TNF-α induced VCAM-1 (p=0.931). Prolonged LSS and OSS elicited a significant elevation in the MFI of TNF-α induced fractalkine (p=0.017). Lastly, acute LSS following prolonged OSS had no effect on the % of gated or MFI of cells expressing VCAM-1 and fractalkine.

CONCLUSIONS: Prolonged OSS may increase markers of vascular inflammation. However, an acute period of LSS, utilized as a model of physical activity, does not appear to alter the OSS-induced inflammatory state.

2806 Board #326 June 2 9:30 AM - 11:00 AM
Physical Activity Attenuates NLRP3 Inflammasome-associated Vascular Dysfunction in Obese Mice Heart
Jonghae Lee, Yang Lee, Kwangchan Kim, Eunkyung Park, Junyong Hong, Yoonjung Park. University of Houston, Houston, TX. Texas A&M Health Science Center College of Medicine, College Station, TX.
Email: jlee88@central.uh.edu
(No relationships reported)

Activation of the NLRP3 inflammasome mediates the release of pro-inflammatory cytokine IL-1β and thereby plays a pivotal role in the inflammatory response in vascular pathology. An active lifestyle has beneficial effects on inflammation-associated vascular dysfunction in obesity. However, it remains unclear how physical activity regulates NLRP3 inflammasome-mediated vascular dysfunction in obesity.

PURPOSE: To determine the protective effect of physical activity on NLRP3 inflammasome-associated vascular dysfunction in mice heart, and the potential underlying mechanisms.

METHODS: C57BL/6J male mice were randomly divided into four groups: (1) control low-fat diet (LF-SED), (2) LF diet given free access to a voluntary running wheel (LF-RUN), (3) high-fat diet (HF-SED; 45% of calories from fat), and (4) HF-RUN. Western blotting and immunofluorescence staining determined NLRP3 inflammasome-related signaling pathways and nitric oxide (NO) bioavailability-related pathways in the heart.

RESULTS: Western blotting showed increased protein expression of NLRP3 in HF-SED (31%) compared to LF-SED, but it was reduced by voluntary wheel running. Immunofluorescence staining illustrated significantly higher expression of caspase-1 and IL-1β in coronary endothelial cells and arterioles in HF-SED than LF-SED, LF-RUN, and HF-RUN. Compared to LF-SED, decreased expression of endothelial nitric oxide synthase (eNOS; 32%) and increased NOX2 (NADPH oxidase 2; 51%) expression in HF-SED were normalized to the level of LF-SED by voluntary wheel running.

CONCLUSION: Our findings suggest that voluntary running would oppose high fat diet-induced vascular dysfunction in mice heart by suppressing NLRP3 inflammasome activation and possibly improving NO bioavailability via increased expression of eNOS and reduced oxidative stress.

2807 Board #327 June 2 9:30 AM - 11:00 AM
Vigorous-Intensity Physical Activity May Improve Central Aortic Pressure Response to Glucose Loading in Overweight/Obese Men
Toru Yoshikawa, Hiroshi Kumagai, Kanae Myoenzono, Tomoko Kaneko, Takehiko Tsujimoto, Kiyoo Tanaka, FACSM, Seiji Maeda. University of Tsukuba, Tsukuba, Japan. (Sponsor: Kiyoo Tanaka, FACSM)
Email: yoshikawai@live.jp
(No relationships reported)

Central aortic systolic blood pressure (cSBP) decreases after a meal or glucose challenge, but the response is impaired by obesity-related disorders. We have previously reported that the blunted cSBP response to oral glucose loading in overweight/obese men was normalized after a 12-week aerobic exercise training program. However, the most effective intensity of physical activity to improve the cSBP response to glucose loading is unclear.

PURPOSE: To evaluate the effect of intensity of regular physical activity on cSBP response to oral glucose loading in overweight/obese men.

