The effects of ethanol vary and can depend on the extent of its consumption and environmental context. In addition to its acute effects, ethanol can impede physical performance when its consumption is of a chronically abusive nature, i.e., alcoholism. It has been known for some time that individuals diagnosed with alcohol dependence have displayed various degrees of muscle damage and weakness (26, 37).

The neuroendocrine system, and specifically the interaction of various anabolic (14) and catabolic hormones (2, 19), is part of the response to resistance exercise. This system, which has a role in the maintenance and adaptation of muscular tissue and strength, is also among the many systems affected by acute (1, 27, 32) and chronic ethanol abuse (28, 32).

With regard to the pituitary-adrenocortical axis, increased postexercise cortisol concentrations have been shown consequent to acute resistance exercise (15, 17). Ethanol ingestion results in elevated circulating cortisol concentrations, and there is apparently a dose-related effect (13, 32). Although there is evidence to suggest that the effect of ethanol on this axis occurs via corticotropin-releasing hormone (CRH) secretion from hypothalamic neurons (35) via adrenergic stimulation of higher centers (41), there is evidence to indicate that ethanol’s effect is instead mediated via an increase in ACTH release from the anterior pituitary gland (18, 33).

In the pituitary-gonadal axis of men, testosterone is thought to have a role in the adaptation to resistance exercise, inasmuch as its serum concentration is increased in response to various resistance exercise protocols (15, 17, 22). Serum testosterone concentration is lowered for up to several hours after acute ethanol administration (18, 29). Although the majority of studies involving humans show no ethanol effect on serum luteinizing hormone (LH), some data have demonstrated an increase while others have supported a decrease (16, 29).

Also, as a result of increased adrenomedullary and sympathetic nervous system stimulation, resistance exercise results in increased 5-min postexercise catecholamine concentrations (23, 25). Urinary and plasma concentrations of catecholamines are increased in response to acute ethanol administration (1, 34). In addition to a direct effect on adrenomedullary activity, a centrally mediated effect may contribute to the sympathoadrenal response to ethanol ingestion.

The purpose of this investigation was to examine the pituitary-adrenal axis, the pituitary-gonadal axis, and the sympathoadrenal system over a prolonged time frame after a resistance exercise session and to determine the effect of postexercise acute ethanol intoxication on this response. Findings could have implications for coaches and athletes interested in the effects of ethanol intoxication on physical conditioning.
METHODS

Subjects. Nine 21- to 34-yr-old men (25.0 ± 1.4 yr, 179.4 ± 3.4 cm, 79.7 ± 3.3 kg, 10.8 ± 1.8% fat) participated in the study. They formed a highly selected group by undergoing multiple-level screening to control for potential confounding variables. The subjects were in good health and were cleared for participation by a physician. They were free of pathological or relevant orthopedic conditions. Additionally, they attested that they had not used tobacco products during the last year and were tolerant to venipuncture. Subjects also attested that they had not taken any drugs or medication during the 2 mo before the first treatment, other than headache medicine on an infrequent basis (up to 3 times/wk). Men using certain drugs such as anabolic-androgenic steroids or glucocorticoids within 1 yr before the study were not allowed to participate. Subjects were excluded from participation if they were attempting to lose weight or if their diet was restricted in calories, carbohydrates, or protein. Subjects were also required to meet a set of criteria to be considered recreationally resistance trained for the study.

Furthermore, the subjects were required to complete a series of questionnaires regarding their historic and current use of ethanol. Additional criteria for participation in the study consisted of a lack of clinical signs of ethanol abuse, an 88- to 89-h ethanol restriction period before the first treatment, and an absence of blood ethanol during each laboratory visit. The subjects who met these criteria were considered to be low-to-moderate ethanol consumers according to the US Department of Health and Human Services (38), to not have consumption-induced metabolic tolerance to ethanol, to not be alcohol dependent, and to be able to tolerate the ethanol ingestion in this study without any extreme effects such as nausea or flushing.

Subjects provided written informed consent, and the study was approved by the University Institutional Review Board for Use of Human Subjects. To prevent subjects from anticipating a particular treatment, they were told that they may potentially receive the same type of treatment on as many as two occasions.

