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Effects of acetylsalicylic acid on adiposity in a mouse model of diet-induced obesity

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Obesity is a growing public health problem and its prevalence has reached epidemic proportions in recent decades [1]. Several studies have demonstrated that obesity modifies the metabolic and endocrine functions of adipose tissue and is closely associated with chronic, low grade inflammation [2]. Because inflammation was proposed to be involved in the pathophysiology of obesity [1,2], we decided to evaluate the effects of the antinflammatory drug acetylsalicylic acid (ASA) in a mouse model of diet-induced obesity. We performed the experiments using C57BL/6J female mice fed for three months with Standard Diet (SD) or with High Fat Diet (HFD). At the end of three months, mice fed with HFD were separated in four groups and fed for other two months as follows: one group continued with HFD, one group returned to SD, one group continued with HFD with the addition of 30mg/kg of ASA and, finally, the last group returned to SD with the addition of 30mg/kg of ASA. ASA was administered in the drinking water. The metabolic and inflammatory status was evaluated by histological, molecular and biochemical analysis in all mice. As expected, HFD induced an increase in body weight and insulin resistance with a consequent reduction of glucose tolerance. Measurement of adipocyte size revealed that ASA significantly reduced HFD-induced adipocyte hypertrophy and it was able to revert insulin resistance with amelioration of glucose tolerance. Moreover, gene expression profiles of pro- and anti-inflammatory cytokines as well as the expression of macrophage and lymphocyte markers showed that HFD led to a significant increase in macrophages accumulation and an increase of inflammatory cytokines. However, we observed a significant trend for reduction of these molecules after treatment with ASA. The level of the anti-inflammatory molecules were also significantly increased after ASA administration. In conclusion, our results suggest that ASA can be proposed as pharmacologic option for reducing adipose tissue inflammation associated with obesity.

References

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Keywords