13. **Effect of Carnosic Acid on Tumor Necrosis Factor-α-mediated Inflammation and Insulin Resistance in 3T3-L1 Adipocytes.** Chia-Wen Tsai and Yu-Ru Lin, China Medical University, Taiwan.

Obesity and insulin resistance have been linked to a low-grade chronic inflammatory response. Carnosic acid (CA), found in rosemary, has been reported to have antioxidant, antiinflammation and antiadipogenic properties. Therefore, we examined the effects of CA on inflammation and insulin resistance in 3T3-L1 adipocytes treated with tumor necrosis factor-α (TNF-α). In this study, we found that CA attenuated TNF-α-induced mRNA expression of inflammatory genes including interleukin-6 (IL-6), and monocyte chemoattractant protein-1 (MCP-1). CA attenuated TNF-α-mediated activation of extracellular signal-related kinase (ERK), c-Jun NH 2-terminal kinase (JNK), and nuclear translocation of p65 as well as DNA binding activity of nuclear factor-kappa B (NF-κB). CA or PP242 (mTOR inhibitor) suppresses TNF-α induced protein expression of mTOR, p70S6K, elf4E and IL-6. Moreover, CA attenuated TNF-α mediated the suppression of peroxisome proliferator activated receptor γ (PPARγ), adiponectin and adipocyte protein 2 (aP2). CA reversed TNF-α mediated the suppression of insulin-stimulated glucose uptake, phosphorylation of Tyr632 insulin receptor substrate-1 (IRS-1), Akt and FoxO1, but decreased TNF-α induced phosphorylation of Ser307 IRS-1 as well as total FoxO1. In conclusion, CA attenuates TNF-α-mediated inflammation and insulin resistance in 3T3-L1 adipocytes.

14. **The Novel iFat1 Transgenic Mouse is Capable of Inducible Endogenous Tissue Enrichment of n-3 PUFA.** Shannon Clarke¹, Jing Kang², and David Ma¹, ¹University of Guelph, Canada; ²Massachusetts General Hospital/Harvard Medical School, USA.

The fat-1 transgenic mouse, which endogenously synthesizes n-3 polyunsaturated (PUFA) from n-6 PUFA, is a powerful tool in nutritional research which has facilitated enhanced insight into protective health effects of lifelong tissue n-3 PUFA enrichment. However, the relative impact of timing of n-3 PUFA enrichment on health related outcomes remains poorly defined. To this end, the inducible fat-1 (iFat1) mouse model, which carries a Cre-recombinase dependent version of the C. elegans fat-1 gene, has been developed. The objective of this study was to characterize the utility of the iFat1 transgenic mouse as a model of conditional endogenous n-3 PUFA enrichment. iFat1 transgene function was verified in vitro. For in vivo analysis, iFat1 females were crossed with R26-Cre-ER(T2) males, a tamoxifen inducible Cre expression model. At 6 weeks of age R26-Cre-ER(T2)/iFat1 double hybrid males (n=3/group) were transiently treated with either tamoxifen or vehicle control. Tissues were collected at 9 weeks of age and phospholipid fatty acid composition determined using gas chromatography. Relative to their vehicle treated controls, tamoxifen treated double hybrids experienced an approximate 2-fold, or more, reduction (p<0.05) in the n-6/n-3 PUFA ratio within major phospholipid fractions, phosphatidylethanolamine (PE) and phosphatidylcholine (PC), of each of the liver, kidney and muscle. Total saturated, monounsaturated and PUFA did not generally differ between groups. These results suggest that the iFat1 transgenic mouse has potential application as tool in addressing the temporal effects of n-3 PUFA enrichment in disease prevention and treatment.


This study was to determine anti-inflammatory effect of black raspberry seed (BRS) oil containing abundant α-linolenic acid. Dried black raspberry seeds were ground and oil was extracted with hexane. Corn, olive, canola, soy bean, grape seed and sunflower seed oils were used for comparison. RAW264.7 cell viability was measured by MTT assay. Anti-inflammatory effects were determined by measuring LPS-induced NO, PGE2 and COX-2 productions in RAW264.7 cells treated with the BRS oil and the other vegetable oils. COX-2 expression was determined by western blotting. The oil samples tested in this study had no noticeable cell cytotoxicity. Inhibitory effects of 0.5 mM BRS oil on NO production in the LPS-treated cells significantly increased (p<0.05). The BRS oil at the concentrations of 0.1 and 0.3 mM showed significantly higher inhibitory effects on PGE2 production than other oils except olive oil (p<0.05). COX-2 expression was significantly reduced compared with LPS-treated control at 5 μM oil (p<0.05). However, there were no significantly differences in COX-2 expression between oils. The results suggest that BRS oil have anti-inflammatory activity.