METHODS: Twenty-two college- pre-hypertensive men with a BMI > 25 were assigned to an untreated tart cherry (UTC) or heat-treated tart cherry (HTC) group using a randomized balanced design, with a placebo (PLA) serving as a control in both groups. Each trial was separated by a minimum of 48 hours. Participants entered the lab following an overnight fast, completed a blood draw, consumed the supplement, rested for 1 hour, and the blood draw was repeated. Samples were centrifuged, serum was aliquoted and stored at -80 degree Celsius. Samples were analyzed using an ELISA analysis. All data are presented as mean ± SEM.
RESULTS: Total Antioxidant Capacity in the UTC group changed from 207.67 ± 44.81 pre-treatment to 227.91 ± 46.70 mM one hour post-ingestion and the HTC group changed from 238.91 ± 29.59 pre-treatment to 251.83 ± 30.57 mM post-ingestion. Total Antioxidant Capacity in the placebo condition changed from 213.41 ± 32.12 to 190.84 ± 28.22 mM in the UTC group and from 259.10 ± 32.61 to 247.61 ± 35.18 mM in the HTC group. A repeated measures ANOVA examining change in total antioxidant capacity in the placebo versus control condition revealed that there was no significant effect for treatment group ($F = 0.52$; $p = 0.48$). There was a significant effect for time ($F = 6.07$; $p = 0.03$), but no interaction effect ($F = 0.10$; $p = 0.75$).
CONCLUSIONS: Cherry supplements, both heat-treated and untreated, do increase total antioxidant capacity. However, heat-treated cherries do not have a significantly greater effect on total antioxidant capacity in serum compared to untreated cherries.

849

Branched Chain Amino Acid Supplementation Drives Metabolomic Shifts Downstream Of Serotonin In Endurance Runners

Jesse T. Peach, Dakota Funk, Lizzi Frothingham, McMilin R. Colleen, Brian Bothner, Mary P. Miles, FACSM. *Montana State University, Bozeman, MT.* (Sponsor: Dr. Mary Miles, FACSM)

Elevated serum serotonin concentrations have been linked to exercise and are postulated to influence central fatigue. Serotonin is synthesized in the brain from tryptophan, which crosses the blood-brain barrier via the LAT1 transporter. Branched chain amino acids (BCAA) also use the LAT1 transporter, and increased BCAA concentrations limit tryptophan transport and serotonin production. The downstream effects of serotonin dysregulation are not well understood and the specific metabolites promoting serotonin induced central fatigue are unknown.
PURPOSE: Determine the effects of BCAA on serotonin production and uncover the downstream metabolomic processes regulated by serotonin in endurance runners.
METHODS: Endurance runners participating in at least five hours of endurance training per week (n=10) participated in this double-blind, randomized crossover study. VO2max and treadmill pace at 65% VO2max were determined during an initial visit. In two subsequent visits participants received either a drink containing 11 g of BCAA or a flavor matched control in randomized order. Participants then ran on a treadmill at their predetermined moderate intensity pace for one hour. Blood samples were taken before and after exercise and analyzed via LCMS.
RESULTS: A paired t-test indicated that serotonin was lower ($p<0.05$) after consumption of BCAA at pre-exercise but not post-exercise. An untargeted analysis of the LCMS data revealed four metabolites linked to BCAA supplementation and serotonin metabolism elevated after exercise in the BCAA compared to placebo condition, including leucine and valine from the BCAA beverage along with the metabolites octopamine and ornithine.
CONCLUSION: BCAA supplementation led to an initial decrease in serotonin in endurance runners relative to placebo supplementation. However, after exercising this variation dissipated. Supplementation of BCAA also increased the metabolites octopamine and ornithine, which can be metabolically regulated by serotonin-dependent mechanisms, are members of pathways modulating energy production and fatigue, and provide viable links between serotonin concentration and central fatigue.
Supported by the Kreighbaum Endowment and Montana State University Research Enhancement Funds

---

850

Effect Of Nitrate Supplementation On Motor Unit Functions In Healthy Active Adults

Ozcan Esen, Michael Callaghan, Jamie McPhee. *Manchester Metropolitan University, Manchester, United Kingdom.*

