

THE EFFECT OF POST-EXERCISE ETHANOL CONSUMPTION ON THE ACUTE  
HORMONAL RESPONSE TO HEAVY RESISTANCE EXERCISE IN WOMEN

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The purpose of this study was to examine the hormonal response to acute ethanol ingestion following a bout of heavy resistance exercise in women. Eight resistance trained women completed two identical acute heavy resistance exercise tasks (AHRET). From 10-20 minutes post-AHRET, participants consumed either a grain ethanol or a placebo beverage. Blood was collected before (PRE) and immediately after the AHRET (IP) and then every 20 minutes for five hours. Blood collected after beverage ingestion was pooled into 3 batches (phases: 20-40 minutes, 60-120 minutes, and 140-300 minutes post-exercise) and analyzed for serum total testosterone (TT), free testosterone (FT), insulin-like growth factor-I (IGF-I), human growth hormone (GH), cortisol (COR), and estradiol (E2) concentrations. Circulating concentrations of TT were significantly greater at P20-40 than at PRE, P60-120, and P140-300. Circulating concentrations of FT were significantly greater at P20-40 than at all other times. Circulating concentrations of GH were significantly greater at IP than at PRE, P60-120, and P140-300. Circulating concentrations of COR were significantly greater at P20-40 than at all other times. Additionally, COR concentrations at P140-300 were significantly lower than at all other times. Circulating concentrations of IGF-1 were significantly greater at P20-40 than at P60-120 and P140-300. Circulating concentrations of E2 were significantly greater at P20-40 than at all other times. In summary, the present study demonstrated an acute modulation of the neuroendocrine milieu following a heavy resistance exercise bout in women. Ethanol ingestion appeared to have no significant effect on the characteristics of acute hormonal augmentation in TT, FT, GH, COR, IGF-1, or E2.

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## INTRODUCTION

Athletes, especially student athletes, drink more ethanol (alcohol) and report a greater number of heavy binge drinking episodes than their non-athlete peers (37). The number of National Collegiate Athletic Association athletes who use ethanol increased from 77.5% in 2005 to 83.1% in 2009, with 54% reporting drinking throughout competitive seasons and nearly 50% reporting incidences of binge drinking (5 or more drinks in a sitting) (6). In certain recreational sports settings, it is common for athletes to consume ethanol directly after or in some cases even during these events. Although it is well established that binge drinking behavior can result in numerous negative consequences (10) including impaired recovery from exercise (5), almost 60% of athletes believe that their use of ethanol does not affect their athletic performance or overall health (6). Acute ethanol use might interfere with short-term resistance exercise adaptations (and subsequently athletic performance), but few studies have investigated the effects of acute ethanol ingestion on resistance exercise outcomes and only one of these studies has specifically examined such effects in women.

Adaptations to chronic resistance exercise in men and women are moderated by the endocrine milieu. Acting via hormones, including testosterone, human growth hormone (GH), insulin-like growth factor-I (IGF-1), and cortisol (COR), the endocrine system responds to an acute bout of exercise and has profound effects on the process of recovery and adaptation to exercise stimuli (35). The acute hormonal response to heavy resistance exercise in women is different from that of men (23). Acutely, a few studies have demonstrated increases in total (TT) and/or free testosterone (FT) in response to resistance exercise in women (46, 69), but most studies have demonstrated no significant changes in the concentrations FT and TT while GH and COR concentrations increase acutely with a bout of resistance exercise (31, 32, 38). Results for

circulating IGF-1 have been inconsistent, with increases, decreases, and no changes reported (47). Acute elevations of estradiol (E2) following resistance exercise have also been reported, especially when women are in a hypocaloric state (30). The anabolic milieu is important to resistance training adaptations and the acute hormonal response to resistance exercise might play a role in subsequent physiological adaptations. Although conflicting evidence exist, it has been suggested that an increased acute hormonal response can provide superior chronic adaptations to resistance exercise in men (24, 52). For women, the importance of the acute hormonal response on chronic adaptations has not been established, but it is known that the endocrine environment (especially testosterone) is important to maintaining muscle mass and physiological adaptations in women (44). Despite the potential importance of the acute hormonal response to resistance exercise for women, the vast majority of research in this area has been conducted with men.

Acute ethanol intoxication has dramatic effects on hormonal homeostasis and might have substantial physiological consequences in women (i.e., breast cancer, hirsutism, etc.). Frias et al. (19) examined adolescent women admitted to emergency rooms with acute ethanol intoxication and found significant increases in COR, testosterone, adrenocorticotrophic hormone,  $\beta$ -endorphin, and prolactin as well as decreases in GH. Lavigne (36) demonstrated decreases in circulating serum IGF-1 concentrations in both eumenorrheic and postmenopausal women with daily consumption of ethanol. In addition, ethanol ingestion increases testosterone concentrations acutely in women (57) and substantially depresses nocturnal secretion of GH (50). E2 concentrations can increase in eumenorrheic and postmenopausal women with acute and (51) and chronic (43) ethanol consumption. Ethanol ingestion clearly disrupts the normal hormonal environment in women at rest, but the combined effects of ethanol consumption and other stimuli (e.g., resistance exercise) on the hormonal milieu in women are currently unknown.

