REPLACEMENT SPEAKER AND PRESENTATION TITLE CHANGE

Symposia VII—Friday, November 3 at 8:30—10:45 AM

9:30 AM 20.3 Development of Distinct Adipocyte Populations: Can Exercise Play a Role?  
Kathleen Gavin, Univ. of Colorado Sch. of Med.

MEALS AT THE MEETING

The meeting registration includes the following for each attendee: opening reception, lunch on Thursday and Friday, and dinner on Friday evening. Dinner tickets are needed for the dinner on Friday evening. Get your tickets at the registration desk before 12:00 Noon on Thursday, November 3. Sorry, no refunds on meals that are missed or not attended.

There are no coffee breaks for this meeting. There is a coffee shop in the lobby area of the hotel for you to purchase drinks.

GUIDE TO EXHIBITS

Don’t forget to stop by our exhibit booths during the meeting. Exhibits are located in the Atrium Lobby (2nd floor) by the posters.

Booth #114  
Aurora Scientific, Inc.  

Booth #116  
AEI Technologies, Inc.  
AEI Technologies offers three levels of Metabolic Carts providing cost effectiveness verses budgets. Carts are suitable for teaching, research, and clinical applications as well as for resting or exercise physiology. Our metabolic systems are easily upgraded for EKG, Cardiac Output, and Breath-by-Breath monitoring to accurately measure energy metabolism by indirect calorimetry. www.aeitechnologies.com.

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Please note that photography and video are not permitted in the session rooms or at the posters
Poster Board #112
Exercise Increases Resting-state Brain Activity in Parkinson’s Disease
K. H. Wood1,2, N. A. Kelly2,3, J. B. Allendorfer1,2, M. P. Ford4, C. S. Bickel2,5, J. Marstrander6, A. W. Amara1,2, T. Anthony4, M. M. Bamman2,3, and F. M. Skidmore1,2
1University of Alabama at Birmingham (UAB), Department of Neurology, 2UAB Center for Exercise Medicine, 3UAB, Department of Cell, Developmental, and Integrative Biology, 4Department of Physical Therapy, Samford University, 5UAB, Department Physical Therapy, 6UAB, Department of Engineering.

Purpose: Parkinson’s disease (PD) is a multisystem disorder that results in progressive deterioration of multiple structures in the brain/nervous system. As a result, PD presents clinically with both motor and non-motor deficits. The positive effect of exercise on the human brain, even among older adults, is well established, as are the beneficial effects of exercise on motor and non-motor symptoms in PD. Since physical exercise alters resting-state brain activity in non-PD populations and improves PD symptoms, we assessed the effects of exercise on resting-state brain activity in individuals with PD. The present study was designed to better understand the effects of exercise on resting-state brain activity in PD and potentially elucidate mechanisms behind common symptomatic and functional improvements seen in response to exercise training in PD patients. Methods: Individuals with PD underwent 16 weeks of high-intensity exercise training. Resting-state fMRI was performed post-training (after the 16th week) 1 hour before a single bout of exercise and again 1 hour after the exercise session. Amplitude of the low-frequency fluctuation (ALFF) signal was used to evaluate the effect of exercise on brain activity. Results: Increased resting-state brain activity was observed within the left ventromedial prefrontal cortex (PFC), right dorsolateral PFC, and bilaterally within the substantia nigra (SN). Conclusions: Given the role of the SN and PFC in motor and non-motor symptoms in PD, increases in brain activity within these regions, if repeated frequently over time (i.e., exercise training), may serve as a potential mechanism underlying exercise-induced PD-specific clinical benefits. Future directions: Assess chronic changes in regional brain activity due to long-term exercise training compared to non-PD participants. Investigate changes in resting-state functional connectivity linked to clinical and behavioral outcomes in PD. Acknowledgements: UAB School of Medicine, UAB Center for Exercise Medicine, National Institutes of Health [1T32 HD071866 (KHW, NAK), 5K23NS083620 (FMS) K23NS080912 (AWA)], and the UAB Center for Clinical and Translational Science [UL1 TR000165].

