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Effect of Hydration State on Testosterone and Cortisol Responses to Training-Intensity Exercise in Collegiate Runners

Abstract

Exercise intensity powerfully influences testosterone, cortisol, and testosterone : cortisol ratio (T : C) responses to endurance exercise. Hydration state may also modulate these hormones, and therefore may alter the anabolic/catabolic balance in response to endurance exercise and training. This study examined the effect of running intensity on testosterone, cortisol, and T : C when exercise was initiated in a hypohydrated state. Nine male collegiate runners (age = 20 ± 0 y, height = 178 ± 2 cm, mass = 67.0 ± 1.8 kg, body fat % = 9.8 ± 0.7 %, $\dot{V}O_{2max}$ = 65.7 ± 1.1 $ml \cdot kg^{-1} \cdot min^{-1}$) completed four 10-min treadmill runs differing in pre-exercise hydration status (euhydrated, or hypohydrated by 5 % of body mass) and exercise intensity (70 % or 85 % $\dot{V}O_{2max}$). Body mass, urine osmolality, and urine-specific gravity documented fluid balance; blood samples drawn pre-, immediately post-, and 20 min post-exercise were analyzed for testosterone, cortisol, and T : C. Except for heart rate measured during the 70 % $\dot{V}O_{2max}$ trials, heart rate, $\dot{V}O_2$, and plasma lactate were similar between euhydrated and hypohydrated conditions for a given intensity, suggesting hypohydration did not measurably increase the physiological stress of the exercise bouts. Furthermore, hydration state had no measurable effect on testosterone concentrations before, during, or after exercise at either intensity. Regardless of exercise intensity, cortisol concentrations were greater during hypohydration than euhydration pre-exercise and 20 min post-exercise. Additionally, T : C was significantly lower 20 min post-exercise at 70 % $\dot{V}O_{2max}$ when subjects were initially hypohydrated (T : C = 0.055) versus euhydrated (T : C = 0.072). These findings suggest that depending on exercise intensity, T : C may be altered by hydration state, therefore influencing the balance between anabolism and catabolism in response to running exercise performed at typical training intensities.

Key words
Exercise duration · exercise intensity · fluid balance · hormones · hypohydration

Introduction

Like many hormones, circulating concentrations of testosterone (T) and cortisol (C) are sensitive to exercise. Although an acute bout of endurance exercise normally increases circulating T and C [24] in an intensity-dependent manner [8,32], the responses can be modulated by training status [30], exercise mode [31], and exercise duration [17,23]. Typically, studies have evaluated the hormonal response to prolonged, low intensity exercise; our laboratory previously examined shorter, more intense exercise and found that 10 min of moderate- to high-intensity running increased T but had no effect on C in endurance-trained athletes [21]. Additionally, the T to C ratio (T : C), an important indicator

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of the anabolic/catabolic status of the body [1], was unaffected by this protocol [21].

Hydration status, a key determinant of exercise performance [2,26,27], is also capable of modifying the response of many hormones during exercise [28,29]. Although no previous research has directly examined the effect of hydration alone on T, C, and T : C, several authors have indirectly evaluated the effects of fluid balance on T and C during protocols designed to elicit hypohydration by exercising subjects for prolonged duration in the heat. Mild dehydration (< 2% loss in body mass) induced by heat [22], exercise [20], or exercise-heat stress [18] appears to have no effect on T, however, we know of no previous studies that measured T during a more profound hypohydration. C, on the other hand, significantly increased in subjects who dehydrated (– 3.4%) during prolonged low intensity exercise at 32 °C [9] and was significantly correlated to the degree of hypohydration during very low intensity exercise at 49 °C [11].

While dehydration during exercise is important in modulating the endocrine response, evidence also suggests that initiating exercise in a hypohydrated state significantly alters the hormonal outcome [18,25]. This is especially important because 1) exercising subjects voluntarily replace only ~ 70% of their net water loss [12,19], suggesting that daily strenuous training may result in chronic hypohydration, and 2) daily strenuous training represents a real-life scenario for many soldiers, laborers, and competitive endurance athletes. We previously demonstrated that dehydration to – 3% body mass did not influence T or C during prolonged low intensity exercise at 33 °C [18]. The exercise protocol used in that study, however, was of significantly lower intensity than conventional endurance training, and the effects of dehydration and exercise could not be separated from possible heat-mediated responses. If hydration status affects T, C, or T : C before, during, or after exercise, fluid balance may alter the relationship between anabolism and catabolism that results from physical activity performed at typical training intensities. Therefore, the purpose of this study was to examine the effect of initial hydration state on T and C responses to moderate- to high-intensity exercise in trained endurance athletes.

