AMERICAN ASSOCIATION FOR CANCER RESEARCH
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Cancer Research

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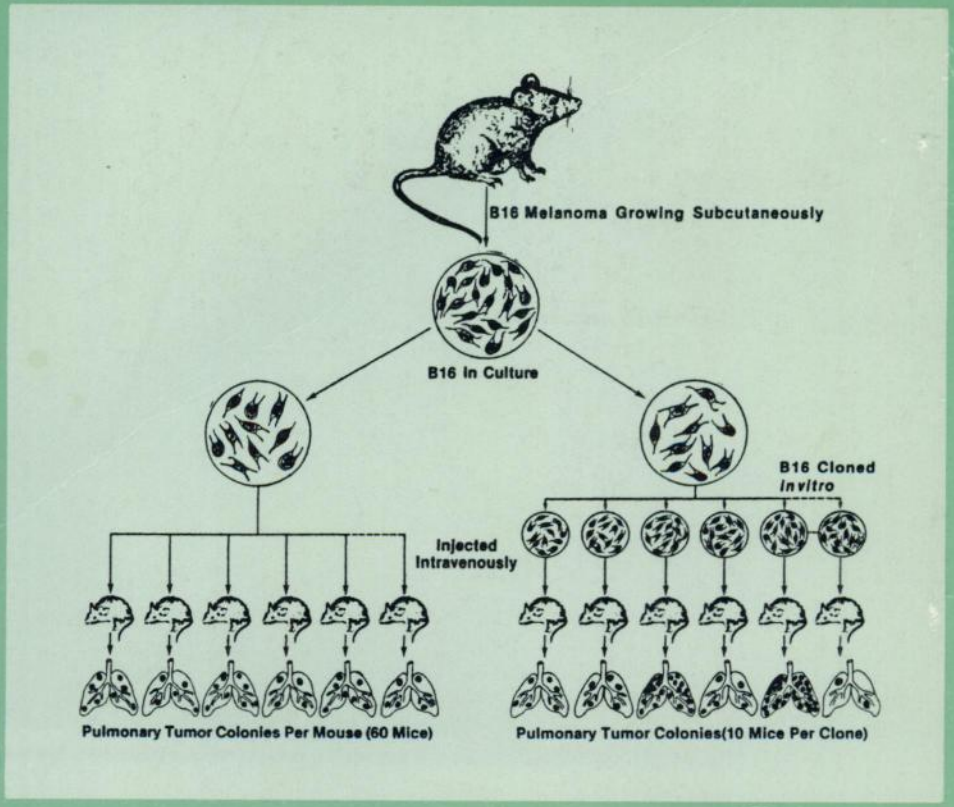

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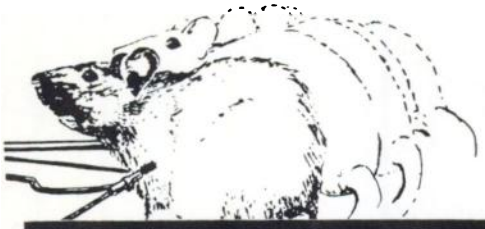

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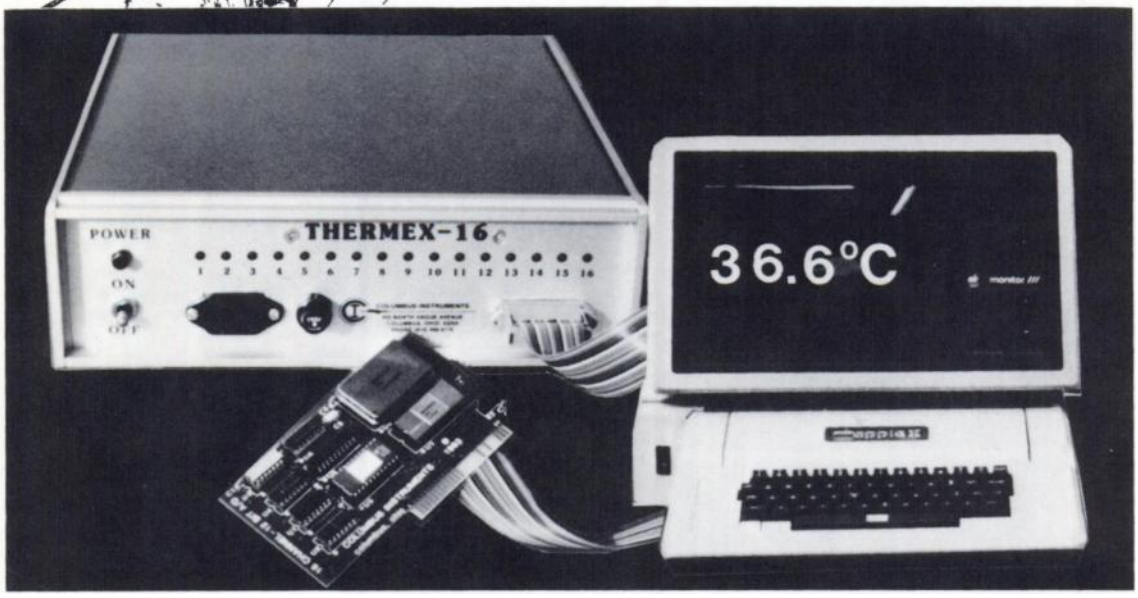

