國立臺灣海洋大學食品科學系碩士班

專題討論書面報告

桑葚果實對高脂飲食誘導肥胖和發炎之保護效果

Protective Effects of Mulberry Against High-Fat Diet-Induced Obesity and Inflammation

已註解 [振全1]:

已註解 [振全2]:

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報告日期：114 年 10 月 22 日

內容 40%	時間掌控 10%	表達能力 30%	投影片 10%	書面資料 10%

指導老師簽名：_____

桑葚果實對高脂飲食誘導肥胖之保護效果

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10/22/2025

大綱

摘要

台灣成年人人口中有約 24% 的人口皆符合衛福部判定的肥胖，並且肥胖與多種代謝疾病、慢性發炎密切相關，因此希望找尋不同對抗肥胖的方法。本研究使用了富含多酚的桑葚果實萃取物 Mulberry fruit extract (ME) 用於改善高脂飲食 (High-Fat Diet, HFD) 誘導的肥胖及代謝異常，而由於多酚類的生物利用度較低，因此也希望透過使用細胞外囊泡 (Extracellular vesicles, EVs) 來增強其對抗肥胖和抗發炎之效果。研究指出，ME 能有效介入 HFD 誘導的脂質生成及發炎，在動物模型中，補充 ME 顯著改善脂質異常，顯著降低血清總膽固醇、低密度脂蛋白及三酸甘油脂，並提升高密度脂蛋白。且脂質生成關鍵基因如 SREBP-1c、PPAR-γ 及 aP2 皆有顯著下降。發炎方面抑制了促發炎轉錄因子 NF-κB 的活化，並抑制其下游的 TNF-α、IL-6、MCP-1 等關鍵發炎因子及趨化因子，並由 CD11c 的表現量下降及 CD163 上升顯示巨噬細胞由促炎的 M1 表型朝向抗炎的 M2 表型 。由於慢性發炎過程會傷害血管導致內皮功能障礙，因此透過使用 ME 的 EVs 觀察對 Human Microvascular Endothelial Cells-1 (HMEC-1) 內皮細胞的保護作用，結果可見雖然不具顯著差異，但仍有保護的趨勢。總結來說，ME 透過其多酚對 HFD 誘導的肥胖模型展現其具有抗肥胖和抗發炎的保護潛力。

已註解 [振全3]:

已註解 [振全4]:

Protective Effects of Mulberry Against High-Fat Diet-Induced Obesity

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Outline

1. Introduction
2. Dried mulberry fruit ameliorates cardiovascular and liver histopathological changes in high-fat diet-induced hyperlipidemic mice
3. Mulberry (*Morus alba L.*) Fruit Extract Ameliorates Inflammation via Regulating MicroRNA-21/132/143 Expression and Increases the Skeletal Muscle Mitochondrial Content and AMPK/SIRT Activities
4. Contribution of Extracellular Particles Isolated from Morus sp. (Mulberry) Fruit to Their Reported Protective Health Benefits: An In Vitro Study
5. Conclusion

Abstract

Approximately 24% of the adult population in Taiwan classified as obese, obesity is intricately linked to metabolic disorders, chronic inflammation, the development of novel and effective therapeutic strategies is imperative. This study aimed to investigate the potential of a polyphenol-rich Mulberry fruit extract (ME) to ameliorate obesity and metabolic dysfunction induced by a high-fat diet (HFD). Cause of the low bioavailability of polyphenols, we explored mulberry-derived extracellular vesicles (EVs) as a potential nanodelivery system to enhance the anti-obesity and anti-inflammatory efficacy of ME. Research indicates that ME effectively mitigates HFD-induced lipogenesis and inflammation. *In vivo*, ME supplementation significantly ameliorated dyslipidemia, evidenced by reduced serum total cholesterol (TC), low-density lipoprotein (LDL), and triglycerides (TGs), alongside elevated high-density lipoprotein (HDL) levels. This was correlated with the significant downregulation of key lipogenic genes, including SREBP-1c, PPAR-γ, and aP2. Regarding its anti-inflammatory properties, ME suppressed the activation of the pro-inflammatory transcription factor NF-κB, which consequently downregulated its downstream targets, including pro-inflammatory cytokines (TNF-α, IL-6) and chemokines (MCP-1). And research indicated by decreased M1 marker CD11c expression and increased M2 marker CD163 expression, suggesting a shift from a pro-inflammatory M1 to an anti-inflammatory M2 state. Given that chronic inflammation precipitates endothelial dysfunction, we also assessed the protective effects of ME-derived EVs on human microvascular endothelial cells (HMEC-1). Though these results did not achieve statistical significance, a protective trend was observed. In conclusion, ME, likely via its polyphenol constituents, demonstrates significant protective potential against HFD-induced obesity and inflammation.