Experimental treatments. Each subject participated in three experimental treatments and thus served as his own control. Treatments were scheduled at 1-wk intervals and were administered in a randomized-block crossover design to minimize interference effects such as order of carryover effects. To minimize anticipatory effects on some of the dependent variables, subjects were blind to the experimental condition selected for that treatment day until its administration. The subjects were not told if they would be participating in a condition on that day that included an exercise session until the start of the exercise period of a treatment day, to not consume caffeine or participate in sexual activity during the previous 24 h, to not exercise as of 10 PM 3 nights earlier (other than some strength testing that was part of another aspect of the project), to not consume any nonstudy ethanol from 4 nights before the first treatment until the end of their participation, and to not take any drugs or medication not approved by the physician overseeing the project. Subjects were also required to not have donated plasma for 96 h and blood for 8 wk before any laboratory visit. Subjects attested that all their records were accurate and that they had adhered to all instructions.

Table 1 provides an overview of a treatment day. Subjects were screened for blood ethanol and via questionnaire for the restrictions regarding diet, sleep, caffeine, sexual activity, exercise, ethanol, and drugs. They brought a 24-h urine collection to the laboratory and answered questions regarding the collection methods and record keeping. They were also questioned regarding any changes in their health status since the medical examination or the previous treatment day. Subjects were asked to sign a form affirming this information. Subjects then drank an appropriate volume of cold water to bring their cumulative (8 AM to reporting time) water intake volume to 1.18 ml/kg (e.g., 770 ml for a 2:30 PM reporting time).

Table 1. Sample treatment day

<table>
<thead>
<tr>
<th>Time Line, min</th>
<th>Time, PM</th>
<th>Activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>−45−90</td>
<td>2:30−3:15</td>
<td>Arrival, Breathalyzer for BEC; change into athletic attire; body mass; questionnaire</td>
</tr>
<tr>
<td>−45</td>
<td>3:15</td>
<td>Cannulation</td>
</tr>
<tr>
<td>−15</td>
<td>3:45</td>
<td>6.0-ml blood sample after 20−30 min of equilibration</td>
</tr>
<tr>
<td>0</td>
<td>4:00−4:45</td>
<td>Exercise or sit quietly</td>
</tr>
<tr>
<td>+3−12</td>
<td>4:48−4:57</td>
<td>Ingestion period</td>
</tr>
<tr>
<td>+20</td>
<td>5:05</td>
<td>10.5-ml blood sample</td>
</tr>
<tr>
<td>≥30</td>
<td>≥5:15</td>
<td>BEC (every 5 min until peak is established)</td>
</tr>
<tr>
<td>+40</td>
<td>5:55</td>
<td>10.5-ml blood sample; BEC</td>
</tr>
<tr>
<td>+60</td>
<td>5:45</td>
<td>10.5-ml blood sample; BEC</td>
</tr>
<tr>
<td>+80</td>
<td>6:05</td>
<td>6.0-ml blood sample; BEC</td>
</tr>
<tr>
<td>+100</td>
<td>6:25</td>
<td>6.0-ml blood sample; BEC</td>
</tr>
<tr>
<td>+120</td>
<td>6:45</td>
<td>6.0-ml blood sample; BEC</td>
</tr>
<tr>
<td>+123−137</td>
<td>6:48−7:02</td>
<td>Standardized meal: moderate-carbohydrate commercially available frozen dinner</td>
</tr>
<tr>
<td>+140</td>
<td>7:05</td>
<td>5.0-ml blood sample; BEC</td>
</tr>
<tr>
<td>+160</td>
<td>7:25</td>
<td>5.0-ml blood sample; BEC</td>
</tr>
<tr>
<td>+180</td>
<td>7:45</td>
<td>5.0-ml blood sample; BEC</td>
</tr>
<tr>
<td>+200</td>
<td>8:05</td>
<td>5.0-ml blood sample; BEC</td>
</tr>
<tr>
<td>+220</td>
<td>8:25</td>
<td>5.0-ml blood sample; BEC</td>
</tr>
<tr>
<td>+240</td>
<td>8:45</td>
<td>5.0-ml blood sample; BEC</td>
</tr>
<tr>
<td>+260</td>
<td>9:05</td>
<td>5.0-ml blood sample; BEC</td>
</tr>
<tr>
<td>+280</td>
<td>9:25</td>
<td>5.0-ml blood sample; BEC</td>
</tr>
<tr>
<td>+300</td>
<td>9:45</td>
<td>5.0-ml blood sample; BEC</td>
</tr>
<tr>
<td>+305</td>
<td>9:50</td>
<td>Body mass</td>
</tr>
<tr>
<td>&gt;305</td>
<td>&gt;9:50</td>
<td>Additional BECs (until departure criterion reached)</td>
</tr>
</tbody>
</table>

