RESULTS: While there were no significant interactions among baseline and postprandial lipids (Total cholesterol, LDL-C, and HDL-C) or in AUC, there were significant differences in TG-AUCs. Furthermore, the 65kcal/kg/FFM TG-AUC was higher (14840.4±3612.2) compared to 45kcal/kg/FFM and 25kcal/kg/FFM (9841.3±2011.2 and 6438.4±3880.3, respectively).
CONCLUSION: These data suggests that in healthy men, overeating over 24hrs results in exaggerated PPL the morning following a single bout of exercise, as compared to being calorically restricted or in a balanced energy availability status.

1435 Board #88 June 2, 9:00 AM - 10:30 AM

Acute State Of Postprandial Lipemia Induces Changes In Heart Rate Variability In Healthy Adults: Clinical Pilot Study

Diana Lorena Camelo-Prieto[1], Luis Andrés Téllez Tinjaca[1], Katherine González-Ruíz[2], Adalberto Palacios-López[2], Jorge Enrique Correa-Bautista[2], Robinson Ramírez-Vélez[3]. *[1]Universidad Santo Tomas, Bogotá D.C, Colombia. [2]Universidad del Rosario, Bogotá D.C, Colombia. [3]Universidad Manuela Beltrán, Bogotá D.C, Colombia.*
Email: robin640@hotmail.com

(No relationships reported)

PURPOSE: Experimental evidence has shown the relationship between heart rate variability (HRV) and cardiac dysfunction. Nevertheless, it is unknown if changes in HRV can be modified after a high fat meal (HFM) intake. The hypothesis of this study was focused on demonstrating that an acute state of postprandial lipemia induces changes in HRV in healthy adults.
METHODS: Prospective interventional study in 14 apparently healthy adults of both sexes, (mean age: 30.71 ± 7.9 years; body weight: 71.53 ± 12.9 kg; BMI 26.2 ± 3.4 kg/m2), with no past medical history of cardiovascular or endocrine disease. The HFM consisted of a breakfast with a total weight of 141g and the following nutritional composition: 31 g fat, 69 g carbohydrate, 31 g protein, and a total of 1171 kcal. Pilot studies confirmed that in a rested state this meal produced a transient impairment in endothelial function. HRV was measured by the mean length of the RR interval (ms), after 10-12h fast (0 min, baseline) and after 60 min, 120 min, 180 min and 240 min postprandial. ANOVA for repeated measures was performed for five times, with Bonferroni correction.
RESULTS: The basal value of mean RR was 925.2 ± 48.9 ms. It was identified that postprandial lipemia decreases the HRV in the first 60 min (826.9 ± 31.1 ms) by 10.6% (p< .261 for all ANOVA measures). Nevertheless, it increases at 120 minute by 10% (909.8 ± 49.25 ms) regarding to min 60. This increase was maintained at 180 min (907.6 ± 55.4 ms increment 9.7% compared to min 60). Finally, another decrease was identified at 240 min postprandial (845.7 ± 81.1 ms decrease of 8.6%) regarding to baseline.
CONCLUSIONS: This study is the first experimental evidence that demonstrates that a high-fat intake changes HRV in healthy subjects. We recommend further studies with larger sample size in order to complement the results found on this study.

1436 Board #89 June 2, 9:00 AM - 10:30 AM

The Effects Of A High-fat Diet On Neuronal Inflammation

David S. Umbaugh, J. Claire Maciejewski, Brianne L. Guilford. *Southern Illinois University Edwardsville, Edwardsville, IL.*

(No relationships reported)

