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Increased macrophage infiltration and TNF- levels in the adipose tissue of obese Zucker rats after habitual exercise-induced stress

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INCREASED MACROPHAGE INFILTRATION AND TNF- α LEVELS IN THE ADIPOSE TISSUE OF OBESE ZUCKER RATS AFTER HABITUAL EXERCISE-INDUCED STRESS

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KEYWORDS: obesity, exercise, stress, TNF- α , macrophages

Metabolic syndrome (MS) is a disorder associated with obesity and constitutes a risk factor for type-2 diabetes mellitus and a state of “low grade inflammation”. The main molecular link between inflammation and obesity is TNF- α . This pro-inflammatory cytokine has been reported to be over-expressed and over-produced in the adipose tissue of rodent models of obesity, and it is also involved in insulin resistance. The sources of this cytokine are mainly macrophages that invade the adipose tissue in obese individuals. Regular moderate exercise is a good non-pharmacological therapeutic strategy in the management of MS since it improves diabetic status, insulin sensitivity, and the immune response. Based on the potential anti-inflammatory effects of exercise, it has been also proposed as a good strategy to control low-grade inflammation. Nevertheless, exercise can also elicit a danger-stress and inflammatory response that may become detrimental to health. Thus, in previous studies we found that an exercise of an inappropriate intensity worsened the dysregulation in the feedback mechanisms between the inflammatory and stress responses in the obese Zucker rat model of the metabolic syndrome, even increasing the systemic concentration of glucose. In the present study we have hypothesized that a program of habitual exercise-induced stress (running, 5 days/week for 35 min at 35 cm/s for 14 weeks) could also contribute to increased infiltration of macrophages and to increased levels of TNF- α in the adipose tissue of obese Zucker rats (fa/fa), which may contribute to the systemic hyperglycemia.

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Infiltrated macrophages and TNF- α expression in the white adipose tissue was determined by lectin histochemistry and immunohistochemistry; noradrenaline (NA) by HPLC, and glucose by standard method. The adipose tissue from obese sedentary rats showed higher levels of infiltrated macrophages and TNF- α than those observed in lean rats (Fa/fa) as reference values. The program of exercise increased the number of infiltrated macrophages and the expression level of TNF- α in the adipose tissue of obese rats, together with an increase in the circulating NA and glucose concentrations. Thus, the intensity of this program of habitual exercise could be considered as an example of "dangerous exercise". It is concluded that inadequate exercise-induced stress can further increase the obesity-induced augmented infiltration of macrophages and the TNF- α overexpression in the adipose tissue of obese animals, thus contributing to the metabolic, inflammatory, and stress disorders associated with the metabolic syndrome.

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