BEC, blood ethanol concentration.
Furthermore, subjects ingested 70 ml of water per 15-min interval during a period of 75 min immediately before the start of the exercise period and per 20-min interval from 140 to 300 min after the exercise period. The same time line was followed at the same time of day for all conditions. Once subjects completed their exercise-or-sit-quietly period, they sat quietly in a chair for the rest of the evening and were not permitted to sleep. Blood samples were drawn and blood ethanol concentration (BEC) was determined according to the time line shown in Table 1. To prevent boredom and anxiety, the subjects were permitted to read or write during times when they sat quietly. Subjects were asked to not become involved in any reading or writing that may cause them to experience any stress or any strong feelings, whether positive or negative. Music was played on the radio throughout the treatments and was restricted to certain “easy-listening/pop” stations.

Resistance exercise protocol. The protocol began with a standardized 5-min cycle ergometer warm-up. Universal machines (Universal Gym Equipment, Cedar Rapids, IA) were then used to exercise major muscle groups of the body. Subjects performed one set of each exercise and proceeded to the next exercise with 1-min rest intervals between sets. Each circuit consisted of the following order: high pull, toe press, bench press, leg press 1, wrist curl, shoulder press, leg press 2. The first circuit consisted of 10-repetition warm-up sets with 50% of the 5-repetition maximum (RM) load. During the remaining three circuits, 5-RM loads were used for all exercises, and subjects were asked to complete as many repetitions as possible per set until concentric fatigue. Forced repetitions, if necessary, were used to complete five repetitions. The subjects attempted to move the load as rapidly as possible during each concentric phase and controlled its descent during a 1- to 2-s eccentric phase for each repetition, depending on the distance involved. No bouncing of the weight stack and no interrepetition pausing were permitted. Target body positions were set and followed to control the range of motion for each exercise among subjects. This uniformity aided in ensuring that a similar total work was performed in the two exercise sessions. Previous research has shown that this type of resistance exercise protocol is well tolerated by recreational-level resistance-trained individuals (22). During the control treatment, subjects sat quietly during the “exercise” period.

During a familiarization exercise session involving a protocol similar to that described above, a 5-RM load was determined for each exercise. Subjects were asked to report in a euhydrated state (cumulative water intake volume of 118 ml/h). A body mass measurement was obtained before the subjects started this exercise session, after water intake status was addressed.

Ethanol ingestion. A peak BEC of 0.10 g/dl (21.75 mmol/l) was targeted as a group goal. A dose of 1.09 ml of 95% USP grade ethanol (Everclear grain alcohol, World Wide Distilled Products, St. Louis, MO) per kilogram of body mass (equal to 0.83 g ethanol/kg body mass) was determined and was fine-tuned for each individual by applying a correction for the amount of fat-free mass above or below an average of 85%. The ethanol dose was diluted to a 30% concentration of absolute ethanol. The mix was a sugar-free, noncaffeinated beverage that was sweetened with NutraSweet. The subjects drank 1 of 10 equal parts each minute during the drink period. During control and Ex, the subjects ingested an isovolumetric drink using the same protocol with the volume of ethanol replaced by water. The majority of subjects achieved their peak BEC within 1 h (57 ± 5 min) after the end of the drink period, and all subjects did so by 90 min. The peak BEC in this investigation was 21.97 ± 1.09 mmol/l (0.101 ± 0.005 g/dl).

Blood ethanol. BEC was estimated from breath ethanol content with an Intoximeter 3000 (Intoximeters, St. Louis, MO). This instrument has a detection limit of 0.001 g/dl (0.22 mmol/l) with an accuracy of ± 0.005 g/dl (1.09 mmol/l). It was calibrated against standards after each breath sample. The schedule of BEC measurements is displayed in Table 1. At no time were subjects allowed to know their BEC.

