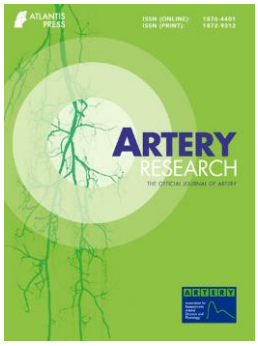

Artery Research

ISSN (Online): 1876-4401 ISSN (Print): 1872-9312
Journal Home Page: https://www.atlantis-press.com/journals/artres

PO-16: BLOOD PRESSURE VARIABILITY AND BARORECEPTOR SENSITIVITY IN NORMOTENSIVE OBESE IN RESPONSE TO AEROBIC EXERCISE

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To cite this article: Georgios Grigoriadis, Kanokwan Bunsawat, Bo Fernhall, Tracy Baynard (2016) PO-16: BLOOD PRESSURE VARIABILITY AND BARORECEPTOR SENSITIVITY IN NORMOTENSIVE OBESE IN RESPONSE TO AEROBIC EXERCISE, Artery Research 16:C, 93–94, DOI: https://doi.org/10.1016/j.artres.2016.08.023

To link to this article: https://doi.org/10.1016/j.artres.2016.08.023

Published online: 7 December 2019

PO-13
SEX DIFFERENCES IN VASCULAR FUNCTION FOLLOWING ANTIOXIDANT SUPPLEMENTATION

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Objectives: Sex differences in cardiovascular disease risk and progression are well established. Estrogen loss following menopause leads to vascular dysfunction, potentially due to elevations in oxidative stress and subsequent decrements in nitric oxide. It is possible a reduction in oxidative stress utilizing an antioxidant supplement could improve vascular function in older females.
Methods: Forty-seven young (27 ± 0.5 years, 23 M and 24 F) and 46 older (59 ± 0.7 years, 23 M and 23 F) subjects underwent measures of vascular function following both placebo and antioxidant supplementation in a randomized, double-blind, crossover study.
Results: Young males displayed higher central and peripheral pressures, stiffer arteries and decreased macrovascular endothelial function when compared to young females, and this was reversed with aging, with females developing stiffer arteries, higher pressures and endothelial dysfunction to match the older male group. Young males were more responsive to AOX and showed improvements in macrovascular function following AOX. In the older group, although both males and females improved FMD% with AOX, females were more responsive and improved significantly more.
Conclusions: These results demonstrate the potential role of oxidative stress in estrogen loss and subsequent arterial dysfunction, possibly due to reductions in nitric oxide bioavailability.

PO-14
PULSE WAVE VELOCITY IS INCREASED WITH EXPERIMENTAL SLEEP RESTRICTION IN HEALTHY HUMANS

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Objectives: Increased carotid-femoral pulse wave velocity is indicative of vascular stiffening of the central arterial tree. Aortic stiffness is a key risk factor for the development of hypertension and cardiovascular disease. Following acute (24-hour) sleep deprivation, healthy adults exhibit an increase in carotid-femoral pulse wave velocity; however, acute sleep deprivation poorly represents sleep patterns observed in everyday life. With this information in mind, we hypothesized a prolonged (9 day) exposure to restricted sleep (4 hours of sleep per night) would result in increases in carotid-femoral pulse wave velocity in healthy humans.
Methods: Seven (3M, 5F) young (23±1 yrs), healthy adults underwent a 4-day period of acclimation followed by 9 days of experimental sleep restriction (4 hours of sleep per night — from 12:30 AM to 4:30 AM). High-fidelity radial arterial pressure waveforms and carotid-femoral pulse wave velocity were assessed using applanation tonometry (SphygmoCor, AtCor Medical). Subjects were studied on Day 2 (Acclimation) and Day 13 (Restriction).
Results: Sleep restriction resulted in an increase in carotid-femoral pulse wave velocity (5.6±0.2 to 5.9±0.2 m/s, $p=0.05$) and a decrease in round trip time (179±8 to 150±11 ms, $p<0.01$) when compared to the acclimation period. A reduction in the Buckberg subendocardial viability ratio (SEVR, indicative of myocardial oxygen supply/demand, $p=0.02$) and an increase in the Pressure-Time Integral Systole (PTI, an index of cardiac load, $p=0.01$) were also observed following sleep restriction.
Conclusions: Prolonged (9-day) exposure to experimental sleep restriction in young healthy humans results in unfavorable changes in central macrovascular function, including an increase in central arterial stiffness and cardiac load. These results may have important implications for the increase in cardiovascular disease risk in individuals experiencing limited sleep.