METHODS: Thirteen overweight/obese (body mass index, BMI ≥25 kg/m²) men (age, 50 ± 8 years; BMI, 27.1 ± 2.5 kg/m²; mean ± SD) completed a 12-week aerobic exercise training program, involving both supervised and unsupervised walking/jogging. Physical activity time (PAT) was measured using a tri-axial accelerometer for 2-3 weeks prior to the exercise program and during 12 weeks of the exercise program, and was classified into low- (<1.5-2.9 METs), moderate- (3.0-5.9 METs), or vigorous-intensity (>6.0 METs). Before and after the program, cSBP was noninvasively estimated using application tonometry of the radial artery with a validated general transfer function at fasting and 120 min after 75 g oral glucose loading.

RESULTS: Moderate- and vigorous-intensity PAT significantly increased during the exercise program, whereas low-intensity PAT did not change (low-intensity: 244 ± 55 to 254 ± 63 min/day, P = 0.39; moderate-intensity: 48 ± 16 to 70 ± 19 min/day, P < 0.01; vigorous-intensity: 0 ± 1 to 12 ± 10 min/day, P < 0.01). Glucose loading significantly decreased cSBP only after the exercise program (109 ± 13 to 104 ± 14 mmHg, P < 0.05), but not before (111 ± 15 to 110 ± 17 mmHg, P = 0.56). Changes in cSBP response to glucose loading after the exercise program were significantly correlated with changes in vigorous-intensity PAT, but not with changes in low- and moderate-intensity PAT (low-intensity: r = 0.03, P = 0.93; moderate-intensity: r = 0.16, P = 0.61; vigorous-intensity: r = -0.78, P < 0.01).

CONCLUSIONS: Vigorous-intensity physical activity during the 12 weeks of aerobic exercise training program may have improved the cSBP response to oral glucose loading in overweight/obese men.

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2808 Board #328 June 2 9:30 AM - 11:00 AM
The Impact of Repetitive Long-duration Water Immersion on Vascular Function
Erin E. Simmons, Elizabeth R. Bergeron, John P. Florian. Navy Experimental Diving Unit, Panama City, FL.
Email: ees06@tamu.edu
(No relationships reported)

While physiological responses to water immersion (WI) are well-studied, the vascular responses after WI are less understood.

PURPOSE: The objective of this study was to quantify the changes in endothelial function and vascular stiffness following repeated six-hour WIs with surface-supplied air.

METHODS: Sixteen healthy subjects (15 male) performed six-hour resting thermonautic water immersions (WI) at 1.35 atmospheres absolute (ATA) for four consecutive days, with follow-up on the fifth day. Measurements included endothelial function and arterial stiffness (peripheral arterial tonometry), beat-to-beat blood pressure (photoplethysmography), heart rate (HR), and plasma volume (PV) calculated from changes in hemoglobin and hematocrit.

RESULTS: The reactive hyperemia index (RHI), a marker of endothelial function, increased with repeated immersions (p<0.008) as did inRHI (p=0.025). By WI 3, RHI and inRHI increased 16% and 17%, respectively, compared to WI 1 values, but no significant differences were detected between WI 4 and WI 1 for either measure. Absolute arterial stiffness (augmentation index, AI) increased by an average of 33% (p<0.001) and AI normalized for HR (AIat75s) by 11% (p=0.12) following each WI. PV decreased significantly by 13.2% (p<0.001) following WI and remained 6.8% lower at follow-up compared to pre-WI. Systolic blood pressure significantly decreased by an average of 2.5% following each WI (p<0.01). HR decreased 4.3% after each WI (p<0.001) but increased overall by 6.6% over the course of repeated WI (p<0.001). Total peripheral resistance increased by an average of 13.1% following WI (p=0.003).

CONCLUSION: Four consecutive days of six-hour WIs while breathing air at 1.35 ATA results in a transient increase in arterial stiffness following each WI as well as an increase in endothelial function on the third day. Additionally, in the context of acute exposure to WI, blood pressure and endothelial function diverge from their usual direct associations with arterial stiffness.

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