Reduction of plasma aldosterone and arterial stiffness in obese pre- and stage1 hypertensive subjects after aerobic exercise

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Abstract

Obesity-related hypertension is associated with increased activity of the renin-angiotensin-aldosterone system (RAAS), increasing arterial stiffness. Aerobic exercise decreases pulse wave velocity (PWV), therefore a treatment option for hypertension and obesity. Assess RAAS activity and PWV before and after 4 weeks of aerobic training in unmedicated, pre-to-stage-1 hypertensives. Ten obese subjects (52±3.2 years, body mass index ¼ 33.5±1.4) performed 30 min of aerobic exercise on a treadmill 3 days per week at 65%of peak oxygen consumption (VO2peak). Descriptive characteristics, systolic and diastolic blood pressure (SBP and DBP), PWV, and a blood draw was performed at baseline, following the 4-week control and training interventions. No differences in descriptive characteristics during the control period were observed, however, a significant decrease in plasma aldosterone (ALDO) (255.4±75 to 215.8±66 pg ml 1, P ¼ 0.001), SBP (140±12 to 136±10.4 mm Hg; P ¼ 0.02), DBP (89±4.2 to 85±6.3 mm Hg; P ¼ 0.03) and central PWV (11.2±0.6 to 9.8±0.8 m s 1; P ¼ 0.04) was shown pre-to-post exercise training. Four weeks of moderate-intensity aerobic training in obese, hypertensives decreases plasma ALDO independently of body weight and is significantly correlated to decreases in PWV reductions.

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Reduction of plasma aldosterone and arterial stiffness in obese pre- and stage1 hypertensive subjects after aerobic exercise

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Obesity-related hypertension is associated with increased activity of the renin-angiotensin-aldosterone system (RAAS), increasing arterial stiffness. Aerobic exercise decreases pulse wave velocity (PWV), therefore a treatment option for hypertension and obesity. Assess RAAS activity and PWV before and after 4 weeks of aerobic training in unmedicated, pre-to-stage-1 hypertensives. Ten obese subjects (52 ± 3.2 years, body mass index = 33.5 ± 1.4) performed 30 min of aerobic exercise on a treadmill 3 days per week at 65% of peak oxygen consumption (VO_{2peak}). Descriptive characteristics, systolic and diastolic blood pressure (SBP and DBP), PWV, and a blood draw was performed at baseline, following the 4-week control and training interventions. No differences in descriptive characteristics during the control period were observed, however, a significant decrease in plasma aldosterone (ALDO) (255.4 ± 75 to 215.8 ± 66 pg ml^{-1}, $P = 0.001$), SBP (140 ± 12 to 136 ± 10.4 mm Hg; $P = 0.02$), DBP (89 ± 4.2 to 85 ± 6.3 mm Hg; $P = 0.03$) and central PWV (11.2 ± 0.6 to 9.8 ± 0.8 m s^{-1}; $P = 0.04$) was shown pre-to-post exercise training. Four weeks of moderate-intensity aerobic training in obese, hypertensives decreases plasma ALDO independently of body weight and is significantly correlated to decreases in PWV reductions.

INTRODUCTION

The incidence of obesity and hypertension is escalating rapidly in the United States. Population data report that there has been a doubling of obese individuals since 1980, with an increase in the number of obese hypertensives presently at 41% of the population.[1] Moreover, research reports that hypertension is six times more frequent in obese than in lean individuals.[2,3] This disconcerting synergism brings serious health related problems as obesity-related hypertension causes hemodynamic alterations through structural adaptations as well as metabolic mechanisms.[4]

It has been shown that compared with lean controls, obese individuals report elevated plasma renin activity (PRA) as well as angiotensin II (Ang II) and plasma aldosterone (ALDO) levels that cause vasoconstriction, ions, water and sodium reabsorption leading to further increases in blood pressure (BP).[5] The higher systemic blood volumes and consequent greater cardiac output reported by obese hypertensives may lead to a concomitant increase in peripheral resistance and related increases in pulse wave velocity (PWV) inducing heart and vascular wall remodeling.[4,6] Therefore, the renin-angiotensin-aldosterone system (RAAS) is an important mechanism regulating fluid balance.