Table: Pressure and vascular response following placebo and AOX supplementation in Young and Older Adults.

	Young (n=47)				Older (n=46)			
	Males (n=23)		Females (n=24)		Males (n=23)		Females (n=23)	
	Placebo	AOX	Placebo	AOX	Placebo	AOX	Placebo	AOX
bSBP (mmHg) #	126±2*	125±2*	106±2	105±2	128±4	127±3	127±4	125±3
bDBP (mmHg) #	71±1*	69±1*$	64±1	65±1	76±2	75±2	77±2	77±2
aSBP (mmHg) #	106 ± 1	105 ± 2	93 ± 1	91 ± 2	119 ± 4	118 ± 3	120 ± 4	119 ± 3
cPWV (m/s) #	6.4±0.2	6.1±0.6	5.9±0.2	6.4±0.6	8.1±0.5	8.5±0.4	8.4±0.5	7.4±0.5
Carotid Arterial Compliance (mm^2/kPa) #	1.1±0.8*	1.1±0.6*	1.5±0.8	1.4±0.6	0.95± 0.59*	0.91± 0.51	0.77± 0.59	0.80± 0.51

Significance $p<0.05$, Mean ± SEM. AOX, antioxidant supplementation; bSBP, brachial systolic blood pressure; bDBP, brachial diastolic blood pressure; aSBP, aortic systolic blood pressure; cPWV, central pulse wave velocity.
*significant sex difference
$ significantly different from placebo
# significant age group differences

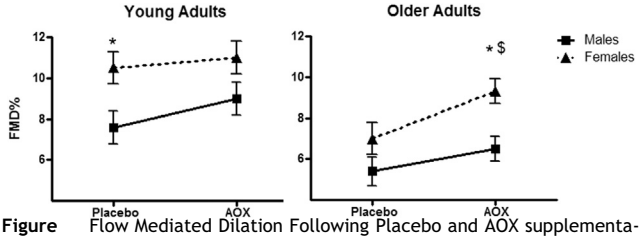

Figure Flow Mediated Dilation Following Placebo and AOX supplementation in Young and Older Adults. There were significant differences between age groups at both placebo and AOX condition. *denotes a significant difference between sexes, $ denotes a significant difference from placebo.

PO-16
BLOOD PRESSURE VARIABILITY AND BARORECEPTOR SENSITIVITY IN NORMOTENSIVE OBESE IN RESPONSE TO AEROBIC EXERCISE

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Background: Autonomic dysfunction, with increased sympathetic activity at rest has been reported in obese individuals. Indices of blood pressure variability

