2023 ACSM President's Cup

Wednesday, May 31, 2023
2:00 – 4:30pm
Hyatt Regency Denver
Centennial F

CENTRAL STATES CHAPTER

Megan D. Jones
University of Arkansas
Faculty Mentor: Michelle Gray, PhD

Health Coaching Improves Body Composition in Males 45 – 75 After 1-year of Intervention

Megan D. Jones[1], Anthony Campitelli[1], Cody L. Diehl[1], Ray Urbina[1], Jordan Glenn[2], Kelsey Bryk[2], Josh Gills[3] Sally Paulson[4], Michelle Gray[1]
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Obesity, defined as having abnormal or excessive fat accumulation, is a disease affecting all populations worldwide and is linked to diabetes mellitus, hypertension, and cardiovascular disease (CVD). Diseases associated with excess body fat is estimated to cause nearly 3 million deaths a year worldwide with CVD being a main cause of premature death among men. It has been established that physical activity and regular exercise can decrease body fat and prevent chronic disease however, only 23% of the adult population meets recommendations for physical activity. Commonly reported barriers for not participating in physical activity include lack of motivation, social support, and knowledge. Health coaching is lifestyle intervention designed to give personalized programming to individuals to help remove these barriers. **PURPOSE:** The purpose of this study was to determine if 1 year of a health coaching intervention would improve body composition in males 45 -75 years. **METHODS:** Males aged 45 -75 years (N: 59: age: 61.4±8.6 years) were randomly assigned to a health coaching (HC) group (N=34) or a health education (HE) group (N=25) with body composition measurements taken at baseline and 1-year. Height and body weight (BW) were collected to determine body mass index (BMI). Dual-energy X-ray absorptiometry (DXA) was used to evaluate total body fat percentage (%BF), android fat (AF), gynoid fat (GF), and lean mass (LM). Individuals in HC received 1-on-1 health coaching every 6-8 weeks targeting improvements in self-selected lifestyle modifications. Those in HE received educational emails on lifestyle modifications once every 2 weeks. **RESULTS:** After 1-year of intervention, those in HC significantly decreased BMI (p<.05) by 2%, AF (p<.05) by 3%, and GF (p=.05) by 4% when compared to those in HE where increases of 1%, 2% and 1% were observed in these variables, respectively. Although not significant, those in HC also displayed decreases in BW (p=.06) by 2% and %BF (p=.08) by 3% when compared to those in HE who observed a 1% increase in BW and no change in %BF. No changes were observed for lean mass for either group. **CONCLUSION:** After 1-year of intervention, health coaching was effective in decreasing body weight and body fat in males aged 45 – 75 years when compared to males who received 1-year of health education.

GREATER NEW YORK CHAPTER

Lindsay Guzzetta
SUNY Plattsburgh
Faculty Mentor: Andreas Stamatis, PhD, FACSM

Strength Versus Deficit Educational-based Mental Toughness Interventions on Mental Health of Female Student-athletes

LINDSAY GUZZETTA1, ZACHARIAS PAPADAKIS2, GRANT B. MORGAN3, AND ANDREAS STAMATIS1
1Exercise and Nutrition Sciences, SUNY Plattsburgh, Plattsburgh, NY, 2Human Performance Laboratory, Barry University; Miami Shores, FL, 3Educational Psychology, Baylor University, Waco, TX

