Analyzing Flavonoid-Induced Apoptosis in Colon Cancer Cells With and Without Functional p53

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Flavonoids, a subclass of polyphenols, are known for their antioxidant and anti-inflammatory properties, potentially reducing the risk of cancer. This experiment tested the ability of five specific flavonoids, catechin-hydrate, genistein, hesperetin, apigenin, and quercetin, one from each of the common subclasses of flavonoids, on colon cancer cells with and without the p53 gene, ultimately to test if various flavonoids use the p53 pathway to induce apoptosis in colon cancer cells. Solutions of the flavonoids ranged from 200μM-5000μM. Cells were cultured until they reached 80-90% confluency and then cells were plated into 96-well plates using a seeding density of .01 X 106 cells/well. After 48 hours, wells were washed with PBS three times to remove all traces of flavonoids, and an MTS assay was performed. Apigenin significantly prevented cell proliferation in both cell lines (ANOVA test and Tukey post-hoc, p=0.00). Quercetin significantly prevented cell proliferation of P53 knock-out (KO) cells (p=0.00), but not in wildtype (p=0.168). Hesperetin significantly prevented cell proliferation in the KO and promoted cell proliferation in the wildtype (p=0.00). Genistein significantly prevented cell proliferation of the wildtype (p=0.00) but not the KO (p=0.14). Catechin-Hydrate did not have any significant difference in both cell lines (p=0.135, p=0.05). Data suggests that specific flavonoids inhibit the growth of colon cancer cells, possibly by inducing apoptosis which was observed through microscopy. Cells were then plated into 96-well plates using a different row order. After 48 hours, an apoptosis assay was performed. Apigenin and Quercetin significantly induced apoptosis in both cell lines.

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