CANCER 3

A COMPREHENSIVE TREATISE

BIOLOGY OF TUMORS: Cellular Biology and Growth

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Preface

As was shown in the first two volumes of this series, great strides have been made in identifying many of the agents or classes of substances responsible for carcinogenesis and in delineating their interactions with the cell. Clearly, the aim of such studies is that, once identified, these agents can be eliminated from the environment. Yet, despite these advances and the elimination of some important carcinogenic agents, one major problem exists. It is a constant monitor of all oncologic study and diminishes the importance of every experiment and of every clinical observation. *As we noted earlier, that problem is our inability to define the malignant cell.* It is through studies of the fundamental biology of tumors that we seek this definition.

A vast amount of information has been gathered which describes *what* this cell does and—to a lesser extent—*how* it does it. But the *why* evades us. We have been unable to define the malignant cell, save in broad terms by comparing it to its normal counterpart. The major problem appears to be that the malignant cell does so much. It is a chimera, mystifyingly composed of normal activities and structures, of phenotypic schizophrenia with embryonic, fetal, and adult characteristics and, occasionally, a hint of an unclassifiable capacity unique to malignant cells.

The clues as to the *why* of cancer function must be derived directly or by induction from the *what* and *how*. Malignant cells replicate when replication is not required. Whether by escaping the normal inhibitory controls of the host, or by suprasensitivity to stimulation, or by some defect other than these, the tumor grows. The growth is noncompensatory and nonfunctional. The malignant cell also lives beyond its normal span. Together, growth and increased life span result in disruptive cellular accumulation. And what is more, malignant cells compete rather than participate with their normal neighbors and then competitively invade and destroy. The malignant cell itself evades destruction by humoral, immunological, and cellular defense mechanisms. It is therefore characterized by autonomous behavior, living off the host rather than with it. Are these abnormal activities the result of a single alteration or many? One integrated pattern or many? And what genetic or epigenetic or genetic–epigenetic alteration is responsible for this successful, this deadly capacity? An examination of the biology of

tumors presented in Volumes 3 and 4 serves many purposes. It may enable us to better understand normal cell biology. It may suggest crucial cellular alterations induced by carcinogenic agents. And, by understanding its aberrations of control and the advantages thus gained by the malignant cell, we may be better able to find a means of reversing them and halting their destructive processes.

New York Frederick F. Becker

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to Volume 3

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Contents

General Concepts

Ectopic Isoenzymes: Expression of Embryonic Genes in Neoplasia 3

WILLIAM H. FISHMAN AND ROBERT M. SINGER

Ectopic Hormone Secretion by Tumors 4

WILLIAM D. ODELL AND ADA WOLFSEN

Tumor Progression 5

DANIEL MEDINA

Growth of Tumors

Cell Division and Tumor Growth 8

CHARLES LIGHTDALE AND MARTIN LIPKIN

Stimulation 9

JOSEPH LOBUE AND MILAN POTMESIL

Endocrine Factors and Tumor Growth 10

KELLY H. CLIFTON AND BHAVANI N. SRIDHARAN

Chalones (Specific and Endogenous Mitotic Inhibitors) and Cancer 11

J. C. HOUCK AND A. M. ATTALLAH

Extracellular Compartments of Solid Tumors 12

PIETRO M. GULLINO

Tumor Angiogenesis 13

JUDAH FOLKMAN

Regulation of Energy Metabolism in Normal and Tumor Tissues 14

CHARLES E. WENNER

DNA-Nucleoproteins 15

GIOVANNI ROVERA

Protein Synthesis 16

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(内部交流)

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