Educational-based psychological skills training (PST) is effective in terms of Mental Health (MH) outcomes. Mental toughness (MT), a Positive Psychology construct, is positively associated with MH. Sports training emphasizes working on the weaknesses of the athlete. Positive Psychology is rooted in strength-based interventions. In Applied Sports Positive Psychology, where females are underrepresented, the two approaches appear contradictory. PURPOSE: To examine the effects of deficit- versus strength-based MT interventions on MH levels of female collegiate athletes. METHODS: Out of the 161 female athletes of a SUNYAC institution, 95 participated. MH scores were collected via the Mental Health Continuum Short Form (MHC-SF) while MT scores were via the eight-item, Mental Toughness Index (MTI). Each MTI question (score range: 1-7) represents one key MT dimension (e.g., Q7: Buoyancy). We had previously created and successfully pilot-tested eight educational PST videos (one per key dimension). MT scores 1-3 were considered low (deficits) and 6-8 high (strengths). Participants were clustered into two groups. Power analysis yielded a sample size of 34. Group 1 (n=18) received intervention in the form of 1-3 videos based on their deficits, whereas Group 2 (n=18) on their strengths. Descriptive statistics, a two-sided t-test, and an analysis of variance (ANOVA) on the gain scores were produced on SPSS 28. RESULTS: Deficit Group MH scores: MPRE=43.2, SD=10.3; MPOST=51.9, SD=12.5. Strength Group MH scores: MPRE=52.2, SD=7.1; MPOST=52.9, SD=9.4. Gain scores: ΔDEFICIT=8.7, SD=11.7; ΔSTRENGTH=0.7, SD=7.2. T-test of deficit group: t(17)=-3.2, p=.01, d=0.84. T-test of strength group: t(17)=-.4, p=.68, d=0.09. ANOVA: F(1,34)=6.1, p=.19, ηp 2=.151. CONCLUSION: Both interventions were effective. Only the deficit-based intervention was significant and of large magnitude. The difference between the groups in the effect of the interventions was also significant and of large magnitude. This is the first study to examine the effectiveness of a telehealth education-based PST strength- versus deficit-based MT intervention on MH.

MID-ATLANTIC CHAPTER

Candace Longoria
Rutgers University
Faculty Mentor: Sara Campbell, PhD, FACSM

Resident Gut Microbiota Mediates Exercise Capacity and Tissue Metabolomes in Mice

Candace R. Longoria, Lee J. Kerkhof, Marko Oydanich, Xiaoyang Su, Eric Chiles, Stephen F. Vatner, Sara C. Campbell, FACSM. Rutgers, The State University of New Jersey

The Regulator of G Protein Signaling 14 knockout (RGS14 KO) mouse has a unique brown adipose tissue (BAT) mechanism mediating its phenotype of improved exercise performance. RGS14 KO mice showed a 51 ± 8% increase in running distance and a 44 ± 7% increase in work to exhaustion compared to wild type (WT). Three days after BAT transplantation from RGS14 KO mice to WT mice, RGS14 KO BAT donors lost their enhanced exercise capacity (EXC), whereas WT BAT recipients gained this EXC. We also found that RGS14 KO mice harbor a distinct gut microbiota and BAT metabolome, suggesting a gut-BAT-muscle axis that may regulate EXC. **PURPOSE:** 1) Examine EXC and tissue (BAT, quadriceps) metabolomes of RGS14 KO mice upon antibiotic treatment (ABX) and 2) begin establishing a gut-BAT-muscle axis that may regulate EXC. **METHODS:** Eight mice (n=4 RGS14 KO, n=4 WT) were used to examine EXC and identify predominant metabolites following ABX. Metabolomics used ultra-high performance liquid chromatography and mass spectrometry to evaluate untargeted metabolites in quadriceps and BAT samples. T-test was used to compare EXC before and after ABX. Two-tailed t-tests were used to compare WT and RGS14 KO metabolite means ($p<0.05$). MetaboAnalyst 5.0 was used to identify significant metabolite pathways and generate pathway plots. **RESULTS:** RGS14 KO running distance fell by 35 ± 7%, and work to exhaustion fell by 41 ± 7%, showing RGS14 KO mice lost their enhanced EXC after ABX. At baseline in RGS14 KO BAT, there were significant increases in the starch and sucrose metabolism pathway ($p=0.004$) and antimicrobial biosynthesis ($p=0.013$); however, no significant BAT pathways were detected after ABX. Further, 7 significant baseline quadriceps metabolite pathways were identified in RGS14 KO mice. There were no significant metabolites or pathways were identified in the quadriceps following ABX. **CONCLUSIONS:** RGS14 KO BAT responds to changes in resident microbiota that are beneficial to EXC and upon removal with ABX, EXC declines. Ablation of the gut microbiota alters metabolite pathways in RGS14 KO compared to baseline suggesting the absence of the resident microbiota changes metabolism. These data support the importance of the gut microbiota in tissue-specific metabolite production and how the gut microbiota may influence BAT and muscle function.

