Cancer and geographic risks

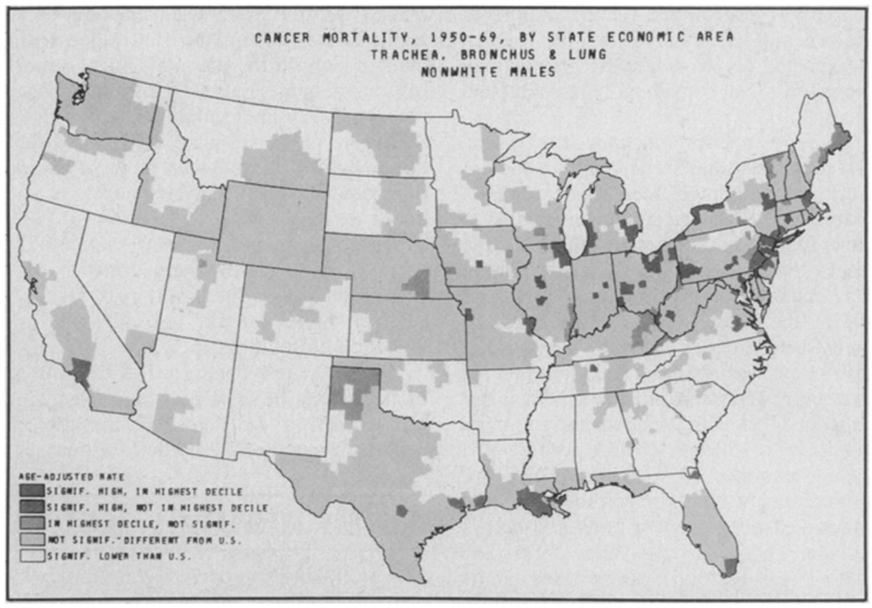
In 1975, National Cancer Institute epidemiologists literally put cancer on the map for the first time, linking death rates from different cancers with various geographic regions of the United States. However, their results, reported in the NCI *Atlas of Cancer Mortality for U.S. Counties 1950–1969*, were only for whites (SN: 5/3/75, p. 286).

Now a similar atlas has been published for cancer deaths among American nonwhites—blacks, American Indians, Chinese and Japanese—entitled *Atlas of Cancer Mortality Among U.S. Nonwhites: 1950–1969*. The authors are Thomas J. Mason, Frank W. McKay, Robert Hoover, William L. Blot and Joseph F. Faumeni Jr.

Comparing findings from both atlases, the authors report that the death rate for all forms of cancer combined for nonwhites is slightly higher than the rate for whites. But what mainly impressed them is the frequent similarity, between whites and nonwhites, of regional death rates from major causes of cancer. For instance, there were certain similarities between whites and nonwhites for cancers of the breast, colon, rectum and esophagus, which showed generally high rates in the North and low rates in the South. Cancers of the larynx, bladder and ovaries also had above-average rates in the North for both groups. What's more, both white and nonwhite males in northern areas experienced high rates of lung cancer, and both white and nonwhite females in rural areas of the South showed above-average rates for cancer of the cervix.

These resemblances in regional cancer death rates, regardless of race or ethnic background, strongly suggest that environmental carcinogens are responsible for them. Some regional differences in cancer mortality rates between whites and nonwhites suggest the same causal relationship. Lung cancer, for example, which is higher among whites along the Gulf and southeast Atlantic coasts, may stem from job discrimination in chemical industries in those areas.

However, certain ethnic groups are more prone to certain cancers than are other groups regardless of geographic location, the atlas reveals. For example, American Indians from diverse geographic locations had relatively high death rates from cancers of the gall bladder, bile ducts and liver. Persons of Japanese ancestry showed above-average death rates from cancer of the stomach, while Chinese had unusually high death rates from cancer of the nasopharnyx—the inner passages of the nose. These statistics suggest a genetic cause for cancer, or a cause due to a particular life-style or diet apart from environmental carcinogens.



"Geographic patterns and associations do not resolve issues of cancer etiology," the authors stress, "but suggest risk factors and communities where further epidemiologic studies should be concentrated." □

Trace elements and cancer

While studying methods of preventing and treating cancer, biologists have generally neglected the effects of essential trace elements—inorganic substances such as zinc, selenium, manganese, and probably even arsenic—that occur in very small amounts as components of living organisms. Now, however, chemists are turning their attention to the influence of trace elements on cancer growth and development. They are finding that some of these substances enhance tumor growth, while others apparently protect against cancer development, according to reports presented at a symposium of the International Association of Bioinorganic Scientists, held in La Jolla, Calif., recently.

A dietary deficiency of the essential nutrient zinc decreases tumor growth in rats and mice. In some of the experiments reported by W. J. Pories of Case Western Reserve University School of Medicine in Cleveland, Ohio, transplanted tumors didn't grow at all in zinc-deficient animals, whereas animals with normal diets developed tumors.

These animal experiments may be relevant to human cancer therapy, since clinical studies indicate that cancer patients with low levels of zinc in their blood fail to respond to anticancer drugs, which usually act when cells are dividing. Pories suggests that zinc therapy might be used in patients to increase tumor cell division temporarily, thereby possibly increasing the responsiveness of the tumors to anticancer drugs.

Contrasting with zinc's stimulation of cancer growth, the trace element selenium apparently prevents tumor development in experimental animals and in people. G. Schrauzer of the University of California at San Diego added different concentrations of selenium to the drinking water of mice that developed mammary tumors spontaneously. With 0.1 part per million (ppm) selenium in their drinking water, 94 percent of mice developed tumors. This is about the spontaneous rate. Among mice with 1 ppm selenium in their drinking water, only 3 percent developed tumors. The protective effect of selenium against tumor development disappeared when the mice also received supplemental zinc.

Might selenium protect people against cancer? Based on analyses of human cancer mortalities throughout the world, Schrauzer finds that selenium intake and cancer mortality are inversely related. In areas where people have relatively high levels of selenium in their blood, or eat diets rich in selenium, overall cancer death rates are lower than in those areas where populations ingest little of this element. Schrauzer said that the optimum amount of selenium in an adult diet is 0.3 milligram per day, and that Americans on the average get half this amount. Cereal grains and seafood contain relatively large amounts of this element.

Further statistical studies of cancer suggest additional relationships between dietary trace elements and cancer mortality. Whereas selenium, and possibly manganese, are associated with low cancer mortality rates, zinc, chromium and cadmium are generally associated with higher cancer mortality. The antagonistic effects of zinc and selenium seen in cancer development in mice, therefore, also seem to apply to people. □