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Resumo	The arcuate and the paraventricular and lateral hypothalamic nuclei, related to hunger and satiety control, are generally compromised by excess fatty acids. In this situation, fatty acids cause inflammation via TLR4 (toll like receptor 4) and the nuclei become less responsive to the hormones leptin and insulin, contributing to the development of obesity. In this work, these nuclei were analyzed in animals fed with high-fat diet and submitted to swimming without and with load for two months. For this, frontal sections of the hypothalamus were immunolabelled with GFAP (glial fibrillary acidic protein), synaptophysin, IL-6 (interleukin 6) and TLR4. Also, proteins extracted from the hypothalamus were analyzed using Western blotting (GFAP and synaptophysin), fluorometric analysis for caspases 3 and 7, and CBA (cytometric bead array) for Th1, Th2, and Th17 profiles. The high-fat diet significantly caused overweight and, in the hypothalamus, decreased synapses and increased astrocytic reactivity. The swimming with load, especially 80 % of the maximum load, reduced those consequences. The high-fat diet increased TLR4 in the arcuate nucleus and the swimming exercise with 80 % of the maximum load showed a tendency of reducing this expression. Swimming did not significantly influence the inflammatory or anti-inflammatory cytokines in the hypothalamus or in plasma. The high-fat diet in sedentary animals increased the expression of caspases 3 and 7 and swimming practice reduced this increment to levels compatible with animals fed on a normal diet. The set of results conclude that the impact of swimming on the damage caused in the hypothalamus by a high-fat diet is positive. The different aspects analyzed in here point to better cellular viability and conservation of the

	synapses in the hypothalamic nuclei of overweight animals that practiced swimming with a load.
Fomento	