Methods

Nine members of the University of Connecticut men's cross country team who trained consistently (~ 100 km · wk^{-1}) for at least the previous three months were subjects for this study (age = 20 ± 0 y, height = 178 ± 2 cm, mass = 67.0 ± 1.8 kg, body fat % = 9.8 ± 0.7%, maximal aerobic power = 65.7 ± 1.1 ml · kg^{-1} · min^{-1}). Prior to their participation, subjects provided written informed consent. All procedures adhered to 45 CFR 46 (federal regulations guiding the use of human subjects in research) and were approved by the Institutional Review Board of the University of Connecticut.

During the initial visit, subjects' age, height, mass, body composition (via hydrostatic weighing), and maximal aerobic power ($\dot{V}O_{2max}$) were determined. The $\dot{V}O_{2max}$ test entailed 10 – 15 min of progressive, incremental running on a motorized treadmill to volitional fatigue. $\dot{V}O_2$ (CPF-S, MedGraphics, St. Paul, MN, USA), heart rate (HR) (Vantage, Polar Electro, Woodbury, NY, USA), and ratings of perceived exertion (RPE) were measured periodically throughout the test. Specific criteria (plateau in $\dot{V}O_2$ with increasing exercise intensity, RPE ≥ 18, and/or HR = age-predicted maximum) ensured that $\dot{V}O_{2max}$ was attained.

To establish appropriate running intensities for subsequent data collection (70% and 85% $\dot{V}O_{2max}$), subjects completed two preliminary treadmill tests on separate days. Tests consisted of three 10-min runs, each separated by 10 min of rest. Running speed was initially based on percentage of HR and further modified by $\dot{V}O_2$ measures taken during minutes 6 – 8 of each exercise bout. Adjustments were made until treadmill speeds eliciting 70% and 85% of $\dot{V}O_{2max}$ were achieved; these values were recorded and represented treadmill speeds used during actual data collection sessions.

Subjects then completed four experimental trials differing in pre-exercise hydration status and exercise intensity: euhydrated (EU) at 70% $\dot{V}O_{2max}$ (EU70), hypohydrated (HY) to ~ 5% body mass loss at 70% $\dot{V}O_{2max}$ (HY70), euhydrated at 85% $\dot{V}O_{2max}$ (EU85), and hypohydrated to ~ 5% body mass loss at 85% $\dot{V}O_{2max}$ (HY85). Trial order was randomized; a minimum of 24 h separated each trial. All sessions for a given subject were standardized for time of day, environmental conditions (23 °C), and subject attire. For all trials, subject masses were obtained 24 h before exercise to obtain a baseline body mass. For HY trials, subjects reported to the laboratory one day prior after a typical breakfast for pre-body weight and dehydration instructions. Subjects were instructed to refrain from any fluid intake during daily living and normal training (≥ 1 h, approximately 75% $\dot{V}O_{2max}$) for necessary body water loss. Food intake for dehydration consisted of a typical breakfast prior to weigh-in, two turkey sandwiches on wheat bread with one teaspoon of mustard, two bagels, and one cup of spaghetti with parmesan cheese. This diet, excluding breakfast, provided 1177 calories (61% carbohydrate, 24% protein, and 15% fat), and a total fluid mass of 301 g water and 130 g in solid food; a nutritionist helped design this diet to minimize water consumption while maintaining the necessary caloric intake with a high concentration of carbohydrates.

On each of the four mornings of testing, subjects arrived in a post-absorptive state and provided an acute urine sample. Urine was immediately analyzed for specific gravity via refractometry (A300CL-E01, Atago, Tokyo, Japan) and osmolality via freezing point depression (Osmette, Precision Systems, Natick, MA, USA) to validate hydration status [3]. Twenty-four hour body mass change was also measured to document hydration status. Subjects then assumed a supine position; a 20-gauge Teflon cannula was inserted into an antecubital vein, maintained patent with isotonic saline. Following a 20-min standing equilibration, a pre-exercise blood sample was taken (PRE). Subjects then ran for 10 min on a motorized treadmill at the prescribed exercise intensity. HR and $\dot{V}O_2$ were measured every two minutes and from minutes 6 – 8, respectively. Further blood samples were obtained immediately post-exercise (IP) and after 20 min of rest (+ 20).