Poster Board #113
Acute Mental Stress and its Impact on Systemic Vascular Endothelial Function in Obese Adults
Julien V. Brugniaux1,2, Danielle Hodson1, Christopher J. Marley1 and Damian M. Bailey1
1Neurovascular Research Laboratory, Faculty of Life Science and Education, University of South Wales, Wales, UK. 2School of Science & Health, Western Sydney University, NSW, Australia.

Background and aims: Everyday life stressors such as acute mental stress can result in transient impairments in vascular endothelial function (2). Similarly, obesity is known to impair arterial smooth muscle function (1). However, their combined effect remains unknown. To investigate this, we determined to what extent a battery of neuropsychometric tests further compounded vascular endothelial dysfunction in obese adults related to non-obese controls. Methods: Eight obese [26 (mean) ± 8 (SD) years old; BMI = 33 ± 4] and 8 non-obese [21 ± 4 years old; BMI = 25 ± 3] male participants were recruited. Brachial artery flow-mediated dilatation (FMD, Acuson P50, Siemens) was measured 1 hour before and immediately following a standardised battery of neuropsychometric tests. Following a 1-min baseline, the occluding cuff (distal) was inflated (>250mmHg) for 5 min and a subsequently released for 5-min into recovery. Brachial artery diameter and flow were recorded continuously throughout the test. Baseline data correspond to the 1-min average pre cuff inflation and peak diameter was measured as the average of the highest 3 values recorded. Data were analysed using automated edge-detection and wall-tracking software (Brachial Tools, Medical Imaging Application). Following confirmation of distribution normality (Shapiro-Wilk W tests), data were analysed using a 2-way (Time x Group) repeated measures ANOVA. Data are expressed as mean ± SD with significance set at P < 0.05. Results: Psychometric stress was shown to impair FMD (Pre: 5.3 ± 1.4% vs. Post: 4.4 ± 1.4%, P < 0.05). The obese group also displayed a lower FMD than their non-obese peers during both FMD tests (P<0.05). The obese further exhibited a decrease in baseline flow from 0.11 ± 0.02 m/sec before to 0.08 ± 0.02 m/sec after acute mental stress (P<0.05). Conclusion: The present results confirmed that acute mental stress impairs systemic vascular endothelial function. Contrary to our original hypothesis, this impairment was not further

Poster Board #114

**Metabolic Benefits of Endurance Exercise Training in Pregnancy for Females Born Small on High Fat Diet**

Glenn D. Wadley, Dayana Mahizir, Kristina Anevksa, Andrew J. Jefferies, Karen M. Moritz & Mary E. Wlodek

1 Institute for Physical Activity and Nutrition, School of Exercise and Nutrition Sciences, Deakin University, Burwood, Victoria, Australia, 2 Department of Physiology, The University of Melbourne, Parkville, Victoria, Australia and 3 School of Biomedical Sciences, University of Queensland, St. Lucia, Queensland, Australia.