Clinical data report that attenuation of PRA through pharmacological blockade or weight loss has been shown to decrease ALDO and consequently resting BP.[7,8] Moderate-intensity aerobic exercise is prescribed as a cornerstone therapy for the prevention and treatment of obesity and hypertension.[9] It has been shown that physical activity can positively affect the vasculature, lowering PWV and improving arterial distensibility reducing resting systolic (SBP) and diastolic (DBP) blood pressure by ~4 mm Hg after only 4 weeks of moderate-intensity aerobic exercise.[10] These BP reductions are of major clinical significance as decreases in resting BP of 3 mm Hg have been shown to reduce the incidence of stroke and all-cause mortality by 3- and 7-fold, respectively.[11] However, there are no known studies linking the beneficial changes in the RAAS with aerobic exercise training in pre-to-stage-1 unmedicated hypertensive humans.

Therefore, the purpose of this original independent prospective investigation was to elucidate the effect of moderate-intensity aerobic exercise on PRA and plasma Ang II and ALDO as well as pulse wave transit times in an unmedicated, pre-to-stage-1 hypertensive population.

MATERIALS AND METHODS

Subjects

Ten obese, unmedicated, pre-to-stage-1 hypertensive[12] individuals (seven males, three females) between 40 and 60 years of age were recruited through local community physicians after they had been identified within the last year with pre- ($n = 4$) or stage-1 essential hypertension ($n = 6$). The inclusion criteria were: (1) no history of diabetes, coronary heart disease or kidney disease; (2) being nonsmokers; and (3) not being on any medications, including antihypertensives or anti-inflammatories as identified by their physicians and by a health history questionnaire. Due to the confounding influence of estrogen on cardiovascular measures, only postmenopausal women (history >12 months of amenorrhea) not currently under hormone replacement therapy were recruited. The present study was approved by the Institutional Review Board and all subjects gave written consent.

Experimental design

Subjects reported to the laboratory at 0700 hours for each of the three visits: at the start of testing for baseline measures (BLs), at the end of the

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4-week, pre-training control period (Pre) and at the end of the 4-week aerobic training period, post exercise training (Post). Subjects received cardiovascular and peak oxygen consumption (VO_{2peak}) evaluations followed by fasted blood marker assessment at 0700 hours within 24 h of the final exercise session.

All subject visits took place at the same time of day to minimize diurnal influences on tested physiological parameters. Pre-testing started with a manual BP assessment and then subjects were asked to rest quietly in the supine position in a dimly lit room for 15 min. Next, PWV was recorded at central and distal sites followed by an electrocardiogram (heart rate) and beat-to-beat BP recordings for a period of 10 min. After the second laboratory visit, each subject underwent a supervised, 4-week aerobic training program. The training prescription was designed using the values attained from the preliminary exercise testing sessions. Aerobic training consisted of 30 min supervised treadmill exercise, 3 days per week at 65% of their VO_{2peak}. At the conclusion of 4 weeks, the subjects reported back to the lab within 24 h of their last exercise session for post-measurements.

Each exercise training session was supervised by a trained exercise physiologist and subject compliance was 100%.

Anthropometric and body composition assessment

Body mass and stature were measured using a beam scale and a stadiometer to the nearest 0.5 kg and 0.5 cm, respectively. Body mass index was calculated as weight (kg) divided by height (m) squared. Body composition assessment was determined by a BodPod whole body plethysmography test (BodPod, Life Measurement Inc., Concord, CA, USA).

