# Adrenocortical Responses to Submaximal Exercise in Postmenopausal Black and White Women

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**The purpose of this study was to determine whether racial differences exist in the dehydroepiandrosterone (DHEA), dehydroepiandrosterone sulfate (DHEAS), and cortisol concentrations of black and white postmenopausal women at rest and in response to submaximal exercise. Twenty-three postmenopausal women (13 white and 10 black) were studied on 2 occasions. On one occasion subjects rested quietly for 4 hours (control day), whereas on the other occasion after 50 minutes of rest, subjects exercised at 70% of $Vo_2$ peak for 30 minutes on a cycle ergometer (exercise day). Blood was sampled at rest, during exercise, and during recovery and assayed for DHEA, DHEAS, and cortisol concentrations. Resting DHEA and cortisol concentrations and integrated area under the curve (AUC) were similar between the black and white women; however, the black women had lower resting DHEAS concentrations compared with the white women (DHEAS, black: 1.32 ± 0.29 *v* white: 2.18 ± 0.25 $\mu mol \cdot L^{-1}$, $P < .05$). Regardless of race, DHEA and cortisol AUC increased significantly above resting values ($P < .01$), but the exercise AUC for DHEA and cortisol were not different between the black and white women (DHEA: 607 ± 133 and 824 ± 108 min × $nmol \cdot L^{-1}$; cortisol: 9,604 ± 1,247 and 8,076 ± 1,093 min × $nmol \cdot L^{-1}$, respectively). No exercise-induced change in integrated DHEAS AUC was found in either group. In conclusion, racial differences exist in the resting DHEAS levels of postmenopausal women, but with no racial differences in resting DHEA and cortisol concentrations. Race had no impact on these adrenal hormone responses to submaximal exercise.**


BOTH DEHYDROEPIANDROSTERONE and dehydroepiandrosterone sulfate (DHEA and DHEAS) are part of adrenocortical secretion and decrease with age, such that low basal DHEA and DHEAS levels are found in postmenopausal women compared with young women.[1] The decrease in DHEA levels is due to a decrease in adrenal output in the zona reticularis region[2] and not to a change in metabolism.[3] Further the deficiency in adrenal androgen production with aging is not likely due to a deficiency of pituitary adrenocorticotropic hormone (ACTH) secretion, because cortisol levels do not decrease with age.[2] The biologic significance of DHEA and DHEAS is unknown, but declining levels have been associated with atherosclerosis, osteoporosis, obesity, insulin resistance, glucose intolerance, increased cancer risk, suppressed immune function, etc.[4-6] Finding an association between these hormone levels and health issues may be complicated by racial differences. Few studies have examined the racial differences in the adrenocortical hormones. One study has reported no differences in resting DHEAS and androstenedione between black and white women,[7] while others[8] found significantly lower resting DHEAS in black women compared with white women. Although differences in these hormones may exist, cortisol levels are similar between black and white women, despite black women having higher adrenocorticotropin-immunoreactivity (ACTH-IR) than white women.[9,10]

The adrenocortical response to exercise has been studied on numerous occasions, with particular attention devoted to cortisol. The cortisol response to exercise has been shown to increase at moderate to high intensities of aerobic and resistance exercise and is affected by time of day, prior nutrition, training level of the subject, etc.[11-15] The DHEA and DHEAS response has been less well studied. Much of the prior research has been conducted on younger individuals, and generally DHEA and DHEAS levels increase with moderate intensity exercise.[16] Velardo et al[17] reported that 1 hour of swimming resulted in increases in DHEAS in young men, while others have found associations between physical activity levels and higher DHEAS levels.[18] An acute bout of resistance exercise has been found to stimulate an increase in DHEA levels,[12] but no change in the resting DHEA levels in response to heavy resistance training.[19] Only one study has examined the exercise-induced responses in postmenopausal women,[20] but racial differences were not discussed. The exercise-induced adrenocortical responses of black and white premenopausal women has been studied[21]; and similar to findings at rest, maximal exercise resulted in higher ACTH-IR levels in black women, but without a concomitant increase in cortisol levels.[9,10] Thus the effect of exercise, as a physiologic stimulus of adrenocortical hormones, has not been examined in postmenopausal women. The purpose of this study was to investigate whether racial differences exist in the DHEA, DHEAS, and cortisol concentrations of black and white postmenopausal women at rest and in response to a submaximal bout of aerobic exercise.

