

## CHIA OIL SUPPLEMENTATION IMPROVES VASCULAR DYSFUNCTION AND INDUCES PERIVASCULAR ADIPOSE TISSUE REMODELING IN OBESE MICE

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**INTRODUCTION:** Excessive accumulation of body fat promotes the expansion and inflammation of perivascular adipose tissue (TAPV), which are involved in developing insulin resistance, endothelial dysfunction, and atherosclerosis in obesity. However, which mechanisms link the expansion of TAPV during obesity to the pathogenesis of cardiovascular diseases (CVD) remains unclear. The nutritional quality of the diet is a pivotal factor in the pathophysiology of obesity. Recently, we demonstrated that dietary supplementation with chia oil (*Salvia hispanica* L.) improves insulin sensitivity and induces adipose tissue remodeling during obesity. **OBJECTIVE:** To evaluate the role of dietary supplementation with chia oil on metabolic and vascular changes resulting from high-fat diet-induced obesity. **METHODS:** Thirty-days-old male C57BL/6 mice were fed a regular chow diet (10% Kcal from fat - C-group) or a high-fat diet (45% Kcal from fat - H-group) for eight weeks. H and C groups were subdivided and supplemented with 1.5% (v/v) of chia seed oil (HC and CC groups, respectively) for a further six weeks. **RESULTS:** The chia oil supplementation significantly restored vasodilator response to acetylcholine and decreased the vasoconstrictor response to norepinephrine in the mesenteric vascular bed from obese animals. Treatment of H mice with chia oil reduced the adipocytes area, IL-1 $\beta$  secretion, and iNOS and HO-1 protein expression in mesenteric PVAT. In contrast, chia oil supplementation increased the production of IL-10 and induced the activation of the AMPK in the PVAT from obese animals. In vitro, endothelial cells incubated with PVAT conditioned medium (CM) derived from HC mice decreased NO production compared to obese mice. **CONCLUSION:** The dietary supplementation with chia oil promoted the morphological and functional remodeling of PVAT, contributing to the improvement of vascular dysfunction in obese animals.

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