Published in final edited form as:

Exp Physiol. 2007 September ; 92(5): 871–879. doi:10.1113/expphysiol.2007.037408.

Differential aerobic exercise-induced changes in plasma aldosterone between African Americans and Caucasians

Jennifer M. Jones¹, Thomas C. Dowling², Jung-Jun Park¹, Dana A. Phares¹, Joon-Young Park¹, Thomas O. Obisesan³, and Michael D. Brown^{1,4}

¹Department of Kinesiology, University of Maryland, College Park, MD, USA

²Department of Pharmacy Practice and Science at the University of Maryland School of Pharmacy, Baltimore, MD, USA

³Department of Medicine, Howard University, Washington, DC, USA

⁴Department of Kinesiology, Temple University, Philadelphia, PA, USA

Abstract

Aldosterone influences the kidney's regulation of blood pressure (BP), but aldosterone can contribute to the pathogenesis of hypertension. Blood pressure is reduced with aerobic exercise training (AEX), but the extent to which plasma aldosterone (PA) levels change is unclear. The purpose of this study was to determine whether 6 months of AEX changed PA levels, 24 h sodium (Na⁺) excretion and BP in prehypertensive and hypertensive subjects and whether these changes differed according to ethnicity. The study was performed in the Kinesiology Department at the University of Maryland, College Park, and 35 (22 Caucasian; 13 African American) sedentary prehypertensive and hypertensive subjects completed 6 months of AEX. Blood samples were collected under fasting and supine conditions, and PA was measured by radioimmunoassay. In total population aerobic exercise training increased maximal oxygen consumption (24 ± 0.8 versus 28 ± 1 ml kg⁻¹ min⁻¹, $P < 0.001$) and decreased PA levels (97 ± 11 versus 72 ± 6 pg ml⁻¹, $P = 0.01$), body mass index (28 ± 0.5 versus 28 ± 0.5 kg m⁻², $P = 0.004$) and weight (85 ± 2 versus 83 ± 2 kg, $P = 0.003$). Aerobic exercise training decreased PA levels (from 119 ± 16 to 81 ± 7 pg ml⁻¹, $P = 0.02$) in the Caucasians but there was no change in BP or Na⁺ excretion. African American participants had no significant changes in PA levels, BP and Na⁺ excretion. Plasma aldosterone levels were 47% lower at baseline ($P = 0.01$) and 30% lower after AEX ($P = 0.04$) in African American participants compared with Caucasians. Baseline ($P = 0.08$) and final PA levels ($P = 0.17$) did not differ between the two groups after accounting for baseline and final intra-abdominal fat, respectively. The reduction in PA levels with AEX appeared to be driven by the change in PA levels in Caucasian participants. Fat distribution contributed to the ethnic differences in PA levels.

Essential hypertension is a chronic disease in which the aetiology is considered to be multifactorial and polygenic. The renin–angiotensin–aldosterone system (RAAS) has received significant attention because of its role in the regulation of blood pressure by the kidney (Hall *et al.* 1989). Aldosterone, a component of the RAAS, plays a necessary role in long-term regulation of blood pressure. However, there is evidence to suggest that aldosterone is involved in the pathogenesis of hypertension via its contribution to the development of endothelial

dysfunction (Schmidt *et al.* 2003; Arima *et al.* 2004; Oberleithner, 2005; Duprez, 2007) as well as through its influence on renal sodium (Na^+) handling (Guyton & Hall, 2000). Recently, Vasan *et al.* (2004) reported that elevated aldosterone levels, within the normal range, can contribute to the incidence of hypertension. Other studies have found elevated plasma aldosterone levels among individuals with essential hypertension compared with normotensive subjects (Zozaya *et al.* 1987; Forrester *et al.* 1997; Cortes *et al.* 2000).

Aerobic exercise training is effective in reducing blood pressure in hypertensive individuals (Roman *et al.* 1981; Hagberg *et al.* 1989; Dubbert *et al.* 1994; Seals *et al.* 1997). The mechanisms underlying exercise-training-induced reductions in blood pressure have yet to be elucidated. One possible mechanism is that alterations in Na^+ handling may be related to exercise-training-induced alterations in plasma aldosterone levels (Kiyonaga *et al.* 1985; Brown *et al.* 1997). Subsequently, only a limited number of studies have reported on changes in plasma aldosterone levels with aerobic exercise training in hypertensive individuals (Hespel *et al.* 1988; Braith *et al.* 1999). The present study was designed to increase our understanding of the influence that aerobic exercise training may have on aldosterone in a population in the early stages of disease progression.

The purpose of the present study was to determine whether 6 months of standardized aerobic exercise training changed plasma aldosterone levels, 24 h Na^+ excretion and blood pressure in Caucasian and African American middle-aged to older prehypertensive and hypertensive subjects and whether these changes differed by ethnicity. We hypothesized that aerobic exercise training would decrease plasma aldosterone levels. Additionally, we hypothesized that there would be a relationship between the change in plasma aldosterone levels and the change in Na^+ excretion and blood pressure.

