Sharma and Terasaki (School of Medicine, The Center for the Health Sciences, Los Angeles, California) compared the ability of lymphocytes from 193 cancer patients and 163 normal persons to become immunized to cultured human tumor cells and lymphoblast-associated antigens. Lymphocytes of patients with various neoplasms had significantly depressed immune responses compared to those of normal individuals. Responsiveness of cancer patients to cultured tumor cells was much lower than that of controls. No significant difference was found between lymphocytes of cancer patients and normal persons in response to human lymphoblast-associated antigens.

In a study of HL-A heterozygosity as a genetic marker of long-term survival, Gerkins et al. (University of Southern California School of Medicine, Los Angeles, California) used blood specimens from 126 healthy persons, from 66 elderly patients with solid tumors, from 148 normal patients less than 36 years old, and from 72 cancer patients with onset of the disease at less than 36 years of age. HL-A typing was done by the microlymphotoxicity test with 115 highly selective antisera defining 25 HL-A specificities. Old, healthy persons had a larger number of HL-A specificities than did cancer patients or young, healthy persons. Young cancer patients had the fewest specificities. The authors concluded that there was a relationship between heterozygosity of the HL-A system, survival to old age, and a decreased susceptibility to cancer.

Papageorge and associates (National Cancer Institute, Bethesda, Maryland) detected the murine sarcoma virus genome and the group-specific antigen in human amnion and lung cell cultures in the absence of complete virions.

To identify the Ph1 chromosome and the related translocation in patients with chronic myelogenous leukemia, Raposa et al. (Stockholm University and Karolinska Institute, Stockholm, Sweden) used the fluorochrome Hoechst 33258, a bis-benzimidazol derivative. This technique clearly distinguished between chromosomes #21 and 22 of group G and
permitted easy identification of chromosomes #1, 9 and 16. The Ph¹ chromosome arose after a deletion in the long arm of chromosome #22, and this deleted part was probably translocated to the long arm of one of the #9 chromosomes.

July

Using data from 11 colleges of veterinary medicine in the United States and Canada from 1964 to 1972, Priester (National Cancer Institute, Bethesda, Maryland) reported pancreatic islet cell tumors in 27 dogs and two cats. Only in standard poodles were the tumors excessive. In dogs, 17 of the neoplasms were functionally active; five were inactive, and five were not specified. Some characteristics of these lesions were remarkably similar to the pancreatic islet cell tumors in 52 people, reported by Broder and Carter (Annals of Internal Medicine 79: 101-107, 1973): equal distribution by sex, 80 percent functionally active, and occurrence at all ages, but most prevalent in middle age.

Litovitz and Lutzner (National Cancer Institute, Bethesda, Maryland) measured the nuclear contour index of lymphocytes from patients with chronic lymphocytic leukemia (CLL) and the Sézary syndrome. The nuclear contour index was determined as the ratio of nuclear circumference to the square root of the nuclear cross-sectional areas.

The authors postulated that, with this index measurement method, it might be possible to distinguish between normal, bone marrow-derived lymphocytes, and thymus-derived lymphocytes of the Sézary cell. The results expressed quantitatively the variation from the nearly circular cross section of the CLL nucleus to the highly irregular cross section of the Sézary syndrome. Rarely, except in the Sézary syndrome, were circulating indexes found with a nuclear contour index greater than 8.6.

For ultrastructural studies, Yeh and associates (National Taiwan University, Taipei, Taiwan, Republic of China) examined 35 skin biopsy specimens from 30 patients with Bowen’s disease caused by chronic arsenicalism.

The most prominent changes of the Bowen’s lesion were disorganization of tonofilaments, increased incidence of mitosis, giant cells, intracytoplasmic desmosomes, and vacuolar degeneration of keratinocytes. The acanthotic epidermis of Bowen’s disease was composed chiefly of less differentiated, basal cell-like elements that could be divided into three types according to the differences in opacity and density of the nuclear matrix.

The studies also detailed the process of dyskeratosis and postulated the mechanisms of developing intracytoplasmic desmosomes and vacuoles.