Blood samples were drawn into 10-mL glass tubes pre-treated with ethylenediaminetetraacetic acid (BD Vacutainer, Becton-Dickinson, Franklin Lakes, NJ, USA), centrifuged for 15 min at

Table **1** Hydration indices during euhydrated and hypohydrated exercise trials

Experimental session	*Urine-specific gravity*	*Urine osmolality ($mOsm \cdot kg^{-1}$)*	*Body mass (kg)*
EU70	1.024 ± 0.002	572 ± 93	67.9 ± 1.6
HY70	1.034 ± 0.001*	1003 ± 22*	65.3 ± 1.5*
EU85	1.023 ± 0.002	562 ± 85	67.8 ± 1.6
HY85	1.033 ± 0.001*	970 ± 33*	65.2 ± 1.7*

Values are means ± SE. * indicates significant difference compared to corresponding EU value. Abbreviations: EU70 – exercise at 70% $\dot{V}O_{2max}$ initiated in a euhydrated condition; HY70 – exercise at 70% $\dot{V}O_{2max}$ initiated in a hypohydrated condition; EU85 – exercise at 85% $\dot{V}O_{2max}$ initiated in a euhydrated condition; HY85 – exercise at 85% $\dot{V}O_{2max}$ initiated in a hypohydrated condition

3000 RPM at 4 °C. Resulting plasma was aliquoted and stored at – 80 °C until analysis. Samples were thawed only once. Plasma lactate (La^-) was analyzed in duplicate via enzymatic techniques (Model 2300, Yellow Springs Incorporated, Yellow Springs, OH, USA). Blood samples were analyzed in triplicate for hematocrit via microcapillary technique and hemoglobin via the cyanmethemoglobin method. Percent changes in plasma volume (PV) were calculated [6]. Circulating T (Diagnostic Products, Los Angeles, CA, USA) and C (Diagnostic Systems Inc., Webster, TX, USA) were assayed in duplicate via radioimmunoassay. Inter-assay coefficients of variation were ± 7.1% and ± 3.4% for T and C, respectively; intra-assays coefficients of variation for both analytes were less than 5%. Hormonal concentrations were the same when corrected for the minor PV shifts (in all cases < 10%); as such, reported values are absolute, not corrected.

Descriptive statistics (means and SE) were calculated for all variables. Data were analyzed with a repeated measures ANOVA. When appropriate, Tukey's post hoc test was used to determine specific pairwise differences. Significance was set at $p < 0.05$.

Results

Table **1** shows measures of hydration state. Subject body mass loss (baseline to IP) was 5.7 ± 0.4% and 5.5 ± 0.3% for the 70% and 85% $\dot{V}O_{2max}$ HY trials, respectively. Urine-specific gravity and urine osmolality were significantly higher before HY trials than EU trials for both exercise intensities.

Measures of physiological strain during exercise are presented in Table **2**. $\dot{V}O_2$, HR, and La^- were significantly greater during the 85% $\dot{V}O_{2max}$ trial than the 70% $\dot{V}O_{2max}$ trial regardless of hydration state. HR was significantly higher during HY70 than EU70, however, hydration state did not alter any other responses to exercise.

Fig. **1** displays the circulating T response to exercise. T at IP was significantly higher than PRE or + 20 during EU85. In all other trials, T was stable and similar throughout. Fig. **2** shows C responses to exercise. At both exercise intensities, C measurements at PRE and + 20 were significantly greater in HY than corresponding EU trials. Measurement time and exercise intensity had no effect on C. T : C (see Fig. **3**) was significantly lower in HY70 than in EU70 at + 20. No other significant differences in T : C were noted regardless of measurement time, exercise intensity, or hydration state.

Discussion

Despite completing identical exercise protocols during HY70 and EU70, the T : C was significantly lower at + 20 during HY70. These results suggest that 1) in highly trained distance runners, initiation of moderate intensity exercise while hypohydrated may increase the catabolic state of the body and 2) hydration state may contribute to this response. As most soldiers, laborers, and athletes do not adequately replace fluid lost during exercise [12,19], they may become chronically hypohydrated (2 – 3%) and potentially predispose themselves to enhanced catabolism resulting from subsequent training-intensity exercise bouts.