Methods

Screening

Participants between the ages of 50 and 75 years were recruited from the College Park, Maryland and the District of Columbia metropolitan area. The Institutional Review Board of the University of Maryland, College Park approved the study, and written informed consent was obtained on all participants. The study conformed to the standards set by the Declaration of Helsinki. Participants were required to be sedentary, have a body mass index (BMI) $< 37 \text{ kg m}^{-2}$, and not have liver disease, diabetes, pulmonary disease, any form of cardiovascular disease (CVD) other than hypertension, or orthopedic conditions that would impair their ability to exercise. Females had to be postmenopausal for more than 2 years and were required to maintain their hormone replacement status for the entirety of the study. Potential participants on more than two antihypertensive medications were excluded.

Participants who met the initial study inclusion criteria provided written informed consent and were scheduled for two screening visits. Participants were excluded if they exhibited a fasting blood glucose $> 126 \text{ mg dl}^{-1}$ or a blood glucose greater than 200 mg dl^{-1} at 2 h of an oral glucose tolerance test (OGTT). Participants were excluded if they had a glomerular filtration rate (GFR) $< 60 \text{ ml min}^{-1} (1.73 \text{ m})^{-2}$, which was estimated by using the Modification of Diet in Renal Disease (MDRD) Study equation (Levey *et al.* 1999), and serum creatinine levels $> 1.5 \text{ mg dl}^{-1}$; both ensured that they did not have evidence of renal disease. The BMI was verified, and three casual blood pressure measurements were taken on each arm using a standard sphygmomanometer and JNC 7 guidelines (Chobanian *et al.* 2003). Participants with an average systolic blood pressure (SBP) < 120 or $> 159 \text{ mmHg}$ and/or diastolic blood pressure (DBP) < 80 or $> 99 \text{ mmHg}$ were excluded from the study.

During the second screening visit, participants performed a physician-supervised maximal treadmill exercise test to screen for cardiovascular, pulmonary or other chronic diseases that would prevent participants from exercise training.

Dietary stabilization/medication tapering

Participants were required to stay within 5% of their study entry body weight throughout the study. Weight stability was required because changes in weight and/or BMI are independently associated with changes in blood pressure (Winnicki *et al.* 2006) and aldosterone levels (Engeli *et al.* 2005). It was important to ensure that any changes to blood pressure or aldosterone were independent of weight changes. Participants attended a 6 week dietary class taught by a Registered Dietician twice a week and followed the American Heart Association (AHA) Step I diet for the entirety of the study. Participants were weighed once a week, throughout the 6 months, at one of their exercise sessions, and their weight was documented. If the participant was below their weight range they were encouraged to slightly increase their caloric intake with heart-healthy foods and if they were above their weight range they were encouraged to reduce their caloric intake. Participants using antihypertensive medications were tapered off of their medications during the 6 week dietary period with written approval from their personal physician. Participants who failed to maintain their blood pressure in the range 120–159/80–99 mmHg during the 6 week dietary period were excluded from the study.

Baseline and final testing

Casual blood pressure was measured in all participants on three separate days according to the JNC 7 guidelines (Chobanian *et al.* 2003). Participants underwent 24 h urine collection to measure 24 h Na⁺ and potassium (K⁺) excretion. To develop an exercise prescription specific for each participant and to assess their cardiovascular fitness, participants performed a physician-supervised maximal oxygen consumption ($\dot{V}_{O_{2max}}$) treadmill test. Participants began exercising on a treadmill at an intensity equivalent to 70% of the peak heart rate that they achieved during their screening exercise test. Participants were fitted with a mouthpiece and a nose clip in order to collect the expired air necessary to measure oxygen consumption. The gradient was increased by 2% every 2 min, and ECG, heart rate and blood pressure were measured every 2 min and after the test. The test was stopped when participants could no longer continue or upon onset of cardiovascular signs and/or symptoms (American College of Sports Medicine, 2000). Oxygen uptake was measured using a computerized on-line $\dot{V}_{O_2}$ system including a gas analyser (Mass Spectrometer MGA-1100, Marquette Electronics Inc., Milwaukee, WI, USA) and a bidirectional turbine flowmeter (Ventilation Measurement Module VMM-2, Interface Associates, Aliso Viejo, CA, USA). Oxygen consumption ($\dot{V}_{O_2}$) was measured continuously and, to ensure that a true $\dot{V}_{O_{2max}}$ was achieved, two of the three criteria (respiratory exchange ratio > 1.1, HR > [220 – age], and < 150 ml min⁻¹ increase in $\dot{V}_{O_2}$ during the last 2 min of the test) must have been met (American College of Sports Medicine, 2000).

Body composition was assessed by total-body dual energy X-ray absorptiometry (model DPX-L, Lunar Corporation, Madison, WI, USA). Participants were instructed to fast for 12 h prior to the start of the test.

