Antineoplastic Activity of Cannabinoids

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Summary

Lewis lung adenocarcinoma growth was retarded by the oral administration of Δ⁹-tetrahydrocannabinol (Δ⁹-THC), Δ⁸-tetrahydrocannabinol (Δ⁸-THC), and cannabidiol (CBD), but not cannabidiol (CBD). Animals treated for 10 consecutive days with Δ⁹-THC, beginning the day after tumor implantation, demonstrated a dose-dependent action of retarded tumor growth. Mice treated for 20 consecutive days with Δ⁸-THC and CBN had reduced primary tumor size. CBD showed no inhibitory effect on tumor growth at 14, 21, or 28 days. Δ⁹-THC, Δ⁸-THC, and CBN increased the mean survival time (36% at 100 mg/kg, 25% at 200 mg/kg, and 27% at 50 mg/kg, respectively), whereas CBD did not. Δ⁹-THC administered orally daily until death in doses of 50, 100, or 200 mg/kg did not increase the life-spans of (C57BL/6 × DBA/2)F₁ (BDF₁) mice hosting the L1210 murine leukemia. However, Δ⁹-THC administered daily for 10 days significantly inhibited Friend leukemia virus-induced splenomegaly by 71% at 200 mg/kg as compared to 90.2% for actinomycin D. Experiments with bone marrow and isolated Lewis lung cells incubated in vitro with Δ⁸-THC and Δ⁸-THC showed a dose-dependent (10⁻⁴–10⁻⁷) inhibition (80–20%, respectively) of tritiated thymidine and ¹⁴C-uridine uptake into these cells. CBD was active only in high concentrations (10⁻⁴).