## MATERIALS AND METHODS

### *Study Subjects*

Thirteen white and 10 black postmenopausal women, 50 to 61 years old, volunteered to participate in this research study. Both parents and grandparents of the subjects had to be American born, and both parents and grandparents were either Caucasian or African American. The Institutional Review Boards of Syracuse University and SUNY Upstate Medical University, Syracuse, NY approved this study, and all subjects signed an informed consent. The subjects were diagnosed as postmenopausal for a minimum of 1 year by their own physician. Ten of the 23

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*Submitted April 14, 2003; accepted May 30, 2003.*

*Supported in part by Grant No. 1RO3AG16370-01 from the National Institute on Aging.*

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*0026-0495/03/5212-0060$30.00/0*

*doi:10.1016/S0026-0495(03)00312-3*

subjects (7 white and 5 black women) were on hormone replacement therapy (HRT, Premarin, 0.625 mg; Wyeth-Ayerst, Philadelphia, PA) or Premarin plus medroxyprogesterone acetate (2.5 mg) and had been on HRT for approximately 4 years. All subjects were healthy, with no major chronic diseases such as diabetes, cardiovascular disease, atherosclerosis, hypertension, dyslipidemia, etc. Subjects who were on $\beta$-blocker medications were excluded from the study. Subjects were nonsmokers, physically inactive, with no recent participation in a regular exercise program.

### *Experimental Procedures*

Subjects visited the Human Performance Lab at Syracuse University on 3 occasions. During the first visit, subjects completed a medical history and a physical activity questionnaire and underwent an exercise stress test in the presence of a cardiologist. The exercise stress test was conducted on a cycle ergometer and the exercise protocol consisted of 2-minute exercise stages, starting at 50 W and increased 15 W every 2 minutes until volitional fatigue. Metabolic data (oxygen consumption, carbon dioxide production, and respiratory exchange ratio [RER]) were collected using standard open spirometric techniques with a previously calibrated Sensormedics 2900 metabolic cart (Anaheim, CA). $V_{O_2}$ peak was determined as the highest oxygen consumption attained during the exercise test. Heart rate, blood pressure, and rating of perceived exertion (RPE) were measured at rest and at the end of each 2-minute exercise stage. To ensure that a maximal effort was given, 2 of the following conditions had to be met: heart rate approaching age-predicted max, rate of perceived exertion (RPE) $>$ 17, and a leveling of $V_{O_2}$.[22] A continuous 12-lead electrocardiogram (ECG) recording was performed throughout the exercise stress test and examined by the cardiologist for any cardiac abnormalities. Subjects were excluded if they had cardiac abnormalities.

At the completion of the stress test, measurement of total body fat was conducted using the hydrostatic weighing method. Body density was measured with the underwater weight taken simultaneously with measurement of residual lung volume.[23] Percent body fat was calculated using age-specific equations.[24]

During visits 2 and 3, subjects were required to come to the Human Performance Lab for a control or an exercise study day, the order of the study days were randomized. Subjects reported to the Human Performance Lab at 7 AM, after an overnight 12-hour fast. Before the start of the experiment, a heparin lock was placed in the anticubital vein of the subjects' arm by a registered nurse and kept patent by a saline flush. After 30 minutes of quiet rest, blood samples were drawn over 4 hours at the following time points $-50$, 0, 10, 20, 30, 40, 50, 60, 180, and 240 minutes. On the exercise study day after 60 minutes of blood sampling, subjects exercised on a cycle ergometer for 30 minutes at 70% $V_{O_2}$ peak. After exercise, subjects rested quietly as they did on the control day.