Although HR statistically differed between HY70 and EU70, no other measures of physiological strain were affected due to the significant hypohydration (Table **2**). These results support previous research examining submaximal exercise in thermoneutral conditions, as up to 5% hypohydration did not affect HR, $\dot{V}O_2$, and La^- during approximately 30 min of progressive submaximal cycling [5] and 60 min of cycling at 60% $\dot{V}O_{2max}$ [4]. Even though 5% hypohydration did not increase the physiological strain through the classic measures of HR, $\dot{V}O_2$, and La^- in the majority

Table **2** Indices of physiological strain during euhydrated and hypohydrated exercise trials

Experimental session	*$\dot{V}O_2$ ($L \cdot min^{-1}$)*	*($ml \cdot kg^{-1} \cdot min^{-1}$)*	*HR ($beats \cdot min^{-1}$)*	*Δ La^- ($mmol \cdot L^{-1}$)*	*Δ PV (%)*
EU70	3.1 ± 0.1†	46.3 ± 1.0†	147 ± 3†	0.7 ± 0.2†	– 7.1 ± 1.4
HY70	3.1 ± 0.1†	47.2 ± 1.2†	155 ± 2*†	0.8 ± 0.3†	– 2.5 ± 1.8
EU85	4.0 ± 0.1	58.6 ± 0.9	170 ± 3	5.2 ± 1.2	– 6.3 ± 2.5
HY85	3.8 ± 0.1	58.9 ± 1.3	170 ± 4	5.3 ± 1.1	– 5.6 ± 2.0

Values are means ± SE. * indicates significant difference compared to corresponding EU value. † indicates significant difference compared to corresponding 85% $\dot{V}O_{2max}$ trial. Abbrevations: EU70 – exercise at 70% $\dot{V}O_{2max}$ initiated in a euhydrated condition; HY70 – exercise at 70% $\dot{V}O_{2max}$ initiated in a hypohydrated condition; EU85 – exercise at 85% $\dot{V}O_{2max}$ initiated in a euhydrated condition; HY85 – exercise at 85% $\dot{V}O_{2max}$ initiated in a hypohydrated condition; $\dot{V}O_2$ – oxygen consumption; HR – heart rate; La^- – lactate; PV – plasma volume

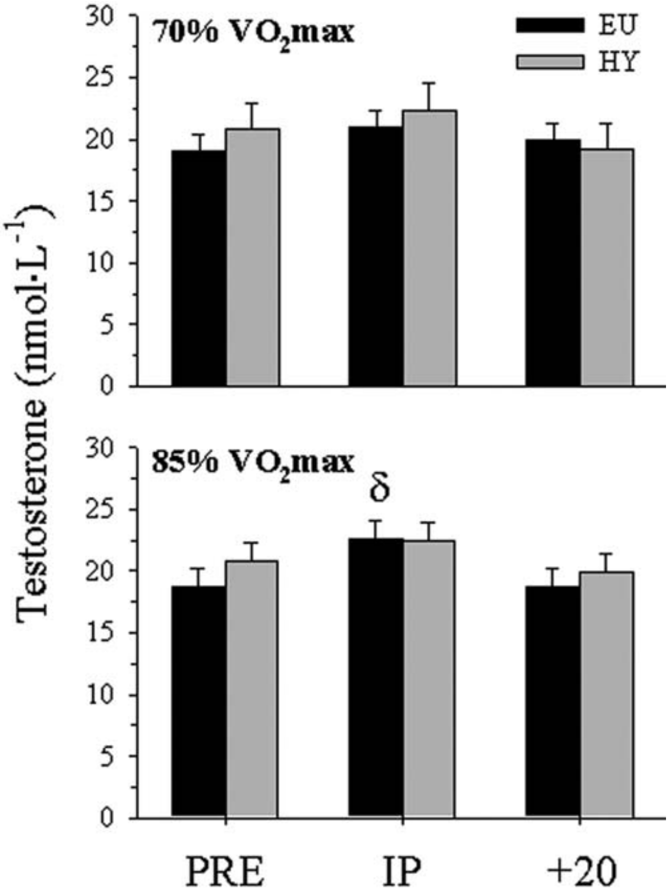

Fig. 1 T response to 10 min of running at 70% $\dot{V}O_{2max}$ (top panel) and 85% $\dot{V}O_{2max}$ (bottom panel) in euhydrated (black bars) and hypohydrated (gray bars) athletes. Values are means ± SE. δ indicates significant difference compared to all other time points within hydration status. Abbrevations: EU – euhydrated, HY – hypohydrated, PRE – pre-exercise, IP – immediate post-exercise, + 20 – 20 min post-exercise.

