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Effect of green tea on hepatic enzyme activities and  
mutagenic transformation of benzo[a]pyrene

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Green tea mainly contains catechins and caffeine. Exposure of rats to green tea (2.5% w/v) and crude catechins (0.25% w/v) for four weeks gave rise to a significant increase in the 7-ethoxycoumarin O-deethylase, caffeine N-1 demethylase, caffeine C-8 hydroxylase, UDP-glucuronyl transferase (UDPGT) and Mn-superoxide dismutase (Mn-SOD) activities. Exposure of rats to caffeine (150 mg/kg/day) by gastric gavage for three days also gave rise to a similar increase in these enzyme activities. But exposure of rats to decaffeinated-crude catechins (800 mg/kg/day) by gastric gavage for three days gave rise only to a significant increase in the UDPGT activity. In western blot analysis, the induction of P-450 1A2 was observed in the microsomes from green tea-, crude catechin- and caffeine-treated rats. Metabolic activation of carcinogens such as benzo[a]pyrene (BP) was catalyzed mainly by P-450 1A and detoxified by UDPGT activity. In Ames-test, the levels of mutagenicity induced by BP markedly increased in the S-9 from green tea- or crude catechin-treated rats. When UDPGA was added to the reaction mixture, the number of revertants decreased to the same level as in the control. In another Ames-test using *S. typhimurium* TA102, which is sensitive to active oxygen species, the addition of crude catechins to the reaction mixture resulted in marked decreases in

the number of revertants dose dependently. With lipid peroxidation, no alteration was observed in the S-9 or in the mitochondria from green tea- or crude catechin-treated rats. In general, induction of P-450 1A2 and UDPGT activities is thought to be mediated by Ah receptor. However, according to a recent paper, caffeine induces P-450 1A2 in an Ah receptor-independent manner. When we exposed of C57BL/6 mice, which are Ah receptor-responsive, and DBA/2 mice, which are the Ah receptor-non-responsive, to caffeine (150 mg/kg/day) by gastric gavage for three days there was a significant increase in P-450 1A2 and UDPGT activities in both strains of mice. Our results indicated that caffeine in green tea increased P-450 1A2 and Mn-SOD activities upon exposure to green tea, and that both caffeine and another substance such as catechins increased UDPGT activity. It is known that green tea inhibits P-4501A activity. Our results suggest that green tea could reduce the residence time of exposure to active metabolites or active oxygen species for target organs by inducing P-450 1A2 and UDPGT, and by scavenging active oxygen species or by directly inhibiting P-4501A activity. The exposure to caffeine, which is the major component of green tea, induces P-450 1A2 and UDPGT activities in an Ah receptor-independent manner.