Gastrointestinal Inflammation Induced by High Fat Diet
Jenna R. Lee & Terry L. Powley
Purdue University

Background
• Negative feedback signals in the gastrointestinal tract inform the central nervous system about consumed nutrients in order to manage food intake (Lam, T.K. 2010).
• Obese individuals are found to have elevated levels of inflammatory molecules throughout the body (Weisberg, S.P. 2003).
• If high fat diets induce chronic inflammation in tissue critical for nutrient feedback, circuits informing the brain about consumed food could be disrupted, therefore inducing altered patterns of meal consumption and weight gain.
• Observing patterns of food consumption, weight gain, and body fat composition while rats are placed on diets of varying fat content, inferences can be made about the connection between inflammatory responses, ingestive behavior, and body composition changes.

Method
• Forty-seven Sprague Dawley rats were given an initial diet of lab chow for one week. The rats were divided into either 10% fat diet, 45% fat diet, or remained on the control chow. Rats were matched based on baseline weight and body fat composition.
• Weight and body fat composition were recorded throughout the study. Weight was recorded every 3 days and body fat composition was recorded every 15 days using a MRI machine.
• Food intake was monitored continuously for a subset of rats in all three groups during the last five weeks of the study using the Biodaq automated computer system.
• After 105 days, gastrointestinal tissue was extracted from three regions of the small intestine: the duodenum, the jejunum, and the ileum.
• Tissue was stained using the immunohistochemistry procedure that employed MPO antibody to selectively stain for monocytes. Monocyte cells serve as markers for inflammation in tissue. Monocyte number in each intestinal region was determined microscopically at 200x with random sampling protocol.
• All slides were counted blind by a single investigator. A subset was counted by a second investigator for a reliability check.
• Patterns of food consumption were analyzed based on a 12 hour dark cycle for meal size, meal number, meal interval, and total caloric intake.

Results
Intestinal inflammation marked by monocyte cell counts. Diet (p=.0002) Location (p<.0001) Diet by location (p=.0006 ) Level of inflammation correlates with diet, location, and diet by location.

Conclusion
• Increased levels of overall inflammation were found in the 10% and 45% fat diet group. The levels of inflammation also varied based on the location along the gastrointestinal tract. Further analysis revealed an interaction between type of diet and area along gastrointestinal tract. Inflammation in the Ileum is correlated with a significant increase in body fat composition, suggesting that the location of inflammation may be important in determining the onset of excess fat gain.
• Intestinal inflammation produced by high fat diets were correlated with significantly altered patterns of food consumption. Compared to intake of chow, both diets high in fat produced larger satiety ratios leading to fewer meals and a reduction in calories consumed during the night phase.
• The results are consistent with the hypothesis that high fat diets disturb food intake control, body fat, and weight regulation by producing inflammation in the gastrointestinal tract.