between cancer and somatic mutation lose much of their significance.

But leaving aside this uncertainty, strong evidence against the somatic mutation hypothesis is comprised in quantitative studies of the induction of cancers by ultraviolet light. Repeated exposures to ultraviolet light are necessary to induce tumors. If it is assumed that each dose of radiation produces one or more mutant cells each of which proliferates by "unlimited cell division" to form a clone of cancer cells, it may be calculated that (if the proliferation rate is comparable to the growth rate measured after the tumors have appeared) only the first few clones would have any appreciable effect on the growth of the tumor. The rapid proliferation of the initial clones would swamp out any late-appearing clones, and hence after the first few exposures ultraviolet light should have no effect on the time of appearance of the tumor. Yet doses of ultraviolet light continue to accelerate tumor appearance long after the time they should be ineffective if we were dealing with somatic mutations. Furthermore, the data indicate that the process of carcinogenesis is continuous throughout the whole period of tumor development from the first dose of ultraviolet radiation to the appearance of the tumor, and that no sharp separation into periods of induction and growth can be made. These findings are difficult to fit with any form of the somatic mutation hypothesis.2

Most of the reasoning regarding the somatic mutation hypothesis disregards the quantitative aspects of tumor growth, and this constitutes an important weakness. Knowledge of the character of the growth rate is essential to the successful extrapolation back in time to the origin of cancer; and in this regard all cancer theories are extrapolations. The lack of such knowledge is a principal hindrance to our understanding the nature of cancer.

Taking these things into account it seems that ² H. F. Blum: On the mechanism of cancer induction by ultraviolet radiation, *Journal of the National Cancer Institute*, 11, 463-495 (1950). The evidence against the somatic mutation hypothesis presented in this paper is derived independently of the author's own hypothesis of progressive acceleration of growth rate, which is also described there.

there is strong evidence against the somatic mutation hypothesis of cancer; that there is a great deal to be learned before we can accept this or any other hypothesis; and that basic studies of growth and differentiation would seem logical (though perhaps far from smooth) avenues of approach to the problem of carcinogenesis.

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In his review, "A Reconsideration of the Somatic Theory of Cancer" in Science, (117, 441-445 [1953]), John C. Fardon fails to call attention to certain recent publications which indicate that the "manifestations of cellular anarchy," which he ascribes to mutations, may in part be consequent upon the activities of a pathogenic microorganism of pleomorphic nature. The presence of this microorganism has been revealed by special methods described in the two papers listed below, which state that some of its forms are of viral dimensions, and that these forms have been demonstrated within the cytoplasm and the nuclei of cancer cells obtained from human as well as from animal specimens.

(1) "Cultural Properties and Pathogenicity of Certain Microorganisms Obtained from Various Proliferative and Neoplastic Diseases," by Virginia Wuerthele-Caspé, M.D., Eleanor Alexander-Jackson, Ph.D., James Hillier, Ph.D., Roy M. Allen, D.Sc., and Lawrence W. Smith, M.D. American Journal of the Medical Sciences, 220, 638-648 (1950).

(2) "Some Aspects of the Microbiology of Cancer," by Virginia Wuerthele-Caspé, M.D., Eleanor Alexander-Jackson, Ph.D., and Lawrence Weld Smith, M.D. Journal of the American Medical Women's Association, 8, 7-12 (1953).

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Insect Physiology. Kenneth D. Roeder, Ed. New York: Wiley; London: Chapman & Hall, 1953. 1100 pp. Illus. \$15.00.

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