underwent specific familiarization with the resistance exercise session before the experimental trials. This likely induced a rapid adaptation in the neuromuscular system (22), so that the subsequent sessions did not inflict large damage to their muscles or caused alterations in neural drive. The low CK levels support this assumption. We chose this design because it seems more relevant for people regularly engaged in exercise and training. By contrast, the 300 eccentric contractions protocol, as applied by Barnes et al. (1,2), has been found to inflict considerable muscle damage and local inflammation (26), thus possibly making the neuromuscular system more prone to the detrimental effects of ethanol.

Ethanol can increase cortisol and reduce testosterone levels by interfering with the hypothalamic–pituitary adrenal and gonadal axes (9,32). In line with this, we found significantly elevated cortisol levels after the high ethanol dose (compared with those after placebo). This corresponds to the findings of Valimeki et al. (36). More importantly, we observed a reduction in the calculated T/C ratio, also only after the high ethanol dose. Interestingly, the reduction was statistically larger than that after the low dose, implying that relatively high ethanol doses are required to induce this effect. The consequences of these hormonal changes are several, but most importantly, cortisol stimulates protein degradation in muscle tissues (12), whereas reduced testosterone levels may hamper muscle growth induced by strength training (17). Reduced T/C ratio is strongly associated with overtraining and reduced physical performance (35). Hence, these effects suggest that high doses of ethanol in the postexercise recovery phase will, over time, exert negative effects on adaption to resistance exercise, i.e., muscle hypertrophy. To support this assumption, animal models have demonstrated drastic adverse effects of ethanol on protein synthesis and muscle growth (29), possibly through inhibition of mTOR (18). Intriguingly, both exercise (*per se*) and testosterone activate mTOR to induce muscle hypertrophy (6,7). Further studies should investigate the long-term effects of ethanol consumption after exercise in humans, and muscle biopsies should be obtained to elucidate the cellular effects.

The present study has an original research design for the topic at hand (e.g., a familiarization session and dose–response approach), but certain limitations should be mentioned. Firstly, the number of participants ($n = 9$) may have been too low to detect subtle effects of ethanol. Secondly, we did not collect blood samples before each exercise session, meaning that we cannot be sure that values of circulating markers reverted to baseline between trials. Previous relevant research indicates, however, that exercise-induced increases in testosterone and cortisol levels are short-lasting and 72 h of rest should be more than adequate (11,30). Also, measures of muscle function did demonstrate recovery between trials. A final limitation was that we did not record sleep quality. Because ethanol consumption can disturb sleep (33), the altered hormone levels (T/C ratio) could plausibly be related to this indirect effect of ethanol. Future studies should take this into account when investigating the interaction between recovery from and adaptation to exercise concomitantly with regular ethanol consumption.

Practical application. The question of whether adaptations to exercise are lost and recovery is prolonged if ethanol is consumed in the recovery period is debated among athletes and those who exercise for recreation and health. We provide evidence that ethanol consumption in the hours after exercise does not adversely affect recovery of muscle performance from a typical resistance exercise session. Importantly, although no clear adverse effects were found, we do not endorse ethanol consumption after exercise. In fact, the literature does indicate interference if combining very strenuous and/or muscle-damaging exercise and large doses of ethanol consumption (≥1 $g \cdot kg^{-1}$ body mass). Moreover, on the basis of the reduced T/C ratio observed in the present study, it is likely that frequent ethanol intake would hamper training-induced muscle growth over time, but this awaits experimental evidence.

CONCLUSIONS

We hypothesized that ethanol consumed in the hours after a session of traditional resistance exercise would impair recovery in a dose–response manner in recreationally

strength-trained individuals. However, neither a low (0.6–0.7 $g \cdot kg^{-1}$ body mass) nor a high dose (1.2–1.4 $g \cdot kg^{-1}$ body mass) of ethanol adversely affected recovery of muscle function during the 24-h period after exercise. Increases in cortisol and reductions in the T/C ratio after the high ethanol dose could translate into long-term negative effects, such as blunted hypertrophy.

The authors declare no conflicts of interest. The results of the present study do not constitute endorsement by the American College of Sports Medicine.

