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Effect of Resistance Training on Biomarkers of Vascular Function and Oxidative Stress in Young African American and Caucasian men

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Abstract

African Americans (AA) have an earlier onset of hypertension and a different vascular profile than their Caucasian (Cau) peers. Research suggests that biological mediators of vascular inflammation are different among these groups in hypertensive populations. Resistance training (RT) is an important exercise modality which improves the vascular profile of young AA men. We examined the role of RT on biomarkers of vascular function and oxidative stress in BMI-matched AA and Cau men. Six weeks of RT elicited significant changes in circulating MMP-9 and 8-Isoprostane (8-IsoP) in young AA men (n= 14 AA; n= 18 Cau; 18–35 yo). MMP-9 was lower and decreased in AA (pre: p=0.02; post: p<0.001) and a time x group interaction forMMP-9 ($F_{1,30}$=4.81; p=0.036)and 8-IsoP($F_{1,24}$=7.09; p=0.014) was detected. 8-IsoP decreased in AA (p=0.026) but did not change in Cau (p=0.309). Notably, the increase in strength (1-RM) was correlated with the decrease in MMP-9 (r= −0.398; p=0.022). Further, these adaptations were independent of any improvement in cardiorespiratory fitness. We demonstrate that RT effectively reduces matrix remodeling proteins and oxidative stress in young AA men. Increasing strength may be beneficial for improving vascular health and offsetting novel CV risk factors of hypertension in young AA men.

Keywords

Resistance training; MMP-9; 8-isoprostane; African American

INTRODUCTION

African Americans (AA) have increased incidence of cardiovascular (CV) risk factors including obesity, hypertension, diabetes, and decreased leisure-time physical activity[1].

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Furthermore, the prevalence of more than one risk factor for cardiovascular disease (CVD) in AA is remarkably high[2]. African Americans also have a less desirable vascular profile with greater reductions in endothelial function, increased arterial stiffness [3] and earlier onset of hypertension compared to Caucasians(Cau)[4]. This suggests that early intervention may be beneficial in limiting vascular dysfunction in young AA.

Pharmacological interventions producing improvements in vascular function are associated with improved clinical outcomes[5, 6], although this has not been specifically addressed in AA. Physical activity is a common lifestyle intervention associated with reductions in CVD risk factors, CVD morbidity and mortality [7]. Both aerobic and resistance exercise training (RT)elicit reductions in central and peripheral blood pressure coupled with improvements in endothelial function[8, 9, 10]. We have previously reported that RT augments arterial function, reduces central blood pressure [11] and C-Reactive Protein (CRP) [12] in young AA men, thus improving their vascular function and decreasing their risk for hypertension. There is also an inverse relationship between muscle strength and aortic stiffness [13] which suggests that muscle strength improvements may also bevital for vascular health. However, we have also observed an increase in brachial arterial stiffening in young AA men, but not in young Cau men, after RT that does not coincide with the improvement in endothelial function [11]. This suggests there may be differential responses to RT in young AA and Cau men. Biomarkers associated with increased risk of arterial dysfunction also differ between AA and Cau individuals and may be related to pathogenesis of hypertension[14].

Inflammation is linked to the pathogenesis of CV disease. Tumor necrosis factor-alpha (TNF-α), a pro-inflammatory cytokine, is responsible for the stimulation of immune and vascular cell types. Intracellular adhesion molecule-1 (ICAM-1/CD54) and vascular cell adhesion molecule-1 (VCAM-1/CD106) both have roles in cell signaling that recruit and facilitate immune cell migration across the vascular endothelium. Their expression in vascular endothelial cells and in immune cells (e.g., lymphocytes and monocytes) is stimulated by TNF-α. Although necessary in vascular repair, this is also a mechanism that directly links inflammation to vascular dysfunction. Matrix metalloproteinase's (MMP's) are enzymes that are involved in arterial tissue remodeling and are highly active in the chronic disease processes[15–17] including vascular remodeling, hypertension, arterial dysfunction[18, 19] and pathogenesis of atherosclerosis[20].