Intrauterine growth restriction programs adult metabolic disease, which is exacerbated with “second hits” such as pregnancy and obesity in females born small. Importantly, exercise is reported to have a positive effect in those born small. This study determined if a high fat diet (HFD) exacerbates metabolic dysfunction in pregnant females born small and whether exercise before and during pregnancy is more beneficial in preventing these complications than exercise during pregnancy alone. The study was approved by the University of Melbourne ethics committee and conformed to the APS Guiding Principles for the Care and Use of Animals. Uteroplacental insufficiency resulting in growth restriction was induced by bilateral uterine vessel ligation (Restricted) or sham (Control) surgery on embryonic day 18 (E18) in Wistar-Kyoto rats. Female offspring consumed a Chow or HFD (43% kcal from fat) from 5 weeks and were mated at 20 weeks. Female rats were exercised on treadmills for 4 weeks before pregnancy and throughout pregnancy or during the last two thirds of pregnancy only. A glucose tolerance test was performed at E18 and plasma, pancreas and skeletal muscle were collected at E20. Control and Restricted rats exposed to a HFD were heavier with higher plasma leptin concentrations compared to Chow-fed rats irrespective of exercise interventions. HFD exacerbated the pre-existing glucose intolerance in Restricted female rats and importantly exercise before and during pregnancy prevented the development of glucose intolerance (p<0.05). These females on a HFD who exercised before and during pregnancy had increased pancreatic β-cell and islet mass (p<0.05). Compared to Control counterparts, exercise before and during pregnancy reduced intramuscular triglyceride in Restricted Chow-fed females (p<0.05). However, no difference in mitochondrial biogenesis markers (peroxisome proliferator-activated receptor gamma coactivator 1-α and citrate synthase activity) were detected across the groups. Metabolic dysfunction was not impacted by exercise in pregnancy alone. In conclusion, females born small are at a greater risk of glucose intolerance when exposed to a HFD and this was prevented by the lifestyle intervention of exercise potentially due to improved β-cell mass but muscle mitochondrial biogenesis was not affected. This study also suggests that exercise prior to and during pregnancy is more beneficial in preventing metabolic disease than exercise during pregnancy only.

Poster Board #115

**Long-term Cardiac Programming by Short-term Juvenile Exercise Training**

Glenn D. Wadley, Mary E. Wlodek, Mary J. Black, Aaron P. Russell, Paul. Soeding and Yasmin. Asif

1 Institute for Physical Activity and Nutrition, School of Exercise and Nutrition Sciences, Deakin University, Burwood, Victoria, Australia, 2 Department of Physiology, The University of Melbourne, Parkville, Victoria, Australia, 3 Department of Anatomy & Developmental Biology, Monash University, Clayton, Melbourne, Victoria, Australia and 4 Department of Pharmacology, The University of Melbourne, Parkville, Victoria, Australia.

Background: Wadley et al. (2016) recently reported a sustained 10% increase in the heart mass of adult rats who underwent a few weeks of endurance training in juvenile life. Therefore, the aim of this study was to investigate if short-term endurance training during juvenile life (5-9 wks of age) results in sustained improvements in adult heart structure, function, and morphology. Methods: The study was approved by the University of Melbourne ethics committee and conformed to the APS Guiding Principles for the Care and Use of Animals. Training was conducted on a motorized treadmill 1h/day, 5 days/wk in either juvenile (training from 5-9 wks of age), or adult (20-24 wks of age) male Wistar-Kyoto rats, and compared to sedentary rats. Cardiac structure and function were assessed at 9 and 24 wks of age using transthoracic echocardiography with hearts being perfusion-fixed for cardiomyocyte analysis or frozen in liquid nitrogen for miRNA analysis by PCR. Results: When compared to 24 wk old sedentary rats, juvenile exercise training led to long-term increases in left ventricle (LV) mass (+18%; P<0.05), wall thickness
(+11%; P<0.05), the longitudinal area (LA) of binucleated cardiomyocytes (P<0.05), cardiomyocyte number (+36%, P<0.05), and doubled the proportion of mononucleated cardiomyocytes (from 7% to 14%; P<0.05). Adult exercise training also increased LV mass (+11%; P<0.05), wall thickness (+6%; P<0.05) and the LA of binucleated cardiomyocytes (P<0.05), despite no change in cardiomyocyte number or the proportion of mono and binucleated cardiomyocytes. Resting cardiac function, LV chamber dimensions and fibrosis levels were not altered by juvenile or adult exercise training (P>0.05). At 9 wks of age juvenile exercise significantly reduced the expression of miR-1 and increased miR-133b. However, at 24 wks of age miR-1 expression was unaltered following juvenile exercise but significantly increased following adult exercise. At 24 wks of age, adult exercise significantly reduced the expression of miR-208b and miR-222. **Conclusion:** The juvenile period of life is a stage of developmental plasticity that is amenable to long-term, beneficial cardiac programming by short-term endurance training. **References:** Wadley, Laker, McConnell & Wlodek. Endurance training in early life results in long-term programming of heart mass in rats. 4(4): 1-14, 2016. Physiological Reports. Supported by the Institute for Physical Activity and Nutrition (IPAN), Deakin University.