Morning sessions. Subjects reported on the morning of days 2 and 3 of each treatment after an overnight fast and without having consumed any caffeine, ethanol, or drugs; having had any physical exercise or sexual activity; or having donated blood or plasma since their laboratory visit of the previous day. They delivered their urine collection and answered questions regarding the urine collection methods and record keeping. They were questioned about any changes in their health status since the previous laboratory visit. Subjects were then screened for blood ethanol and weighed. Blood samples were drawn at a similar time for each subject across the duration of the study. None of the subjects indicated any sign of a hangover according to the hangover sign index. Subjects were not allowed to discuss any aspect of the study (e.g., previous night’s treatment session or sleep) with other subjects or the staff conducting the testing or blood sampling.

Blood collection. On the treatment day, a Teflon cannula (Vascular Access, Becton-Dickinson, Sandy, UT) was inserted in a superficial forearm vein while the subject was seated. Cannula patency was maintained with a saline lock (0.9% sodium chloride USP, Baxter Healthcare, Deerfield, IL), and while the subject was seated, blood was drawn using a single-use injection site (CharterMed, Lakewood, N J) and a single-use syringe (Becton-Dickinson). The injection site and cannula were flushed on two occasions during the exercise period and at 10-min intervals at all other times. Each treatment-day blood sample volume was replaced with saline. The frequency and timing of the blood samples are listed in Table 1.

During the morning sessions, a needle and evacuated tube blood collection system (Vacutainer, Becton-Dickinson, Rutherford, N J) were used to obtain two 1.5-ml blood samples 20 min apart from a superficial forearm vein. Blood was not drawn for ACTH, LH, and catecholamine determination during the morning sessions.

Urine volume. Urine volumes were measured in case they provided an explanation for a greater plasma volume change during ExE. Urine was collected by the subject for two 24-h periods per treatment starting on the morning of the treatment day. For each 24-h period, collection began after the bladder was voided on awakening on the start day and ended with the first urination, inclusive, on the morning of the finish day.

Blood processing. Once a sample was collected, the blood was placed into collection tubes with appropriate preservatives where necessary and processed for the various biochemical analyses. Blood for serum analyses was allowed to clot at room temperature. Blood for plasma ACTH analyses was mixed with an EDTA solution in chilled glass tubes. Blood for plasma catecholamine analyses was collected in a heparinized tube and mixed with 80 µl of an EGTA-reduced glutathione solution in a chilled glass tube. Blood for whole blood analyses was collected in a heparinized tube. All blood for serum or plasma analyses was then centrifuged at 1,500 g for 15 min at 4°C. The resultant serum or plasma was extracted and stored in Eppendorf microtubes. All aliquots requiring storage were kept at −80°C until the time of analysis and were thawed only once. The pretreatment, immediately post-
exercise, and 20-, 40-, 60-, 80-, 100-, and 120-min postexercise blood samples were analyzed individually. The latter six of these values were subsequently averaged into two phases. The nine serum samples drawn every 20 min beginning at 140 min after exercise were pooled before freezing and analysis. Also pooled before freezing and analysis were the two serum samples within each of the day 2 and 3 mornings.