Interestingly, inflammatory cytokines are affected by RT. CRP is decreased in AA but not Cau[12] following RT, and RT also increased anti-inflammatory IL-10 cytokine production after an acute bout of exercise[21]. However, the influence of RT on inflammatory markers known to affect vascular function is not well understood. RT also modulates circulating levels of MMP's [22] and baseline MMPs differ between AA and Cau[14]. Additionally, oxidative stress is markedly elevated in hypertensive AA as evidenced by elevated levels of 8-Isoprostane (8-IsoP)[23]. While aerobic exercise may decrease circulating 8-Isoprostane levels [24], no data are available on the effect of RT.

Recent data suggest that there are in vitro differences in inflammation and oxidative stress between AA and Cau. Markers of both inflammation and oxidative stress were higher in AA human vein umbilical cells (HUVAC), in support of in vivo data[25, 26]. Furthermore, AA

HUVACs were more responsive to shear stress than Cau HUVACs, despite baseline differences. Thus, since RT affects vascular function and can produce vascular remodeling, presumable through shear stress induced improvements in nitric oxide metabolism and potentially inflammation mediated processes[27, 28], it is possible that RT may affect other biomarkers of vascular function, remodeling, inflammation and oxidative stress differentially in AA and Cau.

Therefore, the purpose of this study was to examine the effect of RT on markers of inflammation (TNF-α, IL-10), endothelial function and vascular remodeling (sICAM, sVCAM, MMP-2 and MMP-9), and oxidative stress(8-isoprostane)in young AA and Cau men. We hypothesized that RT would lower circulating levels of TNF-α, increase IL-10, and reduce ICAM, VCAM, MMP-2, MMP-9, and 8-isoprostane in both groups of young men but that these changes would be greater in young AA men.

METHODS

Thirty-two subjects (14AA and 18 Cau young men; aged 18–35 years old) were recruited. Subjects were screened for and did not have diabetes, hypercholesterolemia, and renal disease, did not smoke and did not use medications of any kind (including anti-inflammatory medications). Subjects were sedentary or recreationally active and none were previously endurance or resistance exercise trained. Race was self-reported as AA (i.e., both parents were of African descent) or non-Hispanic white (i.e. both parents were of white European descent). All subjects were recruited from the local university student population and gave written informed consent. The subjects included in this study were a subset of subjects from a larger study we have previously published [12] and participants were matched for body mass index (BMI). The methodological design has been previously described [12] and is briefly described below. This study was approved by the Institutional Review Board of the University of Illinois at Urbana-Champaign.

We obtained our measurements before and following a 6 week resistance training intervention (the larger project also included a 4 week detraining period). For this study, only baseline and post resistance training intervention blood pressures, anthropometric data, muscle strength and cardiovascular fitness data and blood samples were available. At baseline, subjects completed a body composition assessment and a blood test after a12 h overnight fast. During a second visit, conducted 24–48 h after the blood draw, subjects performed maximal aerobic exercise testing and 1-repetition maximum(1-RM) bench press test, in that order. Aerobic exercise testing and strength testing was conducted in a postprandial state (~3 h). Subjects were also asked to refrain from caffeine and alcohol ingestion for 24 h prior to testing. During the resistance training period, subjects were instructed to refrain from any structured aerobic/endurance exercise.

1-RM bench press

One repetition maximum for the bench press was defined as the maximum amount of weight lifted for a single repetition with proper form through a full range of motion. The relative 1-RM value was taken as a measure of upper body strength and used to document a training

effect. The 1-RM value was then divided by lean body mass to express muscular strength relative to lean mass.

Resistance training

All training sessions were supervised by personal trainers/ strength and conditioning specialists and consisted of 3 exercise sessions per week (~60 min per session). The resistance training protocol used was a two-way body part split (legs, back and biceps on one day; chest, shoulders and triceps on a separate day) as previously described [11].

