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Gonchar A

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characteristics of obesity-prone and obesity-resistant female offspring
in mice

Metabolic response to obesogenic diet (OD) varies widely even in mice with the same genetic background, some mice develop obesity (obesity-prone, OP), and others do not (obesity-resistant, OR). Prenatal developmental programming may impact this variability. In mice, elevated maternal leptin during pregnancy is associated with enhanced resistance to obesity in offspring but metabolic pathways affected by maternal leptin are unknown. We examined the metabolic features associated with resistance to diet-induced obesity (DIO) and the effect of hyperleptinemia during pregnancy on these characteristics in OP and OR female offspring. Females born to control C57Bl and C57Bl-Ay (hyperleptinemia during pregnancy) mothers received standard chow after weaning for 12 weeks and then they consumed OD. After eight weeks they were divided into OP or OR according to their body weight. On standard chow, OP and OR mice did not differ in food intake, but OR mice gained less weight than OP mice. Obesity development in OP mice was associated with hyperglycemia and glucose and insulin intolerance. Glucose level and insulin sensitivity were normal in OR mice and insulin receptor (InsR) mRNA expression in the liver and adipose tissue was increased compared to control (standard diet) and OP mice and it can be a mechanism providing resistance to DIO. Maternal hyperleptinemia affected only growth rate after weaning on standard diet in OR females (accelerated) and did not affect other characteristics. Perhaps, resistance to DIO may be associated with the effect of maternal leptin on the growth hormone axis in OR mice.

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Biography

Gonchar A has a Bachelor's degree in Biology and is currently pursuing her Master's degree from the Faculty of Natural Sciences of the Novosibirsk State University. She took part in University and Institute of Cytology and Genetics student conferences. Her area of interest includes: diet-induced obesity, type 2 diabetes, resistance to diet-induced obesity, genetic and epigenetic mechanisms in obesity.

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