

Effect of Exercise at Three Exercise Intensities on Salivary Cortisol

DEAN E. JACKS,¹ JAMES SOWASH,² JOHN ANNING,²
THOMAS MCGLOUGHLIN,² AND FREDRICK ANDRES³

¹Physical Education, Winston-Salem State University, Winston-Salem, NC 27110; ²University of Toledo Exercise Physiology Laboratory, University of Toledo, Toledo, Ohio 43606; ³Eastern Michigan University, Ypsilanti, Michigan 48197.

ABSTRACT

Changes in cortisol concentration in response to exercise at 3 different intensities were quantified. Ten apparently healthy, recreationally active males participated. On 4 separate occasions, subjects were assigned a random order of 1-hour cycle ergometer bouts of exercise at $44.5 \pm 5.5\%$, $62.3 \pm 3.8\%$, and $76.0 \pm 6.0\%$ (mean \pm SD) of $\dot{V}O_{2\text{peak}}$ and a resting control session. Saliva samples were collected before exercise at 10, 20, 40, and 59 minutes of exercise and at 20 minutes of recovery. Differences in cortisol concentration were assessed via multivariate analysis of variance ($\alpha = 0.05$) Tukey post hoc analysis when indicated. During the highest-intensity exercise session, cortisol was significantly higher at 59 minutes of exercise ($p = 0.004$) and at 20 minutes of recovery ($p = 0.016$) than at those same time points during the resting control session. No significant differences in cortisol concentration were noted among resting, low-, and moderate-intensity exercise. Exercise <40 minutes in duration elicited no significant differences at any intensity. These data indicate that only exercise of high intensity and long duration results in significant elevations of salivary cortisol.

Key Words: $\dot{V}O_2$, cycle, duration

Reference Data: Jacks, D.E., J. Sowash, J. Anning, T. McGloughlin, and F. Andres. Effect of exercise at three exercise intensities on salivary cortisol. *J. Strength Cond. Res.* 16(2):286–289. 2002.

Introduction

Cortisol, the principal glucocorticoid in humans, is known to play a major role in metabolism and immune function (5, 15). It is considered catabolic in nature because of its effects on protein and carbohydrate metabolism (21, 24). Stimulation of gluconeogenesis by cortisol spares blood glucose and reduces protein stores. Such diminution of stored protein may lead to a wasting of the skeletal muscle (21).

Previous investigations have assessed cortisol concentration at different points in the circadian rhythm,

after consuming various diets, and at different exercise intensities and durations (2–4, 6, 9, 12–14, 17–19). Although these investigations generally produce some variability in cortisol response to different intensities and durations of exercise, the findings do seem to indicate that cortisol concentration increases in response to long-duration, high-intensity exercise (2–4, 6, 12, 13). However, the response induced by low-intensity and short-duration exercise is not clear (2, 4, 12, 14, 17, 19). None of the aforementioned investigations has simultaneously controlled for diet, duration and intensity of exercise, and diurnal changes. Therefore, changes identified cannot be specifically attributed to the effect of exercise alone.

Cortisol is commonly measured in plasma and serum from blood samples obtained during exercise (2, 4, 6, 9, 12). Maximizing anxiety during exercise has elicited higher concentrations of cortisol than trials performed at the same workload without emotional stress (8, 23). The effect of anxiety on cortisol associated with blood sampling and a high correlation between salivary and serum unbound cortisol ($r = 0.97$) make measurement of salivary cortisol the preferred method (22). The purpose of this investigation was to identify changes in salivary cortisol concentration that are attributed to exercise, utilizing a resting session to control for diurnal variation.

Methods

Subjects

Ten apparently healthy, recreationally active men of age 25.6 ± 4 years, body composition $13.1 \pm 4.4\%$ fat, height 173.6 ± 12.8 cm (mean \pm SD) and $\dot{V}O_2$ 44.5 ± 6.6 ml·kg⁻¹·min⁻¹ (mean \pm SD) were recruited from the local area. Before participation, subjects completed a medical history form and were screened for orthopedic injuries and metabolic disorders. Subjects exercised at least 2 times per week for a minimum of 20 minutes to a maximum of 40 minutes per day during

the previous 6 week period. Following a verbal description of the procedures to be used and the potential risks, subjects provided written informed consent.

Familiarization Trials

Subjects were invited to the laboratory for orientation. Each subject was fitted with a headset, nose clip, and respiratory valve (Hans Rudolph, Inc., Kansas City, MO). Each subject was measured for seat height and body composition via 3-site skinfold (1). Subjects were asked to exercise for 10 minutes on a cycle ergometer (Monark Ergomedic 818 E, Vansbro, Sweden), wearing gas analysis equipment simulating maximal testing conditions.

