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Sustained proliferation in cancer: Mechanisms and novel therapeutic targets

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Proliferation is an important part of cancer development and progression. This is manifested by altered expression and/or activity of cell cycle related proteins. Constitutive activation of many signal transduction pathways also stimulates cell growth. A number of natural compounds have been found to inhibit one or more pathways that contribute to proliferation. Many of these compounds are nontoxic at doses that inhibit tumor growth and/or prevent the appearance of tumor. These include polyphenol compounds, such as curcumin, resveratrol, genistein, and indole-3-carbinol. However, a key to their efficacy involves their earliest possible therapeutic application. This is because their efficacy is likely to be the greatest in target tissue prior to the appearance of tumor where cellular heterogeneity is the least. In addition, many of the steps in carcinogenesis prior to tumor appearance are epigenetic in nature, and are more easily targeted by existing compounds, most of which target wild type molecules. This approach limits adaptive resistance, since early intervention doesn't have to deal with the issues of aneuploidy, loss of heterozygosity in multiple tumor suppressor genes, and point mutations in oncogenes. The contribution of bioinformatics analyses will also be very important for identifying signaling pathways and molecular targets that may provide early diagnostic markers and/or critical targets for the development of new drugs or drug combinations that block tumor formation. Thus, early intervention in pathways and molecules that mediate sustained proliferative signaling will limit adaptive resistance because it targets cells in tissues that have limited genotypic and phenotypic heterogeneity.

Biography

Mark Feitelson received his PhD from the UCLA School of Medicine in 1979 and was then an American Cancer Society Postdoctoral Fellow in the Department of Medicine at Stanford University. In 1982, he was recruited to the Fox Chase Cancer Center by Dr. Baruch S. Blumberg, who won the Nobel Prize for the discovery of hepatitis B virus (HBV), eradication of HBV from the blood supply, and formulation of the first prophylactic vaccine. In 1991, he joined Thomas Jefferson University as an Associate Professor, and then as Professor of Pathology, Anatomy and Cell Biology. At Jefferson, he directed the Molecular Pathology Lab in Microbiology (CAP certified). In 2007, he joined the Department of Biology at Temple University, and more recently became director of the Professional Science Master's Program in Biotechnology at Temple. He has published well over 120 papers in the peer reviewed literature, including two books and numerous invited reviews. His research spans the pathogenesis of chronic hepatitis B and C infections and the relationship of these viruses to the development of liver cancer. His research has been supported all this time by grants from the National Institutes of Health, from private foundations, and from pharmaceutical companies. He has carried out extensive collaborations with scientists from the People's Republic of China.

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