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Molecular Mechanisms of Metformin in Esophageal Carcinoma Therapy

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Introduction

Esophageal carcinoma ranks among the deadliest cancers worldwide, characterized by its aggressive nature, limited treatment options and poor prognosis. Despite advancements in therapy, the overall survival rates for esophageal carcinoma remain low, highlighting the urgent need for innovative treatment approaches. Metformin, an oral hypoglycemic agent, has garnered attention for its potential anticancer effects [1]. In this article, we delve into the molecular mechanisms through which metformin exerts its anticancer activity in esophageal carcinoma and explore its therapeutic implications.

Description

Metformin activates AMPK, a key cellular energy sensor, leading to inhibition of mTOR signaling pathway. AMPK activation by metformin suppresses cell proliferation, induces cell cycle arrest and promotes apoptosis in esophageal carcinoma cells. Metformin disrupts cancer cell metabolism by inhibiting mitochondrial complex I, leading to decreased ATP production and altered cellular metabolism [2]. This metabolic reprogramming attenuates cancer cell growth and survival in esophageal carcinoma. Metformin inhibits the insulin/IGF signaling pathway, which is implicated in tumorigenesis and cancer progression. Suppression of IGF signaling by metformin impedes proliferation, migration and invasion of esophageal carcinoma cells [3,4].

By inhibiting EMT, metformin impedes invasion and metastasis of esophageal carcinoma cells, enhancing treatment efficacy. Metformin holds promise as an adjuvant therapy in esophageal carcinoma, complementing standard treatment modalities such as surgery, chemotherapy and radiotherapy. Clinical trials evaluating the efficacy of metformin as an adjuvant therapy in esophageal carcinoma are underway, with preliminary results showing encouraging outcomes. Combining metformin with conventional chemotherapeutic agents or targeted therapies may enhance treatment efficacy and overcome resistance mechanisms [5]. Synergistic interactions between metformin and other anticancer agents have been observed in preclinical studies, underscoring the potential for combination therapy in esophageal carcinoma. Identification of biomarkers predictive of metformin response can facilitate personalized treatment approaches in esophageal carcinoma. Biomarker-guided selection of patients likely to benefit from metformin therapy may optimize treatment outcomes and minimize unnecessary side effects.

Conclusion

Metformin emerges as a promising therapeutic agent for esophageal carcinoma, exerting anticancer effects through multiple molecular mechanisms. Its ability to target various hallmarks of cancer, including dysregulated metabolism, proliferative signaling and inflammation, underscores its potential as a multifaceted anticancer agent. Further research, including well-designed clinical trials, is warranted to elucidate the therapeutic efficacy and safety profile of metformin in the management of esophageal carcinoma.

Acknowledgement

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Conflict of Interest

None.

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