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GROWTH INHIBITION OF HEPATOCELLULAR CARCINOMA HCC) BY CBD RICH T3/C15 CANNABIS FRACTION IS) MEDIATED VIA THE A3 ADENOSINE RECEPTOR

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Background: The Wnt/β-catenin signal transduction pathway latelpromotes liver tumor cell growth by enhancing hepatic ste ecell activation and survival. Namodenoson, an A3 adenosin receptor (A3AR) agonist, de-regulates the Wnt/β-catenin pathway in hepatocellular carcinoma (HCC) cells, inducing apoptosis. As cannabinoids were found to bind with high estigatevaffinity to A3AR, the goal of the current study was to in udytthe anti-growth effect of cannabinoids on HCC cells and s the molecular mechanism involved. **Methods:** Hep-3b HCC cencells were cultured for 48 hours in the presence and abse of 10 nM CBD-rich THC3/CBD15 (T3/C15) and in the presence and absence of the A3AR antagonist MRS1523. sesy^{3}[H]-thymidine proliferation assay, and Western blot anal were performed. **Results:** CBD-rich T3/C15 significantly This inhibited Hep-3b cell proliferation (56%±5.5 ; $p < 0.05$). response was neutralized by the A3AR antagonist MRS1523. ,Growth regulatory proteins down-stream to A3AR activation llincluding p-Akt, NF- kappa B, GSK-3β and β-catenin were a lityidown-regulated. **Conclusion:** Our findings highlight the ab ,sof CBD-rich T3/C15 to inhibit, in nanomolar concentration fthe growth of HCC via A3AR activation and de-regulation o the Wnt/β-catenin pathway. These findings open a novel therapeutic opportunity in liver cancer with minute CBD . concentrations and low content of psychotropic THC fraction

: Disclosures

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