Few studies exist examining the influence of ethanol on recovery from exercise in women. Acute moderate ethanol consumption in women may interfere with aerobic energy metabolism during submaximal cycle ergometry (72) as well as blunt heart rate recovery and heart rate variability after maximal cycle ergometry (74). The only study that has specifically investigated the response to ethanol consumption with resistance exercise in women found no reduction in muscular performance measures or markers of muscle damage; however, the acute hormonal response was not examined (8). In contrast, several recent studies using men have found that acute high-dose ethanol ingestion ( $1 \text{ g ethanol} \cdot \text{kg}^{-1}$  body mass) disrupts muscle recovery (i.e., force production capability) from a bout of strenuous eccentric resistance exercise (5). Additionally, Vingren et al. (66) found an altered total concentration and bioavailability of testosterone following heavy resistance exercise with ethanol consumption in men. Ethanol ingestion following a bout of resistance exercise could disrupt recovery by reducing the anabolic milieu, possibly leading to compromised adaptations from that exercise bout. Subsequently, chronic training adaptations might be compromised. However, the acute hormonal response from the combination of resistance exercise and ethanol consumption has not been examined in women. As women appear to be more vulnerable to the effects of ethanol than men (40), it is important to establish the relationship between ethanol consumption and the acute response to heavy resistance exercise.

Independently, resistance exercise and ethanol consumption affect circulating hormone concentrations. Despite the potentially important implications of acute ethanol ingestion for individuals involved in a physical conditioning program, few investigations have examined the anabolic and catabolic endocrine response to ethanol ingestion during the recovery from resistance exercise and no investigations have examined the endocrine response to exercise in

women in this context. Thus, the purpose of this study is to examine the hormonal response to acute ethanol ingestion following a bout of heavy resistance exercise in women.

## MATERIALS AND METHODS

A within subjects design was used to examine the effect of ethanol ingestion following resistance exercise on the acute hormonal response in women. Eight resistance trained women completed two identical acute heavy resistance exercise tasks (AHRET) separated by approximately 28 days (to allow for standardization of menstrual phase). From 10-20 minutes post-AHRET, participants consumed either ethanol (EtOH condition) or no ethanol (placebo condition) diluted in an artificially sweetened and calorie free beverage. Blood was collected before (PRE) and immediately after the AHRET (IP; before ethanol/placebo ingestion); and then every 20 minutes for five hours. Blood collected after beverage ingestion was pooled into 3 batches (phases: 20-40 minutes, 60-120 minutes, and 140-300 minutes post-exercise) for biochemical analysis. PRE, IP, and pooled phases samples were analyzed for serum total testosterone (TT), free testosterone (FT), insulin-like growth factor-I (IGF-1), human growth hormone (GH), cortisol (COR), and estradiol (E2) concentrations.

### Participants

Eight women ( $23 \pm 2$  y;  $161 \pm 3$  cm;  $60.5 \pm 6.8$  kg;  $25.9 \pm 3.2\%$  body fat) who were recreationally resistance trained (at least 2 sessions per week including the back squat for the past 6 months) participated in this study. Participants were screened for any medical concerns that could confound the results of the study or place the participants at an elevated risk during the study. Exclusion criteria included: preexisting heart conditions or anomalies, respiratory conditions, blood pressure problems, musculoskeletal problems, herniated inter-vertebral discs or previous orthopedic injuries that would limit the range of motion about the shoulder, elbow, hip, knee or ankle joint. In addition, the participants were required to be eumenorrheic and not

pregnant or trying to become pregnant. To be included in the study, participants could not have used drugs such as glucocorticoids or anabolic-androgenic steroids within one year prior to the start of the study.

The “Young Adult Alcohol Problems Screening Test” (25) and the “Alcohol Use Disorders Identification Test” (3) questionnaires were used to screen participants for clinical signs of ethanol abuse. To be considered for the study, the participants had to be considered low-to-moderate ethanol consumers as defined by the US Department of Health and Human Services (National Center for Health Statistics). Based on the screening, participants were not ethanol dependent, did not have consumption-induced metabolic intolerance to ethanol, and were deemed to be capable of tolerating the amount of ethanol ingestion required in this study without being affected in any extreme manner such as by nausea or flushing. The study was approved by the University Institutional Review Board and the volunteers provided written informed consent to participate.

Anthropometric measurements, familiarization, and 1-repetition maximum test

Approximately one week before the first exercise and ethanol/placebo session, participants reported to the laboratory for anthropometric measurements, familiarization and 1-repetition maximum (1RM) determination. After measurement of height and weight, body composition was measured using Dual-energy X-ray absorptiometry (Lunar Prodigy General Electric Company, Madison, WI). Participants wore only light athletic clothes and no shoes or metals for measurements of height, body mass, and body composition. Participants then performed a standardized warm-up consisting of 5 minutes of cycling on a stationary bike followed by light dynamic stretches (heel kicks, lunges, high knees, high kicks, and unweighted squats). Following warm-up, participants were familiarized with the proper technique for

performing the squat exercise using a Smith machine with no counter-balance weights (the Smith machine allowed only vertical translation of the bar). Once participants demonstrated proper technique in the Smith machine squat exercise, their 1RM strength was measured using the methods described by Kraemer and Fleck (29). Briefly, participants performed squats for 8-10 repetitions at ~50% of their estimated 1RM followed by another set of 2-5 repetitions at ~85% of estimated 1RM. Subsequently, 4-5 one-repetition trials were used to determine the 1RM.

### Experimental treatments

Each participant completed both experimental treatments (EtOH and placebo) and thereby served as her own control. Women were “phased” so that they completed the experimental treatment sessions during their early follicular phase (days 2-7 after the start of menses) in order to account for hormonal variations during the menstrual cycle (12). The treatments were performed approximately 28 days apart and administered in a balanced, randomized, crossover design. The participants and experimenters overseeing the exercise bout were kept blind to the experimental conditions selected for that treatment day. In order to prevent participants from anticipating a particular treatment, they were informed that they could potentially receive the same treatment on both experimental treatments visits. Participants were required to consume the same diet for the morning of each treatment visit (EtOH or placebo visit). Participants completed a diet record prior to the first treatment visit and were instructed to follow the same diet prior to the second treatment visit. All participants attested that they had complied with the diet instructions for their second visit.