Measurement of aldosterone

Participants were instructed to undergo a 12 h fast prior to blood sample collection and to exclude all ibuprofen, aspirin and antihistamines 48 h before the test (Cartledge & Lawson, 2000). Blood samples were collected between 6.30 and 8.30 am, a period of time in which plasma aldosterone levels are shown to be at their peak (Hurwitz *et al.* 2004), and after the participant had rested supine for 15–20 min (Stewart, 1999). Blood samples for the measurement of plasma aldosterone were collected in EDTA tubes and centrifuged at 1600 g

for 20 min at 4°C, and the plasma was aliquoted into 1.5 ml microtubes and stored at -20°C (Kley *et al.* 1985). Radioimmunoassay was used to measure plasma aldosterone levels (^{125}I Coat-A-Count Aldosterone Kit, Diagnostic Products Corp., Los Angeles, CA, USA). All baseline and final plasma samples from each participant were run in the same assay.

Exercise training

Participants underwent a 6 month supervised aerobic exercise training intervention, which was held at the University of Maryland Wellness Research Laboratory. Participants exercised 3 times per week and were given a wrist heart-rate monitor (Model 6124, Polar Electro, Canada) to monitor their prescribed heart rate. During the first week of exercise training, participants exercised for 20 min at 50% of heart rate reserve (HRR). Exercise duration was increased gradually by 5 min per week until the participants were exercising for 40 min. Starting at the sixth week, exercise intensity was increased by 5% of their HRR every week until they were exercising at 70% of HRR. Seated blood pressures were measured at the beginning and end of each exercise session. The participants recorded their blood pressure, heart rate, weight, exercise intensity and duration in a logbook provided for them. These logbooks were analysed to ensure that the participants were adhering to their exercise prescription. Once the participants completed the tenth week of exercise training, they were required to add an extra day of unsupervised exercise for 45–60 min at < 70% of HRR to their current exercise prescription. Only the data from participants that completed > 80% of their exercise training sessions were used in the data analysis.

Data analysis

Statistical analyses were performed using SPSS (version 15.0, Chicago, IL, USA). Dependent sample *t* tests were performed to test for differences between baseline and final values for subject characteristics and the primary outcome variables (plasma aldosterone levels, SBP, DBP and Na^+ excretion) in the total population. Dependent sample *t* tests were performed within each ethnic group to test the differences in the subject characteristics and in percentage body fat, intra-abdominal and subcutaneous fat, and the primary outcome variables. Independent sample *t* test was used to test for differences in subject characteristics and in percentage body fat, intra-abdominal and subcutaneous fat, and primary outcome variables between African Americans and Caucasians. Independent *t* tests were used to test for differences in the primary outcome variables between women and men. In order to determine whether prior use of antihypertensive medication affected how participants responded to the exercise intervention, an independent *t* test was used. Linear regression analyses were also conducted in the total sample and within each ethnic group to determine whether there were relationships between the change values for plasma aldosterone levels, 24 h Na^+ excretion and casual blood pressure.

An ANOVA was performed to test whether ethnicity contributed to the differences in baseline, final and the change in plasma aldosterone levels between African Americans and Caucasians. An ANCOVA was used to determine whether: (1) baseline and final percentage body fat accounted for ethnic differences in baseline and final plasma aldosterone levels; (2) baseline and final intra-abdominal body fat accounted for ethnic differences in baseline and final plasma aldosterone levels; and (3) baseline and final subcutaneous body fat accounted for ethnic differences in baseline and final plasma aldosterone levels. If changes were found within an ethnic group, a linear regression analysis was conducted to determine whether there was a relationship between baseline plasma aldosterone levels and the change in plasma aldosterone levels. An ANOVA was performed to test whether baseline plasma aldosterone levels accounted for the change in plasma aldosterone levels. This was followed by an ANOVA to determine whether the change in percentage body fat and intra-abdominal fat accounted for the change in plasma aldosterone levels. All values are reported as means $\pm$ S.E.M., and ANCOVA

results are reported as adjusted means. A P value of 0.05 was considered statistically significant.

Results

Total sample

Baseline subject characteristics are shown in Table 1. Thirty-five participants (20 males and 15 females) completed the 6 month aerobic exercise training intervention. Of those completing the aerobic exercise training intervention, 22 were Caucasian and 13 were African American. Participants in the study were prehypertensive and hypertensive but the mean blood pressure of the total group was in the prehypertensive range. The effect of aerobic exercise training on the primary outcome variables (blood pressure, Na^+ excretion and plasma aldosterone) was not different between men and women. Fourteen participants were on one or two antihypertensive therapies prior to the start of the study and 21 were not being treated for hypertension. There were no differences in the primary outcome variables between those previously treated *versus* those never treated.

The total group of participants significantly increased $\dot{V}_{\text{O}_{2\text{max}}}$ and reduced plasma aldosterone levels, body weight and BMI after 6 months of aerobic exercise training (Table 1). There were no significant changes in Na^+ and K^+ excretion or blood pressure. The change in plasma aldosterone levels with aerobic exercise training was not independently related to the change in SBP, DBP or 24 h Na^+ excretion.