Maximal Exercise Testing

At least 1 day after completing the familiarization trial, a maximal exercise test was performed. Subjects were fitted with a heart rate monitor (Polar USA, Inc., Stamford, CT), a sphygmomanometer, and the gas analysis equipment described in the aforementioned familiarization trials. Inspired gas was measured via a gasometer (RAM 9200, Rayfield Equipment, Chicago, IL). Expired gas was analyzed for oxygen and carbon dioxide concentrations at 15 second intervals via Applied Electrochemistry SA-3 and CD 3A analyzers (Ametek, Thermomax Instruments Division, Pittsburgh, PA). Incremental increases in workload were applied at 3 minute intervals; the magnitude of the increases depended upon body weight (modified protocol from ACSM's Guidelines for Exercise Testing and Prescription, 5th ed.) (1). Subjects were instructed to maintain a pedal cadence of 75 revolutions per minute on a cycle ergometer. The test was terminated when the subject could no longer maintain a cadence of 75 revolutions per minute.

Resting and Exercise Trials

A random sequence was utilized to determine the order of resting and exercise trials. All trials were completed at the same time of the day (between 1100 and 1400 hours). All testing was conducted on days when the subject woke up at a standard time. Each test was separated by a minimum 1-day rest period. Subjects were instructed to consume one of five 500 kcal breakfast options (approximately 55% carbohydrate, 30% fat, and 20% protein) 3 hours before exercise and not to eat between breakfast and trials. Subjects also completed an activity recall to summarize activity over the previous 36 hours and were instructed to refrain from high-intensity exercise 48 hours before testing. $\dot{V}O_2$ and workload data acquired from the maximal test were plotted, and a line was extrapolated across workloads. Mechanical workloads (kilopounds) for the exercise bouts were estimated to elicit exercise intensities of 45.0, 60.0, and 75.0% of $\dot{V}O_{2peak}$ from the extrapolated line. On 3 separate occasions, subjects completed a 1-hour bout of cycle ergometry (Monark Ergo-

medic 818 E) at a bout of exercise at 44.5 ± 5.5 , 62.3 ± 3.8 , and $76 \pm 6.0\%$ of $\dot{V}O_{2peak}$ and a resting session. Workload was not adjusted during the exercise bout. $\dot{V}O_2$ was assessed via 30 second Douglas Bags obtained at 12, 22, 42, 52 minutes during the exercise trials and reported as averages among those time points. A capillary blood sample was obtained from the fingertip 3 minutes before and 3 minutes after the 1-hour exercise bouts and then measured for blood glucose concentration by GLUCOMETER ELITE (Bayer Corporation, Elkhart, IN). Saliva samples were collected (1 milliliter per sample) before exercise, at the 10th, 20th, 40th, and 59th minute during exercise, and at 20 minutes of recovery. Samples were immediately frozen at -20°C and later assayed in duplicate for cortisol concentration (mean interassay coefficient of variation between duplicates = 2.39%) (Coat-A-Count Cortisol, Diagnostic Products Corporation Los Angeles, CA). Saliva samples were collected during the resting session at the same intervals utilized for the exercise bouts.

Statistical Analyses

Statistical analysis was performed using SPSS 7.5 for Windows (SPSS Inc., Chicago, IL, 1989–1996). Differences in cortisol concentrations were assessed via multivariate analysis of variance ($\alpha = 0.05$). Between trial comparisons of samples at each time point of interest were conducted. Within trial comparisons—across time—were not performed because of normal diurnal variation. When indicated by a significant *F* ratio, a Tukey post hoc test was utilized to identify pairwise differences.

Results

Cortisol concentration was significantly higher at 59 minutes of high-intensity exercise than it was at 59 minutes of low-intensity exercise or at rest ($p = 0.004$ and $p = 0.042$, respectively). Cortisol concentration was also significantly higher at 20 minutes of recovery from high-intensity exercise than it was at rest or after 20 minutes of recovery from low-intensity exercise ($p = 0.016$ and $p = 0.047$, respectively). Diurnal fluctuations at rest were not significant when assessed by analysis of variance at $\alpha = 0.05$. Preexercise and postexercise blood glucose concentrations were not significant when assessed by one-sample *t*-test at $\alpha = 0.05$ (Table 1).