Hemodynamics

Brachial blood pressure (BP) was measured in duplicate in the supine position using an automated oscillometric cuff(HEM-907 XL; Omron, Shimane, Japan). If these values deviated by more than 5mmHg, a third measurement was conducted. The average of the two closest values was recorded and used for analysis.

Peak Oxygen consumption *(VO_2 peak)*

VO_2 peak was assessed using a graded cycle ergometry protocol. Participants began with a warm-up consisting of pedaling at 60–100 rpm at 30 Watts (W) for 30 seconds then starting the test by pedaling at 50W for 2min. Workload was then increased by 30W every 2 minutes until test termination. Heart rate (HR) was measured with a Polar Heart Rate Monitor (Polar Electro, Woodbury, NY). Expired air was analyzed with a Quark b2 breath-by-breath metabolic system (Cosmed, Rome, Italy). The test was concluded when subjects achieved three of the following five criteria: (1) a final rating of perceived exertion score of ≥17 on the Borg scale (scale 6–20), (2) a respiratory exchange ratio >1.1, (3) no change in HR with a change in workload, (4) a “plateau” (increase of no >150ml) in oxygen uptake with an increase in workload,(5) volitional fatigue, defined as an inability to maintain a pedal rate above 60rpm.

Anthropometrics

Body composition was determined using whole body air displacement plethysmography. Height and weight was measured using a stadiometer (to the nearest 0.5 cm) and a beam balance platform scale. BMI was determined by weight (kg) divided by height (m) squared.

Blood Analysis

Baseline and post resistance exercise training concentrations of MMP-2, MMP-9, TNF-α, IL-10, soluble ICAM, soluble VCAM (R & D Systems, Minneapolis, MN) and 8-Isoprostane (Caymen Chemical Company, Ann Arbor, MI) were measured by their respective ELISA’s. 8-Isoprostane was measured from plasma and all other variables were measured from serum samples. Serum and plasma were stored at −80 °C until analysis. IL-10 has detection limits of 3.9–500 pg/ml requiring a 2.5-fold dilution of serum samples (intra-assay coefficient of variation 5.0%). TNF-α has detection limits of 15.6–1000 pg/ml requiring a 3-fold dilution of serum with an intra-assay CV of 5.2%. MMP-2 and MMP-9 have detection limits of 0.78–50 ng/ml requiring a 10-fold dilution of serum (intra-assay CV 2.0%) and 0.312–10 ng/ml requiring a 100-fold dilution of serum (intra-assay CV 5.5%),

respectively. Soluble ICAM has detection limits of 1.56–50 ng/ml requiring a 20-fold dilution of serum (intra-assay CV 5.0%). Soluble VCAM has detection limits of 6.25–200 ng/ml requiring also requiring a 20-fold dilution of serum (intra-assay CV 2.3%). 8-Isoprostane has detection limits of 0.8–500 pg/ml and was measured from plasma(intra-assay CV 11.7%).

Statistical Analysis

Analysis of variance (ANOVA) with repeated measures was used to assess between and within group differences as a result of the intervention. MMP-2 and MMP-9werenot normally distributed thus these values were log transformed to achieve normality. Data for these two variables are presented in raw form (ng/ml) for clinical interpretation. When a significant main or interaction effect was detected at a significance level of $p < 0.05$, Student *t*-tests were used for post hoc comparisons. Analysis of covariance (ANCOVA) was used to assess baseline differences in variables corrected for age. The changes in weight, BMI, body fat percentage and SBP were correlated with the changes in outcome blood variables (MMP-9 and 8-IsoP) to assess their relationship. All results are presented as mean±SEM. Data analyses were performed using Statistical Package for the Social Sciences (SPSS, v 18, SPSS, Inc., Chicago, IL).