MIDWEST CHAPTER

Rafael A. Alamilla
Indiana University – Purdue University, Indianapolis (IUPUI)
Faculty Mentor: NiCole R. Keith, PhD, FACSM

Reducing Physical Activity Disparities Among Vulnerable Minorities – Methods and Preliminary Outcomes

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INTRODUCTION: Vulnerable minorities experience high rates of chronic disease. Physical Activity (PA) is an effective preventive behavior to mitigate multiple diseases. Vulnerable minorities have low PA participation. Finding ways to engage vulnerable minorities in PA is imperative.
PURPOSE: To describe preliminary data from a community-based wait-list pilot PA trial for vulnerable minorities.
METHODS: Forty-five participants from a Midwest urban community were randomized to an experimental (EXP: N = 23; 15 F) or control (CON: N = 22; 15 F) group. Baseline measures are height = 168.5 ± 9.1cm (EXP), and 167.9 ± 7.0cm (CON); weight = 95.8 ± 26.4kg (EXP) and 85.0 ± 19.3kg (CON), age = 39.9 ± 9.7y (EXP) and 48.8 ± 13.2y (CON). EXP participants were counseled to engage in regular PA ($\geq$4d/wk for $\geq$30 mins). EXP participants received a fitness center membership, trainer, and on-site monthly education to help them develop exercise identity and habit formation. The CON group could engage in PA if desired but did not have the same research resources. Both groups completed monthly surveys assessing exercise identity, social support, and habit formation. Baseline data included one week of moderate-to-vigorous PA (MVPA) and health-related fitness (measured by accelerometry and fitness tests, respectively).
RESULTS: Paired-samples T-test were used to make baseline comparisons. Study participants were 73.2% White, 67.4% employed full-time, 56.1% obtained a bachelor's degree or higher, and 32.0% earned >300% of the federal poverty level. MVPA was 127.9 ± 69.8 min/wk (EXP) and 174.7 ± 103.1 min/wk (CON). Other non-significant outcomes included body fat % (EXP: 37.1 ± 10.9%; CON: 32.9 ± 12.0%), 8ft-up-and-go time (EXP: 5.01 ± 0.8s; CON: 5.05 ± 1.10s), and 30s chair stand (EXP: 15.3 ± 6.5; CON: 17.5 ± 5.2), 30s seated arm curl (EXP: R =18.9 ± 5.1, L = 19.2 ± 5.1; CON: R = 21.2 ± 5.7, L = 20.1 ± 5.4). Chair sit-and-reach scores for right (EXP: -0.1 ± 8.3cm; CON: 1.0 ± 12.2cm, p = 0.003) and left (EXP: -0.3 ± 8.2cm; CON: 0.1 ± 11.4cm, p = 0.01) legs were different.
CONCLUSION: Data show baseline measures did not vary between groups and difficulty recruiting vulnerable minorities. Next steps include reporting final outcomes and developing refined recruitment methods.
Funding: Regenstrief Institute Health Equity Research Pilot Grant

NEW ENGLAND CHAPTER

Philip C. Woods
University of Massachusetts Amherst
Faculty Mentor: Mark S. Miller, PhD

Stretch Activation During Fatigue Improves Relative Force Production in Fast-Contracting Mouse Skeletal Muscle Fibers

