minutes after completing the submaximal test. Predicted VO₂ was calculated from a submaximal treadmill test. An SVR exercise response coefficient (ERC) was derived and subjects were then categorized as normal response (NR) to exercise or abnormal response (AR) to exercise. Comparisons between groups were determined using ANOVA.

RESULTS: The AR group indicated a significant blunted or even elevated SVR coefficient (1.092 ± 0.09 p<0.05) compared to the NR group (0.853 ± 0.08). The LAEI was significantly different between the groups (16.25 ± 2.68 mL/min·mmHg x 10 vs 13.58 ± 2.54 mL/min·mmHg x 10). There were also significant differences (p<0.05) in the post exercise responses of ejection time (284.0 ± 107.3 ms vs 114.4 ± 107.0 mmHg), and systolic BP (120.5 ± 10.7 mmHg vs 114.4 ± 10.7 mmHg), and diastolic BP (70.65 ± 7.38 mmHg vs 66.24 ± 7.3 mmHg).

CONCLUSIONS: The lack of response may be due to the poor fitness level. Although fitness was not found to be significant between the groups the fitness levels were poor. Poor fitness due to inactivity decreases vasodilator function in the smooth muscles associated with the smaller arterioles at the tissue level. The results of the lack of vasodilation may be the cause for the reduced ejection time due to the increased afterload.

C-25  Free Communication/Poster - Cardiovascular Responses, Training and Testing
Thursday, May 29, 2014, 7:30 AM - 12:30 PM
Room: WB1

1300  Board #40  May 29, 9:00 AM - 10:30 AM
Activation Of Akt/mTOR Pathway In Response To Acute Exercise In Rats Of Different Trained Status
Yunhong Wang, Hao Wu, Shoufu Yan, Yuan Yao, Lei Liang, Chengcheng Fu, Kun Lang, Dongdong Li, Meiyue Hao. Capital University Physical Education and Sports, Beijing, China.
(No relationships reported)

IGF-1 coupled to the phosphatidylinositol 3-kinase (PI3K)/Akt signaling pathway has been proved to be critical in exercise-induced cardiac hypertrophy. However, the activation pattern of Akt/mTOR signaling pathway in the process of development of exercise-induced cardiac hypertrophy is not well documented.

PURPOSE: To characterize the activation pattern of Akt/mTOR signal pathway induced by exercise training so as to understanding the role of Akt/mTOR signaling pathway in the exercise induced cardiac adaptation.

METHODS: Male 8-week-old Sprague-Dawley rats were randomly divided into either exercise training or control groups. The exercise training groups were subjected to a single bout of exercise, 4-week or 10-week exercise training respectively according to our previously exercise training protocols, and then rats were decapitated at the designated time points (0 hour, 3 hour, 24 hour) after last exercise. The heart was weighed, and then tissues of left ventricle were frozen for determination of the proteins by Western blotting analysis.

RESULTS: The results showed that a single moderate endurance exercise could not induce significant increase in the activation of Akt/mTOR signaling pathway (p>0.05), however, after 4-week training, the basal activation level of Akt/mTOR signaling pathway significantly elevated (p-Akt/Akt ratio increased by 2.46 fold: 0.3891 ± 0.0127 vs 0.9566 ± 0.0394, p<0.05; p-TOR/mTOR ratio increased by 1.20 fold: 0.4076 ± 0.0253 vs 0.4937 ± 0.0148, p<0.05 ), but the heart weight to body weight ratio did not significantly increase (2.89 ± 0.23 vs 3.22 ± 0.19, p>0.05); in contrast, with the exercise training continuously increased in intensity and duration, the basal activation level of Akt/mTOR signaling pathway reversed to the sedentary level, but the heart weight to body weight ratio was significantly increased (2.61 ± 0.16 vs 3.02 ± 0.26, p<0.05).

CONCLUSIONS: The activation of Akt/mTOR signaling pathway changed dependently on the training status, and the sustained activation of Akt/mTOR signaling pathway was observed only at the development stage of cardiac hypertrophy.

1301  Board #41  May 29, 9:00 AM - 10:30 AM
Individual Variability In Cardiac Biomarker Release After High Intensity Rowing In Elite And Amateur Athletes
Luis E. Carranza-Garcia¹, Alejandro Legaz-Arrese², Fernando A. Ochoa-Ahmed¹, César Vinacua-Salvo³, Patricia Muñoz-Iriarte², Oswaldo Ceballos-Gurrola¹, ¹Autonomous University of Nuevo Leon, San Nicolás de los Garza, Mexico. ²University of Zaragoza, Zaragoza, Spain. ³University of Zaragoza, Zaragoza, Spain.
(No relationships reported)

The release of cardiac biomarkers with exercise has been previously suggested, however the influence of training level in continuous strenuous efforts in rowing is currently unknown.

PURPOSE: This study had two specific objectives: 1) to examine the individual release of cardiac troponin I (cTnI) and N-terminal pro-brain natriuretic peptide (NT-proBNP) to high intensity rowing exercise, and 2) to establish the influence of training level on cTnI and NT-proBNP release.

METHODS: We examined cTnI and NT-proBNP in 18 elite and 14 amateur rowers before and 5 min, 1, 3, 6, 12, and 24 h after a 30 min maximal rowing test.

RESULTS: Peak post-exercise cTnI (pre: 0.015 ± 0.030, peak: 0.058 ± 0.091 μg/L, p = 0.000) and NT-proBNP (pre: 15 ± 11, peak post: 31 ± 19 ng/L, p = 0.000) were significantly elevated although considerable heterogeneity in peak cTnI concentration and time-course of release were noted. Peak cTnI exceeded the upper reference limit in 8 elite and 3 amateur rowers. Less heterogeneity was noted in NT-proBNP data with no rower exceeding the URL. There were no significant differences in peak post-exercise cTnI or NT-proBNP between the elite and amateur rowers.

CONCLUSION: In summary, marked individuality in cTnI response was noted to a short but high intensity exercise test. Training status did not seem to mediate cardiac biomarker response to such exercise stimulus.

<table>
<thead>
<tr>
<th>cTnI (μg L^-1) and NT-proBNP (ng L^-1) before and after 30 min of high-intensity rowing exercise</th>
<th>Pre-exercise</th>
<th>5 min post</th>
<th>1 h post</th>
<th>3 h post</th>
<th>6 h post</th>
<th>12 h post</th>
<th>24 h post</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>cTnI</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Elite rowers</td>
<td>0.019 ± 0.038</td>
<td>0.022 ± 0.048</td>
<td>0.030 ± 0.051</td>
<td>0.069 ± 0.055*</td>
<td>0.079 ± 0.116*</td>
<td>0.045 ± 0.073</td>
<td>0.023 ± 0.046</td>
<td>0.000</td>
</tr>
<tr>
<td>Amateur rowers</td>
<td>0.010 ± 0.017</td>
<td>0.008 ± 0.013</td>
<td>0.011 ± 0.018</td>
<td>0.025 ± 0.028*</td>
<td>0.028 ± 0.029*</td>
<td>0.020 ± 0.019</td>
<td>0.007 ± 0.007</td>
<td>0.000</td>
</tr>
<tr>
<td><strong>NT-proBNP</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Elite rowers</td>
<td>14 ± 11</td>
<td>25 ± 18*</td>
<td>21 ± 16*</td>
<td>19 ± 14</td>
<td>18 ± 12</td>
<td>18 ± 11</td>
<td>19 ± 14</td>
<td>0.000</td>
</tr>
<tr>
<td>Amateur rowers</td>
<td>17 ± 12</td>
<td>25 ± 19</td>
<td>25 ± 17</td>
<td>26 ± 18</td>
<td>26 ± 18*</td>
<td>28 ± 18*</td>
<td>27 ± 17*</td>
<td>0.006</td>
</tr>
</tbody>
</table>

* Significantly different from the baseline
Release of Cardiac Troponin in Basketball Players is Independent of Training Level and Maturation Status


1Autonomous University of Nuevo León, San Nicolás de los Garza, Mexico. 2University of Zaragoza, Zaragoza, Spain.

(No relationships reported)

The impact of intermittent exercise on the release of cardiac troponins is controversial, and the influence of several factors, such as training level and maturation status, has not been analyzed.

METHODS: Thirty-six basketball players (12 professional, 12 amateur, and 12 junior) participated in a simulated basketball match with serial assessment of cTnI from blood samples collected at rest, immediately post- and at 1, 3, 6, 12, and 24 h post-exercise.