Biochemical analyses. All samples of a variable for any one subject were analyzed in duplicate within the same assay to eliminate the effect of interassay variance. All inter- and intra-assay variances were well within the laboratory limit of 5%. All samples were decoded only after analyses were completed (blinded analysis). Serum lactate (only immediately after exercise) was analyzed to characterize the metabolic demands of the exercise. An enzymatic-amperometric method (lactate analyzer, Yellow Springs Instruments) was used for serum lactate analysis. Blood was analyzed for Hb by the cyanmethemoglobin method (Sigma Chemical, St. Louis, MO). A small portion of the heparinized whole blood aliquot was analyzed immediately for hematocrit by standard microcapillary technique. Plasma volume change (percent) was calculated using the hematocrit and Hb values (9). Analysis of the various hormone concentrations was performed with 125I RIA techniques and a gamma counter (model 1272 Clinigamma, LKB) and on-line data reduction computer. Concentrations of serum cortisol and testosterone were measured using a single-antibody solid-phase RIA (Diagnostic Products, Los Angeles, CA). Plasma ACTH and serum LH were measured using a liquid-phase RIA with double-antibody technique (Diagnostic Products). Detection limits for the RIAs are 5.5 nmol/l for cortisol, 0.14 nmol/l for testosterone, 1.8 pmol/l for ACTH, and 1.0 IU/l for LH. Plasma catecholamines were determined using a reverse-phase HPLC system with electrochemical detection (BAS 200A, Bioanalytical Systems, W. Lafayette, IN), refrigerated autosampling (Waters 712, Milipore, Milford, MA), and a preliminary aluminum oxide extraction procedure with a Waters 40520 kit (Milipore). Plasma filtrate was injected (60 µl) onto a reverse-phase plasma catecholamine ODS 5-µm column (Bioanalytical Systems, W. Lafayette, IN). Mobile phase (0.15 mmol/l monochloroacetate buffer, pH 3.0, containing 2.0 mmol/l Na2EDTA and 25–30 mg/l sodium octyl sulfate) was pumped at a flow rate of 2.2 ml/min. ChromGraph computer programs (Bioanalytical Systems) were used to process and quantitate the data.

Statistical analyses. Descriptive data (means ± SE) were calculated for all measured variables. One-way ANOVAs with repeated measures were used. Where appropriate, Fisher's protected least significant difference tests were used for all pairwise comparisons of treatment conditions within each timepoint. P ≤ 0.05 was chosen as the α-level of significance.

RESULTS

Data were analyzed within the specific phases of the study. These consisted of averaged values at 20–40 min after exercise (day 1), averaged values at 60–120 min after exercise (day 1), and single pooled-serum values at 140–300 min after exercise (day 1), as well as in the morning on days 2 and 3. Data immediately after exercise are also presented.

Body mass was similar for all treatment and test days and did not change >0.3% from the start to the end of the treatment day (Table 2). The resistance exercise session resulted in a similar response in each of the two treatments involving the exercise. There was no difference between Ex and ExEt immediately after exercise for any of the study variables as well as for serum lactate concentration (12.5 ± 2.1 and 14.0 ± 1.7 for Ex and ExEt, respectively) and plasma volume change (−15.3 ± 2.4 and −13.0 ± 2.6 for Ex and ExEt, respectively). Furthermore, there was no difference among the three treatments for variables measured before exercise.

Serum cortisol concentrations are illustrated in Fig. 1. Compared with the control treatment, the concentration was elevated for Ex and ExEt immediately after exercise and at 20–40 min. Only that for ExEt was elevated at 60–120 min. Plasma ACTH concentration, shown in Fig. 2, was elevated immediately after exercise for Ex and ExEt.

Serum testosterone and LH concentrations are shown in Figs. 3 and 4, respectively. At 60–120 min, testosterone concentration was lower in the Ex than in the control condition. None of the LH concentrations were significantly different among the treatments.

Plasma epinephrine concentration (Fig. 5) was greater for Ex and ExEt than for control immediately after exercise and at 20–60 min. For plasma norepinephrine concentration (Fig. 6), the exercise-induced rise was observed only immediately after exercise.

Table 2. Body mass

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Day</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>−1</td>
</tr>
<tr>
<td>Ex</td>
<td>79.6±3.4</td>
</tr>
<tr>
<td>ExEt</td>
<td>79.8±3.3</td>
</tr>
<tr>
<td>Ctrl</td>
<td>79.8±3.3</td>
</tr>
</tbody>
</table>

Values are means ± SE in kg. Ex, exercise; ExEt, exercise + ethanol; Ctrl, control. Day −1, day before treatment.

Fig. 1. Serum cortisol concentrations immediately after exercise (IP), 20–40 (1A), 60–120 (1B), and 140–300 min after exercise (1C), and in morning on days 2 (2) and 3 (3) for control (Ctrl), exercise (Ex), and exercise + ethanol (ExEt) treatments. Values are means ± SE.