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Stretch activation (F_{SA}) is the delayed increase in muscle fiber force following a rapid stretch and may improve skeletal muscle performance during activities with repetitive cyclical contractions, such as walking or running. Although historically considered minimal compared to cardiac or insect flight muscle, our recent work shows greater levels of inorganic phosphate (Pi) improves relative force production (F_{SA}/F_0, F_0 = calcium-activated force) in soleus skeletal muscle fibers. As peripheral muscular fatigue includes Pi accumulation, our results suggest F_{SA} improves force production in fatigued skeletal muscle. **PURPOSE:** To determine the effects of fatigue (low pH, high Pi) at low and high calcium (Ca^{2+}) concentrations on F_{SA} and F_{SA}/F_0 in myosin heavy chain (MHC) I, IIA, IIX, and IIB expressing fibers compared to control (high Ca^{2+} at resting levels of pH and Pi). **METHODS:** Single fibers from soleus and extensor digitorum longus muscles of female C57BL/6NJ mice were stretched 0.5% of their muscle length at normal and fatigued pH (7.0, 6.2) and Pi (5, 30 mM) as well as at high (pCa 4.5, pCa = -log([Ca^{2+}]) and low (pCa 5.1) Ca^{2+} concentrations. **RESULTS:** F_0 decreased from control to high calcium fatigue to low calcium fatigue conditions for all MHC isoforms, as expected. In MHC IIX and IIB fibers, F_{SA} occurred under all conditions and F_{SA}/F_0 increased from control (17-20%) to high calcium fatigue (32-35%, $p<0.001$) to low calcium fatigue (42-44%, $p<0.001$ vs both). In MHC IIA fibers, F_{SA}/F_0 increased similarly to MHC IIX and IIB fibers in control (14%) and high calcium fatigue (32%, $p<0.001$) but did not increase further under low calcium fatigue (35%, $p<0.001$ vs control). For MHC I fibers, F_{SA}/F_0 was low in control (4%) and was non-existent in high and low calcium fatigue, as no discernable F_{SA} was observed. **CONCLUSION:** Stretch activation is a significant modulator of force production under fatiguing conditions in fast-contracting muscle fibers. This response may be due to the reversal and re-occurrence of myosin power strokes in the presence of a stretch and Pi. This mechanism would reduce fatigue effects in locomotor activities, when the antagonistic muscle rapidly stretches the agonist muscle, if the delayed increase in force due to stretch activation happens when the agonist muscle is shortening.

NORTHLAND CHAPTER

Clayton Thompson
Augustana University
Faculty Mentor: None listed.

The Correlation Between Countermovement Jump Performance and Lead Leg Block

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PURPOSE: Countermovement jump (CMJ) performance is related to fastball velocity (FV). However, it is unknown what aspects of CMJ performance are represented in the baseball pitch which may underlie this relationship. Peak lead knee extension velocity (PLKNEV) has also been previously correlated to FV in collegiate and professional populations, but an overview examining high school and collegiate populations has not been performed.[1] Peak lead knee extension velocity may also be related to CMJ performance because PLKEV could be impacted by an athlete's lower body power. Therefore, the purpose of this study is to examine the relationship between CMJ performance and PLKEV in the baseball pitch in high school and collegiate baseball pitchers. **METHODS:** 54 male baseball players (23 high school and 31 collegiate) participated in this study. Data was evaluated retrospectively from participants who had participated in pitching motion capture sessions in a biomechanics lab under Institutional Review Board approval. The five fastest pitches thrown for each subject were selected for analysis where PLKEV was calculated and averaged across the five trials. Next, three CMJ trials were recorded on two Bertec force plates and metrics were averaged for analysis. The metric chosen to quantify CMJ performance was the modified reactive strength index (mRSI) which is a ratio of the jump height divided by the total time taken from the initiation of movement to the instant of take-off. Subjects were divided into high school and collegiate competition groups before any statistical analysis. Pearson correlation tests were then used to assess for linear relationships in the data for the different populations (α=0.05). **RESULTS:** Pearson correlation tests of the entire testing population revealed significant correlations (p = .005) between FV and PLKEV. Dividing the groups based on competition tiers—high school and collegiate—revealed one significant correlation between FV and PLKEV in the collegiate population. No other significant linear relationships were observed in the data (see Table 1 for non-significant results). **CONCLUSION**: The data indicates that no linear relationship exists between mRSI and PLKEV in high school and collegiate pitchers. This observation supports the idea that PLKEV is the result of better throwing mechanics and is not dependent on CMJ performance. Fastball velocity is created through a variety of physiological qualities and mechanical efficiency. However, it does not seem likely mRSI contributes to mechanical efficiency by influencing PLKEV. More research should be done to determine the relationship between specific biomechanical parameters of the pitching motion and CMJ performance, to determine how which aspects of CMJ performance influence FV.