RESULTS: The basketball match increased cTnI levels (pre: 0.008 ± 0.006; peak post: 0.041 ± 0.057 μg L⁻¹; p = 0.000) (Table 2). There were no differences on the increase of cTnI levels between professionals (0.040 ± 0.066 μg L⁻¹), amateurs (0.012 ± 0.009 μg L⁻¹) and juniors (0.049 ± 0.071 μg L⁻¹) (p = 0.179). 3 professionals and 5 juniors (23%) exceeded the URL of cTnI.

CONCLUSION: The results suggest that basketball match promote a limited release of cTnI not related with training level neither maturation status.

Table 1. Characteristics of the study population by training status

<table>
<thead>
<tr>
<th></th>
<th>Age (years)</th>
<th>Weight (kg)</th>
<th>Height (cm)</th>
<th>VO2peak (ml.kg⁻¹.min⁻¹)</th>
<th>Basketball training history (years)</th>
<th>Basketball training frequency (sessions/week)</th>
<th>Basketball training volume (hours/week)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Professional</td>
<td>27.3 ± 4.1</td>
<td>98.3 ± 12.9</td>
<td>199 ± 7</td>
<td>58 ± 3</td>
<td>17 ± 5</td>
<td>6 ± 0</td>
<td>16 ± 0</td>
</tr>
<tr>
<td>Amateur</td>
<td>29.6 ± 2.9*</td>
<td>83.8 ± 12.9*</td>
<td>184 ± 6*</td>
<td>56 ± 7</td>
<td>13 ± 3*</td>
<td>4.1 ± 1.2*</td>
<td>7.5 ± 3.6*</td>
</tr>
<tr>
<td>Junior</td>
<td>16.6 ± 0.9* &amp;</td>
<td>82.8 ± 10.3*</td>
<td>192 ± 8* &amp;</td>
<td>58 ± 3</td>
<td>8 ± 4* &amp;</td>
<td>4 ± 0*</td>
<td>8.0 ± 0*</td>
</tr>
</tbody>
</table>

* Significant differences between professional and amateur or junior basketball players.
& Significant differences between amateur and junior basketball players.

Table 2. cTnI (μg L⁻¹) before and after the simulated basketball match

<table>
<thead>
<tr>
<th></th>
<th>Pre-exercise</th>
<th>5 min post</th>
<th>30 min post</th>
<th>1 h post</th>
<th>3 h post</th>
<th>6 h post</th>
<th>12 h post</th>
<th>24 h post</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.008 ± 0.006</td>
<td>0.011 ± 0.011</td>
<td>0.018 ± 0.024</td>
<td>0.029 ± 0.043*</td>
<td>0.032 ± 0.044*</td>
<td>0.026 ± 0.038*</td>
<td>0.016 ± 0.026</td>
<td>0.000</td>
<td></td>
</tr>
</tbody>
</table>

* Significant differences compared with the baseline value.
CONCLUSIONS: This study demonstrates that acute and chronic high-intensity aerobic exercise increases the Akt/mTOR signaling pathway and the chronic exercise induces physiological growth of cardiac muscle in aged rats.

The Effects of Salamba Sirsasana on Heart Rate Variability in Advanced Yoga Practitioners

Steven D. Munassi, Silvie Grote. Loyola Marymount University, Los Angeles, CA.

(No relationships reported)

The effects of yoga practice have shown increased vagal modulation but there is limited information on which specific parts of yoga practice enhance autonomic control of the heart.

PURPOSE: The goal of this study was to evaluate effect of acute inversion, salamba sirasana (supported headstand), on heart rate variability (HRV).

METHODS: Advanced yoga practitioners (n = 16; 14 female and 2 male, ages 44.4 ± 13.6 years) completed 3 trials consisting of 5-minute phases of shavasana (resting pose), salamba sirasana, and shavasana. Breathing was paced during both trials of shavasana at 5 breaths per minute, but not during salamba sirasana. The trials were divided between two days within a one-week period. Each trial was carried out in the most comfortable environment for the individual (i.e., yoga studio, office, house etc.) or if unable to utilize those areas, in the Human Performance Lab at Loyola Marymount University. Heart Rate Variability measurements were assessed using the Polar RS800CX G5.

RESULTS: Paired t-test revealed statistically significant increase in HRV in the very low frequency component (VLF) of trial 1 (p = 0.022) and trial 2 (p = 0.021). Statistical significance was also found in the high frequency (HF) component of post phases of 2 trials performed on the same day (p = 0.043). No trends were seen in low-frequency (LF) components.

CONCLUSIONS: Salamba sirasana results in significant increases in VLF component of HRV in advanced yoga practitioners as seen in pre and post assessments of trial 1 and trial 2. Significant increase in HF component in post phases of 2 trials repeated in one day suggests a clinically desirable increase in vagal tone with repeated inversions. Future studies should examine the duration of the effect of inversions on HRV, the result of utilizing different inversions, and the outcome of these trials on non-practitioners.

The Akt/FoxO3a/Atrogin-1 Signaling Pathways Underlying Cardiac Regression after Detraining in a Mouse Heart.

Wanseok Lee1, Yoonjung Park2, Leslie A. Leinwand3, Eunhee Chung1. 1Texas Tech University, Lubbock, TX. 2University of Houston, Houston, TX. 3University of Colorado, Boulder, Boulder, CO. (Sponsor: Jacalyn McComb, FACSM)

(No relationships reported)

Exercise-induced cardiac hypertrophy reverses after cessation of exercise training. The Akt/FoxO3a/Atrogin-1 pathways have been implicated as important players in skeletal and cardiac muscle atrophy. However, little is known about the role of these pathways in cardiac muscle regression after exercise cessation.

PURPOSE: The purpose of this study was to determine the activities of Akt, FoxO, and to measure the levels of the muscle-specific E3 ligase, Atrogin-1 in cardiac muscle which has undergone regression of mass after cessation of exercise training.

METHODS: Three to four month old male C57Bl/6 mice were randomly divided into six groups: sedentary controls, 21 days of exercise, and 4 different time points of detraining after 21 days of voluntary wheel running (ie. 3 days, 5 days, 14 days, and 21 days). Body weight was not significantly different among groups. Exercise training significantly increased the heart weight/body weight ratio compared to sedentary controls (13% increase in HW/BW), but this growth was regressed after 21 days of detraining. We found that phosphorylation of Akt normalized to total Akt was significantly increased in the exercise group, and also in the 3-, 5-, and 14 days of detraining groups, but was significantly decreased in the 21 days of detraining group compared to the sedentary control group. However, the phospho-FoxO3a to total FoxO3a ratio was not different among groups. Interestingly, the level of Atrogin-1 was significantly decreased in 14-, and 21 days of detraining groups. Our results suggest that cardiac regression following exercise cessation is not mediated by up-regulation of Atrogin-1.

CONCLUSION: Taken together, this study demonstrates that cardiac regression occurs after 21 days of exercise cessation and it may be partially mediated by inactivation of Akt.

Racial Differences In Pressure Wave Separation Responses Following An 8 Week Endurance Training Program

Alexander Rosenberg, Tommy Wee, Sushant M. Ranadive, Abbi Lane, Rebecca Kappus, Huimin Yan, Bo Fernhall, FACSM. university of illinois chicago, chicago, IL.

(No relationships reported)

African Americans (AA) have an increased prevalence of hypertension and greater levels of cardiovascular disease morbidity and mortality when compared with Caucasians (CA). Untrained AA do not experience exercise-induced hypotension. It is unknown if exercise training can alter this response.

PURPOSE: To compare the training induced differences in the BP response following a peak bout of aerobic exercise (VO2peak), after 8 weeks of endurance training between AA and CA.

METHODS: Young, healthy sedentary (~25yr) AA (n=21, BMI=29.1 kg/m2) and CA (n=24, BMI=24.7 kg/m2) subjects participated in 40-60 min, 3 times per week for 8 weeks, of aerobic exercise training at 70-80% of peak HR. Individuals had brachial (bSBP, bPP), and aortic (aSBP, aPP) measurements obtained in the supine position at rest, 15 min following a peak bout of aerobic exercise. Applanation tonometry was used to obtain aSBP pressure waveforms. Wave separation analyses were used to produce forward and reflected waves pressures height (FPH, RPH). Repeated measure Analysis of variance (ANOVA) was performed to investigate ethnic differences in post training status.

RESULTS: Resting blood pressure was not changed with training for either group (AA: bSBP 118 vs. 116, aSBP 103 vs. 101 and CA: bSBP 119 vs. 119, aSBP 100 vs. 101). (See table) Exercise training reduced the change from rest to 15 min after acute maximal exercise for bPP, aPP and FPH (**p<0.05), in both groups. However, there was an interaction for RPH (**p<0.05) and the reduced change was significant from baseline only in the AA (p<0.05)

338
CONCLUSION: Exercise training appears to attenuate the changes in blood pressure following acute maximal exercise. This suggests that exercise training reduces post exercise hypotension.