RESULTS

Analyses were completed on thirty-two subjects (14 AA and 18 Cau young men) that were matched for BMI. Caucasian subjects were slightly older (p=0.004) and were not as strong as AA (p=0.003), but there were no other baseline differences between groups.(Table 1)

There was a significant time x group interaction for body weight ($F_{1,30}$=22.23; p<0.001) as body weight increased in Cau (p=0.006) and decreased in AA (p=0.004)after RT. There was a time x group interaction ($F_{1,30}$=11.06; p=0.002) for body fat percentage as it decreased in AA (p=0.001)but did not change in Cau men (p=0.490). There was also a significant group x time interaction for BMI ($F_{1,30}$=24.15; p<0.001) as BMI decreased in AA (p=0.003) but increased in Cau (p=0.006) after RT. VO2peak was unaltered and there was no significant interaction for VO2peak. RT significantly improved strength in both groups (Cau: p<0.001; AA: p<0.001) groups and AA subjects were still significantly stronger following RT (p=0.001), but there was no significant interaction effect. Interestingly, a time x group interaction ($F_{1,30}$=6.02; p=0.020) was detected for systolic blood pressure (SBP) showing a decrease in Cau (p=0.036) but not AA after training. Although the groups were not significantly different, there was a time (training) main effect for diastolic blood pressure (DBP)(p=0.033). (Table 1)

At baseline, Cau and AA subjects did not differ in circulating levels of sVCAM, TNF-α, IL-10, MMP-2, or 8-IsoP. However, baseline sICAM was higher in Cau (p=0.010). There was a significant time x group interaction forMMP-9 ($F_{1,30}$=4.81; p=0.036) as MMP-9 was lower in AA before training (p=0.018) and then decreased significantly after (p<0.001) the intervention only in AA men. A one-way between-subjects ANCOVA was used to examine the effect of the statistically significant age difference between groups on resting pre sICAM and MMP-9differences. Age was not significantly related to the baseline differences in

sICAM ($F_{1,26}$=4.04; p=0.055) or MMP-9 ($F_{1,26}$=0.20; p=0.655)in this analysis. RT did not significantly modify IL-10, TNF-α, sICAM-1, sVCAM-1, or MMP-2 in either group. A time x group interaction showed ($F_{1,24}$=7.09; p=0.014) that RT significantly reduced 8-IsoP in AA(p=0.026) but not Cau (p=0.309) (AA: n=13; Cau: n=16).(Table 2)

To assess if the significant changes in body weight, body mass index, body fat percentage, and fitness (VO_2peak) were related to the changes observed in measured MMP-9 and 8-IsoP, we correlated the change in the above measures (Δ:post-pre)with the change in MMP-9 and change in 8-IsoP separately. These variables were not significantly correlated in AA. There was a significant inverse relationship between fitness (ΔVO_2peak) and ΔMMP-9 (*r=−0.562*: p=0.01) in the Cau group only. Interestingly, the change in strength (1-RM) was also correlated with the ΔMMP-9 (r= −0.398; p=0.022).

DISCUSSION

Our main finding was that RT significantly lowered circulating MMP-9 and 8-isoprostane in AA but not Cau men and this is the first study to report such beneficial effects of RT in young AA men. Although RT is a popular mode of exercise, its effects on biomarkers of vascular health has not been previously investigated. Therefore, we investigated the effects of 6 weeks of RT on circulating biomarkers of vascular inflammation, matrix remodeling proteins, and oxidative stress in young AA and Cau men to explicate potential biological mechanism(s) involved in the alteration of arterial function as a result of RT. The beneficial effects of RT in AA is important as AA have a threefold greater risk of developing CVD and twofold greater mortality and premature CVD death than Cau[29, 30]. Coutinho et al. [14] also recently reported that MMP-2, as well as CRP, were significantly associated with greater pulse pressure in AA but not Cau. We have previously reported that RT can significantly reduce CRP in AA as well as lower central pressures[11, 12]. Thus, based on our current and previous results, coupled with findings from others, it appears that RT can be an effective lifestyle intervention to lower CV risk in AA.