**Poster Board #116**

**Physiological Responses to Treadmill Running in Trained College Aged and Master Level Female Runners**


Central Washington Univ., Dept. of Health Sciences, Ellensburg, WA 98926, Ball State Univ., Human Performance Lab., Muncie, IN. 47306, Central Washington Univ., Dept. of Physical Education, School of Health and Movement Studies, Ellensburg, WA. 98926, Good Samaritan Hosp., Sioux Falls, SD 57108, High Point Univ., High Point, NC 27268, Univ. of Washington, School of Medicine, Dept. of Radiology, Seattle, WA, Central Oregon Community Coll., Bend, OR 97703.

There is a paucity of research on key performance parameters such as maximal oxygen uptake (VO₂ max) and running economy (RE) in highly trained young and masters female runners. Thus, the purpose of this study was to investigate physiological responses in similarly trained, competitive young and masters female runners. As a secondary intent, stride length (SL) and rate (SR) were measured to assess running kinematics. Thirty young (Y; 20.1±0.4 yr) and fourteen masters (M; 45.6±1.1 yr) trained female runners performed a series of submax and one max treadmill run to volitional exhaustion. Metabolic (open-circuit spirometry), HR (telemetry) and blood lactate (finger stick) responses were monitored. SR was measured by timing 50 stride cycles over the last minute of each submaximal effort while SL was computed by dividing running velocity by SR. A similar VO₂ max was noted between groups (2.95±0.11 (M) vs. 2.92±0.10 l min⁻¹ (Y); 49.9±0.9 (M) vs. 48.8±1.0 ml kg⁻¹ min⁻¹ (Y); P>0.05) while a lower HR max (174±3 vs. 193±2 bpm) and peak lactate (8.3±0.4 vs. 10.7±0.9 mM) was found in the masters runners (P<0.05). Maximal O₂ pulse, a surrogate of maximal stroke volume, was greater in the masters runners (~11%; P<0.05). VO₂ (relative to body wt.), blood lactate, HR, SL and SR at common submaximal running speeds was similar between M and Y. Aerobic demand relative to distance covered (ml O₂·kg⁻¹·km⁻¹) at given metabolic efforts (70 to 100% VO₂ max) was independent of running velocity and similar between M and Y (P>0.05). In conclusion, highly trained masters female runners have the same maximal aerobic power and running economy as their younger counterparts of 25 years. Furthermore, both groups displayed the same kinematic profile as noted by similar SL and SR values regardless of submax running velocity. Our findings indirectly suggest that performing a high level of training consistently into the fifth decade of life results in a greater stroke volume to offset the decline in HR max. Lastly, the fitness level of our groups, especially the masters runners, is noteworthy when comparing their VO₂ max to values obtained from cardiopulmonary maximal exercise testing in females (Kaminsky et al. (2015)). Our young and masters runners rank at ~85th and above the 95th percentile, respectively, in measured VO₂ max.