Figure 1 represents mean cortisol concentrations in $\mu\text{g}\cdot\text{dl}^{-1}$ for the resting, low- ($44.5 \pm 5.5\%$ $\dot{V}O_{2peak}$), moderate- ($62.3 \pm 3.8\%$ $\dot{V}O_{2peak}$), and high-intensity ($76.0 \pm 6.0\%$ $\dot{V}O_{2peak}$) trials as a function of time. Table 1 represents the mean blood glucose concentrations for each trial and the corresponding *p* values via dependent *t*-test between preexercise and postexercise at any intensity.

Table 1. Blood glucose concentrations 3 minutes preexercise and postexercise (mean \pm SD).

	Low intensity	Moderate intensity	High intensity
Preexercise	109.1 \pm 24.2 mg·dl ⁻¹	96.9 \pm 15.8 mg·dl ⁻¹	99.6 \pm 15.7 mg·dl ⁻¹
Postexercise	98.2 \pm 12.81 mg·dl ⁻¹	92.2 \pm 14.1 mg·dl ⁻¹	86.4 \pm 16.3 mg·dl ⁻¹
<i>p</i> Value	0.208	0.415	0.094

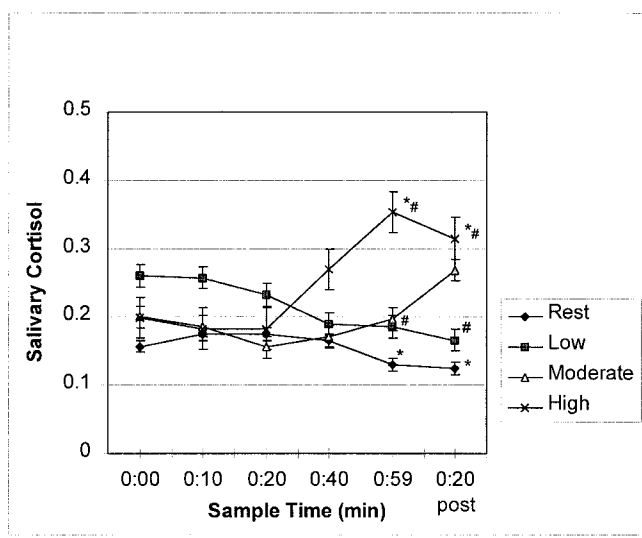


Figure 1. Mean salivary cortisol concentration in micrograms per deciliter (\pm SE) before exercise, during exercise, and at 20 minutes of recovery ($n = 10$). The * symbol denotes significant differences between the high-intensity trial and the resting control trial at 59 minutes of exercise and 20 minutes of recovery ($p = 0.004$ and $p = 0.042$, respectively). The # symbol denotes significant differences between the high-intensity and low-intensity trials at 59 minutes of exercise and 20 minutes of recovery ($p = 0.016$ and $p = 0.047$, respectively).

Discussion

The purpose of this investigation was to identify changes in salivary cortisol concentration that are attributed to exercise at low-, moderate-, and high-intensity. To conclude that changes in cortisol concentration are a response to exercise and not a result of diurnal changes, variations in diet, activity, or stress, well-controlled methods were employed. Previous investigators controlled for some of these variables, but none have controlled for all, nor have they assessed concentrations at low-, moderate-, and high-intensity exercise (2–4, 6, 9, 12–14, 17–19). Statistically significant differences noted in this study conflict with data of previous investigators, particularly exercise of <40 minutes duration and low-intensity exercise (2, 14). The present investigation did not identify any significant decreases in cortisol at any intensity <40 minutes. However, both Davies and Few (2) and Ortega et al. (14) identified significant decreases in cortisol concentration at work rates <50% $\dot{V}O_{2max}$.

In the results observed here, it appears that high-intensity (76.0% $\dot{V}O_{2 peak}$), long-duration (59 minutes) exercise is required to effect significant increases in cortisol concentration. These significant differences during high-intensity exercise are consistent with numerous previous investigations (2–4, 6, 12, 13). However, heavy resistance exercises, even of short duration, appear to elicit significant increases in cortisol concentration (7, 10, 11). These acute increases in cortisol are not associated with protein catabolism. High-intensity resistance exercise stimulates protein anabolism and hypertrophy of skeletal muscle (20). However, to establish the full effect of exercise stimulus on cortisol and subsequent modifications in protein metabolism, measurement of recovery samples is necessary.