Peak aerobic capacity

To assess peak oxygen consumption, a modified Balke protocol was utilized as previously described.[13,14] Briefly, subjects started the test walking at a speed of 3 m.p.h. with a 0% grade for 3 min. Treadmill speed was then increased until a comfortable pace was reached and held constant as grade increased by 2.5% every 3 min until volitional fatigue was reached. Heart rate was measured and recorded every minute throughout the test using a Polar Heart Rate Monitor (Polar Electro Inc., Woodbury, NY, USA). Ratings of perceived exertion were also assessed at the end of each 3-min step.[15] Expired gases were analyzed using a Quark b2 breath-by-breath metabolic system (Cosmed, Rome, Italy). Maximal exercise effort was considered achieved when subjects met three of the following four criteria: (a) no change in heart rate with a change in workload; (b) a final rating of perceived exertion score of 17 or greater on the Borg scale (scale 6–20); (c) an RER greater than 1.15; and (d) a 'plateau' (increase of no more than 150 ml) in oxygen uptake with an additional incremental increase in workload.

Central and peripheral arterial stiffness

PWV measurements were acquired with two MD6 bidirectional transcutaneous Doppler probes (Hokanson, Bellevue, WA, USA) in accordance with the guidelines from the Clinical Application of Arterial Stiffness Task Force 3 and as previously reported by our laboratory.[14,16] Each subject was monitored with an EKG (modified CM5) and heart rate data was gated in phase with the PWV measurements and used as timing markers for PWV identification. Central PWV measures were obtained from the left common carotid artery to the left femoral artery. Distances from the carotid site to the mid-point of the suprasternal notch were subtracted from the carotid-to-femoral artery distance. Peripheral PWV measures were obtained from the left femoral artery to the ipsilateral superior dorsalis pedis artery. The distance between each PWV location was obtained with a tape measure and recorded to the nearest millimeter.

Data were collected in real time by aligning the Doppler waveforms and the ECG tracings on a computer screen (MP100, BioPac Systems, Santa Barbara, CA, USA). All readings were stored and analyzed offline at a later time. PWV was measured from the foot-to-foot flow wave velocity, whereas the foot of the sound wave was identified as the point of systolic upstroke. A minimum of 12 pulse contours were recorded and analyzed as the distances between points and the time delay between proximal and distal foot waveforms were calculated as distance (D) divided by the change in time ($m\,s^{-1}$). One blinded technician analyzed all the data. The intra-class correlation coefficient for PWV calculated using both central and peripheral sites on two separate days was 0.97.

Hemodynamic monitoring

Each subject underwent BP measurements in compliance with the World Health Organization guidelines with the recruiting physician before the start of the study. BP was determined as the average of three measurements using a manual mercury sphygmomanometer and a stethoscope (Korotkoff phases I and IV for SBP and DBP, respectively).

For the acquisition of BP during supine rest, one-minute averages were recorded for 10 min with the subjects in a supine position and attached to a beat-to-beat BP monitoring system via finger plethysmography (Finometer, FMS, Amsterdam ZO, The Netherlands). The Finometer estimates brachial BP using an integrated brachial BP cuff and reconstructs brachial BP waveforms from finger arterial waveforms by applying an inverse transfer function, a waveform filter, a level correction and a level calibration.[17]

Blood analyses

Blood samples for 17beta-estradiol (E2), PRA and plasma Ang II measurements were collected in ice-cold vacutainer tubes containing EDTA (ethylenediaminetetraacetic acid) to inhibit metalloproteases at the following concentrations: 5 mM E2; and 25 mM EDTA for PRA and Ang II. PRA was measured by radioimmunoassay (GammaCoat Plasma Renin Activity, DiaSorin Inc., Stillwater, MN, USA) and was defined as the rate of Ang I generated from endogenous substrate. A previous method was optimized for Ang II recovery and stability.[18] Plasma (0.5 ml) was recovered by centrifugation (3000 $g \times 20$ min, 4 °C), diluted to 2 ml in phosphate buffer (50 mM sodium phosphate, 1 mM EDTA, 0.25 mM thimerosal, 0.25% BSA) containing peptidase inhibitors at above final concentrations and extracted on a phenyl solid-phase cartridge (Agilent Technologies, Santa Clara, CA, USA). After washing columns twice with water (1 ml), Ang II was eluted with methanol (750 µl). The dried eluant was resuspended in 500 µl enzyme immunoassay buffer (Cayman Chemical Co., Ann Arbor, MI, USA) and Ang II was measured by enzyme immunoassay. Assay linearity was between 125 µl and 1 ml of plasma. Blood samples used for plasma ALDO measurements were collected using the anticoagulant heparin (14.3 USP units ml^{-1}) and aldosterone was measured by radioimmunoassay (Coat-A-Count Aldosterone, Diagnostic Products, Los Angeles, CA, USA). The limits of detection of these assays (in $pg\,ml^{-1}$) are: E2, 3; PRA, 25; Ang II, 2 and ALDO, 25. The complete blood count panel was sent to Upstate Medical University Laboratories (Syracuse, NY, USA) for analyses using coded subject identification.