(BPV) and baroreceptor sensitivity (BRS) can provide insight into aspects of autonomic function, particularly following an aerobic exercise bout.
Purpose: To examine BPV and BRS in normotensive obese individuals in response to aerobic exercise.
Methods: Normal-weight (n=8; 25 yr; 23.0 kg/m^2) and obese individuals (n=9; 27 yrs; 32.2 kg/m^2) performed a 60-min leg cycling exercise at 60% of VO_{2peak}. Beat-by-beat blood pressure was recorded at baseline, immediately post-exercise and 30 min into passive recovery using finger plethysmography. R-R intervals were obtained at 1,000 Hz with a digital acquisition system. Power spectral analysis was conducted using WinCPRS software for estimates of BPV (very low and low frequency (VLF, LF), and systolic and diastolic deviation (SDev, DDev)). BRS was estimated using the sequence technique. Natural log-transformed was performed on LF BPV (LnLF) to account for non-normal distribution.
Results: HR increased from baseline similarly in both groups ($p<0.05$). The control group decreased SBP at immediately post-exercise compared to baseline measurements ($p<0.05$), but not the obese group. A main effect of time and group ($p<0.05$) existed for BRS. No group differences were found on DBP, LF, LnLF, VLF, SDev and DDev.
Conclusion: The results showed no difference in the BPV indices between the obese and control groups. The different response in SBP suggests that control group may have better BRS; however, this is not supported by the lower values in BRS. A limitation of this study may be the small number of participants.

	Control			Obese		
	Baseline	Immediate	30min	Baseline	Immediate	30min
HR (bpm)*	66 ± 11	88 ± 12	80 ± 12	60 ± 6	79 ± 11	74 ± 11
SBP (mmHg)*#&	116 ± 11	104 ± 8^{abc}	115 ± 9	122 ± 5	119 ± 6	122 ± 4
DBP (mmHg)	64 ± 10	64 ± 4	68 ± 7	69 ± 5	71 ± 5	72 ± 5
Raw LF (mmHg2)	9.00 ± 5.37	15.91 ± 15.03	15.24 ± 12.29	5.23 ± 4.65	6.89 ± 4.93	9.64 ± 8.13
LnLF (mmHg2)	2.07 ± 0.53	2.40 ± 0.89	2.41 ± 0.89	1.37 ± 0.75	1.73 ± 0.66	1.89 ± 0.98
VLF (mmHg2)	20.83 ± 14.39	29.63 ± 19.77	22.69 ± 13.67	11.91 ± 7.96	18.68 ± 14.70	15.29 ±10.87
BRS (ms/mmHg)*#	15.95 ± 7.92	5.20 ± 3.48	8.05 ± 4.52	19.38 ± 6.79	12.74 ± 8.70	14.49 ± 7.79
SDev (mmHg)	5.61 ± 1.75	7.14 ± 2.71	6.36 ± 2.25	4.77 ± 1.48	5.83 ± 2.42	5.50 ± 2.02
DDev (mmHg)	3.70 ± 1.08	4.06 ± 1.56	3.84 ±.31	3.67 ± 1.33	4.18 ± 1.67	3.90 ± 1.30

All data are mean ± SEM. *Time effect, # Group effect, & time x group effect, a Within-Subjects effect vs Baseline, b Within-Subjects effect vs 30min, c Between-Subject effect vs obese group.