Table 1. Results of the Pearson Correlation Tests for PLKEV r-value (p-value)

Group	mRSI	FV
High School	0.140 (0.523)	0.115 (0.603)
College	-0.044 (0.815)	**0.364 (0.044)**
Entire	0.080 (0.221)	**0.373 (0.005)**

Significant relationships are in **Bold**. Peak lead knee extension velocity (PLNEV). Fastball velocity (FV). Modified reactive strength index (mRSI).

1. Matsuo, T., Escamilla, R. F., Fleisig, G. S., Barrentine, S. W., & Andrews, J. R. (2001). Comparison of Kinematic and Temporal Parameters between Different Pitch Velocity Groups, Journal of Applied Biomechanics, 17(1), 1-13. Retrieved Nov 10, 2022, from https://journals.humankinetics.com/view/journals/jab/17/1/article-p1.xml

NORTHWEST CHAPTER

Emily Larson
University of Oregon
Faculty Mentors: Christopher T. Minson, PhD, FACSM & John R. Halliwill, PhD, FACSM

Systemic Cardiovascular and Baroreflex Support of Blood Pressure during Recovery from Passive Heat Stress

Emily A. Larson[1], Brendan W. Kaiser[1], Emma L. Reed[1], Brandon M. Gibson[1], Kieran S.S. Abbotts[1], W. Larry Kenney[2], FACSM, John R. Halliwill[1], FACSM, Christopher T. Minson[1], FACSM
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Heat stress may promote a distinct recovery period marked by sustained reductions in blood pressure. The mechanisms supporting blood pressure regulation in the post-heating recovery period are unknown. **PURPOSE:** Evaluate the systemic cardiovascular and carotid baroreflex support of blood pressure during recovery from whole-body, passive heat stress. **METHODS:** Mean body temperature (MBT), blood pressure, cardiac output (open circuit acetylene wash in), and heart rate were assessed in nine participants (5 F, 23±4 yrs) at baseline (PRE), at the end of 60 min of passive heating (HT), and for 120 min of normothermic recovery (REC). The neck pressure technique was used to assess carotid baroreflex control of heart rate, mean arterial pressure, and brachial vascular conductance. A repeated measures one-way ANOVA was used to compare PRE to HT and REC responses. Values are reported as mean ± SD. **RESULTS:** Heating increased MBT (PRE: 36.6±0.2 HT: 37.7±0.3°C, $p < 0.01$), cardiac output (PRE: 5.0±1.8 HT: 6.7±2.1 L·min^{-1}, $p < 0.01$), systemic vascular conductance (PRE: 60±20 HT: 86±26 mL·min^{-1}·mmHg $^{-1}$, $p < 0.01$) , and heart rate (PRE: 69±7 HT: 106±24 bpm, $p < 0.01$) and tended to reduce mean arterial pressure (PRE: 83±7 HT: 78±8 mmHg, $p = 0.07$). Cardiac (Δ heart rate PRE: +6±5 HT: +4±3 bpm, $p = 0.15$) and integrated (Δ mean arterial pressure PRE: +5±6 HT: +7±3 mmHg, $p = 0.14$) responses to neck pressure were maintained and the brachial vascular response was attenuated during HT (%Δ brachial vascular conductance PRE: -27±17 HT: -12±7%, $p < 0.01$). After 60 min REC, MBT remained elevated (36.9±0.2°C, $p < 0.01$) and mean arterial pressure returned to baseline levels (83±10 mmHg, $p = 0.74$), while cardiac output (5.2±1.7 L/min), systemic vascular conductance (62±16 mL·min^{-1}·mmHg^{-1}) and heart rate (73±9 bpm) did not differ from PRE (all $p > 0.05$). Cardiac (Δ heart rate +7±5, $p = 0.85$), integrated (Δ mean arterial pressure +6±5, $p = 0.81$), and vascular responses (%Δ brachial vascular conductance -28±14%, $p = 0.75$) to neck pressure did not differ from PRE after 60 min REC. After 120 min REC, MBT remained elevated (36.7±0.2°C, $p = 0.01$) and all other variables did not differ from PRE (all $p > 0.05$). **CONCLUSIONS**: Contrary to our hypothesis, blood pressure and carotid baroreflex control did not differ from baseline values throughout the post-heating recovery period.