Statistical analysis

Student's *t*-test was employed to determine if statistical differences existed in the descriptive characteristics. A 1×3 (BL, Pre and Post) ANOVA with repeated measures using SPSS v18 (Chicago, IL, USA) and if a significant interaction was found, a Bonferroni correction was applied to all dependent variables. To delineate the effects of BP on PWV, we divided MAP into PWV and tested if the adjusted values were significantly different. Significance was set at an alpha <0.05 and all data were reported as mean ± s.e.m.

RESULTS

The descriptive characteristics of the subjects were reported in Table 1. All female subjects had E_2 levels below 7.5 $pg\,ml^{-1}$, thus confirming their postmenopausal status ($<10\,pg\,ml^{-1}$). All male

Table 1. Descriptive characteristics at baseline, pre and post 4 weeks of aerobic training

Variable	*BL*	*Pre*	*Post*
Age (years)	54.1 ± 2.8	54.1 ± 2.8	54.1 ± 2.8
Height (m)	1.62 ± 0.08	1.62 ± 0.08	1.62 ± 0.08
Weight (kg)	73 ± 3.0	73.0 ± 5.0	72.5 ± 3.0
Fat %	43 ± 4.96	42.85 ± 4.89	42.96 ± 4.95
Fat weight %	45 ± 6.13	44.3 ± 4.73	45.5 ± 6
Lean weight %	58.89 ± 5.3	58.85 ± 5.37	59.7 ± 5
BMI ($kg\,m^{-2}$)	33.5 ± 1.4	33.5 ± 1.4	33.5 ± 1.4

Abbreviations: BL, baseline measure; BMI, body mass index. Values are mean ± s.e.m.

and female subjects were in the pre- to-stage-1 hypertensive range (Table 2) as defined by the Joint National Committee for the Prevention, Detection, Evaluation and Treatment of High Blood Pressure (JNC 7) criterion.[12]

Four weeks of aerobic exercise training did not affect body mass index (Table 1) and blood panel analysis of sodium, potassium, chloride, carbon dioxide, aniongap, calcium, blood urea nitrogen, creatinine and plasma glucose pre-to-post training and between BL and Pre conditions (Table 3). No significant differences were found in any of the Pre and BL values, therefore, we used the Pre values when making comparisons with Post values. Results reported a significant reduction in ALDO ($P = 0.04$) between Pre and Post conditions (Figure 1). No significant differences in PRA and Ang II were found between all conditions examined (Table 2). Four weeks of aerobic exercise resulted in a mean decrease of 4 mm Hg (SBP and DBP) ($P = 0.042$) (Table 2) and a significant reduction in central PWV ($P = 0.038$, Figure 2) between Pre and Post training. We employed a Pearson's correlation to determine the level of correlation between ALDO and PWV and BP changes and found that ALDO was moderately correlated to PWV ($r = 0.45$) yet weakly correlated to SBP decreases ($r = 0.33$). No significant differences in PRA were detected between men and the age-matched postmenopausal women at BL, Pre or Post; however, when comparing Pre with Post, the PRA levels approached significance ($P = 0.08$). Further, a Pearson's correlation coefficient between significant groups was employed to determine the level of correlation.