* Different from corresponding Ctrl value: P ≤ 0.05.
DISCUSSION

This investigation was conducted to determine the effect of postexercise ethanol intoxication on the response of selected aspects of the neuroendocrine system to a resistance exercise (Ex) session. The major findings were that ethanol did not increase circulating cortisol concentration above that caused by the resistance exercise, but ethanol appeared to have a more prolonged effect. Future study should further explore this area with different BECs. Also, the response of the pituitary-gonadal axis and of circulating catecholamine concentrations to the resistance exercise was similar with or without intoxication.

Serum cortisol and plasma ACTH concentrations were determined to examine the effect of ethanol intoxication on the response of the pituitary-adrenocortical axis to resistance exercise. The resistance exercise protocol caused an average increase of 67% in circulating cortisol immediately after exercise above the control concentration at the same time of day. The plasma volume shift accounted for only 14% of this change, indicating a perturbation in the balance between the release and clearance of cortisol. For the exercise-only treatment (Ex), this effect extended to 20–40 min after exercise. Although a variety of resistance exercise protocols have resulted in increased peripheral concentrations of cortisol (15, 17), a protocol similar to that used in the present study (5 RM, 1-min rest) did not have this effect compared with other protocols in men (20) but did have this effect compared with similar protocols in women (21). Differences in response may be attributed to vari-

Fig. 2. Plasma ACTH concentrations immediately after exercise (IP) and 20–40 (1A), 60–120 (1B), and 140–300 min after exercise (1C) for Ctrl, Ex, and ExEt treatments. Values are means ± SE. *Different from corresponding Ctrl value: P ≤ 0.05.

Fig. 3. Serum testosterone concentrations immediately after exercise (IP), 20–40 (1A), 60–120 (1B), and 140–300 min after exercise (1C), and in morning on days 2 (2) and 3 (3) for Ctrl, Ex, and ExEt treatments. Values are means ± SE. *Different from corresponding Ctrl value: P ≤ 0.05.

Fig. 4. Serum luteinizing hormone concentrations immediately after exercise (IP) and 20–40 (1A), 60–120 (1B), and 140–300 min after exercise (1C) for Ctrl, Ex, and ExEt treatments. Values are means ± SE. *Different from corresponding Ctrl value: P ≤ 0.05.

Fig. 5. Plasma epinephrine concentrations immediately after exercise (IP) and 20–60 (1A) and 140–300 min after exercise (1C) for Ctrl, Ex, and ExEt treatments. Values are means ± SE. *Different from corresponding Ctrl value: P ≤ 0.05.
neogenesis via activation of gluconeogenic enzymes and blood glucose, the mechanisms include increased glucose metabolism and the release of glucagon and epinephrine, which also have gluconeogenic functions. It has been postulated that the catabolic nature of cortisol in skeletal muscle (2, 19) also plays a role in muscle protein remodeling during the recovery and adaptation from resistance exercise.

The implications of a possible longer-sustained cortisol response after ExEt may include an accentuated catabolic effect. Although this difference would not result in the same muscle atrophy observed with chronic hypercortisolemia, the effect may hinder the normal adaptation of muscle hypertrophy and increased strength during a resistance training program.

Unlike the common effect of resistance exercise on the pituitary-gonadal axis, ethanol ingestion may result in a rebound decline in Ex testosterone rather than any direct effect of ethanol or its metabolites on CRH release from the hypothalamus or corticosterone in the anterior pituitary gland (18, 33), or on cortisol synthesis in the adrenal cortex (4, 5). Nonetheless, a central mechanism may be involved, with stress resulting in neural stimulation from the brain causing hypothalamic CRH release into the portal circulation, although the subjects in the present study reported no incidence of gastrointestinal distress.

A prominent role of elevated cortisol concentrations during and after any type of exercise, including resistance exercise, is to meet the greater demand for energy. To increase plasma free fatty acids, cortisol activates lipolysis in adipose tissue. To provide more blood glucose, the mechanisms include increased gluconeogenesis via activation of gluconeogenic enzymes and absorption of glucose from the gut. Glucocorticoids also stimulate gluconeogenesis from tissues such as skeletal muscle, where cortisol has a permissive effect on gluconeogenesis, which also have gluconeogenic functions. It has been postulated that the catabolic nature of cortisol in skeletal muscle (2, 19) also plays a role in muscle protein remodeling during the recovery and adaptation from resistance exercise.

