Too Much of a Good Thing? A Novel Role of Osteopontin as an Anti-Obesity Cytokine and Its Implication in Obesity-Related Disease

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With world-wide rates of obesity increasing, understanding the biochemical changes associated with the progression of obesity has become increasingly important. Osteopontin (OPN) levels have been found to increase in obesity, and OPN transgenic mice, which over-express osteopontin in their lymphocytes were observed to weigh 17% more than wildtype mice (measurements by mentor). The goal of this study was to understand the changes in metabolism and gene expression occurring in male OPN transgenic mice, using mouse tissue and serum provided by the lab. The transgenic mice displayed hepatic steatosis (assays carried out by qualified scientist) and had significantly higher serum insulin, leptin, and HDL cholesterol levels. The Mouse Fatty Liver PCR array, which was used to look for differentially expressed genes related to obesity, revealed that there were several differentially expressed genes in OPN transgenic mice. Genes in the liver involved in up-taking fats from the bloodstream and fatty acid breakdown were upregulated while genes involved in fat production decreased. Data suggests that osteopontin, may act to limit the effects of obesity by inhibiting adipocytes from expanding—preventing uptake of glucose and fats. Increased expression of genes that promote fat efflux from the adipocytes and genes that increase the liver's capacity to up-take fats and break them down suggest that OPN may cause efflux of fat from adipocytes to the liver where it can be metabolized. Increased osteopontin expression appears to limit fat buildup in adipocytes but is not sufficient to prevent the insulin resistance and metabolic diseases associated with obesity today.

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