<table>
<thead>
<tr>
<th></th>
<th>AA change from rest 15 min post acute exercise</th>
<th>CA change from rest 15 min post acute exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre-training</td>
<td>post-training</td>
</tr>
<tr>
<td>Aortic PP (mm Hg)*</td>
<td>-6.8±1.3</td>
<td>-2.4±1.3</td>
</tr>
<tr>
<td>Brachial PP (mm Hg)*</td>
<td>-5.6±1.6</td>
<td>-0.2±1.7</td>
</tr>
<tr>
<td>FPH (mm Hg)*</td>
<td>-5.0±1.2</td>
<td>-0.7±1.1</td>
</tr>
<tr>
<td>RPH (mmHg)**</td>
<td>-5.0±0.7</td>
<td>-2.8±0.6</td>
</tr>
</tbody>
</table>

Many studies have identified the occurrence of post-exercise hypotension, however the determinants of blood pressure reduction after a single exercise session remains unclear.

PURPOSE: To combine the exercise results and apply the meta-analytic model to identify the influences of regular aerobic exercise on post-exercise hypotension in normotensive and hypertensive subjects and to verify the effect of other possible modulators such as clinical condition, duration/volume and intensity.

METHODS: A systematic review with meta-analysis. The outcomes compared were: Systolic and diastolic blood pressure, considering the differences between regular practice of aerobic exercise, clinical status, duration and intensity. We evaluated the standardized mean difference given the size of the hypotensive effect. Searches were conducted in Medline, Scientific Electronic Library Online (SciELO), Lilacs, EMBASE, SPORTDiscus and EBSCO until September 2013.

RESULTS: Eighty-eight studies were included in the present systematic review, nine were included in the meta-analytic model. The post-exercise hypotensive effect (systolic blood pressure reduction) was identified at 60 min for active (-0.78, 95%CI -1.28 to -0.27 [P=0.003]) and sedentary (-0.33, 95% CI -0.55 to -0.10 [P=0.004]) and 90 min for active (-1.36, 95%CI -2.44 to -0.28 [P=0.013]). The effect size of hypotension was not different according to the regular practice of aerobic exercises. Hypertensive subjects had greater hypotensive effect compared to normotensive (-1.24, 95%CI -1.61 to -0.87 vs -0.33, 95% CI -0.52 to -0.15 - for diastolic blood pressure, respectively). The magnitude of hypotension was not modulated by duration and intensity of the workout.

CONCLUSION: The magnitude of post-exercise hypotension is not modulated by regular aerobic exercise. However, hypertensive subjects have higher postexercise hypotensive effect when compared with their normotensive peers.

BACKGROUND: The mechanismically relation between low oxygen metabolism and poor health remains unresolved.

PURPOSE: To measure mitochondrial oxygen consumption in the left ventricle (LV) of 24 female rats artificially inbred for low- and high running capacity (LCR; HCR, respectively) and randomized to either sedate (LCRsed; HCRsed) or exercise training.

METHODS: Oxidative phosphorylation (OXPHOS) capacity was assessed in chemically permeabilized fibres using high-resolution respirometry and corrected for wet-weight. Activity of the TCA cycle enzyme citrate synthase (CS) was measured to indicate mitochondrial density. Exercise training consisted of aerobic interval training (AIT) sessions 5 times a week for 1 month and then 2 times a week for 8 months (LCRext; HCRext). Statistical analyses were performed using a two-way ANOVA.

RESULTS: Baseline results for LV in LCRsed compared to HCRsed showed reduced complex II linked respiration (102.45±8.77 vs. 126.44±6.43; p<0.05), maximal OXPHOS (132.00±5.32 vs. 161.53±12.46 pmol O2/min±1*mgww-1; p<0.05). AIT improved maximal OXPHOS such that there was no difference between LCRext and HCRext (160.62±16.55 vs. 152.56±17.42 pmol O2/min±1*mgww-1; p>0.05). AIT had no effect on CS-activity in neither LV nor HCR (1.05±0.35 and 1.18±0.13 pmol*mg-1*min-1, respectively; p>0.05), suggesting primarily qualitative adjustments to exercise training in LV. Expressing mass-specific respiration relative to maximal observed flux indicated that fat oxidation (29±8 vs. 37±8% in LCR; 26±3 vs. 37±8% in HCR; p>0.01) and complex I linked respiration (46±6 vs. 53±13% in LCR; 40±5 vs. 52±11% in HCR; p>0.05).独立 mitochondrial density and inbreeding.

CONCLUSIONS: Sedentary rats that contrast in intrinsic low- and high aerobic capacity differ significantly in maximal OXPHOS in LV. Nine months of AIT was able to reverse this initial impairment in mitochondrial function in the heart, mainly through qualitative adjustments. These findings might explain some of the poor health features of low capacity rats and suggest training-induced plasticity within LV.
PURPOSE: Obesity-induced cardiovascular disease is also associated with myocardial insulin resistance and impaired on the Mammalian Target of Rapamycin (mTOR) pathway signaling. Exercise activating the mTOR signaling pathway has been largely shown in skeletal muscle, but insufficiently in myocardial tissue. Actually, it is known that mTOR modulation can occur by many molecules that converge at the level of the Tubrous Sclerosis Tumor Suppressor Proteins 1/2 (TSC1/2). With this in mind, we evaluated P38 Mitogen-Activated Protein Kinases (P38MAPK) phosphorylation and REDD1 (or RTP801, regulated in development and DNA damage responses 1) and 14-3-3 protein levels in the myocardial tissue of obese rats sedentary or physical exercise.

METHODS: After installation/achievement of diet-induced obesity and insulin resistance, Wistar rats were divided in two groups: obese rats sedentary; obese rats performed a treadmill running (50 min/day, 5 days per week velocity of 1.0 km/h for 2 months). Lean rats of the same age were used as control group. Forty eight hours after the final physical exercise, the rats were death and myocardial tissue removed for western blot analysis.

RESULTS: Diet-induced obesity increased REDD1 and TSC protein levels and reduced mTOR, P70S6k (p70 ribosomal S6 protein kinase) and 4EBP1 (4E-binding protein 1) phosphorylation. Interestingly, physical exercise reduced REDD1 and TSC protein levels and increased 14-3-3 protein levels and P38MAPK, mTOR, P70S6k and 4EBP1 phosphorylation.

CONCLUSION: Our results showed that P38MAPK/REDD1/14-3-3 via may be involved in the mTOR activation induced by physical exercise in myocardium of obese rats.

Metabolic responses, such as blood lactate (BL) dynamics, and heart electrical activity, such as heart rate variability (HRV), have not been studied during ultra-distance relay run. It was documented the product of anaerobic glycolysis (lactate) is increased in greater alterations in BAFMD in relation to control (ES=0.19). Training induced a significant improvement of exercise heart rate recovery (HRR) at 1, 3 and 5 min following maximal treadmill exercise and 5 min of HRV change might be used as an indication of the central fatigue.

PURPOSE: To analyze BL levels and monitor HRV in male runners before, during and after long-distance relay run.

METHODS: Six experienced runners (31±5.5 yrs) were monitored during 317 km relay run. Participants were rotating and each completed 6 legs during the race. Average distance of the race leg was 8.8±4.1 km. Capillary blood samples were taken and BL analyzed: Pre; Post; and Post-1 min; 3 min; 5 min; 10 min; 15 min. HRV was monitored with Actiheart 24 h before, during, and 24 h after the race. HRV evaluated using root mean square of successive differences (rMSSD).

RESULTS: BL levels immediately after each race leg were (3.08±0.84 mmol/L) significantly higher (p=0.03) than before (2.23±0.52 mmol/L) during ultra-distance relay run. Beat-to-beat variation in the R-R interval on the electrocardiogram is known as HRV. BL increases during the race legs and is observed after each leg, however, BL levels decrease as race legs progress. rMSSD pre-race (109.1±18.3) was higher than rMSSD post-race (95.4±20.1). Although post-race BL was lower, it wasn’t significant (p=0.067) probably because due to the small sample size (n=6). HRV does not decline from stage 1 to 6, though, it declined from pre-stage to post-stage recovery.

CONCLUSION: BL increases during the race legs and is observed after each leg, however, BL decreases as race legs progress. This might be due to increased Lactate Dehydrogenase enzyme activity. The load experienced from the ultra-distance relay run doesn’t have a great effect on HRV during the race but may suggest accumulative fatigue when comparing pre- and post-race day HRV.

Over the last decade, many studies have examined the influence of exercise training on vascular function. PURPOSE: To conduct a meta-analysis on existing studies which have examined the impact of exercise training on brachial artery flow-mediated dilation (BAFMD).

