OBESITY: A GROWING CONCERN ON FETAL NUTRITION

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ABSTRACT

Obesity is a growing epidemic and a major health concern in the United States and elsewhere. The Fetal Basis of Adult Disease (FBAD) theory holds that events that occur during development can permanently alter gene expression throughout the lifetime of the individual. A link has emerged between fetal nutrition, birth weight, and metabolic profile in adulthood. Metabolic diseases represent a host of conditions relating to abnormal "programming" of nutrient management. We have developed a CD-1 mouse model which shows that fetuses within two body weight ranges at birth are at increased risk for developing metabolic diseases such as obesity, hypertension, cardiovascular disease, and Type II diabetes. It is not just individuals that are born with abnormally high birth weights (macrosomia) that are at risk for becoming overweight later in life. Paradoxically, this risk also applies for individuals who are born with extremely low birth weights, which are termed intrauterine growth restricted (IUGR). Mice identified with IUGR at birth show a dramatic increase in body weight during the first week post-weaning, which results in adult obesity and an altered metabolic profile for the remainder of their adult life. Preliminary studies indicate that when examined in adulthood, adipocytes in males identified with IUGR or macrosomia at birth showed evidence of differences in "programming" of expression of genes involved in fat cell differentiation and function. Certain xenoestrogens in the environment are known to interrupt the process of cell differentiation. Utilizing this model, we will be able to test the effects of

these chemicals on individuals that already have a metabolic deficit. By examining the relationship of fetal nutrition and growth with endocrine disruption, greater evidence may emerge for the need of regulation of endocrine disrupting chemicals.