**Poster Board #117**

**Decreased HETE-12 and -15 are Associated with Glucose Regulation Following High Intensity Exercise in People with Prediabetes**

Steven K. Malin1,2,3,4, Corey A. Rynders5, Judy Y. Weltman3, Eugene J. Barrett2,4, Jerry Nadler6, and Arthur Weltman1,2,3

1Department of Kinesiology, University of Virginia, Charlottesville VA, 2Division of Endocrinology and Metabolism, University of Virginia, Charlottesville VA, 3Exercise Physiology Core Laboratory, University of Virginia, Charlottesville VA, 4Robert M. Berne Cardiovascular Research Center, University of Virginia,
Energy Substrate Oxidation During Cycling and Running in the Cold and Heat
Dominique D. Gagnon, Lina Perrier, Michelle Laurence, Sandra Dorman, Céline Larivière, Olivier Serresse
Introduction: The examination of energy substrate oxidation under thermal stress has previously demonstrated an increase in lipid utilization during walking and running in the cold (Gagnon et al. 2013) but not during cycling (Layden et al. 2002) at fixed intensities. No exercise thermophysiology study has comprehensively examined metabolic differences between exercise modalities nor has addressed the fundamental metabolic changes across exercise intensities. The aim of the study was to examine the influence of environmental temperature on energy substrate oxidation in a cold and warm environment across a range of exercise intensities during running and cycling. Methods: Nine male subjects (21.2±1.4 yrs, 84.0±14.6 kg, 1.76±0.07 m, 10.6±5.7 %BF) dressed in shorts and t-shirt, completed 4 trials, one week apart, during which they performed an incremental maximal oxygen consumption (VO2max) test on a cycle ergometer or treadmill in a cold (4.6°C) or warm environment (34.1°C) which was designed to include 6 to 15 three-min stages. VO2max, maximal fat oxidation rate (MFO), and exercise intensity where MFO occurs (Fat max) were assessed. Data was analyzed using a two-way ANOVA (factors: temperature, exercise modality) for repeated measures with significance at p<0.05. Results: VO2max in the treadmill condition was 47.2 ± 5.9 mlO2 • kg⁻¹ • min⁻¹ and 39 ± 6.8 mlO2 • kg⁻¹ • min⁻¹ in cycling. MFO was greater in the cold vs. warm (0.55 g•min⁻¹ vs. 0.38 g•min⁻¹; p=0.016) as well as during running vs. cycling (0.57 g•min⁻¹ vs. 0.37 g•min⁻¹; p=0.028). Analyses further revealed that environmental temperature had no influence on MFO during cycling (p=0.071). Fat max was also greater in the cold vs. warm (60% vs. 37% of VO2max; p=0.004) with no factorial interactions within groups (p=0.451). Conclusions: We examined thermal influences on energy metabolism across a wide range of intensities during running and cycling. We demonstrated that running in a cold environment increases maximal fat oxidation rate which peaked at a higher exercise intensity compared to a warm environment. These results further validate the implication of environmental temperature on energy metabolism during exercise and could serve as a reference for training designs. This study was supported by an NSERC Discover Grant and a Laurentian University Research Grant. References: Gagnon DD, et al. (2013). Front. Physiol. 4 (99), Layden et al. (2002). Med. Sci. Sports Ex. 34 (5), 774-779.