METHODS: Data used in the analysis were obtained via a literature search of PubMed. Search terms included: “flow-mediated dilation (FMD)”, “vasodilation” and “exercise”. Inclusion criteria were: (1) exercise training ≥1 week;; (2) FMD conducted on the brachial artery with pre- and post-training values reported; and (3) the use of human subjects.

RESULTS: The final dataset consisted of 64 individual studies, totaling 1,803 intervention and 644 control subjects. The subject pool was composed of men (N=1365) and women (N=408), range 8-81y, and “healthy” and “diseased” participants. The overall training effect size (ES) was 1.57 (range; 0.01-10.02) with 70 of the 85 intervention groups showing a significant improvement in BAFMD (ES>0). Of the 15 training intervention groups that did not reach statistical significance, 6 had a negative ES. Studies examining “at risk/diseased” subjects demonstrated a greater ES compared to healthy subjects (1.92 and 0.51, respectively; P<0.005). The ES for combined aerobic and resistance training had the largest effect on BAFMD (N=10; ES=1.91; P=0.005), followed by aerobic training (N=58; ES=1.53; P=0.001), and resistance training (N=18; ES=1.47; P=0.005) compared to controls (N=34; ES=0.19). Training intensity significantly predicted BAMBDF ES (p=0.407, P trend=0.001) as Low-Moderate (ES=1.18; P=0.005) and High (ES=1.91; P=0.001) intensity exercise resulted in greater alterations in BAFMD in relation to control (ES=0.19). Moderate relationships were found between BAFMD ES and the effect of age (r=0.375; P=0.001), training intensity (r=−0.407; P=0.001) and exercise training duration (r=−0.311; P=0.005).

CONCLUSION: On the basis of these data, exercise training produces a large change in BAFMD, a biomarker of vascular health. Moderators to the magnitude of the change in BAFMD with exercise training include health status and training modality.
1313  Board #53  May 29, 9:00 AM - 10:30 AM  
Myocardial Blood Flow Heterogeneity In Highly Endurance-trained Athletes And Untrained Control Subjects  
Ilkka Heinonen1, Joonas Hakala1, Dirk J. Duncker2, Juhani Knutti1, Kari K. Kalliokoski1. 1Turku PET Centre, University of Turku and Turku University Hospital, Turku, Finland. 2Erasmus MC, University Medical Center Rotterdam, Rotterdam, Netherlands.  
(No relationships reported)  
PURPOSE: The effects of acute exercise and endurance training on human myocardial blood flow heterogeneity are largely unknown.  
METHODS: In the present study we measured myocardial blood flow in 17 different segments of the left ventricle in highly endurance-trained male athletes and matched untrained control subjects at rest, during exercise (n=13+13), and adenosine-induced vasodilatation (n=10+10). Myocardial blood flow was measured by positron emission tomography, regional myocardial blood flow analyzed by a novel Carimas image analysis software, and heterogeneity calculated as coefficient of variation of the segments.  
RESULTS: Myocardial blood flow was lower in endurance athletes compared to untrained control subjects in every condition studied. Blood flow was in general the highest in apex, and lowered towards the base. In response to acute exercise (100 watts supine cycling), myocardial blood flow heterogeneity did not change in endurance athletes (23±11% at rest and 19±8% during exercise), but was increased in untrained subjects (18±6% at rest and 30±4% during exercise, p=0.0005). Myocardial blood flow was also more homogeneous during exercise in endurance athletes compared to untrained controls, (p<0.01). However, blood flow heterogeneity did not change from rest to adenosine-induced vasodilatation, and was not different between the groups at rest or during adenosine infusion.  
CONCLUSIONS: Myocardial blood flow heterogeneity increases from rest to exercise in normal healthy untrained men. Additionally, blood flow heterogeneity is more homogenous in highly endurance-trained athletes during exercise at the same absolute external workload, but does not change in response to pharmacologically-induced vasodilatation.  

1314  Board #54  May 29, 9:00 AM - 10:30 AM  
The Effect Of An Increase In Skeletal Muscle Capillarization On O2 Extraction And Blood Flow In The Exercising Leg  
Stefan P. Mortensen1, Mads Madsen2, Jonas B. Hansen2, Gregers D.W. Munch2, Ylva Hellsten1. 1Rigshospitalet, Copenhagen, Denmark. 2 University of Copenhagen, Copenhagen, Denmark.  
(No relationships reported)  
A higher capillary density is thought to be a primary mechanism underlying the increase in skeletal muscle O2 extraction in training, but evidence for this is lacking.  
PURPOSE: To investigate the role of capillary density on skeletal muscle O2 extraction and blood flow.  
METHODS: We measured leg hemodynamics at rest and during one-legged knee-extensor exercise (12 and 24W) and obtained muscle biopsies in 10 healthy young men before and after 4 weeks of treatment with a α1-antagonist (Terazosin, 0.5-1 mg/day) to increase capillarization. Data were analysed by repeated measures two-way ANOVA and Tukey’s honestly significant difference (HSD) post hoc procedure. Statistical significance was accepted at P<0.05.  
RESULTS: Resting leg blood flow was increased throughout the 4 weeks of Terazosin treatment (P<0.05). The capillary to fibre ratio was 1.68±0.07 before Terazosin treatment and it increased to 1.89±0.08 after Terazosin treatment (P<0.05). Resting leg blood flow was lower after the Terazosin treatment (P<0.05), whereas arterial blood pressure was unchanged. After Terazosin treatment, leg blood flow during exercise was lower (12W and 24W, P<0.05), O2 extraction higher (12W, P<0.05) and femoral venous lactate levels lower (24W, P<0.05).  
CONCLUSIONS: These results demonstrate that a 4 week increase in vascular wall shear stress induces capillary growth in humans. An increase in capillarisation increases O2 extraction in exercising leg and lowers blood flow.  
Supported by the Danish Ministry of Culture.  

1315  Board #55  May 29, 9:00 AM - 10:30 AM  
Aortic Size in National Football League Scouting Combine Participants  
Andrew E. Lincoln1, Andrew M. Tucker2, Robert A. Vogel1, Richard J. Kovacs3, Reginald E. Dunn1, Lon W. Castle1. 1MedStar Health Research Institute, Baltimore, MD. 2MedStar Union Memorial Hospital, Lutherville, MD. 3University of Colorado, Denver, CO. 4Indiana University, Indianapolis, IN. 5Cleveland Clinic, Westlake, OH.  
(No relationships reported)  
Aortic rupture is an uncommon, but feared, cause of sudden death in athletes. A goal of pre-participation screening programs is to identify athletes at risk for aortic rupture. Normative aortic size data currently exist for the general population based on sex, height, age and other factors. However, there is a lack of normative data for individuals of the age, size and activity level similar to NFL players.  
PURPOSE: To examine the distribution of aortic size among NFL Scouting Combine participants and identify the prevalence of aortic size greater than 4.0 cm.  
METHODS: We systematically screened draft-eligible college football players at the NFL Scouting Combine over three years (2011-2013) for aortic size prior to participation. Each player underwent a resting echocardiogram as part of a thorough physical examination. Aortic size was measured from the two dimensional parasternal long axis view according to the criteria of the American Society of Echocardiography. Measurements were performed by two expert technicians and confirmed by an expert echocardiographer from the Indiana University Health System.  
RESULTS: 983 draft-eligible Combine players were screened over the three years. On average, players were 23.5 years of age (SD=1.3), 244.3 pounds (SD=46.0), 74.0 inches tall (SD=2.7), and had a BMI of 31.4 kg/m2 (SD=4.5). Two-thirds (66.7%) of players were black and 25% were white. One-third (34.3%) were linemen and 30.7% were receivers/defensive backs. There were no players with inadequate images to measure aortic size. The 75th percentile for aortic size was 2.99 cm; the 90th percentile was 3.22 cm; the 95th percentile was 3.37 cm; and the 99th percentile was 3.63 cm. Only one player had an aorta greater than 4.0 cm (0.1%), which was confirmed by MRI.  
CONCLUSION: An aorta greater than 4.0 cm is uncommon even in very large, elite athletes. Athletes of any size whose aorta exceeds 4.0 cm may be at risk for aortic rupture and their participation in sports must be carefully considered.  
This research was sponsored by the National Football League.  

