Consequences of prenatal stress on appetite control and the energy expenditure pathway

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Established research has illustrated that moderate exposure to stress in the womb influences both adult phonotype and genotype for several physiological pathways, especially in males. Proposed explanations include adaptions made by the fetus resulting from a limited supply of nutrients, referred to as the "thrifty phenotype". In this study, we examine this fetal programming effect on the appetite control and energy expenditure pathways in prenatally stressed adult male offspring. Subjects were male rats born from time-mated female rats exposed to unpredictable, variable prenatal stress (UVPS) throughout gestation. An analysis of the adult male rat offspring genetic expression of epididymal fat pads and the plasma concentrations of hormones involved in appetite control and energy expenditure pathways showed a significantly diminished expression of leptin and adiponectin compared to unstressed controls. Leptin and adiponectin are both major hormones involved in the appetite control and energy expenditure pathways, with leptin regulating energy balance due to its function as an inhibitor of hunger, and adiponectin modulating glucose levels and fatty acid breakdown. We observed higher leptin concentrations within the prenatally stressed male plasma, and lower expression of leptin (OB) and adiponectin (ADIPOQ) genes from the epididymal fat pads. We suggest that elevated leptin in the plasma elicited a negative feedback effect on OB expression levels, decreasing their quantification compared to control animals. Further analysis will include plasma quantification of insulin and glucose, as well as expression of ghrelin, a peptide which acts on the central nervous system and the body's perception of hunger.