Poster Board #120
Randomized, Four-Arm, Dose-Response Clinical Trial to Optimize Resistance Exercise Training for Older Adults with Age-Related Muscle Atrophy
Brandon M Roberts, PhD,1,2 Michael J Stec, PhD,1,2 Anna Thalacker-Mercer, PhD,1,2 David L Mayhew, MD, PhD,1,2 Neil A Kelly, PhD,1,2 S Craig Tuggle, MA,1,2 Edward K Merritt, PhD,1,2 Cynthia J Brown, MD, MSPH,1,3,6 Samuel T Windham, MD,1,2 Louis J Dell’Italia, MD, MD,1,5,7 C Scott Bickel, PT, PhD,1,2 Kristina M Vaughn, DPT,1,2 Irina Isakova-Donahue, PhD,1,2 Gina Many, PhD,1,2 and Marcas M Bamman, PhD1,2,6
1UAB Center for Exercise Medicine, University of Alabama at Birmingham, Birmingham, AL 35294, 2Department of Cell, Developmental, and Integrative Biology, University of Alabama at Birmingham, Birmingham, AL 35294, 3Division of Gerontology, Geriatrics, and Palliative Care, Department of Medicine, University of Alabama at Birmingham, Birmingham, AL 35294, 4Department of Surgery, University of Alabama at Birmingham, Birmingham, AL 35294, 5Division of Cardiovascular Disease, Department of Medicine, University of Alabama at Birmingham, Birmingham, AL 35294, 6Geriatric Research, Education, and Clinical Center, Veterans’ Affairs Affairs Medical Center, Birmingham, AL 35233, 7Research Service, Veterans’ Affairs Affairs Medical Center, Birmingham, AL 35233, 8Department of Physical Therapy, University of Alabama at Birmingham, Birmingham, AL 35294
Background. There are numerous consequences of age-related muscle atrophy including reduced muscular strength, power, and mobility; increased risk of falls, disability, and metabolic disease; and compromised immune function. Ultimately, aging muscle atrophy results from a loss of myofibers and atrophy of the remaining type II myofibers. The purpose of this trial (NCT02442479) was to titrate the dose of resistance training (RT) in older adults in an effort to maximize muscle regrowth and gains in muscle function. The goal was to identify a prescription that sufficiently loaded muscle to promote myogenic processes, while allowing adequate recovery between bouts to limit muscle inflammation signaling. Methods. The design was a randomized, four-arm efficacy trial in which four, distinct RT prescriptions varying in intensity, frequency, and contraction mode/rate were evaluated: (1) high-resistance concentric-eccentric training (H) 3 d/wk (HHH); (2) H training 2 d/wk (HH); (3) 3 d/wk mixed model consisting of H training 2 d/wk separated by 1 bout of low-resistance, high-velocity, concentric only (L) training (HLH); and (4) 2 d/wk mixed model consisting of H training 1 d/wk and L training 1 d/wk (HL). We hypothesized the prescriptions involving 2 d/wk H training (HH, HLH) would yield greater muscle regrowth than HHH (insufficient recovery / inflammatory burden) and HL (insufficient H frequency) and further, the HLH prescription would yield overall superior gains in muscle function and mobility indices due to the insertion of 1 d/wk L training. Results: The HLH prescription maximized gains in thigh muscle mass and total body lean mass. HLH also showed the greatest gains in knee extension maximum isometric strength, and reduced cardiorespiratory demand during.
steady-state walking. HHH was the only prescription that led to increased muscle expression of pro-inflammatory cytokine receptors and this was associated with a lesser gain in TMM and total body lean mass compared to HLH. **Conclusions.** Considering the results in total, it appears the HLH prescription offers some distinct advantages over the other doses, while the HL program is subpar for many of the outcomes studied here. We can conclude from this randomized dose-response trial that older adults benefit greatly from 2 d/wk high-intensity RT, and may further benefit from inserting an additional weekly bout of low-load, explosive RT. **Funding Sources:** P2CHD086851 (Bamman), T32HD071866-04 (BR)

**Poster Board #121**

**Adverse Ultrastructural Remodeling and Mitochondria Dysfunction of Skeletal Muscle in Heart Failure**

Michael Rogowski1, Pamela Powell2, Lianwu Fu3, Jim Collawn1, Marcas Bamman4, and Louis Dell’Italia1,2.

1. School of Medicine, Cardiovascular Disease, University of Alabama Birmingham, Birmingham AL, 2. Department of Veterans Affairs Hospital, Birmingham AL, 3. School of Medicine, Cell Development and Integrative Biology, University of Alabama Birmingham, Birmingham AL, 4. School of Medicine, Center for Exercise Medicine, University of Alabama Birmingham, Birmingham AL