A resting trial was incorporated to compare cortisol concentrations at specific times of the day with cortisol concentrations during exercise. These data from the resting trial served as a basis for comparison of data collected during the exercise trials. Therefore, significant changes that occurred during the high-intensity trial at 59 minutes of exercise and 20 minutes of recovery can be attributed to the stimulus of exercise.

Comparison of resting and low-intensity trials produced no significant differences. Two previous groups of researchers concluded that cortisol concentration decreases during low-intensity exercise (2, 14). Ortega et al. (14) collected data at 0800 hours, a time when the hormone is expected to decrease during nonexercise conditions. Davies and Few (2) attempted to control for diurnal changes by collecting samples between 1100 and 1400 hours, but they did not include a resting trial to isolate exercise-induced changes. Also, they did not control for diet or utilize well-defined training intensities.

Consistent with previous research, in the present study, cortisol concentration did not change significantly during the moderate-intensity exercise trial (2, 14, 17, 18). In contrast, our results show that high-intensity exercise elicited significant increases in cortisol concentration at 59 minutes of exercise and 20 minutes postexercise compared with the resting and low-intensity trials at those same time points. These findings at rest and low-intensity exercise are consistent with prior research (2–4, 6, 12, 13). However, in contrast with Davies and Few (2), no significant differences occurred at 40 minutes of high-intensity exercise. These authors concluded that 40 minutes of

high-intensity exercise (65–90% of $\dot{V}O_{2\max}$) elicited an increase in cortisol concentration. This difference could be attributed to the variance of exercise intensities utilized during Davies and Few's high-intensity trial.

Blood glucose measurements assessed preexercise and postexercise did not differ significantly at any intensity of exercise. The lowest mean postexercise concentration among exercise trials was $86.3 \text{ mg}\cdot\text{dl}^{-1}$. Investigations by Tabata et al. (19) and Sotsky et al. (16) concluded that when glucose concentrations decreased to $59.0 \text{ mg}\cdot\text{dl}^{-1}$, cortisol concentration increased during exercise. Thus, changes in salivary cortisol detected in the present investigation can be attributed to the effect of exercise and not hypoglycemia.

Practical Applications

The present findings indicate that significant increases in salivary cortisol concentration occur in response only to long-duration, high-intensity exercise. Individuals who participate in exercise at any intensity for up to 1 hour may do so without concern for the catabolic effects of increasing cortisol concentration. Therefore, increased caloric expenditure during exercise can be met, utilizing carbohydrate and fat stores, consequently sparing protein. Thus, exercise within the previously mentioned parameters should not reduce lean body mass. However, these findings should not be generalized to heavy resistance training, where even short bouts of exercise may stimulate cortisol elevations (7, 10, 11).