### *Blood Sampling and Analysis*

Each blood sample was collected in one 5-mL EDTA tube, was centrifuged, and the plasma was aliquotted and frozen at a temperature of $-80$°C and later assayed for DHEA, DHEAS, and cortisol concentrations. Determination of DHEAS and DHEA concentrations were performed using a radioimmmunoassay procedure developed by Diagnostic Products (Los Angeles, CA) and Diagnostic Systems Lab (Webster, TX), respectively. Cortisol concentrations were measured with an enzyme immunoassay (EIA) developed by Diagnostic Systems Lab. To avoid any changes in assay variability, all hormone samples of the 2 visits of each subject were analyzed in the same assay. Inter- and intra-assay coefficients of variation were, respectively, 8.3% and 4.5% for DHEA, 9.1% and 5.2% for DHEAS, and 6.1% and 3.4% for cortisol.

**Table 1. Subject Characteristics**

| Variable | White (n = 13) | Black (n = 10) |
|---|---|---|
| Age (yr) | 54.6 ± 0.9 | 53.8 ± 1.4 |
| Weight (kg) | 67.6 ± 3.6 | 88.3 ± 5.4* |
| Height (m) | 1.61 ± 0.00 | 1.62 ± 0.00 |
| BMI ($kg \cdot m^{-2}$) | 25.9 ± 1.0 | 33.3 ± 1.8* |
| Body fat (%) | 36.8 ± 1.8 | 39.4 ± 2.4 |
| $V_{O_2}$ peak ($mL \cdot kg\ FFM^{-1} \cdot min^{-1}$) | 23.9 ± 0.4 | 25.49 ± 1.0 |

*$P < .05$ between groups.

### *Statistical Analysis*

The physical characteristics of the 2 groups were statistically compared using an independent $t$ test. Calculation of the integrated hormone concentration for DHEA, DHEAS, and cortisol (area under the curve [AUC]) was determined by the use of a trapezoidal method (GraphPad Prism, San Diego, CA, version 2.01) and is reported in absolute values (baseline hormone concentration: y = 0). A 2 × 2 analysis of variance (ANOVA) with repeated measures was performed (race × study day) to determine the effects of race and/or exercise on the DHEA, DHEAS, and cortisol AUC. An analysis of covariance (ANCOVA) was performed, with total body fat as the covariate, to control for the effect of differences in fat mass between the 2 races on the DHEA, DHEAS, and cortisol concentrations. The data are reported as mean ± SE, and significance was determined at an $\alpha$ level of 0.05.

## RESULTS

Table 1 shows the subject characteristics of the black and white women. The groups were similar in age, height, and percent body fat (39.4% ± 7.5% and 36.1% ± 6.4%, respectively), but the black women were heavier, had a greater fat mass, and body mass index (BMI) ($P < .01$). Aerobic capacity ($V_{O_2}$ peak) was similar between groups. We found no differences in the resting or exercise levels of DHEA, DHEAS, and cortisol of women who were on HRT and those who were not; thus all data were included in the analysis.

Comparison of the 2 resting blood samples on each study day showed no racial differences in the resting concentrations of DHEA and cortisol between the black and white women (DHEA, black: 24.0 ± 5.6; white: 29.1 ± 4.8 $nmol \cdot L^{-1}$; cortisol, black: 374.6 ± 67.9, white: 334 ± 59.5 $nmol \cdot L^{-1}$). However, black women were found to have significantly lower resting DHEAS concentrations compared with the white women (DHEAS, black: 1.32 ± 0.29 *v* white: 2.18 ± 0.25 $\mu mol \cdot L^{-1}$, $P < .05$). Both groups worked at a similar exercise intensity that was equivalent to approximately 70% of their $V_{O_2}$ max. Exercise caused a significant increase in the DHEA levels in both the black and white women ($P = .05$, Fig 1). The integrated DHEA concentrations increased significantly from a resting value of 756 ± 93 on the control day to 824 ± 108 $min \times nmol \cdot L^{-1}$ on the exercise day in the white women, and from 428 ± 106 on the control day to 607 ± 133 $min \times nmol \cdot L^{-1}$ on the exercise day for the black women. No racial differences were found for this exercise response. The integrated AUC for cortisol significantly increased from 5,920 ± 912 (control day) to 8,076 ± 1,093 $min \times nmol \cdot L^{-1}$ (exercise day) for the white women, and from 6,068 ± 1,040 (control day) to 9,604 ± 1,247 $min \times nmol \cdot L^{-1}$ (exercise day) for the black women ($P < .001$; Fig 2). The exercise day produced a