Caucasian participants

The Caucasian participants significantly increased $\dot{V}_{\text{O}_{2\text{max}}}$, and reduced body weight, BMI and percentage body fat after 6 months of aerobic exercise training (Table 2). There were no significant changes in 24 h Na^+ and K^+ excretion, and SBP and DBP with aerobic exercise training (Table 2). Aerobic exercise training significantly reduced plasma aldosterone levels (Fig. 1). Baseline plasma aldosterone levels were related to the change in plasma aldosterone levels ($P < 0.001$) but after accounting for baseline plasma aldosterone levels the change in plasma aldosterone levels remained significant ($P < 0.001$). The change in plasma aldosterone levels with aerobic exercise training was not independently related to the change in SBP, DBP or 24 h Na^+ excretion in Caucasians.

African American participants

The African American participants also significantly increased their $\dot{V}_{\text{O}_{2\text{max}}}$ with aerobic exercise training (Table 2). In contrast to the Caucasian participants, there was no significant change in BMI, but body weight tended to decrease ($P = 0.06$) after aerobic exercise training (Table 2). There were no significant changes in SBP, DBP (Table 2) and plasma aldosterone levels (Fig. 1) with aerobic exercise training.

Ethnic differences

There was a significant effect of ethnicity on baseline plasma aldosterone levels ($P = 0.01$), in which baseline plasma aldosterone levels in African Americans were 47% lower compared with Caucasians (Fig. 1). There was also a significant effect of ethnicity on final plasma aldosterone levels ($P = 0.04$), in which final plasma aldosterone levels were 30% lower in African Americans compared with Caucasians (Fig. 1). Ethnicity tended to be related to the change in plasma aldosterone levels ($P = 0.11$). The change in SBP was different between the two ethnic groups ($P = 0.04$). There were no ethnic differences in the change in DBP and 24 h Na^+ excretion.

Independent samples *t* tests showed no significant differences in percentage body fat or BMI before and after aerobic exercise training between the two ethnic groups (Table 2). African Americans had lower body weight after aerobic exercise training compared with Caucasians (Table 2, $P = 0.03$). An ANCOVA indicated that after accounting for baseline percentage body fat, baseline ($P = 0.09$) and final ($P = 0.06$) plasma aldosterone levels were no longer significantly different between the two ethnic groups. Intra-abdominal fat was greater in Caucasians compared with African Americans before and after aerobic exercise training (Fig. 2A). After accounting for baseline intra-abdominal fat, baseline plasma aldosterone levels were no longer significantly different between the two ethnic groups ($P = 0.08$). After accounting for final intra-abdominal fat, final plasma aldosterone levels were no longer significantly different between the two ethnic groups ($P = 0.17$). Subcutaneous fat was greater in African Americans compared with Caucasians but the difference was not significant (Fig. 2B). Similarly, after accounting for baseline subcutaneous fat, baseline plasma aldosterone levels were no longer significantly different between the two ethnic groups ($P = 0.11$) and after accounting for final subcutaneous fat, final plasma aldosterone levels were no longer significantly different between the two ethnic groups ($P = 0.90$). In Caucasians only, when accounting for the change in percentage fat, intra-abdominal fat and baseline plasma aldosterone levels, the change in plasma aldosterone levels remained significant.

Discussion

The purpose of the present study was to determine whether 6 months of aerobic exercise training changed plasma aldosterone levels, 24 h Na^+ excretion and blood pressure among middle-aged to older prehypertensive and hypertensive subjects and whether these changes differed by ethnicity. Participants experienced a significant reduction in plasma aldosterone levels with aerobic exercise training; however, this reduction may have been driven by the change in plasma aldosterone levels in the Caucasian participants. Ethnicity appeared to influence plasma aldosterone levels before and after exercise training, but not the change in plasma aldosterone levels, Na^+ excretion and blood pressure with aerobic exercise training. Additionally, ethnic differences in plasma aldosterone levels were partly explained by ethnic differences in percentage body fat, intra-abdominal and subcutaneous body fat.

Only a few studies have found a change in plasma aldosterone levels with aerobic exercise training (Hespel *et al.* 1988; Braith *et al.* 1999). Hespel *et al.* (1988) were among the first to report that in a normotensive population, greater reductions in plasma aldosterone levels were associated with an increase in physical work capacity with aerobic exercise training. Braith *et al.* (1999) demonstrated that among heart failure patients, plasma aldosterone levels significantly decreased by approximately 50 pg ml^{-1} after 16 weeks of aerobic exercise training. The reduction in plasma aldosterone levels among the participants in the present study was smaller (25 pg ml^{-1}) than the plasma aldosterone reduction in the study of Braith *et al.* (1999). This was possibly due to higher baseline plasma aldosterone levels among the participants in the study of Braith *et al.* (1999) compared with those of the present study. Passino *et al.* (2006) also investigated the change in plasma aldosterone levels with aerobic exercise training in heart failure patients but found no change in plasma aldosterone levels after 9 months. One of the confounding factors contributing to the lack change may have been that these patients were taking RAAS blocking drugs.