## Treatment sessions

Participants were instructed to refrain from: 1) eating or drinking anything (except for water and non-caffeinated diet drinks) during the 2 hours leading up to each treatment visit, 2) any ingestion ethanol for 84 hours prior to each treatment visit, 3) consuming large amounts of caffeine on test day (no more than 1 cup of coffee allowed), 4) engaging in sexual activity for 24 hours prior to each treatment visit, and 5) performing any resistance exercise or intense aerobic exercise for 96 hours prior to each visit. It was also required of the participants that they had not donated blood within 8 weeks or plasma within 96 hours of the laboratory visits. The participants attested that they had adhered to all instructions and that their records were accurate. All treatment visits for a participant began at the same time of day for each participant (~0900-1100h arrival time) to avoid circadian influences.

Upon arrival at the laboratory on each treatment day, participants were screened for the presence of blood ethanol with a breathalyzer (Alcomate Accucell, AK solutions, Palisades Park, NJ) and were queried regarding compliance with study guidelines regarding diet, caffeine, sexual activity, ethanol, drugs and exercise. Participants were asked to report to the laboratory in a euhydrated state. Upon arrival at the laboratory, hydration status was measured using urine refractometry; if participants presented with urine specific gravity  $\geq 1.020$ , they were provided with cold water to drink. After hydration status was assessed, body mass was measured. Sixty five minutes prior to the AHRET, participants consumed a standardized meal replacement drink (Ensure Plus®) containing 8 kcal per kg body mass. After the meal replacement drink was ingested, a teflon catheter was inserted into an antecubital vein. The catheter remained in the vein for the remainder of the session and was kept patent with saline.

Participants completed a standardized warm-up 15 minutes before the AHRET (50 minutes after the meal) consisting of 5 minutes of ergometer cycling at a low intensity followed by dynamic stretches (10 lunges, 20 heel kicks, 20 high knees, 10 body weight moving squats). Participants then completed 10 warm-up squats at 50% of 1RM followed by the AHRET (68, 69). The AHRET consisted of 6 sets of 10 repetitions of Smith machine squats starting at 80% of 1RM; 2 minutes of rest were given between sets. If participants were not able to complete 10 repetitions on their own, they were assisted by the researchers to do so and the load was reduced for subsequent sets. The same load for each set was used for both AHRETs. After the completion of the AHRET, the participants sat quietly in a chair for the remainder of the test period and were not allowed to sleep.

#### Blood ethanol concentration

The BEC was determined from serum using an automated analyzer (Chemwell-T, New Jersey) via enzymatic assay (Catalog# A7504, Pointe Scientific, Canton, MI) BEC was measured upon arrival at the laboratory, every 10 minutes from 40 to 100 minutes post-AHRET, and every 20 minutes from 100 to 300 minutes post AHRET for both the EtOH and placebo condition. At no time were the participants allowed to know their BEC or whether they had received any ethanol during their visit. Due to the obvious physiological and psychological effects of ethanol ingestion, some participants were generally aware of when they had received ethanol; however, they were not easily able to discern when they had not received ethanol. Participants were released to the care of a sober designee after a minimum of 5 hours rest following beverage ingestion and once their BEC, as measured using the breathalyzer, was  $\leq 0.03 \text{ g}\cdot\text{dl}^{-1}$ .

### Ethanol ingestion

From 10-20 minutes post-AHRET, participants consumed either grain ethanol (EtOH condition) or no ethanol (placebo condition) in an artificially sweetened and calorie free beverage. For the EtOH condition, a dose of 1.09 g of ethanol (vodka, 40% v/v ethanol) per kg fat free body mass was used. The ethanol was diluted to a concentration of 15 % v/v absolute ethanol; for the placebo condition, the ethanol was substituted with an equal volume of water. The participants consumed 1/10 of the drink each minute during the 10-minute ingestion period. To reduce the ability of the participants to differentiate between the drinks (i.e., taste of EtOH) participants wore a nose-clip during drink ingestion and the rim of the glass was smeared with a small amount of ethanol for both conditions.

### Blood collection

On treatment days, a Teflon coated cannula (Vascular Access, Becton-Dickerson, Sandy, UT) was inserted in a superficial vein of the forearm while the participants were seated. Cannula patency was maintained with sterile saline (0.9% sodium chloride inj., USP, Hospira Inc. Lake Forest, IL). Blood samples were collected while participants were seated: 30 minutes before the AHRET (PRE), immediately after the AHRET (IP), and every 20 minutes for the 300 minutes following the AHRET.

### Blood processing

Blood was allowed to clot at room temperature (~21 °C) and subsequently centrifuged at 1,500 g at 4 °C for 15 minutes. The resultant serum was stored in several aliquots at -80°C until analysis. Following the procedures of Koziris et al. (28) and Vingren et al. (67), samples for PRE and IP were stored individually; whereas, the remaining samples were pooled into 3 batches

(phases: 20-40 minutes [P20-40], 60-120 minutes [P60-120], and 140-300 minutes post-exercise [P140-300]) for later hormone analysis.

### Biochemical analysis

Circulating concentrations of TT, FT, GH, COR, E2 (Monobind, Inc., Lake Forest, California), and IGF-1 (DRG International, Springfield, New Jersey) were determined using commercially available enzyme-linked immunosorbent assays according to manufacturer instructions. The samples were not decoded until after the analysis was completed (blinded analysis). All samples for a particular time point/phase were analyzed in duplicate within the same assay batch to eliminate potential inter-assay variance for a particular variable. Coefficient of variation for each assay were as follows: TT: 11.0%, FT: 12.7%, IGF-1: 4.6%, GH: 10.2%, COR: 8.7%, and E2: 20.4%.

