Inhibition of AFB1-induced liver cancer and induction of increased microsomal enzyme activity by dietary constituents

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Abstract:

A long-term study, using male Wistar rats, was initiated to determine whether the effects of dietary constituents on AFB1-induced liver cancer could be associated with altered microsomal enzyme activity. They were maintained on mice pellets mixed with specific dietary constituents for 7 days and then given a single carcinogenic dose of AFB1 (500 micrograms/rat). After three months, the dietary constituents were discontinued and the animals were left on mice pellets and drinking water only for a period of about 20 months. At the end of the trial period, it was observed that dietary mixtures containing small quantities of either beta-carotene, ascorbic acid, GSH, vitamin E, selenium salt, or uric acid, effectively inhibited the development of AFB1-induced liver cancer and induced increased microsomal enzyme activity. Whereas beta-carotene and uric acid were the most effective inhibitors, vitamin E was the least, yet a significant inhibitor of liver cancer. Hepatic levels of cytochrome P-450, aniline hydroxylase and chlorpromazine demethylase were significantly induced in rats fed fortified food followed by AFB1 treatment than in control animals. The inhibition of liver cancer by dietary factors was probably due to their ability to induce the activity of hepatic microsomal enzymes. Increased enzyme activity could lead to rapid activation of AFB1 metabolism, resulting in loss of activated AFB1 metabolites that attack cell components. Inhibition of liver cancer is therefore associated with induction of increased microsomal enzyme activity.