There are data to support differences in aldosterone levels between Caucasian and African American individuals (Pratt *et al.* 1989; Langford *et al.* 1991; Fisher *et al.* 1994; He *et al.* 1998; Suh *et al.* 2004; Grim *et al.* 2005). In the present study, separate analyses within each ethnic group were conducted to determine whether such a difference existed in the present data. The African American participants had lower baseline and final plasma aldosterone levels compared with the Caucasian participants. It has been suggested that lower plasma aldosterone

levels among African Americans compared with Caucasians may be attributed to Na⁺ retention among African Americans (He *et al.* 1998). For this reason, 24 h Na⁺ excretion was measured in the present study; however, lower Na⁺ excretion among African Americans compared with Caucasians could not explain their lower plasma aldosterone levels.

We previously showed (Jones *et al.* 2006) that ethnic differences in baseline plasma aldosterone levels can be dependent on baseline percentage body fat. The present paper adds to our previous findings in that the differences in baseline percentage body fat between the two ethnic groups, and not ethnicity *per se*, accounted for ethnic differences in plasma aldosterone levels after aerobic exercise training. Specifically, the plasma aldosterone differences were associated with ethnic differences in intra-abdominal and subcutaneous fat. There is evidence of an interrelationship between aldosterone and adiposity (Goodfriend *et al.* 1999; Ehrhart-Bornstein *et al.* 2003). Additionally, there is evidence of upregulation of renin, angiotensin converting enzyme (ACE) and angiotensin II type 1 receptor (AT1R) gene in obese hypertensive subjects, as well as greater ACE and AT1R gene expression in intra-abdominal *versus* subcutaneous fat (Giacchetti *et al.* 2002). The greater intra-abdominal fat and lesser subcutaneous fat in Caucasians may have contributed to their greater plasma aldosterone levels before and after aerobic exercise training.

There is evidence of reductions in aldosterone levels with reductions in adiposity (Engeli *et al.* 2005). There was a significant reduction in percentage body fat and a non-significant reduction in intra-abdominal fat with exercise in Caucasian participants. Neither of these changes accounted for the change in plasma aldosterone level with exercise training in Caucasians, creating the possibility that other mechanisms may have been involved in the exercise-induced reduction in plasma aldosterone levels. Aerobic exercise training has been shown to suppress plasma renin activity (PRA) in normotensive and hypertensive individuals (Hespel *et al.* 1988; Dubbert *et al.* 1994; Kohno *et al.* 1997), leading to a reduction in angiotensin II and to a decrease in aldosterone biosynthesis (Hespel *et al.* 1988; Braith *et al.* 1999; Guyton & Hall, 2000). Sympathetic stimulation, which is known to activate renin release, leading to the release of angiotensin II (DiBona, 1989; Guyton & Hall, 2000), has been shown to decrease with aerobic exercise training in hypertensive individuals. Therefore, it can be suggested that the reduction in sympathetic stimulation with aerobic exercise training may be a possible mechanism by which aldosterone biosynthesis decreases (Hespel *et al.* 1988).

It is possible that the lack of change in aldosterone levels is a consequence of the lower baseline plasma aldosterone levels among African Americans. The mechanisms contributing to this phenomenon among African Americans require further research. African Americans tended only to reduce BMI and weight with exercise training. As noted in the Methods, a large change in BMI or weight, despite the intense exercise, was not expected in this study because participants were required to be weight stable.

After 6 months of aerobic exercise training, there was no significant reduction in SBP and DBP in the total sample and in either ethnic group. Significant reductions in SBP and DBP have been reported with similar aerobic exercise training programmes in which baseline blood pressures were at hypertensive levels (Hagberg *et al.* 1989; Seals & Reiling, 1991). The mean baseline blood pressures for participants in the present study were below hypertensive levels, possibly contributing to the lack of change with aerobic exercise training. Previous aerobic exercise training studies have supported this finding (Gilders *et al.* 1989; Cononie *et al.* 1991). There was a significant difference in the change in SBP between the two ethnic groups, which resulted from the slight reduction SBP among the Caucasian participants and increase in SBP among the African American participants.

There was no relationship between the change in plasma aldosterone and the change in BP, 24 h Na⁺ and K⁺ excretion in the total population or in either ethnic group. This finding is supported by previous research in hypertensive populations (Zozaya *et al.* 1987). The lack of contribution of the change in plasma aldosterone to the change in BP and Na⁺ and K⁺ excretion does not discount the beneficial effects of the reduction in aldosterone, based on its influence on the vasculature and development of endothelial dysfunction (Takeda *et al.* 1995; Oberleithner, 2004; Fiebeler & Luft, 2005). Therefore, the reduction in plasma aldosterone levels with aerobic exercise training in a prehypertensive and hypertensive population may be beneficial in slowing the progression of development of hypertension.

The present study did not use a control group to compare the changes in plasma aldosterone levels, and the rationale for this was based on the population under study. The JNC 7 guidelines specify that a prehypertensive population should be treated with lifestyle modification and that a hypertensive population should be treated with lifestyle modification and first-line antihypertensive therapy if needed. Both of these populations were included in the present study; therefore, to include an untreated control group with similar blood pressure levels to the treated group would have been unethical. Secondly, including a control group treated with the JNC 7 recommended first-line therapy diuretic would have been problematical, because it may have led to changes in aldosterone levels owing to its influence on fluid volume. Therefore, a quasi-experimental design, such as in the present study, with pre-/post-intervention testing can be justified (Radosevich, 2005).

