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Carcinogenicity and Modification of the Carcinogenic Response by bha, Bht, and Other Antioxidants

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Abstract

V. SUMMARY

Carcinogenicity tests showed that addition of the antioxidant BHA to the diet of F344 rats induced high incidences of papilloma and squamous cell carcinoma of the forestomach of both sexes. Male hamsters given BHA for 24 weeks also developed papilloma showing downward growth into the submucosa of the forestomach. These results indicate that BHA should be classified in the category of "sufficient evidence of carcinogenicity" as judged by IARC criteria. The 3-*tert* isomer of BHA seemed to be responsible for the carcinogenicity of crude BHA in the forestomach of rats. BHT was not found to be carcinogenic in rats or mice. In two-stage carcinogenesis in rats after appropriate initiation, BHA enhanced carcinogenesis in the forestomach and urinary bladder of rats, but inhibited carcinogenesis in the liver. BHT enhanced the induction of urinary bladder tumors and inhibited that of liver tumors, but had no effect on carcinogenesis in the forestomach. BHT could be a promoter of thyroid carcinogenesis. Sodium L-ascorbate enhanced forestomach and urinary bladder carcinogenesis. Ethoxyquin enhanced kidney and urinary bladder carcinogenesis, but inhibited liver carcinogenesis. Thus, these antioxidants modify two-stage chemical carcinogenesis in the forestomach, liver, kidney, urinary bladder, and thyroid, but show organ-specific differences in effects.

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