REFERENCES

1. Barnes MJ, Mundel T, Stannard SR. Acute alcohol consumption aggravates the decline in muscle performance following strenuous eccentric exercise. *J Sci Med Sport*. 2010;13(1):189–93.
2. Barnes MJ, Mundel T, Stannard SR. Post-exercise alcohol ingestion exacerbates eccentric-exercise induced losses in performance. *Eur J Appl Physiol*. 2010;108(5):1009–14.
3. Barnes MJ, Mundel T, Stannard SR. A low dose of alcohol does not impact skeletal muscle performance after exercise-induced muscle damage. *Eur J Appl Physiol*. 2011;111(4):725–9.
4. Barnes MJ, Mundel T, Stannard SR. The effects of acute alcohol consumption and eccentric muscle damage on neuromuscular function. *Appl Physiol Nutr Metab*. 2012;37(1):63–71.
5. Barnes MJ, Mundel T, Stannard SR. The effects of acute alcohol consumption on recovery from a simulated rugby match. *J Sports Sci*. 2012;30(3):295–304.
6. Basualto-Alarcon C, Jorquera G, Altamirano F, Jaimovich E, Estrada M. Testosterone signals through mTOR and androgen receptor to induce muscle hypertrophy. *Med Sci Sports Exerc*. 2013;45(9):1712–20.
7. Bodine SC. mTOR signaling and the molecular adaptation to resistance exercise. *Med Sci Sports Exerc*. 2006;38(11):1950–7.
8. Clarkson PM, Reichsman F. The effect of ethanol on exercise-induced muscle damage. *J Stud Alcohol*. 1990;51(1):19–23.
9. Cobb CF, Van Thiel DH. Mechanism of ethanol-induced adrenal stimulation. *Alcohol Clin Exp Res*. 1982;6(2):202–6.
10. Edwards RH, Hill DK, Jones DA, Merton PA. Fatigue of long duration in human skeletal muscle after exercise. *J Physiol*. 1977;272(3):769–78.
11. Fry AC, Kraemer WJ, Ramsey LT. Pituitary-adrenal-gonadal responses to high-intensity resistance exercise overtraining. *J Appl Physiol (1985)*. 1998;85(6):2352–9.
12. Glass DJ. Signaling pathways perturbing muscle mass. *Curr Opin Clin Nutr Metab Care*. 2010;13(3):225–9.
13. Guignard MM, Pesquies PC, Serrurier BD, Merino DB, Reinberg AE. Circadian rhythms in plasma levels of cortisol, dehydroepiandrosterone, delta 4-androstenedione, testosterone and dihydrotestosterone of healthy young men. *Acta Endocrinol (Copenh)*. 1980;94(4):536–45.
14. Heikkonen E, Ylikahri R, Roine R, Valimaki M, Harkonen M, Salaspuro M. The combined effect of alcohol and physical exercise on serum testosterone, luteinizing hormone, and cortisol in males. *Alcohol Clin Exp Res*. 1996;20(4):711–6.
15. Heikkonen E, Ylikahri R, Roine R, Valimaki M, Harkonen M, Salaspuro M. Effect of alcohol on exercise-induced changes in serum glucose and serum free fatty acids. *Alcohol Clin Exp Res*. 1998;22(2):437–43.
16. Junge A, Cheung K, Edwards T, Dvorak J. Injuries in youth amateur soccer and rugby players—comparison of incidence and characteristics. *Br J Sports Med*. 2004;38(2):168–72.
17. Kvorning T, Andersen M, Brixen K, Madsen K. Suppression of endogenous testosterone production attenuates the response to strength training: a randomized, placebo-controlled, and blinded intervention study. *Am J Physiol Endocrinol Metab*. 2006;291(6):E1325–32.
18. Lang CH, Frost RA, Deshpande N, et al. Alcohol impairs leucine-mediated phosphorylation of 4E-BP1, S6K1, eIF4G, and mTOR in skeletal muscle. *Am J Physiol Endocrinol Metab*. 2003;285(6):E1205–15.
19. Lang CH, Pruznak AM, Nystrom GJ, Vary TC. Alcohol-induced decrease in muscle protein synthesis associated with increased binding of mTOR and raptor: comparable effects in young and mature rats. *Nutr Metab (Lond)*. 2009;6:4.