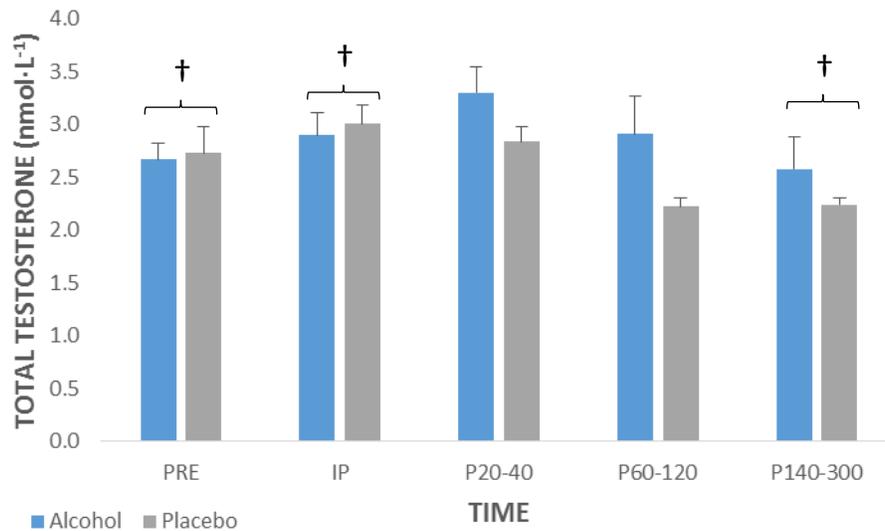
### Statistical analysis

Data were analyzed using a two-way ANOVA (treatment x time point) with repeated measures on both factors (IBM SPSS Statistics version 22, Chicago, IL). Results indicated that sphericity was violated for COR and therefore a Greenhouse-Geisser correction was used. The level of significance was set at  $p < 0.05$ . Where appropriate, Fisher's LSD *post-hoc* test was used to determine pair-wise differences. Data are presented as mean  $\pm$  standard deviation (SD) unless otherwise noted.

## RESULTS

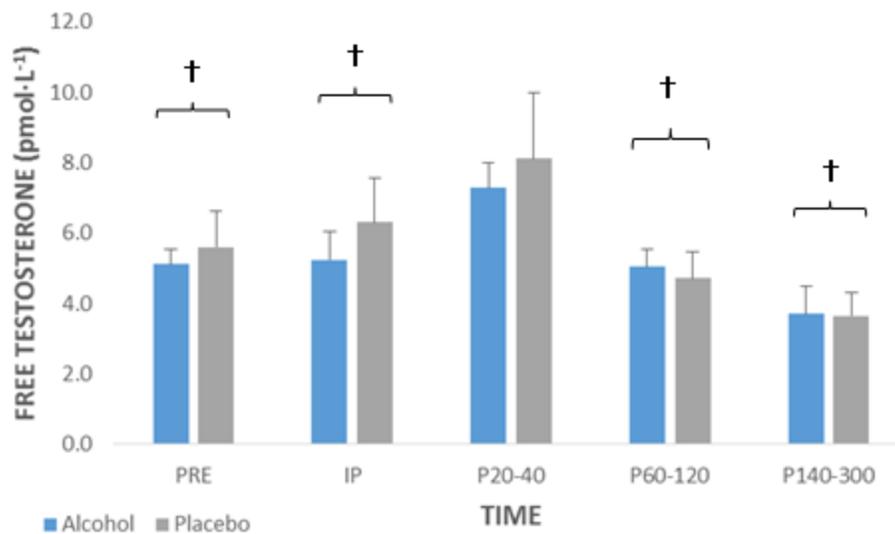
Two of the eight women presented for one of their experimental visits with E2 concentrations outside the normal range expected for the early follicular phase; additionally, those E2 values were markedly higher than their other experimental visit (>10 fold). It was thus determined that these two participants had not completed both exercise visits in the early follicular phase of their menstrual cycle and these participants were thus their data were omitted from analyses and results. Peak BEC was  $0.108 \pm 0.016 \text{ g}\cdot\text{dl}^{-1}$  and was achieved 60-90 min after alcohol ingestion.

Results for TT are presented in Figure 1. A significant main effect of time was found for TT,  $F(4, 20) = 4.62$ ,  $p = 0.008$ ,  $\eta^2 = 0.214$ . Circulating concentrations of TT were significantly greater at P20-40 than at PRE, P60-120, and P140-300. No main effect of condition was found,  $F(1, 5) = 1.78$ ,  $p = 0.239$ ,  $\eta^2 = 0.063$ , nor was an interaction effect found between time and condition,  $F(4, 20) = 1.84$ ,  $p = 0.161$ ,  $\eta^2 = 0.085$ .