Diabetic neuropathy is a common and debilitating complication of diabetes. Overweight humans with dyslipidemia develop neuropathy before developing overt diabetes. In addition, recent evidence indicates a high-fat diet induces signs of neuropathy in rodents and may contribute to the development of neuropathy in pre-diabetic and/or diabetic humans, but mechanisms underlying high-fat diet induced neuropathy have not been elucidated.
PURPOSE: Identify neuronal inflammation as a potential mechanism underlying the pathogenesis of high-fat diet-induced neuropathy. This experiment tested the hypothesis that a HF diet induces neuronal inflammation.
METHODS: Male C57Bl/6 mice were randomized into two groups and fed a standard (Std, n = 11) or high-fat diet (HF, n = 12) for 8 wks. The lumbar dorsal root ganglia were harvested and inflammatory mediators (IL-1α, IL-1β, IL-2, IL-3, IL-4, IL-5, IL-6, IL-10, IL-12p70, IL-17, MCP-1, IFN-γ, TNF-α, MIP-1α, GMCSF, and RANTES) were analyzed using Multiplex ELISA. Neuropathy was characterized by the von Frey test for hindpaw mechanical sensitivity at baseline and every other week thereafter.
RESULTS: At end study, HF fed mice had greater bodyweight (33.3 ± 1.0 vs. 26.7 ± 0.5 g, p < 0.001) and fasting blood glucose levels (160.3 ± 9.4 vs. 138.5 ± 3.4 mg/dl, p < 0.01) compared to Std fed mice. Hindpaw mechanical sensitivity was not significantly different between groups at any time point. However, hindpaw mechanical sensitivity trended toward an increase from baseline to wk 8 in HF (baseline: 56.3 ± 0.05% vs. wk 8: 70.8 ± 0.06%, p = 0.055) whereas there was no increase in Std (baseline: 56.9 ± 0.05% vs. wk 8 : 61.4 ± 0.07%, p = 0.50). MCP-1 was significantly higher in HF compared to Std (18.8 ± 3.8 vs. 10.5 ± 1.9 pg/mg, p < 0.05). There were no other significant differences in inflammatory mediators between groups.
CONCLUSION: Although hindpaw mechanical hypersensitivity is characteristic of HF feeding in mice, the mild increase in hindpaw mechanical sensitivity did not reach statistical significance in this cohort. HF fed mice exhibited elevated MCP-1 levels compared to Std fed mice which is suggestive of diet-induced inflammation. MCP-1 is understood to play a crucial role in recruitment of inflammatory factors, which suggests diet-induced inflammation may play a role in establishing neuropathy.

1437 Board #90 June 2, 9:00 AM - 10:30 AM

Predictors of Maximum Fat Oxidation during Progressive Cycling to Exhaustion in Active Men and Women

Amy Clark[1], Ross Edmunds[2], Rachael M. Gallant[1], Leesa King[3], Anthony Fischer[1], Samantha Namm[1], Kimi Wood[1], Todd A. Astorino[1]. *[1]California State University San Marcos, San Marcos, CA. [2]SUNY-Stony Brook, Stony Brook, NY. [3]National College of Natural Medicine, Portland, OR.*
Email: clark170@cougars.csusm.edu

(No relationships reported)

The workload coincident with maximum fat oxidation (MFO) has been shown to widely vary across individuals (Venables et al. 2005). Determining MFO is paramount considering its relationship to weight status and metabolic health (Robinson et al. 2015). Significant predictors of MFO include fitness level measured by maximal oxygen uptake (VO_2max), fat-free mass (FFM), and dietary fat intake. The majority of existing data were obtained in highly active populations performing progressive treadmill exercise to exhaustion and not cycle ergometry, which has been shown to elicit lower fat oxidation versus treadmill exercise (King et al. 2015).
PURPOSE: To determine significant predictors of MFO during progressive cycling in recreationally-active men and women.
METHODS: 49 men and women (age, body fat, and VO_2max=23.7±4.9 yr, 16.2±6.6 %, and 40.6±5.5 mL/kg/min) performed a graded VO_2max test after an overnight fast and abstention from exercise for 36 h. Subjects cycled for 7 minutes at 30 or 40 Watt followed by a 20 Watt increase in work rate every 3 min until respiratory exchange ratio (RER) = 1.0 for an entire stage, after which power output was increased by 20 Watt/min until fatigue. Oxygen uptake and carbon dioxide production were averaged from the last 90 s of each stage to determine fat and CHO oxidation using the Frayn (1983) equation. Demographic characteristics including body composition via 3-site skinfolds and anthropometry were also measured.
RESULTS: Across participants, MFO was equal to 0.30±0.08 g/min and 5.31±1.43 mg/kg FFM.min^{-1} and occurred at intensities equal to 21.8±8.6% Wmax, 33.6±6.5% VO_2max, and 57.6 ± 6.6 %HRmax, respectively. Fat free mass, VO_2max, RER during stage 1 of exercise, and waist circumference accounted for 81.5% of MFO (p<0.05). Bivariate correlation analyses showed that VO_2max (r = 0.42, p=0.001), FFM (r=0.41, p=0.002) and RER in stage 1 (r=-0.76, p<0.001) were significant correlates of MFO.
CONCLUSION: Data demonstrate that fitness level and body composition account for much of the variance in MFO. Clinicians should emphasize the need to improve cardiorespiratory fitness as it is related to capacity for fat oxidation and potentially metabolic health.