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A major problem in cancer research has been that of correlating the results of carcinogenesis studies in animals to humans. The traditional practice of estimating carcinogenic risk by extrapolating animal tumorigenicity data to humans is fraught with assumptions and potential inaccuracies. During the past 10 years, Curtis Harris and coworkers Herman Autrup, John Lechner, Gary Stoner, and Benjamin Trump have made significant strides toward the development of alternative approaches for assessing the effects of environmental agents on humans. Initially, they developed culture systems for the long-term maintenance and growth of human epithelial tissues and cells from various organ sites including lung, colon, bladder, pancreas, esophagus, breast, kidney, mesothelium, and prostate. These tissues, obtained both from surgery and from autopsies, were shown to be capable of metabolizing a series of chemical carcinogens including polycyclic aromatic hydrocarbons, nitrosamines, a fungal toxin, and aromatic amines. Importantly, humans were found to vary markedly in their ability to metabolize carcinogens into forms that cause DNA damage and to repair that damage. This variation could explain, in part, the observed differences in carcinogenic susceptibility among humans. In addition, human tissues were generally found to metabolize carcinogens in much the same way as animal model tissues in which these carcinogens were observed to induce cancer. These results add credence to the use of animal bioassays for assessing carcinogenic risk to humans.

Harris and colleagues were among the first to use biochemical and immunochemical methods to monitor genetic damage in humans exposed to specific environmental chemicals. For example, aflatoxin-DNA adducts were found in higher quantities in the urine of Africans living in a region of Kenya in which there is a high occurrence of liver cancer than in a region with a low occurrence of the disease. These results suggest the importance of aflatoxin exposure in the etiology of liver cancer in Africa. In addition, coke oven workers exposed to high levels of polycyclic aromatic hydrocarbons had higher levels of benzo-(a)pyrene-DNA adducts in their peripheral blood lymphocytes than non-coke oven workers, confirming the relationship between industrial exposure to carcinogens and genetic damage in the exposed individuals. Antibodies reacting with these DNA adducts were also found in human sera. This new field of biochemical and molecular epidemiology shows potential for the identification of individuals at high risk of cancer through exposure to environmental chemicals.

Recent developments by Harris and colleagues have contributed importantly to our understanding of the biology of human epithelial cells and of the response of these cells to various carcinogens, tumor promoters, toxins, and several growth regulatory factors and differentiation agents. Asbestos and nickel sulfate were shown to induce cytotoxic effects and premalignant changes in lung cells such that these cells became nonresponsive to normal growth and differentiation regulatory signals. The tumor promoter 12-O-tetradecanoylphorabol-13-acetate (TPA) enhanced the tendency of human bronchial epithelial cells to undergo squamous differentiation as opposed to stimulating their growth. A recent report on the malignant transformation of human bronchial epithelial cells after transfection with a single oncogene, c-H-ras, is exciting and holds promise for the identification of the molecular events leading to human cell transformation and the relationship of these events to factors controlling cellular growth differentiation.

Pictured are: Curtis C. Harris (top), Herman N. Autrup, John F. Lechner, Gary Stoner, and Benjamin F. Trump (from left to right along bottom). We are indebted to Dr. Gary Stoner for the material and illustrations.