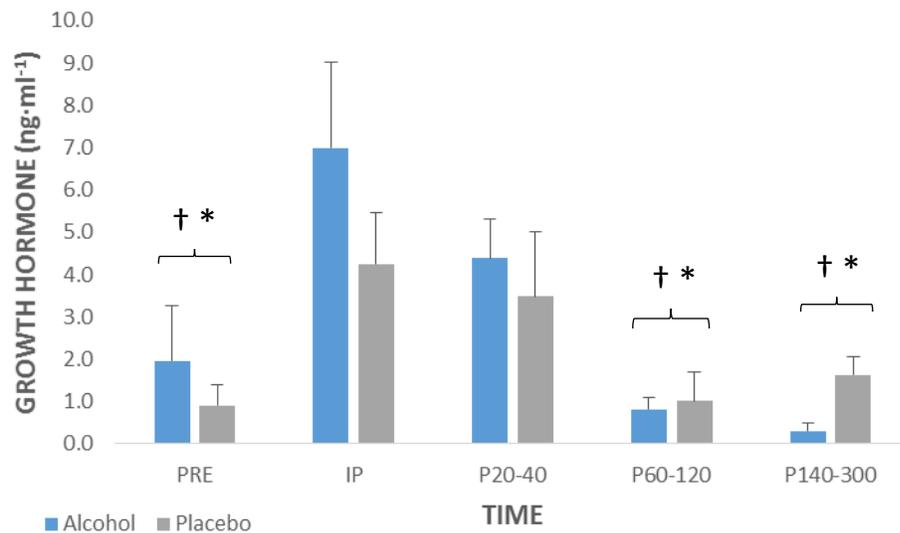
*Figure 1.* Total testosterone concentrations prior to exercise (PRE), immediately post exercise (IP), 20-40 minutes post exercise (P20-40), 60-120 minutes post exercise (P60-120), and 140-300 minutes post exercise (P140-300). † indicates main effect of time, significantly different ( $p \leq 0.05$ ) from P20-40. Data presented as Mean  $\pm$  SE.

Results for FT are presented in Figure 2. A significant main effect of time was found for FT,  $F(4, 20) = 7.53$ ,  $p = 0.001$ ,  $\eta^2 = 0.377$ . Circulating concentrations of FT were significantly greater at P20-40 than at PRE, IP, P60-120, and P140-300. No main effect of condition was found,  $F(1, 5) = 0.017$ ,  $p = 0.698$ ,  $\eta^2 = 0.009$ , nor was an interaction effect was found between time and condition,  $F(4, 20) = 0.810$ ,  $p = 0.535$ ,  $\eta^2 = 0.015$ .



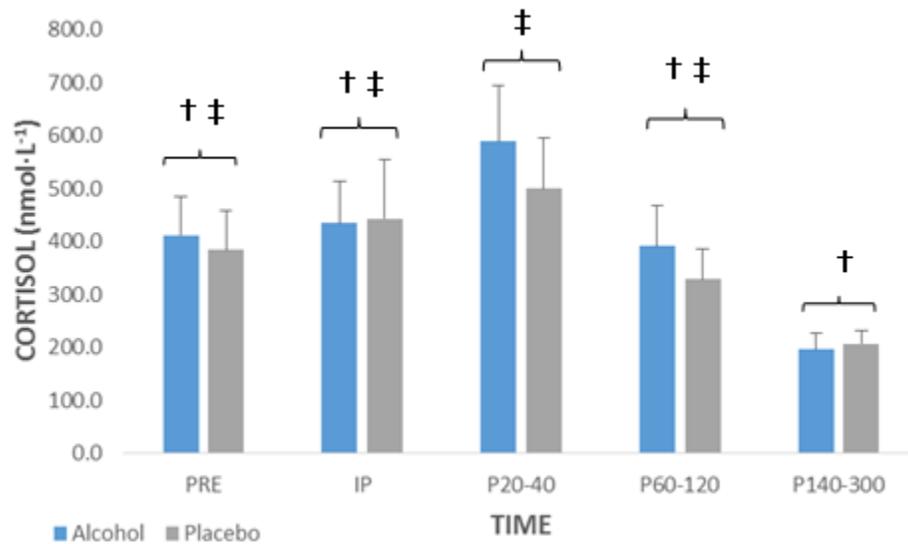
*Figure 2.* Free testosterone concentrations prior to exercise (PRE), immediately post exercise (IP), 20-40 minutes post exercise (P20-40), 60-120 minutes post exercise (P60-120), and 140-300 minutes post exercise (P140-300). † indicates main effect of time, significantly different ( $p \leq 0.05$ ) from P20-40. Data presented as Mean  $\pm$  SE.

Results for GH are presented in Figure 3. A significant main effect of time was found for GH,  $F(4, 20) = 8.669$ ,  $p < 0.001$ ,  $\eta^2 = 0.468$ . Circulating concentrations of GH were significantly greater at IP than at PRE, P60-120, and P140-300. Additionally, GH concentrations at P20-40 were significantly greater than at PRE, P60-120, and P140-300. No main effect of condition was found,  $F(1, 5) = 4.743$ ,  $p = 0.081$ ,  $\eta^2 = 0.013$ , nor was an interaction effect found between condition and time,  $F(4, 20) = 1.734$ ,  $p = 0.182$ ,  $\eta^2 = 0.061$ .



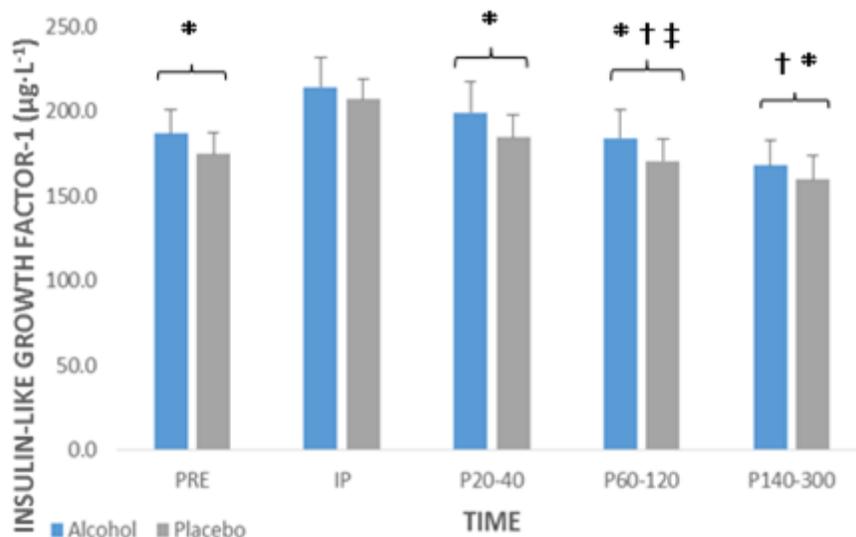
*Figure 3.* Human growth hormone concentrations prior to exercise (PRE), immediately post exercise (IP), 20-40 minutes post exercise (P20-40), 60-120 minutes post exercise (P60-120), and 140-300 minutes post exercise (P140-300). \* indicates main effect of time, significantly different ( $p \leq 0.05$ ) from IP; † indicates significantly different from P20-40. Data presented as Mean  $\pm$  SE.

