The Toxicity of Kavalactones and Flavokavain A and B from Different Plant Areas of Kava on HepG2 Liver Cells

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Although used by Pacific Islanders for thousands of years, Kava toxicity has been the concern of both the World Health Organization and many European nations, who have banned the import and sale of kava from the United States since 1998. The purpose of this project was to find out the concentrations of kavalactones and flavokavain A & B from the different parts (leaves, stems, lateral roots, and adventitious roots) of two Hawaiian kava plant varieties. This project was also designed to test which part of the kava plant was the most toxic to HepG2 liver cells. The main experimental question was how does the concentration of kavalactones and flavokavains A & B from different plant areas of kava affect the cell death rate in HepG2 liver cells. It was hypothesized that there would be increased levels of Flavokavain A & B in the above ground parts of the plant causing faster HepG2 liver cell death. The experiment was composed of three different parts: 1) HPLC analysis of kava compounds, 2) a Calcein-AM liver cell viability assay, and 3) connecting the HPLC extract concentrations to cell viability as compared to the chemically pure standards. The hypothesis was partially supported by the fact that the leaves had the highest levels of Flavokavain A and B, as well as elevated concentrations of dihydromethysticin, which was the total opposite of all of the other plant areas (stems, adventitious and lateral roots). When converting all of the plant extract concentrations to micromolar concentrations in order to compare them to the standards, this experiment showed that the leaves only had 1/6 of the concentration necessary to cause cell death. This leads to further questions about what else is causing the liver cell death.

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