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1. Environmental Pollution and Cancer

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Further research needs in environmental carcinogenesis are much to be desired. Currently, the methodology for identification, quantitation, and assay of some environmental carcinogens is available, but there remains a pressing need for additional fundamental knowledge of the carcinogenic process at the molecular level. The best hope for the control of cancer lies in prevention and chemotherapy, and this in turn depends upon the generation of basic information not yet available. The history of medical science reveals that quantum advances in control and prevention of disease have been direct consequences of research applications derived from pools of existing knowledge. It is hoped that the major efforts on specific problems of environmental carcinogenesis and substantial support of research to obtain new knowledge will prove to be equally rewarding.

2. Protection against Environmental Chemical Carcinogens with Antioxidants: Studies on the Anticarcinogenic Mechanism Using Liver as a Model Tissue

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The overall metabolism of environmental chemicals and the tumor formation by chemical carcinogens, depend greatly on activities of enzyme that metabolize them. The metabolism of chemical carcinogens has been divided into two general phases: (a) *metabolic activation* by such reactions as oxidation, and (b) *metabolic deactivation* by conjugation reactions. Current theories indicate that reactive (electrophilic) metabolites produced by the activation reactions interact with nucleophilic sites in critical macromolecules such as DNA, RNA and proteins. Some of these reactions may lead to carcinogenesis and mutagenesis. However, the ultimate fate of these reactive metabolites and their capacity to initiate the neoplastic process depend not only upon their ability to reach and to react with critical macromolecules, but also upon the competing enzymatic reactions by which the reactive metabolites are converted to nontoxic polar products and excreted.

The dietary administration of butylated hydroxy-anisole (BAH) butylated hydroxytoluene (BAT) and some other food additive antioxidants protect against tumor induction by chemical carcinogens

of diverse structures in a variety of rodent tissues. Recent studies particularly dealing with BHA have demonstrated that the feeding of BHA causes: (a) decreases in overall rate of metabolic activation and alterations in metabolic profiles of carcinogens such as benzo (a) pyrene (BP) so that less of the reactive metabolites are produced, and (b) marked increases of several enzyme activities involved in conjugations and deactivation reactions.

These biochemical observations obtained with fractionated liver tissues were extended not only in isolated hepatocytes, but also with isolated perfused livers. Furthermore, the causative relationship between these *in vitro* biochemical observations and their *in vivo* biological effects have been confirmed by using the model carcinogen BP. Thus, it was demonstrated that in hepatocytes isolated from rodents fed BHA and BP, there were dramatic decreases in the amounts of BP metabolites bound to hepato cellular and other tissue DNA *in vivo*. This demonstration of a causative relationship support, at least in part, the BHA dependent reductions in mutagenic and carcinogenic effects of BP and of other chemical carcinogens.

3. Interaction among Cell Organelles

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