Results for COR are presented in Figure 4. A significant main effect of time was found for COR,  $F(1.301, 6.504) = 13.57$ ,  $p = 0.007$ ,  $\eta^2 = 0.572$ . Circulating concentrations of COR were significantly greater at P20-40 than at PRE, IP, P60-120, and P140-300. Additionally, COR concentrations at P140-300 were significantly lower than at PRE, IP, P20-40, and P60-120. No main effect of condition was found,  $F(1, 5) = 0.484$ ,  $p = 0.518$ ,  $\eta^2 = 0.012$ , nor was an interaction effect found between condition and time,  $F(4, 20) = 1.337$ ,  $p = 0.291$ ,  $\eta^2 = 0.018$ .



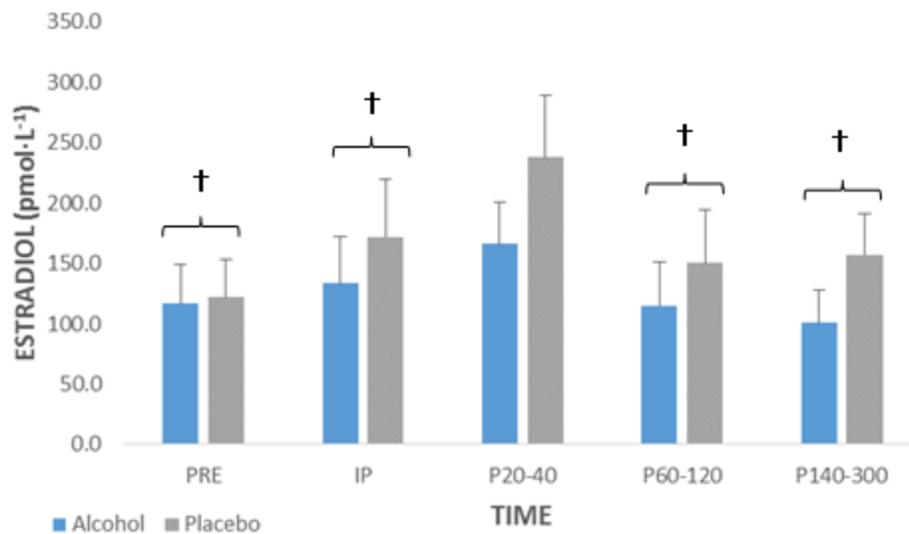
*Figure 4.* Cortisol concentrations prior to exercise (PRE), immediately post exercise (IP), 20-40 minutes post exercise (P20-40), 60-120 minutes post exercise (P60-120), and 140-300 minutes post exercise (P140-300). † indicates main effect of time, significantly different ( $p \leq 0.05$ ) from P20-40; ‡ indicates significantly different from P140-300. Data presented as Mean  $\pm$  SE.

Results for IGF-1 are presented in Figure 5. A significant main effect of time was found for IGF-1,  $F(4, 24) = 25.810$ ,  $p < 0.001$ ,  $\eta^2 = 0.601$ . Circulating concentrations of IGF-1 were significantly greater at IP than at PRE, P20-40, P60-120, and P140-300. Additionally, IGF-1 concentrations at P20-40 were greater than at P60-120 and P140-300. Finally, IGF-1 concentrations at P140-300 were significantly lower than at IP, P20-40, and P60-120. No main effect of condition was found,  $F(1, 5) = 2.360$ ,  $p = 0.185$ ,  $\eta^2 = 0.075$ , nor was an interaction effect was found between time and condition,  $F(4, 20) = 0.638$ ,  $p = 0.641$ ,  $\eta^2 = 0.006$ .



*Figure 5.* Insulin-like growth factor-1 concentrations prior to exercise (PRE), immediately post exercise (IP), 20-40 minutes post exercise (P20-40), 60-120 minutes post exercise (P60-120), and 140-300 minutes post exercise (P140-300). \* indicates main effect of time, significantly different ( $p \leq 0.05$ ) from IP; † indicates significantly different from P20-40; ‡ indicates significantly different from P140-300. Data presented as Mean  $\pm$  SE.

Results for E2 are presented in Figure 6. A significant main effect of time was found for E2,  $F(4, 20) = 18.263$ ,  $p < 0.001$ ,  $\eta^2 = 0.298$ . Circulating concentrations of E2 were significantly greater at P20-40 than at PRE, IP, P60-120, and P140-300. No main effect of condition was found,  $F(1, 5) = 2.446$ ,  $p = 0.179$ ,  $\eta^2 = 0.147$ , nor was an interaction effect was found between time and condition,  $F(4, 20) = 1.631$ ,  $p = 0.205$ ,  $\eta^2 = 0.043$ .



*Figure 6.* Estradiol concentrations prior to exercise (PRE), immediately post exercise (IP), 20-40 minutes post exercise (P20-40), 60-120 minutes post exercise (P60-120), and 140-300 minutes post exercise (P140-300). † indicates main effect of time, significantly different ( $p \leq 0.05$ ) from P20-40. Data presented as Mean  $\pm$  SE.

