The effect of GWP42006, a cannabinoid extract on MCF-7 human breast carcinoma cells

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Introduction:

In recent years, the anti-tumour potential of cannabinoids has highlighted the importance of this system in the generation of new anti-cancer therapies (Freimuth et al., 2010; Patsos et al., 2005). The aim of the present study was to investigate the potential anti-tumour activity of a cannabinoid extract rich in cannabidivarin on breast tumour cells.

Methods:

MCF-7 cells (American Type Culture Collection) were grown and maintained in RPMI 1640 medium supplemented with 10% fetal bovine serum at 37oC, 5% CO2 .The cells were plated in 96-well culture plates at a density of 1x104 cells/well and allowed to adhere at 37oC for 24 hours. The following day, various doses of extract in the absence and presence of AM251, SR144528 and capsazepine, were added to the cells and further incubated for 4 days. Then the supernatant was removed and MTT (3-(4,5-dimethylthiazol-2-yl)-2, 5-diphenyltetrazolium bromide) was added for 4 hours. The ability of cells to form formazan crystals by active mitochondrial respiration was determined by using a Microplate reader after dissolving the crystals in DMSO. Cytotoxicity was expressed as a relative percentage of the absorbance measured at 540 nm in the control and extract-treated cells. Data were presented as the mean ± s.e.mean and analysed using ANOVA followed by Dunnet's t-test; Each point represents the mean of 4 separate experiments of 8 readings for each dose.

Results:

The extract induced dose-dependent cytotoxic effects on MCF-7 cells with an IC50 of 0.067 mg/ml. Pre-treatment with AM251, SR144528 and Capsazepine, CB1, CB2 and TRPV1 receptor antagonists, respectively, did not reverse the cytotoxicity afforded by the extract. Interestingly, the cytotoxicity was potentiated by the application of AM251 with an IC50 of 0.017± 0.01 mg/ml. Single application of antagonists alone or vehicle did not affect the survival rate of the MCF7 cells. (Figure1).

Conclusion:

The data suggest the unlikely involvement of CB1, CB2 and TRPV1 receptors in mediating extract-induced anti-tumour activity in MCF-7 tumour cells. Further experiments are required to investigate the receptor type/subtypes involvement and the mechanism of cell death.

Acknowledgement:

We thank GW Pharmaceuticals for providing the extract.

Reference:

Freimuth N et al., (2010) J of Pharmacology and Exp Ther 332: 336-344.

Patsos HA et al., (2005) Biochem Soc Trans 33: 712-714.

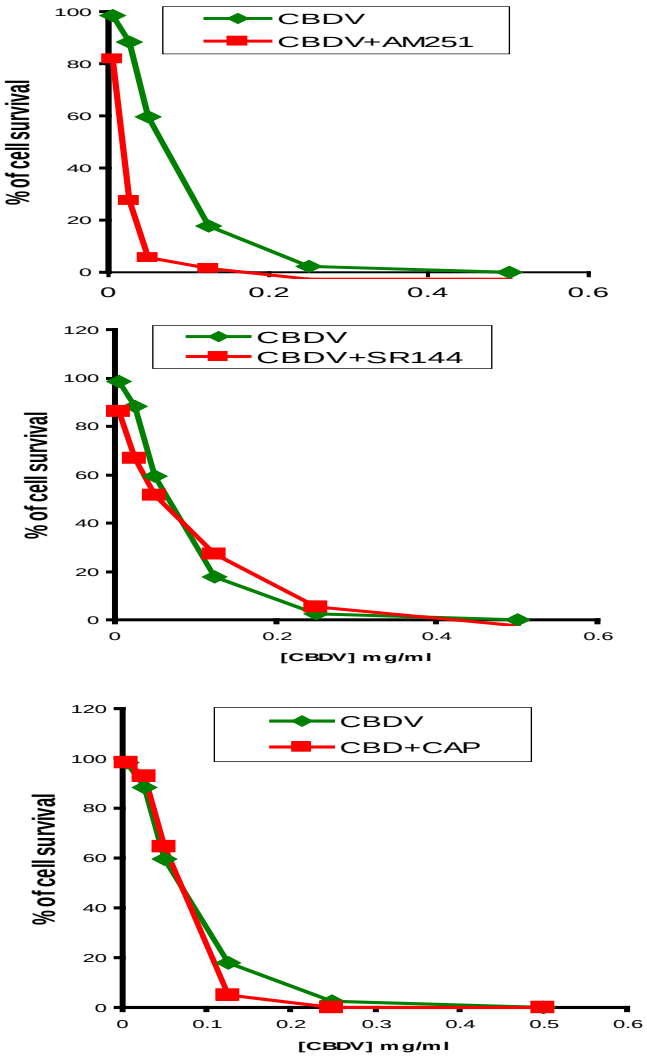

Figure 1. The effect of CBDV on MCF-7 in the absence and presence of CB antagonists.