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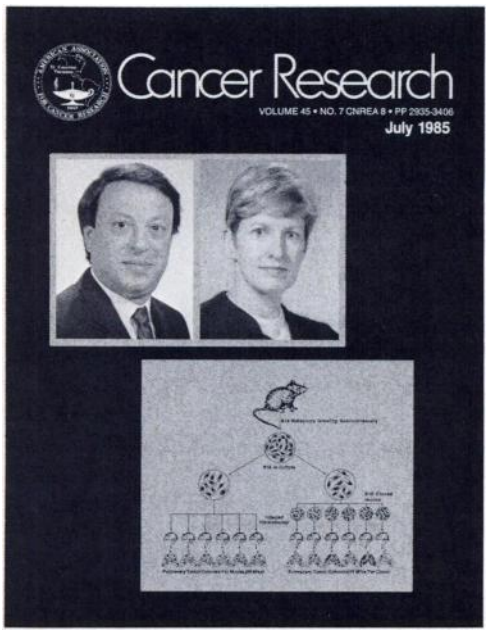

The most devastating aspect of cancer is the propensity of cells from malignant neoplasms to disseminate from their primary site to distant organs, where they develop into metastases. Despite remarkable advances in surgical treatment of primary neoplasms and aggressive adjuvant therapies, most cancer patients die of metastatic disease. The most formidable obstacle to the successful treatment of disseminated cancer may well be the fact that the cells of a tumor are biologically heterogeneous. This phenotypic diversity, which allows selected variants to develop from the primary tumor, means not only that primary tumors and metastases can differ in their responses to treatment but also that individual metastases differ from one another. This diversity can be generated rapidly even when the tumors originate from a single transformed cell.

One of the important goals of today's cancer research is to better understand the mechanisms responsible for the spread of neoplastic cells and the generation of phenotypic diversity in primary and secondary neoplasms.

The first direct evidence that metastases do not result from the random survival of cells released from the primary tumor but, instead, derive from the selective outgrowth of specialized subpopulations of cells comes from the work of Dr. Isaiah J. Fidler and Dr. Margaret L. Kripke [Science (Wash. DC), *197:* 893, 1977], who applied a modification of the classical fluctuation analysis of Luria and Delbruck [Genetics, *28:* 491, 1943] first to the murine B_{16} melanoma and then to other murine tumors of recent origin. Fidler and Kripke reasoned that, if tumors were populated by cells with similar metastatic potential, then isolated clones would produce equal numbers of metastatic foci. In contrast, if tumors were populated by cells with different metastatic potential, then different clones would produce different numbers of metastases. They injected cells obtained from mass culture and from isolated single cell clones into syngeneic mice and found that the cloned sublines differed markedly from the parent tumor and among themselves in the number of pulmonary tumor colonies produced. Control subcloning experiments proved that this diversity was not a result of the cloning procedure *per se* and established that populations of cells with different metastatic capacities existed within the original tumor. By the time of diagnosis, many malignant neoplasms are heterogeneous and contain subpopulations of cells with different biological characteristics.

The generation of biological diversity in malignant neoplasms within and among their metastases has profound implications for research on the pathogenesis of cancer metastasis as well as the design of any successful approach to the treatment of this disease.

Pictured are: Isaiah J. Fidler, D.V.M., Ph.D., who was born in Jerusalem in 1936 and obtained the Doctor of Veterinary Medicine degree from Oklahoma State University in 1963 and the Ph.D. in Pathology from the University of Pennsylvania in 1970; and Margaret L. Kripke, Ph.D., who was born in California in 1943 and obtained her Ph.D. in Immunology from the University of California in Berkeley in 1970. Drs. Fidler and Kripke are married.

The chart shows that neoplasms are heterogeneous and contain cells with different biological properties [Science (Wash. DC), *197:* 893, 1977].

M. B. S.

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