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Publicado por: Imprensa da Universidade de Coimbra

URL persistente: URI:<http://hdl.handle.net/10316.2/44160>

DOI: DOI:https://doi.org/10.14195/2182-7087_ex2018_97

Accessed : 26-Apr-2023 10:58:19

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β₂ ADRENERGIC REGULATION OF THE PHAGOCYtic ACTIVITY OF MONOCYTES IN OBESE MICE: EFFECT OF AN ACUTE INTENSE EXERCISE

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KEYWORDS: acute exercise, adrenergic receptor, obesity, phagocytosis

Obesity is a worldwide epidemic and it is associated with comorbid conditions that involve alterations of the innate/inflammatory immune response. Obesity is also correlated with changes in the activity of the sympathetic nervous system (SNS) and the hypothalamic-pituitary-adrenal (HPA) axis. Catecholamines are important immunoregulatory molecules, and adrenergic agonists interfere with the inflammatory response. On the other hand, physical activity, besides activating the SNS and HPA axis, is an event that can reverse pathologies arising from obesity⁽¹⁾. However, the influence of obesity on the adrenergic regulation of the immune system, and the influence of exercise on the mechanisms underlying this regulation in obesity, is still not well understood. The aim of this study was to investigate the influence of obesity and the effects of a single bout of acute exercise (running, 35 min at 16 m/min) on the β₂ adrenergic regulation of the phagocytic capacity of monocytes. A group of 20 ten-week old C57BL/6J mice were fed a diet containing either 5% (lean group, n=10) or 40% of fat –60% of the kcal from fat– (obese group, n=10) for 18 weeks. Then, 5 animals from each group were subjected to the acute exercise. The monocytes' phagocytic capacity was evaluated by flow cytometry in whole blood collected by cardiac puncture, in the presence or absence of the β₂ adrenergic agonist terbutaline. First, obese mice showed a lower phagocytic monocyte percentage than lean mice, and the acute exercise decreased the phagocytic monocyte percentage (mainly in lean mice) and increased their phagocytic activity in both groups. Terbutaline decreased the phagocytic percentage in both lean and obese mice, but it did not change the phagocytic activity. However, animals that performed

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the bout of acute exercise (both lean and obese), presented an increased phagocytic activity in response to terbutaline. To conclude, it seems that, although there was an overall lower phagocytic capacity of monocytes in obesity, both obese and lean mice showed a similar response to acute exercise and a similar β_2 adrenergic regulation of phagocytosis. However, this β_2 adrenergic regulation seemed to be different after acute exercise than at rest, since the β_2 adrenergic stimulation induced an increased phagocytic activity only during acute exercise.

This investigation has been supported by “Ministerio de Economía y Competitividad” (DEP2015-66093-R). We would like to thank the STAB (UEX) for technical and human support.

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