DISCUSSION

The present study has shown that 4 weeks of aerobic training decreased BP, central PWV and plasma ALDO without significant decreases in body composition in unmedicated middle-aged men and age-matched postmenopausal women. It is well known that aerobic exercise confers a protective effect on cardiovascular diseases in adults with concomitant reductions in BP and arterial stiffness. In accordance with our study, previous researchers report that 1–4 weeks of moderate-intensity aerobic training reduced BP[10,19] and markers of arterial stiffness, without associated changes in aerobic fitness or body composition.[20] Similar to our findings, Kohno *et al.*[21] found that individuals with elevated baseline PRA and hypertension showed the greatest decreases in BP after 3 weeks of moderate-intensity training. Conversely, there is a lack of studies addressing RAAS reductions in an unmedicated, hypertensive group following aerobic training. Jones and colleagues (2007) reported that 6 months of aerobic training lowered plasma ALDO without changes in BP and loss of intra-abdominal fat in pre-hypertensive and hypertensive subjects; however, unlike our study, some of their subject population was treated pharmacologically.[22] Interestingly, the significance of the reduction in cardiovascular variables such as BP and PRA may depend highly on the timing of data collection. Zhang *et al.*[23] has shown that 1–4 weeks of low-to-moderate-intensity leg ergometry led to significant decreases in BP, PRA and Ang II, whereas data collection of the same variables from weeks 4–10 showed a plateau in these data. Taken together, this suggests that the timing of data collection for exercise studies in individuals with elevated BP is crucial to capture BP and hormone changes that are unrelated to humoral factors and plasma volume changes.

It is well known that increases in plasma ALDO concentrations lead to increases in PWV transit times within an adult hypertensive population. This is likely a result of localized mineralocorticoid receptor activity within the endothelium[24,25] as a significant association exists between serum ALDO and central PWV, but not peripheral PWV measurements.[26] This result may indicate that ALDO is more influential in central pressure regulation than in peripheral vascular beds which is congruent with current data showing ALDO receptors more highly positioned in the large conduit than in the muscular peripheral arteries decreasing the influence of ALDO on the peripheral arteries.[27]

The substantial reduction in ALDO following aerobic exercise training is the most significant finding of our study. Plasma ALDO concentrations, along with PRA and plasma Ang II have been reported to be higher in obese subjects when compared with a leaner population.[28] Overexpression of PRA and plasma ALDO is common in obese subjects and elevated levels increase blood volume and vascular resistance.[5] At present, it has been shown that elevated levels of ALDO are positively correlated with increased aortic and arterial stiffness, resulting in deleterious effects on large arteries.[29] Matsui *et al.*[29] has recently shown that pharmacological treatment with thiazide diuretics significantly correlated with decreases in plasma ALDO and aortic PWV in hypertensive individuals. Exercise is a common therapy prescribed to decrease obesity status and BP. Few studies exist regarding the correlation of plasma ALDO on BP following exercise in an obese, hypertensive population. Our study suggests reduced plasma ALDO contributes independently to the reduction in SBP and DBP and central PWV.

This is the first study to match such findings in an obese, unmedicated hypertensive cohort. Using a wait-listed control period increases the power of the study and also enables the researchers to evaluate reliability of assays and measurements while the subjects act as their own controls. It is well known that weight loss is the primary reason for the reduction in PRA and plasma ALDO, resulting in lowered extracellular volume, sympathetic nervous system activity and improved insulin resistance.[30,31] Exercise training has been shown to reduce both systolic and diastolic BP in normotensive and hypertensive adults via changes in body composition and weight loss.[32,33] One study compared racial differences in plasma ALDO and BP showing six months of aerobic exercise moderately lowered plasma ALDO but not BP in a medicated and unmedicated cohort.[22] However, the investigators reported a mean baseline BP below the hypertensive level, which may have been resultant of the medications the individuals were prescribed and body composition reductions. It is important to note that our duration and intensity of exercise training did not result in significant changes in body composition or plasma volume. Our study is in congruence with Whelton *et al.*[34] as they showed BP reductions following aerobic training in both normotensive and hypertensive subjects with minimal weight loss. Combined with our results, these findings suggest that the ALDO decrease following moderate-intensity aerobic exercise training was independent of decreases in body weight. Previous studies have indicated factors associated with weight loss as mediation for improved vascular smooth muscle tone and artery compliance.[35] However, our blood analyses revealed no significant alterations in PRA, Ang II or any other blood markers analyzed,

