ENCRYPTION METABOLISM WAS INHIBITED ON HYPOTHALAMUS OF MICE SUBMITTED TO HIGH-FAT DIET-INDUCED OBESITY MODEL

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Introduction: Obesity is defined as the accumulation of abnormal or excessive fat that may impair health. Considering the great impact of obesity on health and the gaps that still exist in its pathophysiology, researchers are in the quest to better understand the role of the central nervous system. Studies have demonstrated associations between obesity and abnormalities in the hypothalamus. They have also shown that obesity is associated with alterations in mitochondrial functioning and metabolic flux. Information about these changes within the hypothalamus is scarce.

Objectives: This study evaluated the activity of complex I, II and IV of the mitochondrial respiratory chain and creatine kinase activity in the hypothalamus of mice fed a high-fat diet.

Materials and Methods: Male Swiss mice, about 40 days old, were divided into two groups: one group was fed a standard diet, and the other was fed a highly saturated fat diet for 10 weeks. Body weight was measured weekly. At the end of the experiment, the animals were weighed and euthanized by decapitation. The adipose tissue of mesenteric, epididymal and retroperitoneal areas was removed and weighed to evaluate visceral fat. The brain was also removed, and the hypothalamus was isolated for biochemical analysis.

Discussion and Results: The animals that received high-fat diet had body weight and visceral fat significantly higher than the group that received standard diet. The biochemical analysis of the hypothalamus showed that mice submitted to animal model of obesity presented inhibition of complex I, II and IV activity of the mitochondrial respiratory chain as well as inhibition of creatine kinase activity.

Conclusions: Our results support other findings that show that a saturated-fat diet may damage the hypothalamus, and reveal that high-fat diets, and subsequent obesity, lead to inhibition of energy metabolism in the hypothalamus, which contributes to reduction of ATP production.

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Key Words: obesity; hypothalamus; energy metabolism