In conclusion, the results from the present study indicate that the reduction in plasma aldosterone levels with aerobic exercise training among the total population appeared to be driven by the reduction in plasma aldosterone levels among the Caucasian participants. Basal plasma aldosterone levels, as well as the change in plasma aldosterone levels, differed between African American and Caucasian prehypertensive and hypertensive subjects, but regional fat distribution between African Americans and Caucasians may have contributed to these findings. The unaffected plasma aldosterone levels among the African American participants may have been the result of their low baseline plasma aldosterone levels. Further research is needed to uncover the mechanism responsible for the lack of change in plasma aldosterone levels in African Americans and to determine whether this phenomenon is exclusive to African Americans or whether it exists in other ethnic groups.

References

- American College of Sports Medicine. ACSM's Guidelines for Exercise Testing and Prescription. Vol. 6th edn.. Baltimore: Williams & Wilkins; 2000.
- Arima S, Kohagura K, Xu HL, Sugawara A, Uruno A, Satoh F, Takeuchi K, Ito S. Endothelium-derived nitric oxide modulates vascular action of aldosterone in renal arteriole. *Hypertension* 2004;43:352–357. [PubMed: 14691200]
- Braith RW, Welsch MA, Feigenbaum MS, Kluess HA, Pepine CJ. Neuroendocrine activation in heart failure is modified by endurance exercise training. *J Am Coll Cardiol* 1999;34:1170–1175. [PubMed: 10520808]
- Brown MD, Moore GE, Korytkowski MT, McCole SD, Hagberg JM. Improvement of insulin sensitivity by short-term exercise training in hypertensive African American women. *Hypertension* 1997;30:1549–1553. [PubMed: 9403581]
- Cartledge S, Lawson N. Aldosterone and rennin measurements. *Ann Clin Biochem* 2000;37:262–278. [PubMed: 10817239]
- Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, Jones DW, Materson BJ, Oparil S, Wright JT Jr, Roccella EJ. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. *JAMA* 2003;289:2560–2572. [PubMed: 12748199]

- Cononie CC, Graves JE, Pollock ML, Phillips MI, Sumners C, Hagberg JM. Effect of exercise training on blood pressure in 70- to 79-yr-old men and women. *Med Sci Sports Exerc* 1991;23:505–511. [PubMed: 2056908]
- Cortes P, Fardella C, Oestreicher E, Gac H, Mosso L, Soto J, Foradori A, Claverie X, Ahuad J, Montero J. Excess of mineralocorticoids in essential hypertension: clinical-diagnostic approach [in Spanish]. *Rev Med Chil* 2000;128:955–961. [PubMed: 11349499]
- DiBona GF. Neural control of renal function: cardiovascular implications. *Hypertension* 1989;13:539–548. [PubMed: 2661424]
- Dubbert PM, Martin JE, Cushman WC, Meydrech EF, Carroll RG. Endurance exercise in mild hypertension: effects on blood pressure and associated metabolic and quality of life variables. *J Hum Hypertens* 1994;8:265–272. [PubMed: 8021906]
- Duprez DA. Aldosterone and the vasculature: mechanisms mediating resistant hypertension. *J Clin Hypertens (Greenwich)* 2007;9:13–18. [PubMed: 17215650]
- Ehrhart-Bornstein M, Lamounier-Zepter V, Schraven A, Langenbach J, Willenberg HS, Barthel A, Hauner H, McCann SM, Scherbaum WA, Bornstein SR. Human adipocytes secrete mineralocorticoid-releasing factors. *Proc Natl Acad Sci U S A* 2003;100:14211–14216. [PubMed: 14614137]
- Engeli S, Bohnke J, Gorzelniak K, Janke J, Schling P, Bader M, Luft FC, Sharma AM. Weight loss and the renin-angiotensin-aldosterone system. *Hypertension* 2005;45:356–362. [PubMed: 15630041]
- Fiebeler A, Luft FC. The mineralocorticoid receptor and oxidative stress. *Heart Fail Rev* 2005;10:47–52. [PubMed: 15947891]
- Fisher ND, Gleason RE, Moore TJ, Williams GH, Hollenberg NK. Regulation of aldosterone secretion in hypertensive blacks. *Hypertension* 1994;23:179–184. [PubMed: 8307626]
- Forrester T, McFarlane-Anderson N, Bennett FI, Wilks R, Cooper R, Rotimi C, Morrison L, Ward R. The angiotensin converting enzyme and blood pressure in Jamaicans. *Am J Hypertens* 1997;10:519–524. [PubMed: 9160762]
- Giacchetti G, Faloia E, Mariniello B, Sardu C, Gatti C, Camilloni MA, Guerrieri M, Mantero F. Overexpression of the renin-angiotensin system in human visceral adipose tissue in normal and overweight subjects. *Am J Hypertens* 2002;15:381–388. [PubMed: 12022238]
- Gilders RM, Voner C, Dudley GA. Endurance training and blood pressure in normotensive and hypertensive adults. *Med Sci Sports Exerc* 1989;21:629–636. [PubMed: 2626084]
- Goodfriend TL, Kelley DE, Goodpaster BH, Winters SJ. Visceral obesity and insulin resistance are associated with plasma aldosterone levels in women. *Obes Res* 1999;7:355–362. [PubMed: 10440591]
- Grim CE, Cowley AW Jr, Hamet P, Gaudet D, Kaldunski ML, Kotchen JM, Krishnaswami S, Pausova Z, Roman R, Tremblay J, Kotchen TA. Hyperaldosteronism and hypertension: ethnic differences. *Hypertension* 2005;45:766–772. [PubMed: 15699471]
- Guyton, AC.; Hall, JE. *Textbook of Medical Physiology*. Vol. 10th edn.. Vol. 19. Philadelphia: W. B. Saunders Company; 2000. p. 201–209.
- Hagberg JM, Montain SJ, Martin WH 3rd, Ehsani AA. Effect of exercise training in 60- to 69-year-old persons with essential hypertension. *Am J Cardiol* 1989;64:348–353. [PubMed: 2756880]
- Hall JE, Coleman TG, Guyton AC. The renin-angiotensin system. Normal physiology and changes in older hypertensives. *J Am Geriatr Soc* 1989;37:801–813. [PubMed: 2666488]
- He FJ, Markandu ND, Sagnella GA, MacGregor GA. Importance of the renin system in determining blood pressure fall with salt restriction in black and white hypertensives. *Hypertension* 1998;32:820–824. [PubMed: 9822438]
- Hespeel P, Lijnen P, Van Hoof R, Fagard R, Goossens W, Lissens W, Moerman E, Amery A. Effects of physical endurance training on the plasma renin-angiotensin-aldosterone system in normal man. *J Endocrinol* 1988;116:443–449. [PubMed: 3280723]
- Hurwitz S, Cohen RJ, Williams GH. Diurnal variation of aldosterone and plasma renin activity: timing relation to melatonin and cortisol and consistency after prolonged bed rest. *J Appl Physiol* 2004;96:1406–1414. [PubMed: 14660513]
- Jones J, Park JJ, Dowling T, Phares D, Park JY, Brown M. Role of potassium excretion and percent body fat on ethnic differences in plasma aldosterone levels. *Ethn Dis* 2006;16S4–10–54–14

