

Study on the Effect and Mechanism of Emodin in Anti-nasopharyngeal Carcinoma

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Abstract

Objective: Due to the poor prognosis of nasopharyngeal carcinoma (NPC), there is an urgent need for novel therapeutic agents. This study aimed to screen effective anti-NPC candidates from Traditional Chinese Medicine (TCM) monomers and explore their underlying mechanisms. **Methods:** Thirty TCM monomers were initially screened using CCK-8 and trypan blue staining assays with human NPC 6-10B cells. The antitumor efficacy of Emodin was evaluated in vitro and in a nude mouse xenograft model. RNA-Seq was performed to identify differentially expressed genes, and the PI3K/Akt/FOXO3A pathway was validated via qRT-PCR, Western Blot, and immunohistochemistry. **Results:** Five active monomers (Magnolol, Artesunate, Platycodin D, Emodin, and Shikonin) were identified, with Emodin exhibiting the most significant inhibitory effect. Emodin suppressed 6-10B cell proliferation in a dose- and time-dependent manner and induced apoptosis in vitro. RNA-Seq analysis revealed 3,513 differentially expressed genes, which were enriched in the FOXO signaling pathway. Mechanistically, Emodin downregulated PI3K, CyclinB, p-AKT, and p-FOXO3A, while upregulating FOXO3A, Bim, and P19. In vivo, Emodin significantly inhibited tumor growth and promoted apoptosis, consistent with its in vitro regulatory effects on the PI3K/Akt/FOXO3A pathway. **Conclusion** Emodin inhibits nasopharyngeal carcinoma (NPC) cell proliferation and induces apoptosis by modulating the PI3K/Akt/FOXO3A signaling pathway, suggesting its potential as a novel therapeutic agent for NPC.

Keywords

Nasopharyngeal Carcinoma, Traditional Chinese Medicine Monomers, Emodin, 6-10B Cells, RNA-Seq, PI3K/Akt/FOXO3A Signaling Pathway