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Elena Makarova, Denisova E and Kozhevnikova V
Institute of Cytology and Genetics, Russia

Maternal obesity increases the risk of obesity in offspring. Leptin is increased in obese animals, and elevation of maternal leptin may affect the metabolic phenotype of the offspring. We explore the effects of leptin elevation during mid-pregnancy on the offspring metabolic phenotypes, fetal growth, and placental gene expression. C57BL mice received a single injection of leptin or saline on pregnancy day 12. Body weight (BW) was measured weekly in offspring, which consumed standard chow or palatable food and the mRNA expression of glucose and amino acid transporters, insulin-like growth factor 2 and its receptor was measured 3 hours and the placental and fetal weights were measured 24 hours after the injection. The offspring born to leptin-treated mothers exhibited growth retardation before and catch-up growth after weaning and mature male offspring had an increased BW on a standard diet. Prenatal exposure to leptin did not influence the obesity development but prevented the development of obesity-associated hyperglycemia. The leptin injection decreased the fetal weight by 5% and the placental mRNA level of amino acid transporter SNAT2. The results suggest that elevation of maternal leptin in mid-pregnancy has positive effect on glucose metabolism in mature offspring and this effect is associated with leptin influence on fetal growth and amino acid transporter expression in placenta.

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Elena Makarova is currently a Senior Researcher in the Laboratory of Physiological Genetics in the Institute of Cytology and Genetics, Novosibirsk, Russia. Her researches focus on the studies of sex-specific influence of maternal leptin on metabolic characteristics in progeny of rodents. She with her colleagues found maternal leptin retarded obesity development in male progeny and improves glucose metabolism in progeny of both sexes in mice.

enmakarova@gmail.com

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