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ROCKY MOUNTAIN CHAPTER

Darby Easterday
Colorado State University
Faculty Mentor: Daniel Lark, PhD

CD81 + Skeletal Muscle Derived Extracellular Vesicles Promote Adipogenesis In Vitro

Darby S. Easterday, Zackary J. Valenti, Thomas J. LaRocca and Daniel S. Lark

Metabolically active tissues such as skeletal muscle (SkM) and white adipose tissue (WAT) secrete extracellular vesicles (EVs) that may serve as biomarkers for metabolic health, further leveraging the interest in EVs as a therapeutic target to treat age-related metabolic diseases. Our lab demonstrated that SkM secretes ~100x more EVs than WAT ex-vivo, and SkM-derived EVs are able to reach the circulation in-vivo. More precisely, we find that SkM preferentially secretes EVs expressing the tetraspanin protein CD81 – a hallmark of exosomes. The function(s) of CD81 + EVs are poorly defined, but others have shown that deletion of CD81 in adipocyte progenitor cells (APCs) prevents the formation of "beige" adipocytes. Therefore, the purpose of this study was to test the hypothesis that SkM EVs promote beige adipogenesis in vitro. METHODS: The inguinal WAT (iWAT) and hind limb SkM tissue was collected from young (3 mo. 14 days) C57BL/6J mice of both sexes. APCs were isolated from the iWAT and SkM-derived CD81 + EVs were isolated from the SkM tissue. The APCs were cultured on Corning® BioCoat™ 8 well collagen-I coated culture slides in the presence or absence of CD81 + SkM EVs, and with or without beiging agonists (norepinephrine and rosiglitazone). Cells were formalin fixed then incubated with HCS LipidTOX™ Deep Red neutral lipid stain, a fluorescent lipid droplet stain, then imaged on an EVOS M7000 Cellular Imaging System to measure lipid droplet abundance and diameter. Differences between conditions were determined using a two-way ANOVA. RESULTS: Wells treated with exogenous SkM-derived CD81 + EVs contained more lipid droplets than APCs that were not treated with SkM-derived CD81 + EVs ($p=0.0287$). This finding is true in both the absence and presence of norepinephrine and rosiglitazone. There was a trend ($p=0.0586$) for a smaller lipid droplet diameter in APCs treated with SkM-derived CD81+ EVs compared to untreated controls. CONCLUSION: These data support our hypothesis that SkM-derived CD81+ EVs enhance adipogenesis in vitro. Our ongoing studies are looking at the function of CD81+ EVs in the context of metabolic disease if skeletal muscle naturally secretes CD81+ EVs targeted to adipose tissue to combat obesity.