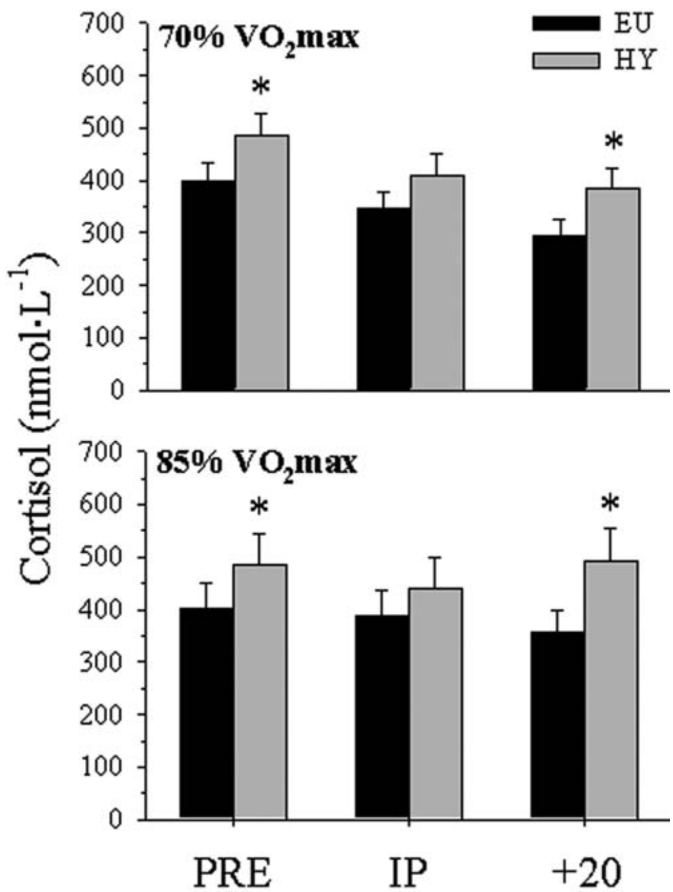

Fig. 2 C response to 10 min of running at 70% $\dot{V}O_{2max}$ (top panel) and 85% $\dot{V}O_{2max}$ (bottom panel) in euhydrated (black bars) and hypohydrated (gray bars) athletes. Values are means ± SE. * indicates significant difference between hydration status. Abbrevations: EU – euhydrated, HY – hypohydrated, PRE – pre-exercise, IP – immediate post-exercise, + 20 – 20 min post-exercise.

of cases, dehydration significantly altered T : C at 70% $\dot{V}O_{2max}$, reinforcing the importance of hydration state in maintaining the anabolic : catabolic balance during training-intensity exercise.

Results from this study suggest that T is insensitive to significant dehydration but is exercise intensity-dependent. At both 70% and 85% $\dot{V}O_{2max}$, and at all times measured, HY and EU trials resulted in statistically similar T values. Previous research has shown a lack of T stimulation at milder levels of dehydration [18,20,22], however, we believe this study is the first to examine T responses to significant hypohydration. The increased T seen IP during EU85, but not EU70 or HY70, suggests that T may have an exercise intensity threshold. It is unclear why similar increases were not seen in the HY85 trial; this may be related to an interaction of dehydration and exercise that we were unable to detect or the potentially significant influence of routine endurance training on endocrine responses to exercise. Chronically endurance-trained men present T concentrations 55 – 85% lower than sedentary men [7,14]. Although our highly conditioned subjects were within the clinically normal range [33], resting, euhydrated T concentrations (~ 18.8 nmol · L^{-1}) were similar to those seen in endurance-trained athletes with altered hypothalamic-pituitary-gonadal activity [13,14,16]. Moreover, endurance athletes often respond to T-sensitive stimuli differently than normal subjects [14,15]. Thus the role of exercise intensity in dictating T response is somewhat unclear, however, the statistical similarity between EU and HY trials at each intensity indicates that in highly trained endurance athletes, hydration state does not modulate plasma T.