PO-17
ROLE OF NITRIC OXIDE IN β_2-ADRENERGIC MEDIATED VASODILATION IN POSTMENOPAUSAL WOMEN

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Objectives: Postmenopausal (PM) women have a blunted β_2-adrenergic receptor-mediated responsiveness when compared to young premenopausal women in part due to a reduction in the relative contribution of nitric oxide (NO) to β_2-adrenergic mediated vasodilation. Hence, we tested the contribution of NO to β_2-adrenergic receptor-mediated vasodilation during terbutaline infusion.
Hypothesis: We hypothesized that the contribution of NO to β_2-adrenergic mediated vasodilation would be attenuated in PM women as compared to young women.
Methods: Venous occlusion plethysmography was used to measure forearm blood flow (FBF) in 7 healthy young premenopausal women and 9 healthy PM women (mean age = 27 ± 1 and 60 ± 1 years, respectively). FBF was measured at baseline and during terbutaline infusion at 0.1, 0.5, 1.0, 2.0 μg/100ml tissue/min before (with saline co-infusion) and during NO synthase inhibition with L-NMMA. Forearm vascular conductance was calculated from FBF and mean arterial pressure.
Results: In young women, there was a significant L-NMMA effect on forearm vascular conductance during terbutaline infusion with and without L-NMMA (1.7 ± 0.14, 3.56 ± 0.41, 7.13 ± 1.11, 7.87 ± 0.74, 10.54 ± 1.81 versus 2.08 ± 0.28, 5.54 ± 0.50, 9.32 ± 1.10, 10.77 ± 1.49, 13.29 ± 1.94 ml/100ml tissue/min/mmHg, respectively). However, there was no effect of L-NMMA in PM women during terbutaline infusion with and without L-NMMA (1.34 ± 0.26, 2.37 ± 0.32, 5.21 ± 0.99, 4.71 ± 0.99, 6.43 ± 1.37 versus 1.62 ± 0.31, 3.11 ± 0.55, 5.41 ± 1.12, 6.26 ± 1.38, 7.26 ± 1.44 ml/100ml tissue/min/mmHg, respectively).
Conclusions: These data suggest that NO contributes to β_2-adrenergic mediated vasodilation in young premenopausal women. In contrast, no contribution of NO to β_2 mediated vasodilation was observed in PM women. These data suggest a lower β_2-adrenergic responsiveness in PM women may be due to a reduced contribution of NO.

PO-19
ASSOCIATIONS OF WALKING WITH SARCOPENIC OBESITY AND CARDIOVASCULAR DISEASE RISK FACTORS IN OLDER ADULTS

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Objectives: To investigate the associations of walking (steps/day) with sarcopenic obesity (SO) and cardiovascular disease (CVD) risk factors in older adults.
Methods: This cross-sectional study included 297 older adults aged ≥65 years (mean age 72, ranged 65-95). Walking was assessed using an accelerometer (Omron HJ-321) and categorized into thirds (tertile) based on the average daily steps. SO was defined based on physical function (gait speed), muscle strength (handgrip strength), and muscle mass (appendicular lean mass [ALM] index) according to the Foundation for the National Institutes of Health Sarcopenia Project diagnostic criteria, and % body fat (obesity as ≥25% in men and ≥30% in women) using Dual Energy X-Ray absorptiometry.
Results: Each 10,000 steps/day increase was associated with improved SO variables and CVD risk factors, specifically with 0.008 faster gait speed (m/s), 0.006 higher muscle mass index (ALM/BMI), 0.59 lower % body fat (%), and 0.68 lower fasting glucose (mg/dl)(all $p<0.05$) in the linear regression after adjusting for age, sex, smoking status, and alcohol intake. Compared to low walking group, odds ratios (ORs)(95% confidence intervals [95% CIs]) in moderate and high walking groups were 0.18 (0.02-1.54) and 0.22 (0.03-2.01) for slow walking, 0.42 (0.14-1.30) and 0.34 (0.09-1.29) for weak handgrip strength, 0.45 (0.23-0.87) and 0.44 (0.22-0.88) for low muscle mass, 0.58 (0.13-2.57) and 0.46 (0.11-2.06) for high % body fat, and 0.62 (0.17-2.28) and 0.21 (0.02-1.78) for SO, respectively, in the multivariable logistic regressions. Compared to individuals without SO, ORs (95% CIs) in individuals with SO were 2.04 (0.58-7.18) for hypertension, 1.27 (0.39-4.22) for hypercholesterolemia, and 1.87 (0.37-9.45) for type 2 diabetes in the multivariable logistic regression. However, these associations appeared to be weaker after further adjustment for walking (steps/day).
Conclusion: This study suggests that walking in older adults is associated with lower risks of SO and CVD risk factors.

PO-20
A HYDROGEN SULFIDE PRODRUG AUGMENTS ANGIOGENESIS IN A SWINE MODEL OF CRITICAL LIMB ISCHEMIA VIA A NITRIC OXIDE DEPENDENT MECHANISM

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Introduction: Despite advances in revascularization, treatments for critical limb ischemia (CLI) have been largely unsuccessful. Hydrogen sulfide (H_2S)