1316  Board #56  May 29, 9:00 AM - 10:30 AM  
Comparison of Cardiac Vagal Modulation From the Orthostatic Stress Test Between Untrained Individuals and Athletes.  
Gabriel D. Rodrigues, Thiago Rodrigues Gonçalves, Sandro Conceição De Souza, Pedro Paulo Da Silva Soares. Federal Fluminense University, Niterói/RJ, Brazil.  
(No relationships reported)  
Heart rate variability (HRV) and global cardiac vagal modulation are associated with physical capacity in healthy individuals. However adjustments in the cardiac vagal modulation caused by orthostatic (ORT) test between individuals of low physical capacity (non-athletes) and athletes are still unclear.  
PURPOSE: To investigate the variation (Δ) of cardiac vagal modulation from supine (SUP) to ORT positions between healthy individuals with low and high physical capacity (athletes).  
METHODS: Participated of the present study 18 individuals, divided in two groups: 9 athletes (20 ± 4 years; VO2max = 48.7 ± 6.7 ml/kg·min−1) and 9 non-athletes (25 ± 5 years; VO2max = 34.0 ± 3.4 ml/kg·min−1). Heart rate (HR) was recorded beat-by-beat during 20min at SUP position and 20min at ORT position. High-frequency component (HF, 0.15- 0.40Hz) was presented in normalized unit (n.u.), and also were observed the RR intervals (RR) and the square root of the differences of successive RR squared (RMSSD) as indexes of HRV. We utilized for statistical analysis mean ±SD and Student t-test unpaired (p ≤ 0.05).
RESULTS: ΔHFr showed significant difference between athletes and non-athletes (-32.3 ± 16.6 vs. -19.5 ± 10.4, p=0.02) respectively. However, there was no significant difference to HF-SUP between the groups (51.4 ± 17.1 vs. 45.3 ± 14.7, p=0.43), as well as to HF-ORT (16.1 ± 10.4 vs. 25.8 ± 20.3, p=0.21). The ΔRR was significantly higher in athletes (280.5 ± 130.0 ms) than in non-athletes (148.0 ± 124.7 ms, p = 0.04), but not at RR-SUP (1036.5 ± 153.5 vs. 885.9 ± 97.5 ms, p = 0.57) and RR-ORT (756.0 ± 105.0 vs. 737.9 ± 89.6 ms, p = 0.45). RMSSD not demonstrated significant differences between groups at SUP (53.8 ± 31.0 vs. 62.8 ± 35.3 ms, p = 0.57), and ORT (26.8 ± 10.6 vs. 31.4 ± 14.7 ms, p = 0.45) positions. ARMSD also not presented significant difference (~27.0 ± 32.4 vs -31.4 ± 35.5 ms, p = 0.78) between athletes and non-athletes, respectively.

CONCLUSION: The variation of cardiac vagal modulation from SUP to ORT position is higher in athletes than in individuals of low physical capacity. Supported by CAPES, CNPq and FAPERJ.

During incremental exercise to volitional exhaustion, it is evident that stroke volume (SV) increases and plateau at submaximal work rates, although a linear increase in SV has been revealed in trained athletes progressively to maximal oxygen uptake (VO2max). However, some data show that at near-maximal intensities, SV declines before attainment of VO2max. This phenomenon has been observed in trained individuals completing traditional incremental protocols (RAMP) and supramaximal constant-load exercise. The effect of exercise structure, such as the self-paced and decremental protocols which have been shown to augment VO2max, on central hemodynamics remains unknown. Furthermore, the influence of fitness level on the SV response to maximal exercise tests has not been examined.

PURPOSE: To observe hemodynamic responses to completion of traditional RAMP and two novel maximal exercise protocols in individuals of various fitness level. METHODS: Thirty men and women (age and %BF = 26.0 ± 5.0 yr and 15.7 ± 7.1%) initially completed RAMP followed by a decremental test (DEC) on a subsequent day. Over the next two sessions, which were randomized and separated by 48 h, participants performed a self-paced (SP) and an additional DEC test. During exercise, gas exchange data were measured via indirect calorimetry and hemodynamic function was assessed via thoracic impedance. Repeated measures ANOVA was used to examine differences in VO2, HR, SV, and cardiac output (CO) during exercise and across protocol. RESULTS: The SP protocol elicited a significantly higher (p < 0.05) CO2max (21.6 ± 3.7 L/min), and HR2max (188.2 ± 11.2 b/min) versus RAMP (20.4 ± 3.4 L/min and 185.6 ± 11.2 b/min). Although there was no effect of protocol on SV2max, participants with VO2max > 45 mL/kg/min displayed a significant (p < 0.05) decline in SV (9 ± 17 mL/beat) prior to attainment of VO22max across all protocols. Increased CO2max in SP trial mediated a significantly greater (p < 0.001) VO22max compared to RAMP (52.7 ± 9.6 vs. 49.7 ± 10.2 mL/kg/min, respectively). CONCLUSION: Data confirm that SV declines suddenly prior to attainment of VO22max in persons of moderate to high fitness. Protocol-induced increases in CO2max augmenting VO22max suggest that O2 delivery is a key limitation to maximal exercise.

Recently, a self-paced (SP) and decremental protocol (DEC) demonstrated higher values for VO2max versus the traditional ramp protocol (RAMP). PURPOSE: The primary aim of the current study was to examine potential differences in VO2max between the RAMP and SP and DEC protocols. METHODS: Thirty men and women (mean age and body fat = 26.0 ± 5.0 yr and 15.7 ± 7.1%) with divergent fitness level (low, moderate, and high fitness = VO2max from 35 – 45, 45 – 55, and > 55 mL/kg/min, n = 10 per group) initially completed a RAMP protocol to volitional exhaustion to determine VO2max, with work rate beginning at 50 – 80 W and increasing by 25 – 40 W/min. On a subsequent day, they performed DEC consisting of 60 s of cycling at 105 %Wmax followed by 10 s reductions in power output during subsequent stages lasting 45, 60, 75 s, and then to exhaustion. Over the next two sessions, which were randomized and separated by 48 h, they performed SP (2 min of cycling at RPE = 11, 13, 15, 17, and 20) and an additional DEC protocol which were followed by a final RAMP 48 h later. During exercise, gas exchange data were obtained using indirect calorimetry, and thoracic impedance was utilized to assess hemodynamic function. Repeated measures analysis of variance was used to examine differences in maximal determinations of VO2, HR, and cardiac output (CO) across protocol.

RESULTS: Results demonstrated significantly lower (p < 0.001) VO2max in RAMP (49.7 ± 10.2 mL/kg/min) compared to SP (52.7 ± 9.6 mL/kg/min) and DEC (51.2 ± 9.8 and 51.2 ± 9.0 mL/kg/min, respectively), with a significant interaction (p = 0.047) seen for fitness level. Eighty percent of participants revealed higher VO2max in SP versus RAMP, with this difference ranging from -1.3 - 11.0 %. Maximal HR and CO (p = 0.02) were higher in response to SP versus other protocols.

CONCLUSIONS: These data show that the traditional ramp protocol may underestimate VO2max in persons of moderate to high fitness. Protocol-induced increases in CO augmenting VO2max suggest that O2 delivery is a key limitation to maximal exercise.

METHOD: An exercise wheel system was used for the exercise paradigm. Male (C57BL/6) wild type (WT) and AT1aKO mice were randomly assigned to four groups: WT control (n=6), WT exercise (WTEX, n=8), AT1a KO (KO) control (n=5), and AT1a KO exercise (KOEX, n=8). Mice were forced to run at a velocity of 8 m/min for 1 hour, 3d/wk, for 7 wks. Echocardiography was conducted at baseline and during 7 wks. Lactate was measured during several exercise sessions. Electrolytes, blood pH, and cardiac histology were assessed post-exercise. Results showed a significant increase in ejection fraction (EF%) in KOEX (72.5 ± 5.1%) vs. WTEX and KO control (64.1 ± 12.2% and 63.2 ± 22.2%, respectively). Mitral valve area assessment revealed a marked increase in E-wave velocity in WTEX compared to WT control at baseline (74.1 ± 9 vs. 90.3 ± 5.3 cm/s, p<0.05), while E/A wave ratio remained unchanged. Cardiomyocyte diameter was larger in WTEX compared to KOEX (29.5 ± 0.7 vs. 25.8 ± 0.5 μm, p<0.05). Heart to body weight ratio was significantly higher in WTEX vs. KOEX (5.5 ± 0.2 vs. 4.3 ± 0.1 mg/g, p<0.001). Masson’s Trichrome staining revealed higher collagen levels in WTEX myocardium compared to WT control and KOEX (16% vs. 5%). Blood lactate accumulation values were greater at 5 and 60 min of wheel running in WTEX (4.2 ± 0.4 and 3.9 ± 0.5 mmol/l) vs. KOEX (3.0 ± 0.2 and 2.9 ± 0.2 mmol/l). A basic metabolic panel revealed higher [HCO3-] in KOEX vs. WTEX (21.7 ± 0.6 vs. 13.7 ± 1.8 mmol/l). Blood pH was significantly lower in WTEX as compared to KOEX (7.28 ± 0.41). In conclusion, mice lacking AT1a receptor exhibited improved cardiac function without myocardium hypertrophy, higher exercise endurance, and enhanced metabolic response under chronic exercise stimulation. Findings indicate that the AT1a receptor is an important mediator of exercise induced cardiac dysfunction and acid-base imbalance during exercise training.
Acute Oxygen Consumption And Heart Rate Responses During A Stationary Running Game

Gusthavo Augusto A. Rodrigues, Danilo S. Felipe, Wagner Z. de Freitas, Elisangela Silva, Fabiano F. da Silva, Renato A. de Souza. Instituto Federal de Educação, Ciência e Tecnologia do Sul de Minas - Campus Macaúbas, Macaúbas, Brazil. (No relationships reported)

Recently, a new class of video games called exergames (EXG) has used virtual reality to provide to the user the possibility of perceptual and performance emulation with potential for sensory and motor abilities development. To date there are insufficient scientific descriptions about acute cardiovascular responses during physical exertion imposed by EXG.

