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Acetylsalicylic acid modulates inflammation and insulin resistance in a mouse model of diet-induced obesity

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Extensive scientific evidence indicates that non-optimal diet and sedentary lifestyle constitute some of the behaviors/risk factors associated with the onset of many diseases with significant societal impact, such as obesity. Chronic over-nutrition dramatically remodels adipose tissue architecture, driving adipocyte hypertrophy, oxidative stress and immune cell infiltration, followed by increased production of proinflammatory adipokines and cytokines that contribute to the progression of a chronic, low-grade inflammatory state (1,2). Obesity is associated with this inflammatory phenotype and increases the risk of chronic disease, including cardiovascular disease, as well as insulin resistance that predisposes to the development of type 2 diabetes (3). These obesity-associated diseases are subsequently linked to premature death, and reinforce the need to further define the complex relationship between inflammation and adipose tissue dysfunction. Reducing inflammation may represent a feasible disease-prevention strategy for obesity. Here we evaluate the effects of acetylsalicylic acid (ASA), a commercial small-molecule anti-inflammatory drug, in a mouse model of diet-induced obesity (DIO). The metabolic and inflammatory status and adipose tissue changes were evaluated by immunohistochemistry and Real time PCR in mice fed with high fat diet (HFD) compared with mice fed with standard diet (SD). We also analyzed how these events were modified as a result of treatment with ASA. Our results demonstrate that ASA not only displays anti-adiposity effects by reducing adipocyte hypertrophy and reversing insulin resistance, but that it also modulates adipose tissue inflammation. This could aid the optimization of clinical interventions and lifestyle changes aimed at improving human health.

References

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Keywords

Obesity; ASA; inflammation; insulin resistance.