- Kiyonaga A, Arakawa K, Tanaka H, Shindo M. Blood pressure and hormonal responses to aerobic exercise. *Hypertension* 1985;7:125–131. [PubMed: 2984115]
- Kley HK, Schlaghecke R, Kruskemper HL. Stability of steroids in plasma over a 10-year period [in German]. *J Clin Chem Clin Biochem* 1985;23:875–878. [PubMed: 3831227]
- Kohno K, Matsuoka H, Takenaka K, Miyake Y, Nomura G, Imaizumi T. Renal depressor mechanisms of physical training in patients with essential hypertension. *Am J Hypertens* 1997;10:859–868. [PubMed: 9270080]
- Langford HG, Cushman WC, Hsu H. Chronic effect of KCl on black-white differences in plasma renin activity, aldosterone, and urinary electrolytes. *Am J Hypertens* 1991;4:399–403. [PubMed: 2069772]
- Levey AS, Bosch JP, Lewis JB, Greene T, Rogers N, Roth D. A more accurate method to estimate glomerular filtration rate from serum creatinine: a new prediction equation. Modification of Diet in Renal Disease Study Group. *Ann Intern Med* 1999;130:461–470. [PubMed: 10075613]
- Oberleithner H. Unorthodox sites and modes of aldosterone action. *News Physiol Sci* 2004;19:51–54. [PubMed: 15016902]
- Oberleithner H. Aldosterone makes human endothelium stiff and vulnerable. *Kidney Int* 2005;67:1680–1682. [PubMed: 15840012]
- Passino C, Severino S, Poletti R, Piepoli MF, Mammini C, Clerico A, Gabutti A, Nassi G, Emdin M. Aerobic training decreases B-type natriuretic peptide expression and adrenergic activation in patients with heart failure. *J Am Coll Cardiol* 2006;47:1835–1839. [PubMed: 16682309]
- Pratt JH, Jones JJ, Miller JZ, Wagner MA, Fineberg NS. Racial differences in aldosterone excretion and plasma aldosterone concentrations in children. *N Engl J Med* 1989;321:1152–1157. [PubMed: 2677724]
- Radosevich, D. Designing an Outcomes Research Study. In: Kane, R., editor. *Understanding Health Care Outcomes*. Vol. 2nd edn. Jones & Bartlett Publishers, Inc.; 2005. p. 23–57.
- Roman O, Camuzzi AL, Villalon E, Klenner C. Physical training program in arterial hypertension. A long-term prospective follow-up. *Cardiology* 1981;67:230–243. [PubMed: 7248998]
- Schmidt BM, Oehmer S, Delles C, Bratke R, Schneider MP, Klingbeil A, Fleischmann EH, Schmieder RE. Rapid nongenomic effects of aldosterone on human forearm vasculature. *Hypertension* 2003;42:156–160. [PubMed: 12860833]
- Seals DR, Reiling MJ. Effect of regular exercise on 24-hour arterial pressure in older hypertensive humans. *Hypertension* 1991;18:583–592. [PubMed: 1937660]
- Seals DR, Silverman HG, Reiling MJ, Davy KP. Effect of regular aerobic exercise on elevated blood pressure in postmenopausal women. *Am J Cardiol* 1997;80:49–55. [PubMed: 9205019]
- Stewart PM. Mineralocorticoid hypertension. *Lancet* 1999;353:1341–1347. [PubMed: 10218547]
- Suh A, DeJesus E, Rosner K, Lerma E, Yu W, Young JB, Rosa RM. Racial differences in potassium disposal. *Kidney Int* 2004;66:1076–1081. [PubMed: 15327401]
- Takeda R, Hatakeyama H, Takeda Y, Iki K, Miyamori I, Sheng WP, Yamamoto H, Blair IA. Aldosterone biosynthesis and action in vascular cells. *Steroids* 1995;60:120–124. [PubMed: 7792796]
- Vasan RS, Evans JC, Larson MG, Wilson PW, Meigs JB, Rifai N, Benjamin EJ, Levy D. Serum aldosterone and the incidence of hypertension in nonhypertensive persons. *N Engl J Med* 2004;351:33–41. [PubMed: 15229305]
- Winnicki M, Bonso E, Dorigatti F, Longo D, Zaetta V, Mattarei M, D'Este D, Laurini G, Pessina AC, Palatini P. Effect of body weight loss on blood pressure after 6 years of follow-up in stage 1 hypertension. *Am J Hypertens* 2006;19:1103–1109. [PubMed: 17070419]
- Zozaya J, Vilorio M, Castro A. Essential arterial hypertension: plasma and urinary aldosterone alterations. *Southern Med J* 1987;80:1224–1227.