SOUTHEAST CHAPTER

Paulo H. C. Mesquita
Auburn University
Faculty Mentor: Andreas N. Kavazis, PhD, FACSM

Effects of Resistance Training on Mitochondrial Adaptations to Subsequent Endurance Training

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BACKGROUND: While resistance training (RT) has long been appreciated for enhancing muscle mass and strength, it is widely underappreciated and under-utilized by endurance athletes and practitioners. However, several studies have shown beneficial effects of RT on endurance performance, which is usually linked to an improvement of running economy through neuromuscular adaptations. Emerging evidence highlights that RT may also promote positive mitochondrial adaptations, which could ultimately enhance endurance performance. Even though there are several studies investigating the effects of concurrent training, to the best of our knowledge, no study to date has investigated the effects of performing a block of RT-only before initiating endurance training (ET)-only. Therefore, the goal of the present study was to investigate the effects of RT on adaptations to subsequent ET. METHODS: 23 young untrained males were recruited and divided into two groups: 1) ET-only (n=12), which performed 7 weeks of high-intensity interval training; 2) RT+ET (n=11), which performed 7 weeks of RT twice weekly before initiating 7 weeks of the same endurance training performed by ET-only. All participants performed a maximal oxygen uptake test on a treadmill for determination of VO_{2max} and speed at onset of blood lactate accumulation (OBLA) before (PRE) and after (POST) ET. Furthermore, muscle biopsies were obtained from participants' vastus lateralis at PRE and POST and used to determine the levels of proteins involved in mitochondrial remodeling. RESULTS: Both groups significantly increased VO_{2max} and speed at OBLA ($p<0.001$) similarly. Protein levels of mitochondrial complexes I, II, and III, and Mitofusin 2 (Mfn2) increased, while Parkin decreased similarly in both groups (main effect of time, $p<0.05$). Significant interactions were found for complex IV (ET-only (PRE < POST), $p=0.039$) and PGC-1α (ETonly-POST > RT+ET-POST, $p=0.001$). CONCLUSIONS: Our results suggest that performing RT prior to ET had no additional benefit on mitochondrial and endurance performance adaptations to ET in young untrained males. Participant compensation as well as select reagents related to analyses presented herein were funded by a grant awarded by National Strength and Conditioning Association Foundation to Paulo H.C. Mesquita.

SOUTHWEST CHAMPTER

Jeremy Ducharme
University of New Mexico
Faculty Sponsor: Michael Deyhle, PhD

Stimulated Muscle Contractions Regulate Membrane-Bound and Soluble TLR4 to Prevent LPS-Induced Signaling and Myotube Atrophy in Skeletal Muscle Cells

Jeremy B. Ducharme, Zachary J. Fennel, Zachary J. McKenna, Quint N. Berkemeier, & Michael R. Deyhle

Toll-like receptor 4 (TLR4) activation by lipopolysaccharides (LPS) contributes to chronic inflammation and causes upregulation of muscle atrophy signaling pathways. Exercise can suppress LPS/TLR4 axis activation by reducing the expression of TLR4 on immune cells. It is unknown how this regulation occurs, and it is not clear how exercise affects TLR4 on skeletal muscle. **PURPOSE:** To uncover the nature and mechanisms by which exercise affects TLR4 expression and intracellular signaling using cell culture models and human experiments. **METHODS:** C2C12 myotubes were subjected to electrical pulse stimulation (EPS) with and without subsequent treatment with 500 ng/mL lipopolysaccharide (LPS) along with corresponding control conditions. To investigate the effect of muscle contraction on the regulation of TLR4 in-vivo, we analyzed PBMC and serum samples from eight recreationally active men that completed 60-minutes of cycling at a moderate intensity (65% of VO_2max). **RESULTS:** In-vitro, LPS decreased membrane-bound TLR4, increased TLR4 signaling (decreased inhibitor of κBα), and induced myotube atrophy. However, stimulated muscle contractions decreased membrane-bound TLR4, increased soluble TLR4 (sTLR4), and prevented LPS-induced signaling and myotube atrophy. In human participants, a single bout of moderate-intensity exercise decreased membrane-bound TLR4 on PBMCs and increased serum-borne sTLR4. **CONCLUSION:** These experiments support exercise may exert a novel anti-catabolic/ anti-inflammatory effect by increasing sTLR4 and decreasing TLR4 expressed on the muscle membrane. These results could help improve interventions for conditions associated with TLR4-mediated inflammation and muscle atrophy, such as diabetes, sarcopenia, and cancer cachexia.