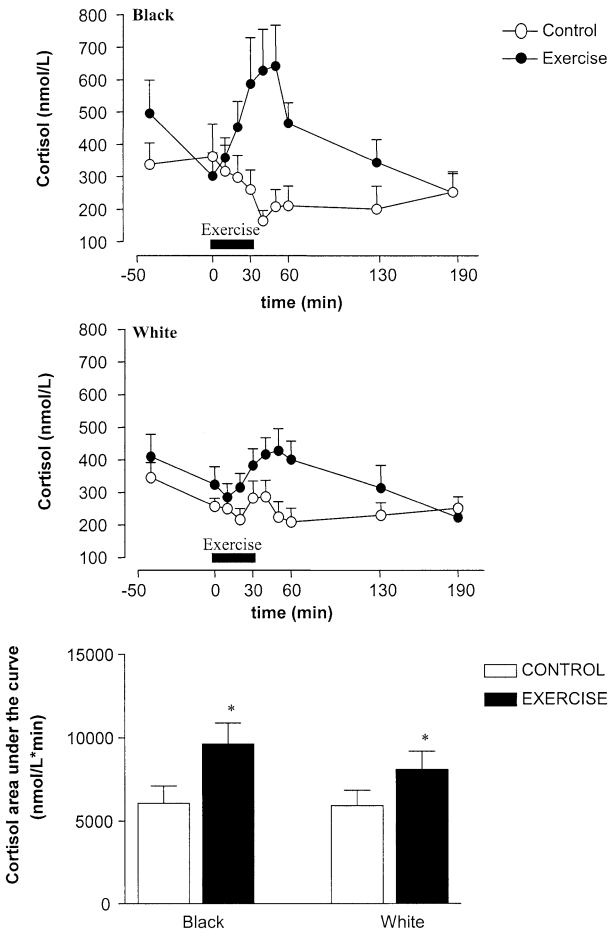


**Fig 1. Cortisol concentration and integrated AUC for the control and exercise session in black and white women *$P < .05$ difference between control and exercise session.**

higher peak cortisol concentration compared with the control day in both groups, and the peak cortisol concentration was significantly higher in the black women compared with the white women (black: 886 ± 96.19 *v* white: 604.9 ± 84.3 nmol · $L^{-1}$, $P < .001$). There were no racial differences in the exercise response. No significant interaction between race and study day was found for DHEA, DHEAS, and cortisol levels ($P > .05$).

Although resting DHEAS levels were lower ($P < .05$) in the black women, peak DHEAS concentrations and integrated AUC were not significantly different between the control and exercise day or between races (Fig 3). Adjusting DHEA, DHEAS, and cortisol concentrations for fat mass and HRT did not alter the results.

## DISCUSSION

Previous work has shown that DHEAS levels are lower[8] and ATCH-IR levels are higher in black women than in white women, but that free cortisol levels are not different.[9,10] The purpose of this study was to investigate whether racial differences exist in the adrenocortical hormones of black and white postmenopausal women at rest and in response to submaximal exercise. We found no difference in resting DHEA and cortisol concentrations in these women, although DHEAS levels were approximately 50% lower in the black women than the white women at rest. In younger women, Williams et al[25] reported that increased amounts of truncal fat and decreased amounts of fat on the legs were associated with increased serum DHEAS concentrations. Our subjects had a similar percent body fat, but the black women had greater total body fat. Adjusting for total fat did not alter the findings of our resting data (data not shown). The black women had lower resting DHEAS concentrations regardless of absolute levels of fat.

Although racial differences have been reported in the literature for a number of health variables between black and white women,[4-6] only 2 studies have studied the racial differences in resting DHEAS levels in women. Supporting our findings, Manson et al[8] observed significantly lower resting DHEAS concentrations in black women in comparison to white women, while others[7] reported no differences in the resting DHEAS and androstenedione concentrations between black and white women. Discrepancies in the findings could be potentially attributed to differences in menopausal status in the populations