## DISCUSSION

This study is the first to examine the acute hormonal response to ethanol ingestion following a heavy resistance exercise bout in women. Resistance exercise induced significant increases in circulating concentrations of TT, FT, COR, GH, IGF-1, and E2 measurements within the first 40 minutes into recovery; subsequently, concentrations decreased until at or below PRE concentrations by the end of the visit (5 hours following exercise). No significant differences were found between placebo and ethanol ingestion conditions for any of the hormones measured; only small to trivial effect sizes were noted between conditions. Thus, ethanol ingestion did not appear to affect the acute hormonal response to the resistance exercise bout in women.

Some, but not all, studies have demonstrated an acute increase in TT following heavy resistance exercise in women (9, 22, 46). Nindl et al. (46) found a significant increase in serum TT following heavy resistance exercise in women. The present study used a heavy resistance exercise protocol identical to Nindl et al. (six sets of ten repetitions of smith machine squats with 2 minutes of rest) and found a post-exercise increase in TT at P20-40, although smaller in percent change than that found by Nindl et al. (~10% vs. 25%). However, other studies (32, 38, 41) have found no acute increase in TT following heavy resistance exercise (although it is important to note that the exercise protocols in these studies varied and was not the same protocol used in the present study). The inconsistency of findings for post-resistance exercise changes in TT for women has not yet been resolved. In contrast to men who have designated glands (Leydig cells) for testosterone production, testosterone in women is largely derived from spillover from production of other hormones in the adrenal glands and ovaries; thus there is no dedicated tissue to provide for large increases in TT with resistance exercise. Furthermore, it is

likely that differences in research design (i.e., selection among the acute program variables) and low sample sizes (69) contributes to these divergent findings. Several studies have reported acute elevations in TT following ethanol consumption without exercise in women (19, 27, 56) in comparison to a non-ethanol consuming placebo group. It has been suggested that the increase in the ratio of NADH to NAD<sup>+</sup> concomitant with acute ethanol consumption results in a secondary shift in the androstenedione to TT equilibrium (mediated by 17 $\beta$ -hydroxysteroid dehydrogenase), thus leading to elevated TT (57). In the present study, there was no significant difference between ethanol and placebo conditions for TT concentrations. Based on the independent effects of resistance exercise and ethanol ingestion on TT, an additive effect of ethanol and resistance exercise on TT in women was expected; however, this hypothesis is not supported by the present data as no effect of ethanol ingestion was observed.

The vast majority of testosterone circulates in the blood bound to binding proteins (i.e., sex-binding hormone globulin, albumin) which inhibits testosterone interaction with its cytosolic receptor, the androgen receptor. Only 0.5-2% of TT circulates as FT (unbound); FT is the most biologically active fraction of TT and thus changes in FT might be more meaningful than changes in TT. Only a few studies have examined the FT response to resistance exercise in women (22, 46, 66). Similar to previous findings for acute increases (~22-25%) in FT concentrations following 6 sets of heavy squats in women (46, 69), the present study found that FT was increased approximately 43% (from PRE to P20-40) following the resistance exercise bout with no differences between beverage conditions. In men, some studies have suggested that the FT response mirrors that of the TT response (1, 15, 62). Due to the equivocal findings for acute changes in TT and FT following resistance exercise in women, this relationship has not yet been well established. Although the physiological significance of this acute increase is still under

intense academic debate, in concert with the findings for TT, the present study supports the notion of a mirrored FT/TT response that has been suggested to occur.

Immediately following heavy resistance exercise, the concentration of circulating GH is typically elevated, the magnitude of which is determined by manipulation of the acute program variables (volume, intensity, rest periods, etc.). Generally, it is thought that a chemoreceptive reflex, mediated by intramuscular metaboreceptors in response to decreased pH, potentiates GH release (59, 65). Unsurprisingly, the results of this study corroborate the well-established resistance exercised-induced increase in GH (21, 34); although pH was not measured in the current study, the metabolic demand of the exercise regimen used is known to induce high lactate production (67) and is thus likely associated with a reduction in muscle pH. No effect of ethanol on the GH response to resistance exercise was observed. Since ethanol can have a suppressive effect on the GH axis, potentially at the hypothalamic level (13, 50, 58, 64), it is somewhat surprising to find a lack of difference between conditions. However, it is possible that the temporal relationship of the two divergent responses (GH response to exercise vs. GH response to ethanol ingestion) resulted in a lack of difference in the immediate hormonal milieu. Specifically, it is likely that the exercise-induced sympathetic response, resulting in hypothalamic GH release via chemoreceptive reflex, occurred prior to a potential ethanol-induced suppressive effect on GH release. Additionally, it is possible that the consequences of ethanol ingestion following resistance exercise on the GH-axis might not be fully experienced until after 5 hours following ingestion. Ethanol is known to suppress nocturnal GH-release, even when ethanol is no longer detectable in the serum (13, 17). As resistance exercise also influences nocturnal GH concentrations and patterns of release (45), it is thus possible that ethanol ingestion

following a resistance exercise bout could influence GH-axis characteristics over the course of a longer time period, outside the present study's sampling window.