Increased concentrations of circulating MMP-9 are associated with increased risk of disease and reducing the levels of MMP-9 has been shown to reduce the atherosclerotic burden in mice[31]. In addition, recent data suggest that MMP-9 is associated with arterial stiffness and inflammation in hypertensive patients as well as in healthy controls[16]. This may be a function of degradation of elastin by MMP-9. Further, studies on circulating MMP's[14, 32] suggest that some biological mediators of vascular health, and their role in arterial function, may be different in certain disease states and amongst racial/ethnic groups. Unexpectantly, Cau in our study had significantly higher MMP-9 than AA participants while RT was effective in significantly reducing MMP-9 only in AA. Interestingly though, Vlachopoulos et al. suggest there is an inverse relationship between MMP-9 and arterial stiffness in young healthy individuals[33]. They suggested that increased levels of MMP-9 in healthy populations without underlying low grade inflammation may reflect a different physiologic process and implication for the association between MMP-9 and cardiovascular risk. Nevertheless, inmost individuals, both healthy cohorts and in those with diseases related to cardiovascular health, MMP's have been directly associated with arterial dysfunction. Thus, the decrease in MMP-9 following RT in AA maybe interpreted as a beneficial change likely related to

reduced risk in this population. Our findings on the decrease inMMP-9 in the AA group, are consistent with previous data showing improvements in central pressure and microvascular function in young AA men following RT [11, 12, 34] and add support to the body of literature showing beneficial effects of RT. Notably, the reduction in MMP-9 observed in AA was not associated with changes in body weight, body fat percentage, body mass index, or cardiovascular fitness (VO_2peak).

Oxidative stress has been reported to be greater in AA[35] and is also associated with hypertension. For example, Zhou et al. reported that 8-iso Prostaglandin $F_{2\alpha}$ (8-IsoP) is significantly increased in hypertensive AA compared to their normotensive cohorts[23]. Measurement of 8-IsoPin plasma has been validated as a marker of oxidative stress [24] but it also has vasoactive characteristics[36]. It is well known that acute bouts of intense exercise are associated with increases in 8-IsoP [37]. Conversely, Galassetti et al. [38] has shown that 7 days of intense aerobic exercise training lowered F_2-isoprostanes suggesting that exercise training reduces oxidative stress. However, no studies have examined the role of RT on 8-IsoP. This study is the first to show that RT is effective in reducing 8-IsoP in young healthy AA, but not Cau men, thus ameliorating another risk factor of hypertension in young AA men. The decline in circulating 8-IsoP observed in AA was also not related to post intervention changes in body weight, body fat, BMI or VO_2peak.

We examined the role of RT in altering vascular inflammatory markers TNF-α, sICAM-1, and sVCAM-1 because studies have reported correlations between inflammation, adhesion molecules and hypertension [39]. Although not specific to RT, higher perceived physical fitness levels are positively associated with lower systemic inflammatory markers (lower TNF-α, F_2-prostaglandin; higher IL-10) [40]. RT significantly decreases TNF-α in older individuals and those suffering from chronic inflammatory diseases but studies on RT have not conclusively characterized a uniform response of TNF-α in young healthy individuals[41]. In our study, RT did not significantly reduce TNF-α(Table 2). Further, we did not detect any changes in soluble ICAM-1 or VCAM-1 in either group. Since TNF-α modulates the activity of adhesion cell molecules that facilitate adhesion of inflammatory cells in the vasculature, TNF-α, ICAM-1, and VCAM-1 are likely not involved in the vascular improvements observed in our study. The previously reported increase in arterial stiffness in AA may be related to the reduced MMP-9 reported in our young healthy AA group, as the study by Vlachopoulos et al[33] suggests, but we did not measure arterial stiffness in our current study, thus this cannot be addressed. Further, RT differentially decreased SBP in Cau but not AA men. It would be beneficial to perform this study in young pre-hypertensive individuals to more conclusively uncover the effect of racial background and RT on functional vascular responses.

