

Modification on Glucose Metabolism and Biochemical Parameters Imposed by Over Nutrition and Exercise Interference on Obesity Onset

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Introduction: Children obesity increases risks of adult onset of metabolic diseases. The small litter model imposes a perturbation during suckling period leading to development of short- and long-term modifications in body mass and tissue metabolism. **Objectives:** To investigate glucose metabolism in muscle and adipose tissues and the effect of exercise training on animal model. **Materials and Methods:** Litters were standardized to 10 puppies. At day 3, litters were adjusted: Small Litter (NR 3 puppies) and Normal Litter (NN 10 puppies). At day 21, animals were subdivided: exercised (EX) and sedentary (S). Swimming protocol was applied between 22-90 days. At day 85, animals were insulin tolerance tested. At day 90, animals were orthothanasiated. Triglycerides, cholesterol and glucose were measured. Fat deposits were weighed. Muscle and adipose tissue were incubated with glucose with or without insulin and lactate production was measured. **Results and Discussion:** At day 90, NR-S showed body mass gain compared with NN-S. NN-E and NR-E reduced body mass compared to sedentary controls. Retroperitoneal and mesenteric fat deposits increased in the NR-S compared to NN-S. NN-E and NR-E decreased for retroperitoneal, mesenteric and perigonadal deposits, compared to sedentary controls. NN-E and NR-E gained brown fat compared to sedentary groups. Soleus muscle lactate production increased in NN-E and NR-E. Insulin stimulus increased NN-E lactate production. NR-E perigonadal fat lactate production increased compared to NR-S at baseline condition. In baseline insulin stimulation condition, NN-S and NN-E increased lactate production in comparison to NR-S and NR-E, respectively. There were no differences on blood parameter among groups. Except for triglycerides, NN-E and NR-E had significant reduction compared to controls.

Conclusions: Small litter manipulation stimulated metabolic changes resulting in increased body mass and fat deposits, and altered glucose metabolism in skeletal muscle. Exercise did not interfere in skeletal muscle metabolic programming, but it led to decrease of adiposity, body mass and current triglycerides.

Key words: metabolic imprint, lactation, obesity

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