Nitrate (NO_3^-) supplementation may enhance fatigue resistance and recovery, possibly because its additive effect on muscle blood flow (BF) during muscle contraction. During submaximal contractions, there are numerous ways in which the decrease in performance can be compensated for, such as motor unit (MU) recruitment, or increasing firing rate (MUFR). Given that changes in BF correlates with changes MUFR, the increased BF via NO_3^- supplementation may enhance MUFR during fatigue and recovery.
PROPOSE: to investigate the effect of NO_3^- supplementation on MU Functions during a sustained isometric contraction where BF was occluded in healthy active adults.
METHODS: In a counterbalanced double-blinded manner, 14 young adults completed two 5-day supplement period with either NO_3^--rich (NIT) or NO_3^--depleted (PLA) beetroot juice. Each trial consisted of measuring isometric knee-extension forces, and MUFR and jiggle were measured with an intramuscular concentric needle. After initial 20 sec muscle contraction at 25% maximal voluntary contraction (MVC) in unfatigued state (UF), leg blood flow was occluded for 5 min and then participants held a 25% MVC for 3 min with recording of iEMG throughout, and final 20 sec of that was taken as fatigue value (F). Participants then rested, but with the still occluded, and performed 20 sec contraction at 25% MVC at 45 sec post-fatigue (R1). Occlusion was then released, and 20 sec contraction at 25% MVC were performed after a further 45 sec (R2). Data were analysed using two-way repeated measures ANOVA.
RESULTS: The plasma NO_2^- concentration was increased after NO_3^- (475 ± 93 nmol·L^{-1}) compared to PLA (198 ± 46 nmol·L^{-1}, $p < 0.01$). MUFR decreased with fatigue and remained low in the recovery periods (UF: 9 ± 0.3 Hz; F: 7.3 ± 0.3 Hz; R1: 8 ± 0.2 Hz; R2: 7.7 ± 0.2 Hz, $p < 0.01$). jiggle increased as fatigue develops, remained high after recovery with occlusion, and decreased after recovery without occlusion (UF: 19.7 ± 0.8 %; F: 23.7 ± 1.4 %; R1: 27.7 ± 2.5 %; R2: 23.3 ± 1.5 %, $p < 0.05$). NO_3^- supplementation had no effect on MUFR and jiggle compared with placebo at any condition ($p > 0.05$).
CONCLUSION: NO_3^- supplementation had no ergogenic aid during muscle fatigue development or short-period recovery in healthy active adults, at least for sustained low intensity isometric contractions.

851

Ginger Root Extract Increases Mitochondrial Fission And Mitophagy In Diabetes Mellitus Rats

Casey Robert Appell[1], Nigel C. Jiwan[1], Rui Wang[2], Chwan-Li Shen[2], HuiYing Luk[1]. *[1]Texas Tech University, Lubbock, TX. [2]Texas Tech University Health Sciences Center, Lubbock, TX.*

Diabetes (DM) is accompanied by mitochondrial dysfunction (i.e., mitochondria fission/fusion and mitophagy) in which results in an accumulation of damaged mitochondria and further impaired insulin resistance. Ginger root extract (GRE) has been shown to improve mitochondrial biogenesis and decreased respiratory coefficient in DM model, however, the effect of GRE on the basal mitochondria fission/fusion and mitophagy state is limited.
PURPOSE: To determine the effect of GRE on mitochondria fission/fusion and mitophagy transcript abundance in rats with diabetes induced by high-fat diet (HFD) with streptozotocin (STZ).
METHOD: Sprague-Dawley rats were randomly divided into 3 groups: standard diet (STD; n=11), HFD with 35 mg/kg of STZ (DM; n=9), and HFD+STZ with 0.75% w/w GRE (GRE; n=7) in diet. After 7 weeks, soleus samples were collected and analyzed for gene expression for fission/fusion (*DRP, MFN*) and mitophagy (*PINK1, PARKIN, BECN1, LC3A, LC3B, P62)* markers.
RESULT: A significant ($p<0.05$) condition effect was found for *PINK1, DRP, LC3A, LC3B, P62,* and autophagic flux. For fission/fusion, GRE had significantly greater *DRP* (2.27±0.9-fold vs. 0.47±0.1-fold) than DM and no difference was found for *MFN*. For mitophagy, GRE had significantly greater *PINK1* (1.59±0.55-fold vs. 0.31±0.06-fold), *LC3A* (1.81±0.65-fold vs. 0.13±0.02-fold), *LC3B* (2.71±0.92-fold vs. 0.66±0.25-fold), *P62* (3.25±1.24-fold vs. 0.43±0.12-fold), and autophagy flux (4.5±1.06-fold vs. 2.41±0.36-fold) than DM and greater *LC3B* (2.71±0.92-fold vs. 1±0.06-fold), *P62* (3.25±1.24-fold vs. 1±0.21-fold), and autophagic flux (4.5±1.06-fold vs. 1±0.26-fold) than STD. Meanwhile, no difference was found for *PARKIN* and *BECN1*.
CONCLUSION: In DM rats, GRE consumption increased soleus basal transcription abundance for genes that regulate mitochondria fission, mitochondria degradation tag, and autophagolysosome formation in which would potentially increase the capacity for reducing the accumulation of damaged mitochondria.