Acknowledgements

We wish to thank the participants of the Gene Exercise Research Study. Also a special thanks to Dr Ken Bauer in the Department of Pharmacy Practice and Sciences for the use of his laboratory facilities. This study was supported by NIH/NIA grant no. K01AG019640 and NIH/NIA grant nos AG15384, AG17474 and AG00268 and K23–AG–60980.

Figure 1. Baseline and final plasma aldosterone levels in Caucasian and African American participants

Figure 2. Baseline and Final Fat Distribution in Caucasian and African American participants
 A, baseline and final plasma intra-abdominal fat mass in Caucasian and African American participants. B, baseline and final plasma subcutaneous fat mass in Caucasian and African American participants.

Table 1

Comparison of variables before and after aerobic exercise training in the total sample

Variable	<i>n</i>	Before	After
Age (years)	35	59 ± 1	—
BMI (kg m ⁻²)	35	28 ± 0.5	28 ± 0.5*
Weight (kg)	34	85 ± 2	83 ± 2*
$V_{O_{2max}}$ (ml kg ⁻¹ min ⁻¹)	34	24 ± 0.8	28 ± 1*
Serum creatinine (mg dl ⁻²)	34	1.0 ± 0.03	—
GFR (ml min ⁻¹ (1.73 m) ⁻²)	34	77 ± 3	—
Systolic BP (mmHg)	30	132 ± 2	132 ± 2
Diastolic BP (mmHg)	29	84 ± 1	84 ± 1
Plasma aldosterone (pg ml ⁻¹)	33	97 ± 11	72 ± 6
Na ⁺ excretion (mmol day ⁻¹)	34	121 ± 9	128 ± 10
K ⁺ excretion (mmol day ⁻¹)	34	67 ± 4	70 ± 4

Values are unadjusted means ± S.E.M.

* $P \leq 0.05$.

Table 2
Comparison of variables before and after aerobic exercise training in African Americans and Caucasians

Variable	African American			Caucasian		
	<i>n</i>	Baseline	Final	<i>n</i>	Baseline	Final
BMI (kg m ⁻²)	13	28 ± 1.0	27 ± 0.8	22	29 ± 0.7	28 ± 0.7 [*]
Weight (kg)	13	79 ± 3	77 ± 3 [†]	22	88 ± 3	86 ± 3 [*]
Percentage body fat	10	40 ± 3	39 ± 3	16	36 ± 2	34 ± 2 [*]
<i>V</i> _{O₂max} (ml kg ⁻¹ min ⁻¹)	13	22 ± 1	26 ± 2 [*]	21	26 ± 1	29 ± 1 [*]
Systolic BP (mmHg)	10	130 ± 3	134 ± 4	20	133 ± 3	131 ± 2
Diastolic BP (mmHg)	9	85 ± 2	84 ± 2	20	84 ± 2	84 ± 2
Na ⁺ excretion (mmol day ⁻¹)	13	114 ± 10	118 ± 11	21	126 ± 14	135 ± 15
K ⁺ excretion (mmol day ⁻¹)	13	57 ± 8	69 ± 8	21	73 ± 5	70 ± 4

Values are unadjusted means ± SEM.

^{*} Training effect and [†] ethnic effect, both *P* ≤ 0.05.