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Unlike the common effect of resistance exercise on the pituitary-gonadal axis, ethanol ingestion may result in a rebound decline in Ex testosterone rather than any direct effect of ethanol or its metabolites on CRH release from the hypothalamus or corticosterone in the anterior pituitary gland (18, 33), or on cortisol synthesis in the adrenal cortex (4, 5). Nonetheless, a central mechanism may be involved, with stress resulting in neural stimulation from the brain causing hypothalamic CRH release into the portal circulation, although the subjects in the present study reported no incidence of gastrointestinal distress.

A prominent role of elevated cortisol concentrations during and after any type of exercise, including resistance exercise, is to meet the greater demand for energy. To increase plasma free fatty acids, cortisol activates lipolysis in adipose tissue. To provide more blood glucose, the mechanisms include increased gluconeogenesis via activation of gluconeogenic enzymes and absorption of glucose from the gut. Glucocorticoids also stimulate gluconeogenesis from tissues such as skeletal muscle, where cortisol has a permissive effect on gluconeogenesis, which also have gluconeogenic functions. It has been postulated that the catabolic nature of cortisol in skeletal muscle (2, 19) also plays a role in muscle protein remodeling during the recovery and adaptation from resistance exercise.

The implications of a possible longer-sustained cortisol response after ExEt may include an accentuated catabolic effect. Although this difference would not result in the same muscle atrophy observed with chronic hypercortisolemia, the effect may hinder the normal adaptation of muscle hypertrophy and increased strength during a resistance training program.
a different level, possibly involving binding proteins, cytosolic/nuclear receptors, or gene expression.

The resistance exercise in the present study stimulated the adrenal medulla and the sympathetic nervous system. This was reflected in increased plasma catecholamine concentrations immediately after exercise. Plasma concentrations remained high at 20–60 min after exercise for epinephrine but not for norepinephrine. Previous reports support an increase in circulating catecholamines up to 5 min after exercise (23, 25). The rise observed in serum cortisol may have contributed to that of epinephrine by increasing epinephrine synthesis through induction of phenylethanolamine N-methyltransferase (40).

The use of ethanol in ExEt did not affect the already-increased postexercise catecholamine concentrations. Furthermore, ethanol intoxication did not generate a rise in epinephrine at 140–300 min and in norepinephrine at 20–60 and 140–300 min after exercise. Ethanol has been shown to elevate catecholamine concentrations when exercise is not involved (1, 34), and ethanol metabolism can increase circulating catecholamine concentrations by decreasing the hepatic NAD-to-NADH ratio (7, 8) and result in decreased catecholamine clearance. In addition to this mechanism, the response may also consist of a centrally mediated component.

In summary, the effect of ethanol intoxication was not sufficient to increase circulating cortisol concentrations above those caused by the resistance exercise. The data suggest the possibility of a more prolonged cortisol effect of this level of ethanol intoxication than of the resistance exercise alone. Peripheral cortisol and ACTH concentrations did not seem to have a role beyond the initial 40 min after the resistance exercise or during the subsequent two mornings. Peripheral testosterone and LH concentrations did not seem to have a role during the postexercise period or the subsequent two mornings after the resistance exercise. Finally, ethanol intoxication did not affect the sympathoadrenal system in the context of a prior resistance exercise session. This includes the lack of a rise of catecholamines, both above exercise-induced concentrations early during the postexercise period and above baseline values observed beyond the initial 40 min of the postexercise period.

Within the short-term nature of this investigation, ethanol intoxication after resistance exercise exerts only a minor effect on certain aspects of the neuroendocrine system in the study sample. This should not be seen by athletes and coaches as a license for intoxication after resistance exercise without any extensive attenuation of their physiological conditioning progress; the effect of ethanol on a conditioning program over a period of weeks or months requires study. Additionally, the individual who would like to apply the results of this investigation must consider one more aspect. Confounding factors such as hypohydration were controlled in this study so as to not affect the dependent variables of interest and to allow for the findings to be attributed to the ethanol. There may be secondary effects such as this that may lead to different results for the individual in a less-controlled setting. Furthermore, future studies should involve a BEC that reaches a higher concentration, is maintained for a longer period, or is attained at a different time and should examine these and other endocrine axes via a variety of cellular and molecular mechanisms.

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