Like the GH response, the acute COR response to resistance exercise depends on manipulation of the acute program variables - specifically, a greater quantity of physiological work done in a shorter period of time will elicit a greater COR response. In the present study, COR reached its highest measured concentration at P20-40 and then decreased to below PRE values at P60-120 and P140-300, with no differences between conditions. The physiological consequences of ethanol consumption on COR are not fully understood. While cross-sectional investigations suggest a chronic elevation of cortisol in response to regular ethanol (possibly beverage-specific) consumption (4, 39), some prior research has not demonstrated differences in the acute COR response to ethanol or placebo conditions (26, 55, 71) in men. Vingren et al. (67) examined the effects of ethanol on the acute response to an AHRET (the same exercise protocol used in the present study) in men and found no differences in COR response between conditions. However, Koziris et al. (28) previously noted that ethanol prolonged the post-exercise increase in COR in conjunction with heavy resistance exercise in men. Although similar in overall design (except for exercise protocol) to that of Koziris et al., the present study did not find a prolonged cortisol response. It is important to note that, in contrast to the present study as well as the study conducted by Vingren et al., in the investigation by Koziris et al, the participants completed the exercise visits after an overnight fast and did not consume a standardized meal prior to the exercise session. It has been shown that, when fasted, COR concentrations in the 4h recovery period following endurance exercise are decreased (when compared to a carbohydrate consuming control) (11). Although it is possible that the differences in exercise protocol could explain the differences in the acute COR response, it is likely that the effect of ethanol on COR

concentrations were ablated by the consumption of a standardized meal. Finally, the present study does not intend to suggest that acute ethanol consumption does not affect COR; it is possible that the interaction of ethanol and heavy resistance exercise affected COR production outside of the present study's sampling window, as prior research in the absence of exercise has found delayed elevations in COR in response to ethanol eight hours following consumption (16).

Evolutionarily conserved and structurally related to insulin, the Insulin-like Growth Factor polypeptides, especially IGF-1, are critical for proper somatic growth and development (14). The IGF-1 response to heavy resistance exercise alone remains unclear, where some studies have shown no change in IGF-1 concentrations immediately following resistance exercise (7, 30, 33) and others have shown an acute increase (31, 53). The lack of immediate increases in IGF-1 found in some studies has been attributed to delayed systemic IGF-1 secretion (from liver hepatocytes) following GH-stimulated IGF-1 mRNA synthesis (mediated via ERK, PI3K/AKT, and JAK-STAT pathways acting on the IGF-1 gene) 3-9h after GH-stimulation (32). In the present study, there was a significant increase from PRE to IP (17.0%) in circulating IGF-1 concentrations; no difference between conditions was observed. Given the delayed release of IGF-1 secretion upon stimulation by GH, it is not clear why such increases in IGF-1 were found. However, the present exercise protocol was identical to that used by Rubin et al. (53), and even though they used only men, our data corroborates their findings for IGF-1. Independent of exercise, circulating concentrations of IGF-1 decrease in women (during all phases of the menstrual cycle) as a result of ethanol ingestion (36), although at least one study has found no effect of ethanol intoxication on IGF-1 concentrations (20). In the present study, IGF-1 peaked at IP and decreased to approximately PRE values by P60-120 with no differences between

conditions. The effect of ethanol thus appears to have been abolished by the acute physiological perturbations induced by the resistance exercise bout.

Estrogens, particularly E2, modulate a number of biological processes, in addition to those involved in the female reproductive cycle, and appear to be important in the maintenance of bone density (by reduction of bone resorption) (63) and, while yet somewhat controversial in humans, potentially skeletal muscle size, force generation, and the muscle damage/repair process (see (18, 60) for review on the potential protective effects of estrogens on muscle). Although E2's protective effect on human muscle has not been fully established, mechanisms of this protective effect have been suggested to include: antioxidant properties (E2 possesses a hydroxyl group on its phenol group, which likely donates a hydrogen atom and allows for free radical scavenging during times of oxidative stress) (2); membrane stabilization via intercalation with membrane phospholipids (73); and estrogen receptor modulation of downstream gene/molecular targets (48, 61). Although few studies have examined the acute E2 response to heavy resistance exercise, it appears that, irrespective of the phase of the menstrual cycle, E2 acutely increases following resistance exercise (9, 30, 70). In the present study, E2 was acutely increased at IP, corroborating previous resistance exercise studies' results. In isolation, ethanol ingestion results in acutely increased E2 concentrations (42). As hepatic ethanol metabolism decreases NAD<sup>+</sup> availability, it has been suggested that E2 levels rise as a result of reduced E2 to estrone conversion (NAD<sup>+</sup> is a cofactor in 17 $\beta$ -HSD-mediated E2 to estrone conversion) (49, 54). The present study found no differences in E2 concentrations between conditions. It is not clear why ethanol did not augment the E2 response to resistance exercise. However, there are presently no established mechanisms as to how resistance exercise acutely increases E2 concentrations; thus,

it is difficult to speculate on a potential reason for the lack of an effect of ethanol on the acute E2 response to resistance exercise.

In summary, the present study demonstrated an acute modulation of the neuroendocrine milieu following a heavy resistance exercise bout in women. Ethanol ingestion appeared to have no significant effect on the characteristics of acute hormonal augmentation in TT, FT, GH, COR, IGF-1, or E2. As both resistance exercise and ethanol ingestion have potent, generally disparate, acute effects on hormonal concentrations, the lack of differences between ethanol and placebo conditions was surprising. While the data do not indicate any immediate influence of ethanol consumption on endocrine milieu, these findings should not be interpreted to mean that there are no negative effects of ethanol ingestion following a bout of resistance exercise. More research on the effects of ethanol, especially in women, is needed before conclusions on overall effects of ethanol ingestion on resistance exercise adaptations can be made.

## PRACTICAL APPLICATIONS

This study examined the influence of ethanol ingestion on the acute hormonal response to heavy resistance exercise in women. As ethanol consumption is a common social activity that athletes engage in regularly, it is important for the coach and practitioner to understand its implications for physiological adaptation. In this study, ethanol did not appear to have an acute effect on the hormonal environment following resistance exercise. This lack of findings does not, however, mean that ethanol ingestion following resistance exercise would have no effect on subsequent resistance training adaptations. Thus, the coach should still recommend caution when advising athletes on drinking behaviors following exercise.

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