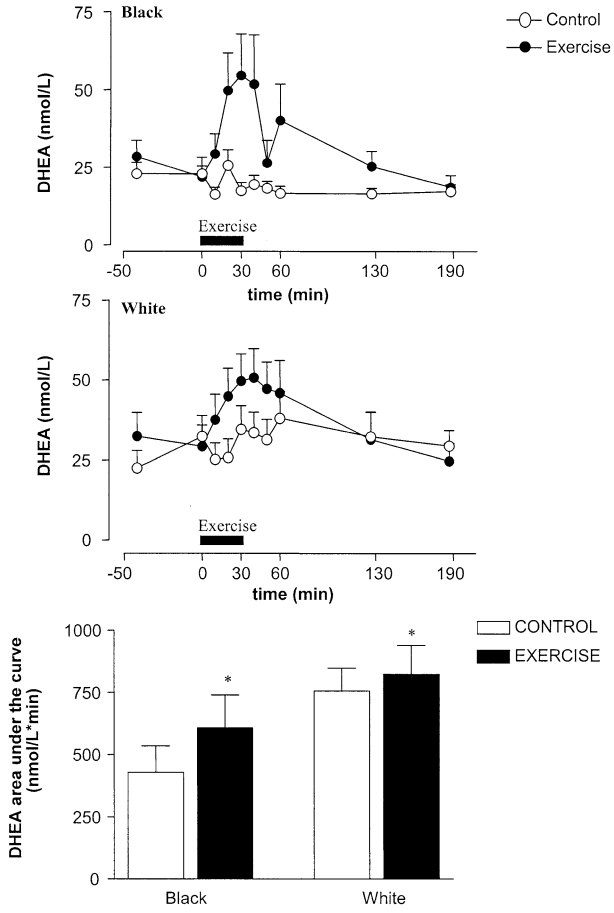


**Fig 2. DHEA concentration and integrated AUC for the control and exercise session in black and white women *$P < .05$ difference between control and exercise session.**

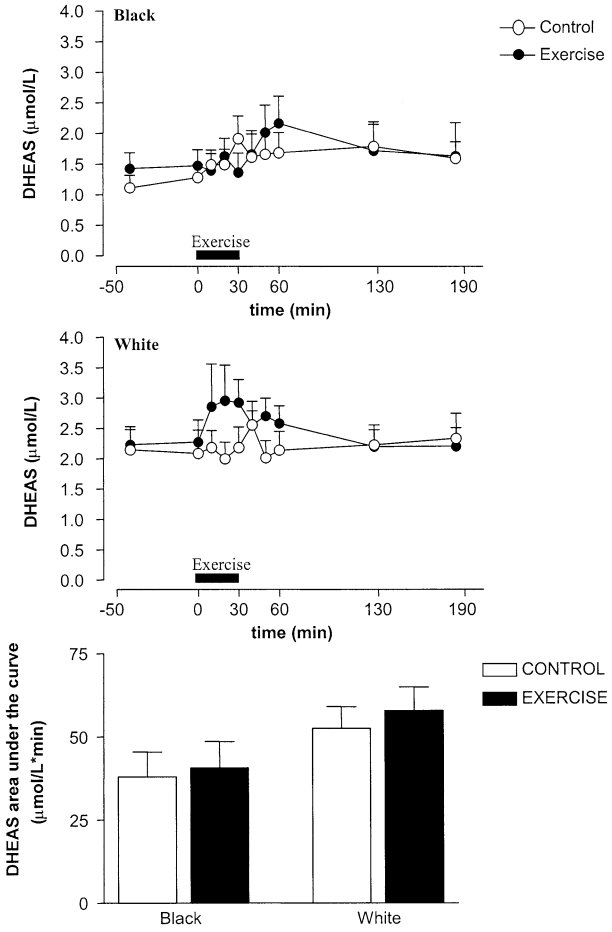


**Fig 3. DHEAS concentration and integrated AUC for the control and exercise session in black and white women.**

studied. In the present study, the women were early menopausal (~4.5 years), while in the Kleerekoper study[7] the women were at least 10 years postmenopause. Manson, on the other hand, studied premenopausal women. Potentially there are discrepancies in DHEAS levels between black and white women in the pre and early menopausal years, and these differences disappear with aging, such that no racial differences exist as the women become older.