Table 2. Cardiovascular and metabolic parameters at baseline, pre and post 4 weeks of aerobic training

Variable	*BL*	*Pre*	*Post*
VO_{2peak} (ml kg^{-1} min^{-1})	32.4 ± 2.4	—	33.7 ± 3.1
PRA (ng ml^{-1} h^{-1})	0.979 ± 0.16	0.845 ± 0.11	0.592 ± 0.10
Ang II (pg ml^{-1})	934 ± 190	933 ± 177	931 ± 107
SBP (mm Hg)	140 ± 10.3	140 ± 12	136 ± 10.4[a]
DBP (mm Hg)	90 ± 5.4	89 ± 4.2	85 ± 6.3[a]
HR (b.p.m.)	66 ± 3	67.8 ± 4.3	64.8 ± 2.9

Abbreviations: Ang II, angiotensin II; BL, baseline measure; DBP, diastolic blood pressure; HR, heart rate; PRA, plasma renin activity; SBP, systolic blood pressure; VO_{2peak}, peak oxygen consumption. Values are reported as mean ± s.e.m. [a]Denotes significance at $P \leqslant 0.05$.

Table 3. Blood panel at baseline, pre and post 4 weeks of aerobic training

Variables	*BL*	*Pre*	*Post*
Sodium ($mg\,dl^{-1}$)	141.4 ± 1.26	141.5 ± 1.85	142.1 ± 1.37
Potassium ($mg\,dl^{-1}$)	4.0 ± 0.13	4.1 ± 0.12	4.2 ± 0.15
Chloride ($mg\,dl^{-1}$)	103.7 ± 1.42	104.6. ± 1.65	104.9 ± 1.97
Carbon dioxide ($mg\,dl^{-1}$)	26.7 ± 2.36	26.9 ± 1.85	26.2 ± 2.39
Aniongap ($mg\,dl^{-1}$)	10.4 ± 1.96	10.0 ± 1.05	10.7 ± 1.95
Serum calcium ($mg\,dl^{-1}$)	9.1 ± 0.29	9.1 ± 0.32	9.2 ± 0.36
Blood urea nitrogen ($mg\,dl^{-1}$)	16.2 ± 2.15	16.3 ± 2.63	15.8 ± 2.44
Creatinine ($mg\,dl^{-1}$)	0.86 ± 0.12	0.88 ± 0.09	0.86 ± 0.12
Free calcium ($mg\,dl^{-1}$)	293.2 ± 2.82	294.3 ± 1.64	295.0 ± 3.33
Glucose ($mg\,dl^{-1}$)	82.4 ± 7.71	83.3 ± 6.57	85.3 ± 10.27
Hemoglobin ($mg\,dl^{-1}$)	15.1 ± 0.55	16.4 ± 1.2	14.2 ± 1.4
Hematocrit ($mg\,dl^{-1}$)	43 ± 0.8	44 ± 1.1	42 ± 2.2

Abbreviation: BL, baseline measure. Values are expressed as mean ± s.e.

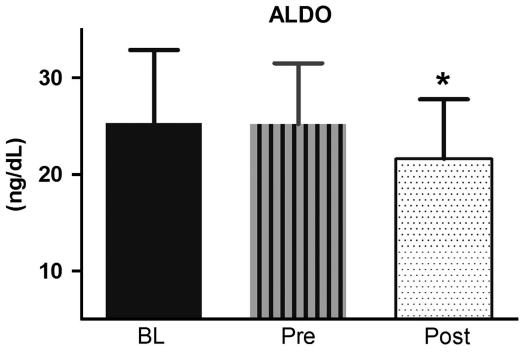

Figure 1. Plasma aldosterone (ALDO) levels at baseline (BL), before (Pre) and after (Post) 4 weeks of aerobic training. Data are presented as mean ± s.e., $*P \leqslant 0.05$ from Pre to Post.

