

改質柑橘果膠於癌症治療中的應用與機制探討

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大綱

一、前言

二、MCP 介導的主動靶向磷酸鈣雜化奈米顆粒用於治療原位耐藥性結腸癌

三、改質柑橘果膠透過靶向缺氧微環境中腫瘤相關巨噬細胞的存活和極化來抑制小鼠 乳腺癌的發展

四、結論

摘要

改質柑橘果膠 (modified citrus pectin, MCP) 是一種由天然柑橘果膠水解而成的低分子量多醣，近年來因其在癌症治療中的潛在應用而備受關注。癌症作為全球主要的健康威脅之一，現行的治療方法如手術、放療及化療雖能延長患者存活，但常受到藥物耐受性與副作用的限制。本文整合以 MCP 為核心的研究，探討其在耐藥性結腸癌與乳腺癌中的應用與作用機制。研究中開發了靶向結腸癌的新型奈米藥物傳輸系統 (PSVII@MCP-CaP)，該系統利用 MCP 作為靶向配體，將難溶性的 Paris saponin VII (PSVII) 包覆於鈣磷酸鹽奈米顆粒 (CaP) 中。研究結果顯示，PSVII@MCP-CaP 能有效抑制耐藥性結腸癌的生長與轉移，並顯著降低 PSVII 所造成的溶血作用，展現其優異的穩定性與生物安全性。另一方面，研究亦探討 MCP 對腫瘤相關巨噬細胞 (tumor-associated macrophages, TAM) 的影響。結果指出，在缺氧腫瘤微環境下，MCP 可透過抑制半乳糖凝集素-3 (galectin-3, Gal-3) 表達與活性氧 (ROS) 水準，降低 TAM 的葡萄糖攝取與 M2 型極化，進而減少乳腺癌的生長與轉移潛能。綜合而言，這兩項研究顯示 MCP 兼具藥物載體與免疫調控的雙重功能，提供一種同時針對抗藥性癌細胞與腫瘤微環境的新興治療策略。

Applications and Mechanistic Insights of Modified Citrus Pectin in Cancer Therapy

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Outline

1. Introduction
2. MCP mediated active targeting calcium phosphate hybrid nanoparticles for the treatment of orthotopic drug-resistant colon cancer
3. Modified citrus pectin inhibits breast cancer development in mice by targeting tumor-associated macrophage survival and polarization in hypoxic microenvironment
4. Conclusion

Abstract

Modified citrus pectin (MCP) is a low-molecular-weight polysaccharide derived from the hydrolysis of natural citrus pectin, which has recently attracted increasing attention for its potential applications in cancer therapy. Cancer remains one of the leading global health challenges, and although current treatments such as surgery, radiotherapy, and chemotherapy can prolong patient survival, they are often limited by drug resistance and severe side effects. This study integrates MCP-based research to explore its therapeutic roles and mechanisms in drug-resistant colon cancer and breast cancer. A novel nanoparticle drug delivery system (PSVII@MCP-CaP) was developed for targeted colon cancer therapy, in which MCP served as a targeting ligand to encapsulate the poorly soluble anticancer compound Paris saponin VII (PSVII) within calcium phosphate nanoparticles (CaP). The results demonstrated that PSVII@MCP-CaP effectively inhibited the growth and metastasis of drug-resistant colon cancer while significantly reducing PSVII-induced hemolysis, indicating excellent stability and biocompatibility. In addition, MCP was shown to regulate the metabolism and function of tumor-associated macrophages (TAMs) within the tumor microenvironment. Under hypoxic conditions, MCP suppressed the expression of galectin-3 (Gla-3) and reduced reactive oxygen species (ROS) levels, thereby decreasing glucose uptake and M2 polarization of TAMs, which in turn attenuated breast cancer progression and metastatic potential. Overall, these findings suggest that MCP possesses dual functionality as both a drug carrier and an immunomodulator, offering a novel therapeutic strategy that simultaneously targets drug-resistant cancer cells and the tumor microenvironment.

Reference

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