The war on cancer engenders a minor revolution

Several years ago, the National Cancer Institute laid on considerable hype concerning the possibility that viruses might be the principle cause of human malignancies. Since the hype meshed with the Nixon war on cancer, the end result was a considerable infusion of research funds engendering tantalizing claims, annoying counter claims, and eventual disillusionment. Some of those funds fortunately found their way into basic research laboratories, however, where they fueled what can only be described as a quiet minor revolution in our understanding of the malignant cell. My purpose today is to instruct you in the particulars of that revolution, to alert you to the fact that we may be on the brink of identifying both the genetic elements whose aberrations initiate the oncogenic process and enzymatic mechanisms that can engineer the malignant phenotype.

Slide

The genesis of tumors in living creatures is a complex affair: the course of events is protracted; and by all accounts, several distinct and sequential events must combine to produce a tumorigenic cell. The same is true of cells in culture, exposed to a chemical carcinogen, as outlined in this diagram. The action opens with an initiating event - a mutation, perhaps, or a chromosomal rearrangement, or even an epigenetic change in the cell. The cell then proceeds through a series of incaemental steps that eventuate - after much time - in the malignant phenotype. Until recently, the biochemical mechanisms that underlie this entire scheme has been an enigma, a black box. But in the past half-dozen years, the use of tumor viruses as models systems has parted the thicket
The view we have gained was embodied in a remarkable philosophy offered up in 1973 by the medical geneticist David Comings. Comings took stock of the mounting evidence that at least certain human tumors can on occasion behave as autosomal dominant genetic burdens - even as autosomal dominant traits. He posited the existence of a family of genes - dubbed Tr - for transformation whose normal role was to direct the growth and development of cells in one or another embryological lineage. The activity of these genes - like that of most other genes - was modulated by other genetic loci Comings called "i" for inhibitor. If the Tr genes were une unleashed as a consequence ....

Further remarkable prediction that viruses ...

The prescience of Comings' proposal is extraordinary - it provides ...

Retroviruses