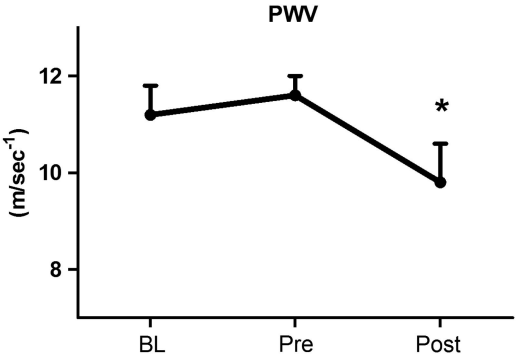

Figure 2. Central pulse wave velocity (PWV) at baseline (BL), before (Pre) and after (Post) 4 weeks of aerobic training. Data are presented as mean ± s.e., $*P \leqslant 0.05$ from Pre to Post.

reinforcing the assumption that decreased ALDO concentrations was the main factor responsible for BP and PWV reductions.

Our study also revealed a trend in PRA (37% decrease, $P = 0.064$) inhibition after moderate-intensity exercise training whereas Martinelli *et al.*[36] found no changes in PRA despite their observed reductions in BP and PWV. They theorized insufficient weight loss as the likely reason for not observing any changes. Dubbert *et al.* (1994); however, reported that 10 weeks of aerobic exercise lowered plasma catecholamines, PRA and arterial BP in untreated hypertensive subjects. Their data analyses on SBP changes were significantly correlated with changes in PRA, but not with body fat, suggesting that the decrement in PRA was the primary cause for lowered SBP.[37] Our changes in PRA could be a contributor to the decreases we realized in PWV and BP; however, PRA decreased slightly after the baseline measurement and PWV increased in the study group, suggesting a threshold may need to be exceeded before PRA contributes to decreases in PWV or our sample size was too small to distinguish between the changes following BLs. In a *post hoc* calculation of sample size, an increase of only two subjects would have given us enough power to determine the differences in baseline and PRA differences.

Pharmacologic inhibition of angiotensin-converting enzyme has been shown to benefit the endothelium in heart failure patients by increasing NO bioactivity or by attenuating reactive oxygen species, which prevents the interaction of superoxide anions which serve to increase endothelial NO synthase expression and activity.[38] Aerobic exercise training has been shown to augment the bioavailability of NO through increases in endothelial NO synthase, leading to beneficial decreases in arterial resistance and BP in normotensives and hypertensive individuals.[39] However, it has been suggested that adipocyte markers, specifically elevated plasma leptin hormone, contribute to increased Ang II levels in an obese population. Their study associates weight loss with appreciable decreases in Ang II levels, which would confer benefits of reduced arterial resistance, in turn lowering BP. As aforementioned, our subjects did not change their body composition as a result of the exercise training, which may explain the insignificant change in Ang II levels. The variation in our Ang II response may have been due to the genetic variability in the modulation of Ang II due to the inter-individual influence of genetic factors on plasma angiotensin and angiotensin-converting enzyme activity.[40] This suggests that moderate-intensity aerobic exercise and the associated depressor response linked to Ang II, obesity and hypertension may be realized only in those individuals that are determined to be 'responders'.

CONCLUSIONS

Independent of genetic factors, the relationship between obesity and hypertension is not fully understood and current research offers only speculation. Until now, the availability of literature with analysis of ALDO concentrations in human hypertensive models was not in existence. The present study has shown that aldosterone is attenuated in unmedicated, pre-to-stage-1 hypertensive individuals independently of body weight following 4 weeks of moderate-intensity aerobic training, resulting as the main factor responsible for SBP, DBP and PWV reductions. Therefore, this exercise practice may offer an alternative therapy for obesity-complicated hypertension.

What is known about topic

- Hypertension is six times more frequent in obese than in lean individuals.
- Compared with lean controls, obese individuals report elevated plasma renin activity as well as angiotensin II and plasma aldosterone, causing further increases in blood pressure.

What this study adds

- Four weeks of aerobic training decreased BP, central PWV and plasma ALDO without significant decreases in body composition in unmedicated middle-aged men and age-matched postmenopausal women.
- The timing of data collection for exercise studies in individuals with elevated BP is crucial to capture BP and hormone changes that are unrelated to humoral factors and plasma volume changes.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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