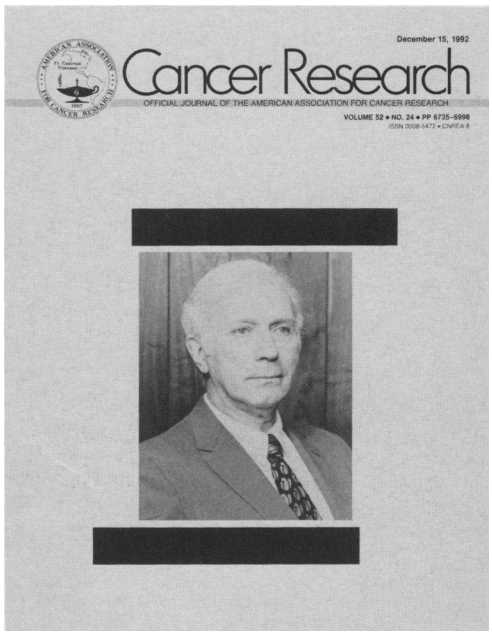
COVER LEGEND



Theodore S. Hauschka, a distinguished early pioneer in the study of the cytogenetics and immunogenetics of cancer, is featured on the cover of this issue of *Cancer Research*. Dr. Hauschka defined cytogenetic parameters in cancer and developmental disorders in mice and humans. His review of tumor immunity appeared in Cancer Res., *12*: 615, 1952; his work on tissue genetics of neoplastic cell populations in Can. Cancer Conf., *2*: 305, 1957; and his nuclear comparison of ontogeny and oncogeny in Cancer Res., *21*: 957, 1961.

Examining the influence of chromosome abnormalities on the histocompatibility and growth rates of mouse ascites carcinomas, sarcomas, and lymphomas, he found that lethal growth of diploid tumors (40 chromosomes) was limited to the inbred strains in which they originated. Heteroploid neoplasms (75 to over 80 chromosomes) transgressed immunogenetic host barriers. Karyotypic imbalance inhibited expression of cellular antigens and reduced or abolished the absorption of H-2 hemagglutinins from sensitive isoantisera (Ann. NY Acad. Sci., *69*: 561, 1957; J. Natl. Cancer Inst., *19*: 13, 1957).

Progressing malignancy requires competition between karyotypically diverse clones, which were microisolated by i.p. injection of single viable cells into newborn mice (Trans. NY Acad. Sci. Ser. II, *16*: 64, 1953; J. Natl. Cancer Inst., *21*: 77, 1958). Nineteen Ehrlich and Krebs-2 ascites clones (E and K) were established in serial passage. Their chromosome numbers were hypotetraploid, except in the case of clone E1 which kept its mode above 80. Extralong, minute, or metacentric chromosomes and hemoglobin content "marked" the different clones. The antigenic diversity of the Ehrlich clones was shown by varied histocompatibility with 129/Rr mice which were killed by E-stock and E1, but survived injection of E5 or E8 cells which immunized them against the otherwise virulent E-stock.

The murine TA-3 ascites, derived from a mammary carcinoma in an A/Ha female, was serially transplanted and lethal only in A/Ha mice. After immunoselection in resistant hosts, it outgrew iso- and heteroimmune barriers and became lethal for mice of seven foreign genotypes and rats of four strains (J. Natl. Cancer Inst., *47*: 343, 1971). Several histocompatibility antigens expressed in the original mouse tumor were irreversibly deleted.

Drug-sensitive mast cell, lymph node, and plasma cell tumors were compared with their resistant and dependent sublines. All of these mouse ascites tumors were diploid. While the altered response to 6-diazo-5-oxonorleucine and cortisone coincided with gross structural chromosome differences, the identical karyotypes of the three plasmacytoma sublines indicated point mutations from amethopterin sensitivity to resistance and dependence [J. Cell. Comp. Physiol., *52*(Suppl. 1): 197, 1958].

Dr. Hauschka proposed Y-linkage of a histocompatibility gene to explain the unexpected rejection of male skin isografts by female C57BL mice (Transplant. Bull., *2*: 154, 1955). Isologous female anti-male sera agglutinated and lysed male leukocytes. In refractory females, tolerance of male isografts was induced by X-ray or by the injection of male cells soon after birth (Genetics and Cancer, p. 271. Austin, TX: University of Texas Press, 1959).

Such studies extended to humans showed that the chromosome constitution of marrow from 34 leukemias was significantly more aneuploid (56%) than that in 60 nonleukemic marrows (12%). Chemotherapy did not enhance chromosome anomalies (Cancer Res., *21*: 678, 1961).

The first XYY man on record, twice married to normal XX women, produced 5 abnormal progeny among his 10 offspring, including instances of amenorrhea and mongolism. This suggested familial predisposition to chromosomal nondisjunction, exhibited quite frequently in cultures of his own skin (Am. J. Hum. Genet., *14*: 22, 1962).

Drs. B. Amos, G. Klein, A. Levan, A. Sandberg, and L. Weiss collaborated in some of the above work. We are indebted to Dr. Hauschka for the information and the photograph.

Dr. Hauschka was born in Austria and, after a classical education, emigrated to the United States and graduated *magna cum laude* from Princeton University in 1935. He earned the M.S. in protozoology at the University of Pennsylvania in 1941 and the Ph.D. in cytogenetics in 1943. He spent the years from 1943 to 1954 at the Institute for Cancer Research (now a part of the Fox Chase Cancer Center), and in 1954 moved to Roswell Park Memorial Institute at Buffalo, NY, as Director of Cancer Research (biology) and Director of the West Seneca Laboratories, the mouse-breeding facilities for the Institute. Dr. Hauschka was chairman of the Academic Program in Biology in the Roswell Park Graduate Division of the State University of New York at Buffalo. Until the time of his retirement from Roswell Park in 1975, he maintained his administrative position as Director of Research as well as his academic rank of Research Professor of Biology.

Dr. Hauschka is a member of numerous professional societies, has served on many committees and task forces, and was President of the AACR in 1959–1960. He received the Sigma Xi award from the State University of New York at Buffalo in 1968 and an honorary doctorate of science from Bates College in 1984. He has published over 100 papers as author and coauthor.

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