Conversely, previous literature [10,11,18] and our present results suggest that C is sensitive and tightly correlated to hypohydration, as indicated by the significantly greater C concentrations for HY than EU at PRE and + 20 for both exercise intensities. Somewhat surprisingly, none of the exercise trials elicited an increase in C. Similar results have been presented by Hoffman et al. [17], who reported a significant decrease in C immediately after short duration, high intensity exercise and hypothesized that the C response to exercise was duration-dependent. Although inconclusive, the current results suggest that during short-term, intense physical activity, exercise-induced reductions in C may eclipse hydration-induced exacerbations in C. In the absence of such an inhibitor, however, circulating C appears to be significantly affected by hypohydration.

The primary finding of this study, the significantly lower T : C at + 20 of HY70 compared to EU70, indicates a greater catabolic stimulus when exercise was initiated in a hypohydrated condi-

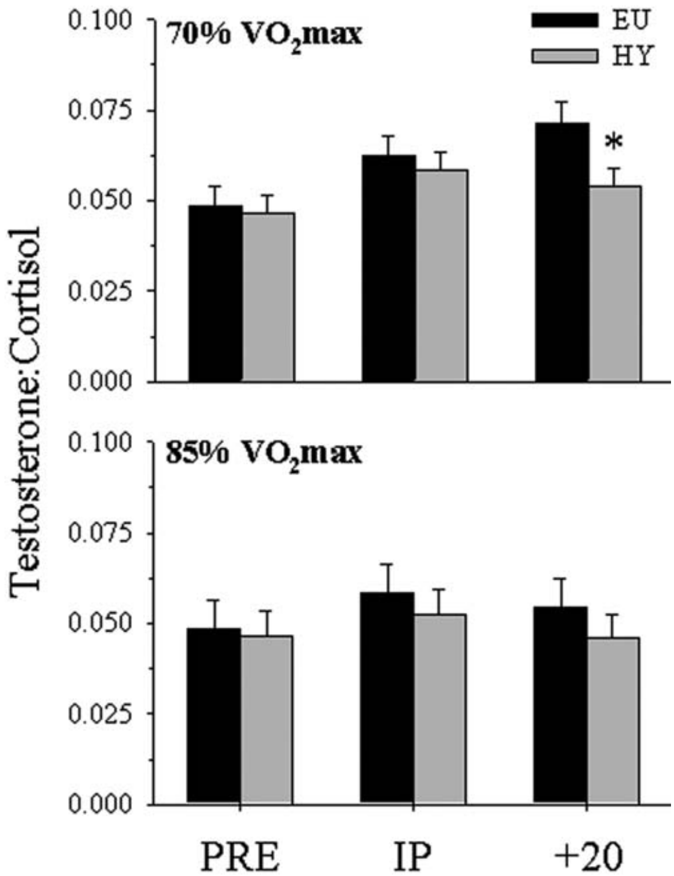

Fig. **3** T : C response to 10 min of running at 70% $\dot{V}O_{2max}$ (top panel) and 85% $\dot{V}O_{2max}$ (bottom panel) in euhydrated (black bars) and hypohydrated (gray bars) athletes. Values are means ± SE. * indicates significant difference between hydration status. Abbrevations: EU – euhydrated, HY – hypohydrated, PRE – pre-exercise, IP – immediate post-exercise, + 20 – 20 minutes post-exercise.

tion. We speculate that similar reductions in T : C did not occur during HY85 because the increased exercise intensity enhanced T stimulation. This hypothesis is somewhat questionable, however, given the lack of temporal alignment between HY85-induced increases in T (IP) and HY70-induced decreases in T : C (+ 20). Given the normal increase in C with hypohydration [10,11,18] and longer exercise duration [8], we propose that greater and more immediate reductions in the T : C may result when typical training intensity and duration exercise is initiated in a hypohydrated state. Although the endocrine responses to 10 min of simulated training are unlikely to have significant deleterious effects, chronic exposure to this hormonal environment may result in increased protein degradation, reduced muscle mass, and subsequent losses in strength and power.

In conclusion, hypohydration and exercise intensity play a significant role in determining the hormonal responses to an exercise bout in highly trained endurance athletes. Our results suggest that T is not sensitive to significant changes in hydration status, but that C is stimulated by hypohydration. Finally, training-intensity exercise that does not evoke a significant T response, if initiated in a hypohydrated state, may lead to detrimental reductions in the T : C ratio and a shift in the body's circulating hormonal balance between anabolism and catabolism.

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