20. Martin A, Carpentier A, Guissard N, van Hoecke J, Duchateau J. Effect of time of day on force variation in a human muscle. *Muscle Nerve*. 1999;22(10):1380–7.
21. Maughan RJ. Alcohol and football. *J Sports Sci*. 2006;24(7):741–8.
22. McHugh MP. Recent advances in the understanding of the repeated bout effect: the protective effect against muscle damage from a single bout of eccentric exercise. *Scand J Med Sci Sports*. 2003;13(2):88–97.
23. Murphy AP, Snape AE, Minett GM, Skein M, Duffield R. The effect of post-match alcohol ingestion on recovery from competitive rugby league matches. *J Strength Cond Res*. 2013;27(5):1304–12.
24. O'Brien KS, Ali A, Cotter JD, O'Shea RP, Stannard S. Hazardous drinking in New Zealand sportspeople: level of sporting participation and drinking motives. *Alcohol Alcohol*. 2007;42(4):376–82.
25. Overgaard K, Fredsted A, Hyldal A, Ingemann-Hansen T, Gissel H, Clausen T. Effects of running distance and training on Ca2+ content and damage in human muscle. *Med Sci Sports Exerc*. 2004;36(5):821–9.
26. Paulsen G, Crameri R, Benestad HB, et al. Time course of leukocyte accumulation in human muscle after eccentric exercise. *Med Sci Sports Exerc*. 2010;42(1):75–85.
27. Paulsen G, Mikkelsen UR, Raastad T, Peake JM. Leucocytes, cytokines and satellite cells: what role do they play in muscle damage and regeneration following eccentric exercise? *Exerc Immunol Rev*. 2012;18:42–97.
28. Poulsen MB, Jakobsen J, Aagaard NK, Andersen H. Motor performance during and following acute alcohol intoxication in healthy non-alcoholic subjects. *Eur J Appl Physiol*. 2007;101(4):513–23.
29. Preedy VR, Peters TJ. The effect of chronic ethanol feeding on body and plasma composition and rates of skeletal muscle protein turnover in the rat. *Alcohol Alcohol*. 1988;23(3):217–24.
30. Raastad T, Glomsheller T, Bjoro T, Hallen J. Recovery of skeletal muscle contractility and hormonal responses to strength exercise after two weeks of high-volume strength training. *Scand J Med Sci Sports*. 2003;13(3):159–68.
31. Raastad T, Hallen J. Recovery of skeletal muscle contractility after high- and moderate-intensity strength exercise. *Eur J Appl Physiol*. 2000;82(3):206–14.
32. Rivier C, Bruhn T, Vale W. Effect of ethanol on the hypothalamic-pituitary-adrenal axis in the rat: role of corticotropin-releasing factor (CRF). *J Pharmacol Exp Ther*. 1984;229(1):127–31.
33. Roehrs T, Roth T. Sleep, sleepiness, and alcohol use. *Alcohol Res Health*. 2001;25(2):101–9.

34. Sierksma A, Sarkola T, Eriksson CJ, van der Gaag MS, Grobbee DE, Hendriks HF. Effect of moderate alcohol consumption on plasma dehydroepiandrosterone sulfate, testosterone, and estradiol levels in middle-aged men and postmenopausal women: a diet-controlled intervention study. *Alcohol Clin Exp Res*. 2004;28(5):780–5.
35. Urhausen A, Gabriel H, Kindermann W. Blood hormones as markers of training stress and overtraining. *Sports Med*. 1995; 20(4):251–76.
36. Valimaki M, Tuominen JA, Huhtaniemi I, Ylikahri R. The pulsatile secretion of gonadotropins and growth hormone, and the biological activity of luteinizing hormone in men acutely intoxicated with ethanol. *Alcohol Clin Exp Res*. 1990;14(6):928–31.
37. Vella LD, Cameron-Smith D. Alcohol, athletic performance and recovery. *Nutrients*. 2010;2(8):781–9.
38. Vermeulen A, Verdonck L, Kaufman JM. A critical evaluation of simple methods for the estimation of free testosterone in serum. *J Clin Endocrinol Metab*. 1999;84(10):3666–72.