Though a secondary feature of chronic heart failure (HF), significant muscle loss is a profound morbidity that directly results in decreased capacity to complete activities of daily living, contributing to loss of independence and decreased quality of life. Despite its frequency as a clinical feature of HF, the pathological process and underlying mechanisms of skeletal muscle (SM) wasting in this disease remain elusive. To this end, we sought to examine the specific pathology at the ultrastructure level in order to better characterize the degradation of SM in HF. Using an in vivo rat model, we induced volume overload HF through the creation of an aorto-caval fistula. The descending aorta is punctured through the vena-cava, allowing arterial flow into the vena-cava resulting in dramatic increase in venous return to the heart, causing volume overload and subsequent HF over 12 weeks. Surgical shams that omit the aorto-caval puncture serve as controls. Upon sacrifice, gastrocs were extracted and fixed appropriately for immunohistochemical or transmission electron microscopy analysis, as well as flash frozen for mitochondria analysis. Electron microscopy of SM with HF revealed extensive disruption of sarcomere morphology, increased glycogen deposition, and aberrant mitochondria organization and morphology. SM fiber cross sectional area was significantly decreased (18 %) in HF. Decreased protein expression levels of Drp1, Hsp60, and LC3II suggest diminished mitophagy in SM due to HF, contributing to mitochondria dysfunction. Mitochondria function was assessed for Complex I, Complex IV, and citrate synthase activity in skeletal muscle that trended towards significant decrease in Complex IV (p=.10) in total homogenate fraction in HF. Cardiomyocyte degeneration in volume overload HF is induced by excess chronic mechanical stretch, yet we observe similar morphological degeneration in skeletal myocytes as well. This suggests that the physiologic environment induced by the failing heart condition results in an overlapping pathological degradation in the skeletal muscle. In addition to atrophy, this adverse cytoskeletal remodeling also appears to negatively impact mitophagy and mitochondrial function in skeletal muscle. Further study is needed to resolve the mechanisms by which cellular processes initiate ultrastructure degradation skeletal muscle during HF. Funding by NIH 1T32HD071866 and P01 HL051952.

**Poster Board #122**

**Mitochondrial Protein Content is Enhanced by Lifelong Physical Activity**


1. School of Sport, Exercise and Rehabilitation Sciences, University of Birmingham. 2. Centre of Human and Aerospace Physiological Sciences, Kings College London. 3. Division of Medical Sciences and Graduate Entry Medicine, University of Nottingham.* Joint corresponding authors.

Sedentary ageing is associated with progressive declines in muscle mass, strength and oxidative capacity. Whilst the age-associated reduction in muscle mass is well characterised, less is known regarding the mechanisms responsible for the decline in oxidative capacity. The purpose of the current study was therefore to (1) investigate the effect of ageing on mitochondrial protein content, and (2) examine whether life-long physical activity in the older adult (i.e. masters athletes) results in enhanced mitochondrial protein content compared to age-matched old sedentary and young active adults. Muscle biopsies were obtained from the vastus lateralis of 8 young (YG: 22 ± 3 years, 24 ± 3 kg/m²) and 8 old (SED: 67 ± 2 years, 27 ± 3 kg/m²) adults not previously engaged in a formal exercise training programme and 8 master athlete (MST: 65 ± 5 years, 26 ± 2 kg/m²) male adults. Immunoblotting was used to assess mitochondrial and mitochondrial-related protein abundance in skeletal muscle. Comparison between YG and SED did not reveal any age-associated declines in mitochondrial protein content. In contrast, the protein content of
electron transport chain (ETC) complexes I, II and IV were significantly greater in MST compared to both YG and SED (p<.001). In parallel, protein content of PGC-1α, a purported mediator of a mitochondrial biogenesis was 3- and 5- fold greater in MST compared to YG and SED respectively (p<.001). Further, the abundance of proteins related to fat and carbohydrate metabolism were greater in MST. VLCAD protein content was greater in MST compared to SED (p<.05), whilst PDH was increased in MST compared to both YG and SED (p<.05). Based on our results, we conclude that ageing is not associated with a decline in protein content of ETC I, II, IV and PGC-1α. Further, we show that mitochondrial related protein content was substantially increased in master athletes, suggesting that exercise can maintain mitochondrial content into later life.

