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Lung tumorigenesis suppressing effects of a commercial kava extract and its selected compounds in A/J mice.

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Abstract

Lung **cancer** is the most deadly malignancy in the US. Chemoprevention is potentially a complementary approach to smoking cessation for lung **cancer** control. Recently, we reported that a commercially available form of **kava** extract significantly inhibits 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK) and benzo(a)pyrene (BaP)-induced lung tumorigenesis in A/J mice at a dose of 10 mg per gram diet. In the present study, we examined the dose-dependent lung tumor inhibitory activities of **kava** and investigated potential active constituent(s). Mice treated with carcinogen alone contained 12.1±5.8 lung adenomas per mouse 22 weeks after final carcinogen administration. Mice that were fed diets containing **kava** at dosages of 1.25, 2.5, 5, and 10 mg/g of diet had 8.4±3.5, 6.6±3.5, 4.3±2.4, and 3.8±2.3 lung adenomas per mouse, respectively. This corresponds to a reduction of 31%, 46%, 65% and 69% in tumor multiplicity, which were all statistically significant ($p < 0.05$). Analyses of lung adenoma tissues derived from **kava**-treated animals revealed that **kava** significantly inhibited adenoma cell proliferation while it had no detectable effect on cell death, indicating that **kava** primarily suppressed lung tumorigenesis in A/J mice via inhibition of cell proliferation. Flavokawains A, B, and C, three chalcone-based components from **kava**, demonstrated greatly reduced chemopreventive efficacies even at concentrations much higher than their natural abundance, suggesting that they alone were unlikely to be responsible for **kava**'s chemopreventive activity. **Kava** at all dosages and treatment regimens did not induce detectable adverse effects, particularly with respect to liver. Specifically, **kava** treatment showed no effect on liver integrity indicator enzymes or liver weight, indicating that **kava** may be potentially safe for long-term chemopreventive application.

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