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МАТЕРИАЛЫ КОНФЕРЕНЦИИ И ШКОЛЫ

FATTY TISSUE HORMONE LEPTIN AS A FACTOR OF ADAPTATION TO EATING CONDITIONS IN RELATED GENERATIONS

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Introduction. The environment conditions via an organism of a pregnant female affect the developing offspring and their adaptability in the future. The blood level of adipocyte hormone leptin decreases with exhaustion of fat stores and increases with their increase. It was shown that an increase above the physiological norm of the level of leptin in the blood during pregnancy improved offspring metabolic characteristics when consuming high-calorie foods. The work is devoted to the study of molecular and physiological mechanisms mediating the adaptive effect of maternal leptin on the reaction of offspring on the consumption of high-calorie foods.

Methods. The effects of leptin administration to pregnant mice on the expression of genes in fetuses at the end of pregnancy, on the offspring growth and food intake under standard conditions, on the development of obesity, taste preferences and liver and muscles gene expression in offspring consuming a high-calorie diet (standard food, pork lard, sweet cookies) were evaluated.

Results. The leptin administration on pregnancy days 11–13 affected gene expression in the fetal liver at the end of pregnancy: increased mRNA levels of insulin-like growth factor 1, DNA methyl transferase (DNMT) 3b, increased mRNA level of DNMT3a in males and decreased it in females. Leptin administration to pregnant females reduced the body weight of female offspring when kept on a standard diet. When the offspring were on a high-calorie diet, leptin administration retarded the development of obesity in males, increased standard food and decreased cookies consumption, and increased muscle expression of genes encoding insulin receptor and glucose transporter 4 in the offspring of both sexes.

Conclusion. The results suggest that maternal leptin increases resistance to obesity in offspring by shifting taste preferences in favor of a balanced diet and maintaining muscle sensitivity to insulin. Perhaps this is due to its effect on fetal DNA methylation.

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