PURPOSE: To analyze the differences of relative (%) VO2 max and HR max using stationary running game of Nintendo Wii in the following experimental situations: (A) with the volunteers running on Wii Balance Board, and (B) with the volunteers running on jump platform.

METHODS: Nine healthy male (age: 20±2 yrs) with no prior experience with Wii were recruited. Firstly, the VO2 max and HR max were determined on a treadmill test by measuring respiratory gases coupled with a heart frequency meter. After 24 hours, the subjects were randomly submitted the experimental protocols A and B with 30 minutes of rest between them. During both protocols the VO2 and HR were monitored during the entire test (6 minutes). Mean and standard deviation of % VO2 max and % HR max were compared using T-Student test considering significance p<0.05.

RESULTS: Both variables analyzed had increase over baseline. Comparing the A and B protocols, it was show that while in A situation the % VO2 max and % HR max achieve 47.0 and 64.3, respectively, during B situation these values were significantly higher, 65.1% and 76.8%, respectively (p<0.05).

CONCLUSION: The data analyzed show that the proposed intervention generated a significant increase in VO2 and HR variables, reaching levels close to indices of moderate and vigorous exercises. Thus, the running game of Wii may be modulating with use of accessories to reach levels according to the ACSM recommendation for health promotion.

1321
Board #61
May 29, 9:00 AM - 10:30 AM
Increase in Exercising Leg Blood Flow Improves Endurance Performance during Dynamic Planter Flexion Exercise
Massashi Ichinose1, Tomoko Ichinose1, Takeshi Nishiyasu2. 1Meiji University, Tokyo, Japan; 2University of Tsukuba, Tsukuba, Japan. (No relationships reported)

The reduction of blood flow to active skeletal muscles during dynamic exercises decreases oxygen supply and enhances accumulation of metabolites in the active muscles and diminishes endurance exercise performance. However, it is unknown whether active muscle blood flow is a limiting factor of the endurance performance under normal blood flow conditions.

PURPOSE: To investigate this point, we examined the effects of the increase in exercising leg blood flow (LBF) on endurance performance, cardiovascular and respiratory responses during dynamic plantar flexion exercise.

METHODS: 8 healthy subjects performed dynamic plantar flexion exercise at 80% of peak workload with the lower thigh enclosed in a negative pressure box. In control conditions (CON), the box pressure was kept at ambient pressure. In negative pressure conditions (NP), beginning 1 min before the start of the exercise, the box pressure was decreased to -70 mmHg. The exercise was continued until voluntary exhaustion, or terminated if subjects could continue the exercise for 30 min.

RESULTS: In CON, all of the subjects reached exhaustion within 15 min and average exercise tolerance time was 59±7 sec. In NP, all subjects could tolerate the exercise longer than in CON and 5 subjects could continue the exercise for 30 min. In CON, exercising LBF, heart rate (HR), mean arterial blood pressure (MAP), cardiac output (CO) and oxygen consumption (VO2) gradually increased with exercise duration. Application of negative pressure significantly increased exercising LBF (at 1st min of exercise: 902±95 vs. 1183±103, at last 1 min in CON vs. corresponding time in NP: 1244±64 vs. 1392±45 mmHg, p<0.05). In addition, the cardiovascular and respiratory responses were significantly diminished in NP compared to CON (HR: 82±4 vs. 69±4 beats/min, MAP: 102±3 vs. 84±2 mmHg, CO: 7.6±0.3 vs. 7.0±0.3 L/min, VO2: 437±19 vs. 346±11 ml/min, p<0.05).

CONCLUSIONS: Our results demonstrated that the increase in exercising LBF improves endurance performance and attenuates cardiovascular and respiratory responses during dynamic plantar flexion exercise. These results indicate that active muscle blood flow is a limiting factor of the endurance performance as well as an important determinant of the cardiovascular and respiratory responses under normal blood flow conditions.

1322
Board #62
May 29, 9:00 AM - 10:30 AM
The Acute Cardiovascular and Inflammatory Responses to Ultramarathon and Marathon
Bridge E. Durocher1, Morton H. Harwood2, Scott N. Drum, FACSMD, Phillip B. Watts, FACSMD, Marguerite T. Moore1, John J. Durocher1. 1Northern Michigan University, Marquette, MI; 2Michigan Technological University, Houghton, MI. (No relationships reported)

Central arterial stiffness (AS) is shown to be increased during the first hour post ultramarathon (ULT), but it is presently unknown if AS remains elevated 24 hours after an ULT or marathon (MAR).

PURPOSE: To determine the effects of ULT and MAR on C-reactive protein (CRP), brachial blood pressure (BP), aortic BP, central AS and aortic augmentation index (AIx). We hypothesized that CRP, central AS and Aixs would be elevated post-Race.

METHODS: Non-invasive estimates of central AS, aortic BP, and AIx were measured via applanation tonometry in 12 endurance athletes (8 men and 4 women) approximately 24 hours before and after an ULT or MAR. Plasma CRP, resting BP, and heart rate variability (HRV) were also assessed during the same times. Runners were 36 ± 2 years, 172 ± 3 cm, 66 ± 3 kg, and had a mean body mass index of 22 ± 1 kg/m². Data for the ULT and MAR were pooled because there were no time × distance interactions when distance was entered as a between subjects factor in a repeated measures ANOVA statistical analysis. Because there were no time × distance interactions, we compared all major variables pre vs. post with paired t-tests.

RESULTS: CRP was significantly (p < 0.05) elevated (0.5 ± 0.1 vs. 6.8 ± 1.1 mg/L), while resting aortic systolic BP (111 ± 3 vs. 101 ± 3 mmHg) and aortic diastolic BP (73 ± 3 vs. 64 ± 3 mmHg) were significantly lower 24 hours post-race compared to 24 hours pre-race. Likewise, brachial systolic BP (128 ± 4 vs. 121 ± 4 mmHg) and diastolic BP (83 ± 14 vs. 73 ± 11 mmHg) were lower 24 hours post-race. Resting HR and low-frequency to high-frequency HRV were similar the day before and the day after the race. Finally, central AS (via pulse wave velocity) was similar 24 hours post-race compared to 24 hours pre-race, while AIx was lower (p = 0.05) 24 hours post-race (13 ± 4 vs. 6 ± 3%).

CONCLUSION: Our findings reveal that systemic inflammation was increased 24 hours post-race, while at the same time brachial BP and aortic BP were decreased. Reductions in AIx and increases in CRP may contribute to the acute exercise-induced central and peripheral hypotension in long-distance runners 24 hours post-race.

This project was funded by an Excellence in Education Research Grant from Northern Michigan University to Bridge Durocher.

1323
Board #63
May 29, 9:00 AM - 10:30 AM
Hemodynamic and Energy Expenditure Responses to Low-Intensity Walking Exercise with Different Blood Flow Restriction Pressures
Murat Karabulut1, Jose A. Leah1. 1University of Texas at Brownsville, Brownsville, TX; 2Texas Woman’s University, Houston, TX. (No relationships reported)

PURPOSE: The purpose of the study was to examine the acute effects of walking exercise with varying blood flow restriction (BFR) on oxygen consumption (O2CON), heart rate (HR), and systolic (SBP) and diastolic blood pressure (DBP).