Overall, these data provide evidence for RT being an effective mode of exercise in modulating matrix remodeling proteins and oxidative stress, thus strengthening the role of RT in the potential prevention of the early onset of hypertension in young AA men. Moreover, this study provides insight into a potential mechanism of the cardioprotective effect of RT that is independent of increases in cardiopulmonary function but may be dependent on methods of improving strength in AA as these data suggest. Future studies should be directed toward providing more evidence to explore racial differences in

mediators of vascular health, as well as on the effects of RT (both positive and negative) in pre-hypertensive and hypertensive individuals, especially in AA where studies are lacking and significant health disparities exist.

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Table 1

Subject Characteristics

Variable	African American			Caucasian American		
Age, yr	21.9 ± 0.38			25.2 ± 0.78*		0.004
Height, cm	180.7 ± 1.92			179.3 ± 1.15		
	PRE	POST	***P***	PRE	POST	***P***
Weight (kg)	95.8 ± 4.79	94.5 ± 4.69*	***0.004***	89.2 ± 3.78	90.1 ± 3.71*	***0.006***
Body mass Index (kg/m2)	29.3 ± 1.29	28.9 ± 1.24*	***0.003***	28.1 ± 1.09	28.4 ± 1.07*	***0.006***
Body Fat (%)	23.7 ± 2.12	22.1 ± 2.12*	***0.001***	25.6 ± 1.9	25.9 ± 1.84	
Peak Oxygen Uptake (ml-kg-min)	29.3 ± 1.86	28.2 ± 1.6		28.4 ± 1.09	29.0 ± 1.14	
1-Rep Max (1 RM)	105.9 ± 6.48*	113.1 ± 6.27*	***0.001***	81.5.1 ± 4.14	87.9 ± 3.94*	***0.001***
1-Rep Max (1 RM)/kg	1.13 ± 0.08	1.23 ± 0.08		0.93 ± 0.05	0.99 ± 0.04	
Systolic Blood Pressure (SBP)	132.1 ± 2.46	133.7 ± 2.05		131.5 ± 2.25	127.2 ± 2.36*	***0.036***
Diastolic Blood Pressure (DBP)	76.5 ± 2.1	74.9 ± 1.97		76.8 ± 1.72	73.0 ± 1.74	

Values are Average ± SEM; *p* values are post hoc analysis from significant ANOVA results

*Significant time x group interaction

Table 2

Group means of measured blood variables

Variable	African American			Caucasian American	
	n= 14			***n=18***	
	PRE	**POST**	***P***	**PRE**	**POST**
MMP2 (ng/ml)	235.1 ± 9.6	230.8 ± 9.3		236.3 ± 15.7	268.8 ± 18.8
MMP9(ng/ml)	276.3 ± 31.6*	171.1 ± 18.3*	***0.001***	408.3 ± 41.8	393.0± 41.4
IL-10 (pg/ml)	33.2 ± 2.4	28.3 ± 2.3		29.0 ± 1.4	29.1 ± 1.3
	n= 13			***n=16***	
TNF-α (pg/ml)	105.9 ± 17.4	96.4 ±19.7		100.9 ± 20.3	107.9 ± 11.2
sICAM (ng/ml)	178.1 ± 16.7	184.7 ± 18.9		237.3 ± 12.9*	208.5 ± 13.2
sVCAM (ng/ml)	529.2 ± 36.6	559.3 ± 37.3		596.3 ± 24.5	577.5 ± 40.6
8-IsoP (pg/ml)	349.8 ± 28.9	299.8 ± 34.9*	***0.026***	279.3 ± 42.1	309.6 ± 36.4

Values are Average ± SEM; *p* values are post hoc analysis from significant ANOVA results

*Significant time x group interaction