References

1. ACSM. *ACSM's Guidelines for Exercise Testing and Prescription* (5th ed.). Baltimore: Williams and Wilkins, 1995. p. 93.
2. DAVIES, C., AND J. FEW. Effects of exercise on adrenocortical function. *J. Appl. Physiol.* 35:887–891. 1973.
3. DEL CORRAL, P., A. MAHON, G. DUNCAN, C. HOWE, AND B. CRAIG. The effect of exercise on serum and salivary cortisol in male children. *Med. Sci. Sports Exerc.* 26:1297–1301. 1994.
4. DUCLOS, M., J. CORCUFF, M. RASHEDI, V. FOUGERE, AND G. MANIER. Trained versus untrained men: Different immediate post-exercise responses of pituitary adrenal axis. A preliminary study. *Eur. J. Appl. Physiol.* 75:343–350. 1997.
5. GUYTON, A., AND J. HALL. *Textbook of Medical Physiology* (9th ed.). Philadelphia: W.B. Saunders Company, 1996. pp. 945–966.
6. HACKNEY, A., M. PREMO, AND R. MCMURRAY. Influence of aerobic versus anaerobic exercise on the relationship between reproductive hormones in men. *J. Sports Sci.* 13:305–311. 1995.
7. HAKKINEN, K., AND A. PAKARINEN. Acute hormonal responses to heavy resistance exercise in men and women at different ages. *Int. J. Sports Med.* 16:507–513. 1995.
8. KACIUBA-USCILKO, H., S. PORTA, K. NAZAR, M. TONDERSKA, E. TITOW-STUPNICKA, A. ZIEMBA, AND J. CHWALBINSKA-MONETA. Effect of mild psychological stress on physiological responses to exercise in men. *J. Physiol. Pharmacol.* 45:429–439. 1994.
9. KRAEMER, R., S. BLAIR, G. KRAEMER, AND V. CASTRACANE. Effects of treadmill running on plasma beta-endorphin, corticotropin, and cortisol levels in male and female 10 K runners. *Eur. J. Appl. Physiol.* 58:845–851. 1989.
10. KRAEMER, W., A. CLEMSON, N. TRIPLETT, J. BUSH, R. NEWTON, AND J. LYNCH. The effects of plasma cortisol elevation on total differential leukocyte counts in response to heavy-resistance exercise. *Eur. J. Appl. Physiol.* 73:93–97. 1996.
11. KRAEMER, W., K. HAKKINEN, R. NEWTON, M. MCCORMICK, B. NINDL, J. VOLEK, L. GOTSHALK, S. FLECK, W. CAMPBELL, S. GORDON, P. FARRELL, AND W. EVANS. Acute hormonal responses to heavy resistance exercise in younger and older men. *Eur. J. Appl. Physiol.* 77:206–211. 1998.
12. MCCARTHY, D., I. MACDONALD, M. GRANT, M. MARBUT, M. WATLING, S. NICHOLSON, J. DEEKS, A. WADE, AND J. PERRY. Studies on the immediate and delayed leucocytosis elicited by brief (30-min) strenuous exercise. *Eur. J. Appl. Physiol.* 64:513–517. 1992.
13. NIEMAN, D., A. MILLER, D. HENSON, B. WARREN, G. GUSEWITCH, R. JOHNSON, J. DAVIS, D. BUTTERWORTH, AND S. NEHLSSEN-CANNARELLA. Effect of high- versus moderate-intensity exercise on natural killer cell activity. *Med. Sci. Sports Exerc.* 25:1126–1349. 1993.
14. ORTEGA, E., M. COLLAZOS, M. MAYNAR, C. BARRIGA, AND M. DE LA FUENTE. Stimulation of the phagocytic function of neutrophils in sedentary men after acute moderate exercise. *Eur. J. Appl. Physiol.* 66:60–64. 1993.
15. PERDERSON, B., H. BRUUNSGAARD, M. KLOKKER, M. KAPPEL, D. MACLEAN, H. NIELSON, T. RHODE, H. ULLUM AND M. ZACHO. Exercise-induced immunomodulation—possible roles of neuroendocrine and metabolic factors. *Int. J. Sports Med.* 18(Suppl. 1):S2–S7. 1997.
16. SOTSKY, M., S. SHILO, AND H. SHAMOON. Regulation of counter-regulatory hormone secretion in man during exercise and hypoglycemia. *J. Clin. Endocrinol. Metab.* 68:9–16. 1989.
17. STAESSEN, J., R. FIOCCHI, R. FAGARD, P. HESPEL, AND A. AMERY. Carotid baroreflex at rest during exercise is not influenced by opioid receptor antagonism. *Eur. J. Appl. Physiol.* 59:131–137. 1989.
18. STEPHENSEN, L., M. KOLKA, R. FRANCESCONI, AND R. GONZALEZ. Circadian variations in plasma renin activity, catecholamines and aldosterone during exercise in women. *Eur. J. Appl. Physiol.* 58:756–764. 1989.
19. TABATA, I., F. OGITA, M. MIYACHI, AND H. SHIBAYAMA. Effect of low blood glucose on plasma CRF, ACTH, and cortisol during prolonged physical exercise. *J. Appl. Physiol.* 71:1807–1812. 1991.
20. TESCH, P. Skeletal muscle adaptations consequent to long-term heavy resistance exercise. *Med. Sci. Sports Exerc.* 20:S132–S134. 1993.
21. URHAUSEN, A., H. GABRIEL, AND W. KINDERMAN. Blood hormone as markers of training stress and overtraining. *Sports Med.* 20:251–276. 1995.
22. VINING, R., R. MCGINLEY, J. MAKSVYTIS, AND K. HO. Salivary cortisol: A better measure of adrenal cortisol function than serum cortisol. *Ann. Clin. Biochem.* 20:329–335. 1983.
23. VOIGT, K., M. ZIEGLER, M. GRUNERT-FUCHS, U. BICKEL, AND G. FEHM-WOLFSORF. Hormonal response to exhausting physical exercise: The role of predictability and controllability of the situation. *Psychoneuroendocrinology* 15:173–184. 1990.
24. WILMORE, J.H., AND D. COSTILL. *Physiology of Sport and Exercise*. Champaign: Human Kinetics, 1994. pp. 133–134.

Address correspondence to Dr. Dean E. Jacks, jacksde@wssu.edu.