TEXAS CHAPTER

Frank Wojan
The University of Texas at Austin
Faculty Mentor: Sophie Lalande, PhD

Impaired Erythropoietin Response to a Single Session of Intermittent Hypoxia in Patients with Type 2 Diabetes

FRANK WOJAN, STEN STRAY-GUNDERSEN, JIAHUI ZHAO, & SOPHIE LALANDE
Clinical Exercise Physiology Laboratory; Department of Kinesiology and Health Education; The University of Texas at Austin; Austin, TX

Patients with type 2 diabetes (T2D) exhibit, on average, a 20% decline in maximal oxygen consumption when compared to healthy adults. Hemoglobin mass strongly correlates to maximal oxygen consumption. A reduced total blood volume has been observed in patients with T2D, suggesting that a reduced hemoglobin mass contributes to the decreased maximal oxygen consumption in this population. Hypoxia stimulates the release of erythropoietin (EPO), the hormone regulating red blood cell production. We previously showed that intermittent hypoxia, consisting of alternating short bouts of breathing hypoxic and normoxic air, increases EPO levels. **PURPOSE**: To determine the effect of a single session of intermittent hypoxia on serum EPO levels and hemoglobin mass in patients with T2D. We hypothesized that a single session of intermittent hypoxia would raise serum EPO levels and lead to an increase in hemoglobin mass in patients with T2D. **METHODS**: Ten patients with T2D (4 women, age: 53 ± 10 years, body mass index: 36.2 ± 8.5 kg/m^2, HbA1c: 7.2 ± 1.2%) were exposed to an intermittent hypoxia protocol consisting of eight 4-min cycles at a targeted oxygen saturation of 80% interspersed with normoxic cycles to resaturation. Air was made hypoxic by titrating nitrogen into a breathing circuit. Pulmonary gas exchange, oxygen saturation, and hemodynamics were continuously measured throughout the protocol. EPO levels were measured before and 4.5 hours after the beginning of the protocol. Hemoglobin mass was assessed via carbon monoxide rebreathing before and seven days following intermittent hypoxia. **RESULTS**: Intermittent hypoxia lowered oxygen saturation (97 ± 2 to 81 ± 2%, $p<0.01$), which resulted from a lower fraction of inspired oxygen (20.8 ± 0.1 to 11.1 ± 1.0%, $p<0.01$). There was no significant change in EPO levels following exposure to intermittent hypoxia (11.9 ± 5.3 to 12.1 ± 4.3 mU/ml, $p=0.83$). There was also no change in hemoglobin mass in response to intermittent hypoxia (864 ± 152 to 850 ± 150 g, $p=0.64$). Intermittent hypoxia did not affect mean arterial pressure (94 ± 5 to 97 ± 7 mmHg, $p=0.18$) but increased cardiac output (9.1 ± 2.7 to 9.8 ± 2.8 L/min, $p=0.03$) due to an increase in heart rate (78 ± 9 to 84 ± 10 bpm, $p<0.01$). **CONCLUSION**: A single session of intermittent hypoxia did not increase serum EPO levels or hemoglobin mass in patients with T2D. These findings suggest an impaired EPO response to decreased oxygen levels in patients with T2D, which may contribute to the reduced hemoglobin mass and total blood volume observed in this population.