This is the first study in postmenopausal women to demonstrate that moderately high intensity exercise (70% $Vo_2$ max) results in an increase in DHEA and cortisol levels regardless of races. A 120% increase in DHEA and a 50% increase in cortisol concentrations were observed before these levels returned towards baseline. Similarly a prior study[20] has reported that a single bout of aerobic exercise resulted in significant elevations of DHEA and cortisol concentrations. Exercise is known to stimulate the hypothalamus to increase corticotrophin-releasing hormone (CRH) secretion and, in turn, increase ACTH secretion.[26] Yanovski et al[21] have observed differences in the hypothalamic-pituitary-adrenal axis and in the plasma ACTH-IR of black and white women, with no differences in plasma cortisol levels. Additionally, ovine CRH stimulation has resulted in greater ACTH-IR in black than white men.[10] Using a physiologically relevant stress stimulus (a maximal exercise test), these investigators noted a greater ACTH-IR in black women than white women, with no racial difference in cortisol levels at the end of exercise or 10 minutes postexercise. Although we did not measure ACTH-IR, our findings are similar in that integrated cortisol concentrations during and postexercise were not different between races, but the black women did have higher peak cortisol levels. Despite the higher peak levels in black women, over time the cortisol levels diminished resulting in similar total hormone released over the blood-sampling period between races.

Both DHEA and DHEAS levels have been reported to be lower in older women than in younger women,[1,2,27] and this adrenal androgen deficiency most likely is in the zona reticularis where DHEAS and DHEA are formed.[2] The deficiency is due to changes in the functional and morphologic integrity of the zona reticularis. However, our data indicates that with exercise, DHEA levels increase, suggesting that the adrenals produce more DHEA, but need a profound physiologic stressor to release it. Thus, with aging the ability of the adrenals to respond to the stress has not been completely lost. In contrast to the differences observed with exercise in the integrated DHEA and cortisol concentrations and peak concentrations, we did not find any significant exercise-induced DHEAS response in either group of women. This contrasts a previous report[20] of an elevation in integrated DHEAS concentrations in white postmenopausal women. The lack of observable increase in DHEAS in our study could be attributed to the considerably longer half-life and higher concentrations of DHEAS relative to DHEA[20] and the time duration of the study may not have been long enough to see increases in DHEAS levels. Further compared with young adults,[28] DHEA sulfotransferase is reduced in older adults, possibly explaining the lack of increase in DHEAS levels with exercise of 30 minutes in duration.

Previous work[14] has indicated that HRT results in a higher DHEA and cortisol response to exercise. In contrast, we found no influence of HRT on DHEA, cortisol, or DHEAS levels either at rest or during exercise. Our finding is supported by the findings of Cumming et al[29] who have shown that long-term estrogen replacement in postmenopausal women had no influence on the decline of DHEAS, although others have reported that estrogen does suppress DHEA concentrations, but more so in young women than in older women.[30]

The overall physiologic significance of increases in DHEA levels with exercise is unclear. Previously published studies concerning the administration of oral DHEA in an older population has provided conflicting results. One study has shown that DHEA supplementation may restore neuroendocrine control of $\beta$-endorphin secretion possibly playing a role in improving well-being in postmenopausal women.[31] In young men receiving DHEA supplements reduced body fat, increased muscle mass, and reduced serum low-density lipoprotein cholesterol levels was reported.[32] Increased insulin-like growth factor-I (IGF-I) levels and self-reported increase in well-being[33] have also been reported. Yet, recently it was reported that DHEA supplementation did not increase exercise capacity, fasting glucose, and insulin levels or body composition in patients with

adrenal insufficiency.[34] In the present study, we demonstrated that a single bout of aerobic exercise resulted in an elevation of DHEA concentrations both in our black and white women, yet the effect of chronic exercise on circulating DHEA levels is not known. Potentially the exercise-induced DHEA increases may play a direct or indirect role in the health and well-being benefits associated with exercise. More research is needed to determine the effects of exercise, acute and chronic, on DHEA and to establish the role that DHEA plays on the various health improvements reported with exercise in older populations.

In conclusion, racial differences in resting DHEAS levels were found in this study, however the adrenocortical hormones respond similarly to exercise stress in black and white postmenopausal women. More research is needed to determine if the increases seen in these hormones with exercise provide any of the beneficial health and well-being effects associated with these hormones.

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