

**Chemopreventive Effects of Plant Polysaccharides (*Aloe barbadensis* Miller, *Lentinus edodes*, *Ganoderma lucidum*, and *Coriolus vesicolor*)**

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Plant polysaccharides have traditionally been used around the world as a folk remedy for various diseases due to their multiple biological properties including anti-inflammation, wound healing, antihepatitis, antiulcer and antineoplastic effects. Hence, chemopreventive effects of plant polysaccharides (*Aloe barbadensis* Miller (APS), *Lentinus edodes* (LPS), *Ganoderma lucidum* (GPS), and *Coriolus vesicolor* (CPS)) were compared using *in vitro* short-term screening methods associated with both initiation and promotion processes in carcinogenesis. In benzo[a]pyrene-DNA adduct formation, APS (180 µg/ml) was the most effective in the inhibition of B[a]P binding to DNA in mouse hepatocytes. Oxidative DNA damage (8-OHdG) was not significantly decreased by plant polysaccharides. In glutathione S-transferase (GST) activity induction, GPS was found to be the most effective among plant polysaccharides. In screening anti-tumor promoting effects, APS (180 µg/ml) significantly inhibited PMA-induced ornithine decarboxylase (ODC) activity in Balb/3T3 cells. In addition, APS significantly inhibited PMA-induced tyrosine kinase (TK) activity in human leukemic (HL-60) cells. APS and CPS significantly inhibited superoxide anion formation. These results suggest that some plant polysaccharides produced both antigenotoxic and anti-tumor promoting activities in *in vitro* tests and therefore may be considered as potential agents for cancer chemoprevention.

In another study, the antigenotoxic effect of *Aloe barbadensis* Miller (polysaccharide fraction) on benzo[a]pyrene (B[a]P)-DNA adducts was investigated *in vitro* and *in vivo*. Aloe showed the time-course and dose dependent inhibition of [<sup>3</sup>H]B[a]P-DNA adduct formation in primary rat hepatocytes (1x10<sup>6</sup> cells/ml) treated with [<sup>3</sup>H]B[a]P (4 nmol/ml). At concentrations of 0.4 to 250 µg Aloe/ml, the binding of [<sup>3</sup>H]B[a]P metabolites to rat hepatocyte DNA was inhibited by 9.1 to 47.9%. Also, in rat hepatocytes cultured for 3 h to 48 h with Aloe (250 µg/ml) and [<sup>3</sup>H]B[a]P (4 nmol/ml), [<sup>3</sup>H]B[a]P-DNA adduct was significantly reduced by 36% compared with the [<sup>3</sup>H]B[a]P alone. Aloe also inhibited the cellular uptake of [<sup>3</sup>H]B[a]P in a dose-dependent manner with the range of 0.4-250 µg/ml by 6.3-34.1%. After a single oral administration of B[a]P to male ICR mice (10 mg/mouse), benzo[a]pyrene-diol-epoxide-I (BPDE-I)-DNA adducts formation and persistence for 16 days following daily treatment with Aloe (50 mg/mouse) were quantitated by enzyme linked immunosorbent assay (ELISA) using monoclonal antibody 8E11. In this animal model, BPDE-I-DNA adduct formation was significantly inhibited in various organs (liver, kidney, forestomach, and lung) (P <0.001). When mice were pretreated with Aloe for 16 days before B[a]P treatment, inhibition of BPDE-I-DNA adduct formation and persistence was enhanced. By enzyme assay, glutathione S-transferase activities were slightly increased in the liver but cytochrome P-450 contents were not affected by Aloe. These results suggest that the inhibitory effect of Aloe on BPDE-I-DNA adduct formation might have a chemopreventive effect by inhibition of B[a]P absorption.