Poster Board #123
Effects of Heightened Inflammation on Arginine Transporters and Metabolic Enzyme Expression in Differentiated Primary Human Progenitor Cells
Thalacker-Mercer A., Blum J., Roman H., Riddle E., Gupta D.
Cornell University, Division of Nutritional Sciences, Ithaca, NY
Background: The underlying etiology of skeletal muscle atrophy with aging (i.e. sarcopenia) is multifactorial and appropriate therapies are ambiguous. Recently, our lab identified Arginine and Proline Metabolism as the metabolic pathway most strongly associated with skeletal muscle index (SMI) among older adults; seven circulating metabolites were positively correlated with SMI in both untrained and resistance exercise trained older adults. Arginine attenuates skeletal muscle wasting induced by diseases with heightened inflammation. However, the relationship between arginine and proline metabolism and skeletal muscle health is largely unknown. Recognizing that older muscle is susceptible to heightened inflammation that induces skeletal muscle deterioration and pathological remodeling, we hypothesized that inflammation disrupts arginine metabolism in skeletal muscle. Objective: Determine the effects of inflammation on arginine transporter and metabolic enzyme gene expression in differentiated primary human progenitor cells. Methods: Skeletal muscle biopsies were collected from younger men and women after an overnight fast. Primary human skeletal muscle progenitor cells harvested from the biopsies were cultured in growth media for 7 days, switched to differentiation media (DM) for 3 days, and then incubated for 2 days in DM in the presence or absence of 10 ng/mL of the proinflammatory cytokine TNFα. Gene expression of the arginine transporters (CAT1 and CAT2) and metabolic enzymes was measured using quantitative PCR and a PCR array, respectively, on RNA collected from the cultured cells. Results: Incubation with TNFα increased the expression of NFKB, IL6, and CAT2 (p<0.05), suggesting increased arginine uptake with inflammation. Additionally, inflammation decreased the expression of five metabolic genes (ornithine decarboxylase-1, pyrroline-5-carboxylate reductase-1, spermine synthase, creatine kinase, and ornithine aminotransferase) and increased the expression of prolyl-4-hydroxylase subunit alpha-1. Reduced expression of metabolic enzymes with inflammation (in vitro) was consistent with lower circulating levels of the related metabolites among older adults (in vivo) that we previously observed. Conclusions: Overall these results suggest that elevated TNFα, such as that observed in older adults, alters skeletal muscle arginine transport and metabolism and may be associated with skeletal muscle deterioration and pathological remodeling. Funding: Cornell University, Affinito-Stewart Grant.

Poster Board #124
Skeletal Muscle Phenotypes in Mkx-Null Mice
Takayuki Akimoto1,2, Yutaka Kano3, Kazutaka Nakamura2, Shigeru Miyaki4, Hiroshi Asahara5, Takashi Ushida2
1Laboratory of Muscle biology, Waseda University, 2 The University of Tokyo, Graduate School of Medicine, 3The University of Electro-Communications, 4Hiroshima University Hospital, 5Tokyo Medical and Dental University. Contact: axi@waseda.jp (T.A.).
Tendons are fibrous connective tissues attaching muscles to bones. Tendons serve to move the bones or structures through muscles. Recently, we have found a homeotic gene, Mohawk homeobox (Mkx) as a regulator for development and/or homeostasis of tendons (Ito et al., PNAS, 2010). The Mkx gene also expressed in skeletal muscle, suggesting the Mkx plays some roles in skeletal muscle. The purpose of this study was to determine muscle phenotypes in Mkx-null mice. As a result, the Mkx-null mice showed an increase in muscle mass compared with WT mice. We also found an increase of MyHCIIa fibers in the null-mice. There was no difference in muscle strength between WT mice and the null-mice, while the null-mice showed higher muscle fatigue resistance. These results suggest that Mkx gene serves as a regulator of skeletal muscle function.