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Cannabinoid WIN55,212-2 induces cell cycle arrest and inhibits the proliferation and migration of human hepatocellular carcinoma BEL7402 cells

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Hepatocellular carcinoma (HCC) is the leading cause of cancer deaths worldwide, however, only limited therapeutic treatments are available. Hence, we investigated the role of cannabinoids as novel therapeutic targets against HCC. This study was conducted to understand the mechanistic basic of WIN55, 212-2, a synthetic cannabinoid on the BEL7402 HCC cell line. The results showed that WIN55, 212-2 could lead to an arrest of BEL7402 cells in the G0/G1 phase of the cell cycle via cannabinoid receptor 2-mediated down-regulation of p-ERK1/2 expression, up-regulation of p27 expression, down-regulation of cyclin D1 and CDK4. We further observed inhibition of CB2 with CB2 antagonist AM630 abrogated the cell cycle arrest by WIN55, 212-2 treatment. Inhibition of ERK1/2 alone also results in cell cycle dysregulation and arrest of cells in G0/G1 phase subsequently resulting in cell growth inhibition. Furthermore, we also observed significant reduction in MMP-9, pRb and E2F1 expression and migration inhibition. Based on these data, we suggest that cananbinoid receptor agonists should be considered as novel targets for the management of HCC..

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