

Morris Hepatomas: Mechanisms of Regulation (Advances in Experimental Medicine & Biology (Springer))



In 1960, Dr. Van R. Potter and Dr. Henry Pitot (at McCardle Laboratory in Madison, Wisconsin), Dr. Tetsuo Ono (then at McCardle Laboratory and now at the Japanese Foundation for Cancer Research in Tokyo, Japan) and Dr. Harold P. Morris (then at the National Cancer Institute and now at Howard University, Washington, D. C.) decided that an experimental cancer model would be an invaluable tool to examine neoplastic changes in cells. Since they were studying the various highly specific metabolic processes which are unique to liver tissues, they determined that a transplantable liver cancer model would be the ideal system to work with. This system would provide for comparison of normal liver tissue of the non-tumor bearing animal, the tumor bearing animals (host) liver and the liver cancer. Dr. Morris undertook a series of rat studies employing several chemicals known to cause liver cancer. Soon the first Morris hepatomas (#3683, 3924A, 5123) were being studied by several labs. During the next 18 years, Dr. Morris developed and transplanted numerous strains of hepatomas of which no two were identical. These tumors ranged from the very slowly-growing, highly differentiated cancer tissues, e. g. , 9618A which is a diploid tumor containing glycogen and a nearly normal complement of enzymes, to a large group of rapidly-growing, poorly differentiated cancer tissues, e. g.

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