METHODS: Eleven sedentary but healthy females (age= 22.7±2.8) participated in this study. On the first day, initial screening, anthropometric measures, and familiarization with testing procedures were completed. Subjects returned to the lab after an overnight fast of at least 8 hours to complete five randomized testing sessions on five different days. Each subject’s resting HR, BP, and O2CON were measured before, during, and after testing using HR monitor, sphygmomanometer, and metabolic cart, respectively. Subjects
exercised for 15 minutes on a treadmill at 3.5 mph and 0% incline. During BFR testing days, elastic cuffs were placed at the upper thighs and the initial restrictive pressure (IRP, pressure created by the tightness of cuffs before inflation with air) was set at ~40 mmHg or ~60 mmHg IRP, and then was inflated with air to reach the final restrictive pressure (FRP; 160 or 200 mmHg). The pressures for four BFR testing days were as follows: IRP-40&FRP-160, IRP-40&FRP-200, IRP-60&FRP-160, and IRP-60&FRP-200 mmHg. During the control day, subjects performed the walking exercise with no BFR (CON).

RESULTS: There were significant main effects for condition and time (p<0.02) for total caloric expenditure, HR, and SBP. Significant condition*time interactions (p<0.01) were detected for total energy expenditure, HR, and SBP. Post-hoc comparisons showed significantly higher caloric expenditure for IRP-60&FRP-160 mmHg compared to CON and IRP-40&FRP-200 and SBP for IRP-60&FRP-160 mmHg compared to CON (p<0.02). HR was significantly lower for the CON session compared to the other conditions (p<0.04) except for the condition using IRP-40&FRP-160 mmHg.

CONCLUSIONS: Results suggest that IRP may be an independent factor producing variations in peripheral resistance and venous return. It is likely that varying IRPs cause different demands on the cardiorespiratory system generating changes in the level of activity of the heart and perhaps respiratory muscles. These variations may ultimately result in changes in caloric expenditure and hemodynamic responses.

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**Acute Vascular Effects of Aquatic and Land Treadmill Exercise**

Dustin P. Joubert1, Peter W. Grandjean, FACSM2, Stephen F. Crouse, FACSM3. 1Texas A&M University, College Station, TX. 2Baylor University, Waco, TX.

**Values represent mean ± SD; P-Value from dependent sample T-Test on Change variables.**

<table>
<thead>
<tr>
<th></th>
<th>FMD</th>
<th>PWV</th>
<th>SBP</th>
<th>DBP</th>
<th>MAP</th>
<th>HR</th>
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<tbody>
<tr>
<td>ATM</td>
<td>Pre-Ex</td>
<td>9.5 ± 4.2</td>
<td>7.1 ± 1.7</td>
<td>116 ± 7</td>
<td>62 ± 7</td>
<td>80 ± 7</td>
</tr>
<tr>
<td></td>
<td>Post-Ex</td>
<td>6.5 ± 1.7</td>
<td>7.6 ± 2.0</td>
<td>113 ± 9</td>
<td>62 ± 7</td>
<td>79 ± 7</td>
</tr>
<tr>
<td></td>
<td>Change</td>
<td>-3.0 ± 4.6</td>
<td>0.6 ± 1.0</td>
<td>-2.9 ± 5.2</td>
<td>0.1 ± 1.1</td>
<td>-0.9 ± 2.0</td>
</tr>
<tr>
<td>LTM</td>
<td>Pre-Ex</td>
<td>6.4 ± 2.7</td>
<td>7.1 ± 0.8</td>
<td>116 ± 8</td>
<td>62 ± 7</td>
<td>80 ± 7</td>
</tr>
<tr>
<td></td>
<td>Post-Ex</td>
<td>7.1 ± 3.3</td>
<td>6.5 ± 1.1</td>
<td>116 ± 7</td>
<td>60 ± 8</td>
<td>79 ± 8</td>
</tr>
<tr>
<td></td>
<td>Change</td>
<td>0.7 ± 4.2</td>
<td>-0.6 ± 0.8</td>
<td>-0.3 ± 7.4</td>
<td>-1.9 ± 4.6</td>
<td>-1.4 ± 4.9</td>
</tr>
</tbody>
</table>

**P-Value** | 0.237 | 0.078 | 0.581 | 0.239 | 0.821 | 0.535
CONCLUSION: No significant changes found pre and post exercise between modes. While exercise modes were matched for VO\textsubscript{2}, RPE was higher for ATM (14.2 ± 1.7) than LTM (12.7 ± 1.5), potentially indicating varying degrees of sympathetic stimulation. While this could potentially explain the slight reduction in FMD, in the present population of young, recreationally active men studied, no differences can be substantiated between ATM and LTM. Lack of significant findings may have also been due to the small sample size and reduced statistical power. Future research should focus on less active, older populations in response to ATM and LTM exercise.

<table>
<thead>
<tr>
<th>Event</th>
<th>Condition</th>
<th>MAP (mmHg)</th>
<th>CO (L/min)</th>
<th>TVC (ml/min/mmHg)</th>
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</thead>
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<tr>
<td></td>
<td>BJ</td>
<td>90±1 vs. 84±2(^*)</td>
<td>4.7±0.1 vs. 5.1±0.1</td>
<td>53.0±1.4 vs. 60.4±2.3(^*)</td>
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<tr>
<td></td>
<td>PL</td>
<td>85±1 vs. 89±2</td>
<td>4.8±0.2 vs. 4.9±0.2</td>
<td>53.8±2.6 vs. 55.7±3.1</td>
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<tr>
<td></td>
<td>30%</td>
<td>103±1 vs. 96±2(^*)</td>
<td>9.5±0.3 vs. 9.7±0.3</td>
<td>92.5±3.1 vs. 100.8±2.7(^*)</td>
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<tr>
<td></td>
<td>PL</td>
<td>100±2 vs. 102±2</td>
<td>9.8±0.3 vs. 9.2±0.4</td>
<td>98.0±3.2 vs. 90.4±3.6</td>
</tr>
<tr>
<td></td>
<td>60%</td>
<td>119±1 vs. 111±2(^*)</td>
<td>14.4±0.4 vs. 14.4±0.4</td>
<td>121.3±3.0 vs 129.8±2.7(^*)</td>
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<tr>
<td></td>
<td>PL</td>
<td>114±2 vs. 118±2</td>
<td>14.7±0.4 vs. 14.2±0.5</td>
<td>128.9±3.8 vs 120.2±4.2</td>
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<tr>
<td></td>
<td>80%</td>
<td>122±1 vs. 115±2(^*)</td>
<td>16.0±0.4 vs. 15.8±0.5</td>
<td>130.7±3.4 vs 137.2±3.0(^*)</td>
</tr>
<tr>
<td></td>
<td>PL</td>
<td>119±2 vs. 119±2</td>
<td>16.5±0.5 vs. 16.2±0.5</td>
<td>138.2±3.4 vs 135.4±4.4</td>
</tr>
</tbody>
</table>

\(^*\)p<0.05 vs. PL.
CONCLUSIONS: NS alters the cardiovascular responses at rest and dynamic exercise and as workload increased, more dilation occurred in peripheral vasculature causing less increases in MAP. These results suggest that NS may enhance vasoprotection and prevent an exaggerated blood pressure response during exercise in humans.

CrossFit (CF) is rapidly becoming a popular form of exercise training throughout the world. Despite this, basic empirical evidence to describe physiological responses from CF, such as autonomic modulation of the heart, is lacking.

PURPOSE: The purpose of this study was to compare post-exercise vagal-rebound between CF and treadmill (TM) exercise.

METHODS: Ten men (age = 26.4 yrs ± 2.7yrs) with at least three months of CF experience participated. Aerobic power was determined with a maximal graded TM test (GXT). On two separate occasions, each participant completed either the CF workout “Cindy” (20min of as many rounds of 5 pull-ups, 10 push-ups, and 15 squats as possible) or a 20 min bout of TM exercise at 90% of GXT determined maximal heart rate. Exercise bouts were performed in a randomized crossover fashion. Electrocardiography (ECG) was analyzed for 10-minutes before and 60-minutes after each bout. Vagal activity was quantified through changes in the heart rate variability index of log-transformed square root of the mean of the squared R-R (lnRMSSD), which was analyzed 5 minute segments at 5-10min pre-exercise period (PRE), and during the post-exercise period at 15-20min (POST1), 20-25min (POST2), 25-30min (POST3), and 1 hour (POST4).

RESULTS: Means ±SD of lnRMSSD are shown in Table 1. Significant time dependent decreases occurred following both CF and TM (p < 0.05*). Trial dependent differences occurred in post-exercise lnRMSSD measures (p < 0.05*). lnRMSSD at TM POST4 was not significantly different compared to pre (p = 0.17). lnRMSSD at CF POST4 remained significantly lower compared to PRE (p < 0.05).

CONCLUSION: The results of this investigation demonstrated a delayed vagal-rebound following CF compared to TM exercise.

