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Delta9-Tetrahydrocannabinol inhibits epithelial growth factor-induced lung cancer cell migration in vitro as well as its growth and metastasis in vivo.

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Abstract

Delta(9)-Tetrahydrocannabinol (THC) is the primary cannabinoid of marijuana and has been shown to either potentiate or inhibit tumor growth, depending on the type of cancer and its pathogenesis. Little is known about the activity of cannabinoids like THC on epidermal growth factor receptor-overexpressing lung cancers, which are often highly aggressive and resistant to chemotherapy. In this study, we characterized the effects of THC on the EGF-induced growth and metastasis of human non-small cell lung cancer using the cell lines A549 and SW-1573 as in vitro models. We found that these cells express the cannabinoid receptors CB(1) and CB(2), known targets for THC action, and that THC inhibited EGF-induced growth, chemotaxis and chemoinvasion. Moreover, signaling studies indicated that THC may act by inhibiting the EGF-induced phosphorylation of ERK1/2, JNK1/2 and AKT. THC also induced the phosphorylation of focal adhesion kinase at tyrosine 397. Additionally, in in vivo studies in severe combined immunodeficient mice, there was significant inhibition of the subcutaneous tumor growth and lung metastasis of A549 cells in THC-treated animals as compared to vehicle-treated controls. Tumor samples from THC-treated animals revealed antiproliferative and antiangiogenic effects of THC. Our study suggests that cannabinoids like THC should be explored as novel therapeutic molecules in controlling the growth and metastasis of certain lung cancers.

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Publication Types, MeSH Terms, Substances, Grant Support

Publication Types Research Support, N.I.H., Extramural

MeSH Terms Animals Antineoplastic Agents/pharmacology* Carcinoma, Non-Small-Cell Lung/metabolism* Carcinoma, Non-Small-Cell Lung/secondary Cell Line, Tumor Cell Proliferation/drug effects Chemotaxis/drug effects* Epidermal Growth Factor/pharmacology Lung Neoplasms/metabolism* Lung Neoplasms/pathology Mice Mice, SCID Mitogen-Activated Protein Kinases/antagonists & inhibitors* Mitogen-Activated Protein Kinases/metabolism Phosphorylation/drug effects Receptors, Cannabinoid/metabolism Tetrahydrocannabinol/pharmacology*

Substances Antineoplastic Agents Receptors, Cannabinoid Tetrahydrocannabinol Epidermal Growth Factor Mitogen-Activated Protein Kinases

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