<table>
<thead>
<tr>
<th>Variable</th>
<th>CINDY</th>
<th>Treadmill</th>
</tr>
</thead>
<tbody>
<tr>
<td>lnRMSSD PRE</td>
<td>4.06 ± 0.48</td>
<td>4.09 ± 0.42</td>
</tr>
<tr>
<td>lnRMSSD POST1</td>
<td>1.92 ± 0.35*</td>
<td>2.76 ± 0.55*</td>
</tr>
<tr>
<td>lnRMSSD POST2</td>
<td>2.1 ± 0.43*</td>
<td>2.96 ± 0.59*</td>
</tr>
<tr>
<td>lnRMSSD POST3</td>
<td>2.25 ± 0.41*</td>
<td>3.06 ± 0.49*</td>
</tr>
<tr>
<td>lnRMSSD POST4</td>
<td>3.34 ± 0.59*</td>
<td>3.84 ± 0.62*</td>
</tr>
</tbody>
</table>

Exercise-induced muscle damage (EIMD) has been shown reduce VO2 peak and ventilatory threshold, and to alter cardiorespiratory and perceptual responses (muscle pain and RPE) during submaximal exercise. It is unclear whether cardiorespiratory and perceptual responses following a similar time-course of recovery to traditional markers of EIMD.

PURPOSE: To examine the changes in oxygen consumption (VO2), ventilation (VE), heart rate (HR), RER, and ratings of muscle pain and sense of effort (RPE) over the course of a week following EIMD.

METHODS: Data were collected on 5 male and 3 female participants. Participants performed a bout of high-intensity lower body eccentric exercise to induce muscle damage. A graded, maximal exercise test was performed prior to (Pre), immediately following (iPost), and 2 (D2), 4 (D4), and 7 days (D7) after the eccentric exercise protocol. Cardiorespiratory measures were taken throughout the test and rating of quadriceps muscle pain and RPE were taken at the end of each minute of exercise. Data from the first 3 stages (6 minutes) are presented.

RESULTS: Immediately following eccentric exercise isometric voluntary contraction was decreased (MVC, -20.6 ± 11.9% ±SD: ± p<0.05) and ratings of muscle soreness in the quadriceps were increased (37.5 ± 22.2 mm; p<0.05). MVC returned to baseline by D4 (p=0.35) while soreness persisted through D7 (p<0.005). Mean VO2 across the 3 submaximal stages was lower (p<0.001) after EIMD with values falling from 45.7±3.6 ml/kg•1•min-1 to 43.9±4.0, 42.7±3.6, 42.4±3.8, and 41.7±4.3, ml/kg•1•min-1 at iPost, D2, D4, and D7 after eccentric exercise. Ratings of muscle pain were increased (p<0.05) compared to Pre (5.1±1.4 and 5.8±0.9 compared to 2.4±0.7 for iPost and D2, respectively) for 48 hours following EIMD. A similar response was also seen for RPE with elevations found at iPost and D2 compared to Pre (15.6±2.5 and 15.0±1.6 compared to 13.4±1.7; p<0.05).

346
CONCLUSIONS: EIMD resulted in a prolonged improvement in movement economy during submaximal exercise. Muscle damage lead to heightened ratings of muscle pain and a heightened sense of effort for the initial 48 hours of recovery. Perceptual values had returned to Pre levels by 4 days post, likely consequent to decreased muscle soreness.

C-26 Free Communication/Poster - Control of Muscle Mass: Anabolic and Atrophic Effects
Thursday, May 29, 2014, 7:30 AM - 12:30 PM
Room: WB1

Comparison Of Different Leg Press Exercise Volumes On The Akt/mTOR Signaling Pathway

William C. Hawkins. University of Kansas, Lawrence, KS. (Sponsor: Dr. Joseph Weir, FACSM)

(Purpose: To better understand the types of resistance training protocols that are most beneficial to stimulate muscle hypertrophy, we examined the difference between a single set (SS) of resistance exercise to multiple sets (MS) of resistance exercise on the Akt/mTOR signaling pathway and on the expression of insulin-like growth factor-1 (IGF-1) and the receptor for IGF-1 (IGF-1R).

Methods: For this study, sixteen healthy males were divided into two groups of eight. Subjects in each group received three biopsies: 1) baseline, prior to exercise; 2) 15-minutes post exercise; and 3) 180 minutes post exercise. Subjects in the SS group performed one set of leg press exercise at 80% of their predetermined 1RM to volitional fatigue. Subjects in the MS group performed 2 sets of 10 repetitions and one set to volitional fatigue at 80% of their predetermined 1RM, with 3 minutes of rest between each set.

Results: There were no differences between groups in the concentration of Akt signaling proteins. Furthermore, there was no difference in Akt expression in the SS group compared to the MS group 180 minutes post-exercise.

Conclusions: These data indicate that exercise volume may not alter signals associated protein synthesis in recreationally trained individuals.

Nutritional Supplementation Enhances Upstream Anabolic Signals Without Impacting Downstream Effectors During a Concurrent Exercise Bout


(Purpose: To explore the impact of nutritional supplementation on anabolic-catabolic balance during CE. Methods: Highly trained men (n=8; mean±SD 27.4±5.4yr, 15.06±5.09% body fat) completed two randomized CE trials (S and P, 7d apart). Each trial comprised a bout of resistance exercise (RE), 5x5 front squat, 80% 1RM, 2 min rest), 15 min rest, and repeated sprints (RS; 8x10s maximal running sprints, 45s rest). Subjects consumed a nutritional supplement (Trial S; 178kcal, 22g whey protein isolate, 16g CHO, 9g fat) or placebo (Trial P; water) immediately post (IP) RE. We measured blood glucose, insulin, testosterone, and cortisol at 8 time points (pre-exercise, IP, RE, pre-RE, IP-RE, and 5, 30, 60, and 180 min post RS (IP+5, IP+30, IP+60, and IP+180, respectively)). In vastus lateralis samples we measured Akt, P70S6K, AMPK, and 4E-BP1 at 3 time points (pre-exercise, 10 and 180 min post RS (IP+10, IP+180)). Results: Nutritional supplementation increased circulating insulin (µIU·mL⁻¹) shortly after consumption at pre-RE (S>P (p=0.036): 13.06±4.65 > 8.26±3.67). Supplementation did not affect IP-RE insulin (p=0.2), but elevated insulin concentrations at IP+5 (S<P>0.01): 26.80±9.95 > 13.80±1.49), IP+30 (S<P>0.001): 19.70±1.18 > 10.34±4.56), and IP+60 (S<P>0.001): 9.42±4.66 > 10.4±3.33). Supplementation did not affect glucose, testosterone, and cortisol. In muscle tissue, Akt(Ser473) phosphorylation was ~1.85 fold higher with supplementation (S<P>0.017). Results were not significantly different when measured at the 60 min time point. Likewise, supplementation did not affect total Akt, total P70S6K, P70S6K(Thr389), 4E-BP1(Thr37/42), and AMPK(Thr172). Conclusion: Nutritional supplementation may increase upstream anabolic signals, like insulin and Akt(Ser473) phosphorylation, without impacting downstream effectors of protein synthesis during CE. Defining optimal nutritional supplementation may promote anabolic signaling during training despite concurrent exercise-induced signaling inhibition.

Nutritional Responses To Acute And Chronic Resistance Exercise Are Enhanced When Combined With Aquatic Treadmill Exercise

Brad Lambert, Kevin Shimkus, James Fluckey, Steven Riechman, Jessica Cardin, Stephen Crouse, FACSM. Texas A&M University, College Station, TX. (Sponsor: Stephen Crouse, FACSM)

(Purpose: To examine acute and chronic anabolic and general physiological responses to resistance training (RT), concurrent RT & land treadmill training (RT-LTM), and concurrent RT & aquatic treadmill training (RT-ATM).

Methods: Forty-seven untrained volunteers (34±23, 37±11yr, 29.6±4.6 kg·m⁻²) were tested for VO₂max, body comp (DEXA), and strength (7 exercises: 3 lower body, 4 upper body) prior to being randomized into 3 groups: RT, RT-LTM, & RT-ATM. All groups performed 12wks of RT2/wk, 3 x 12@60-80% 1RM). The RT-LTM & RT-ATM groups also performed 12wks of aerobic LTM or ATM training [2/wk (immediately post RT), 1/wk (in isolation), 60/85%VO₂max, 250±50 kcal/session]. Baseline tests were repeated after training. Additionally, 25 subjects volunteered for muscle biopsy prior to, and 24h-post acute exercise before and after training. Stable isotope labeling (1H2O, 70%, 3ml kg) was utilized to quantify 24h-post exercise myofibrillar fractional synthesis rates. Western blot analysis was used to analyze basal content of Akt and mTOR before and after training. A mixed model ANOVA was used to examine all independent variables with analysis of Akt and mTOR content repeated across training.

347