



Cancer and Oncogenesis

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Letter to Editor

The promotion of cell division cycle and resistance to apoptosis are key properties of cancer cells. Genes that encode proteins that induce the cell division or promote resistance to apoptosis and cell survival are called proto-oncogenes. Proto-oncogenes could be converted to oncogenes by activating many kinds of mutations. Over expression of proto-oncogene products are results of mutations on proto-oncogene noncoding regions. For induction the oncogenic effect in a cells, activation of a single allele of proto-oncogene is enough. Activating of mutations in proto-oncogenes are autosomal dominant for promotion of a cancer. There are numerous protooncogene products including, growth factors, growth factors receptors, Geb-like adaptor molecules, Src like tyrosine kinases, kinases of MAP cascades, Cdks, cyclins, CAKs, Cdc25s and transcription factors such as Jun, Fos, Myc and E2F that increase the expression of cell division cycle proteins promote the cell division and survival. Elevation of the anti apoptotic or survival proto-oncogenes such as Bcl-2 and Mdn-2 has been took place in many cancers. In 30% of human cancers, the prominent oncogene is activated ras. Conversely, products of tumor suppressor genes suppress the cell division or promote apoptosis. P53 is the most major tumor suppressor protein which promotes the apoptosis with DNA damage. In human cancers P53 gene is mostly inactivated or deregulated in the human cancer. P21 is another tumor suppressor that in hibit the cell cycle progression on DNA damage. BH proteins including Bax, Bak and Bad are also tumor suppressors. Inactivation of tumor suppressor genes could eliminate their activity and lead to appearance of cancer phenotype in a cell. It is worthy to note that environmental, cultural factors and lifestyle are predominant causes of human cancer. Their effect on cancer incidence is interpreted through a genetic background. Susceptibly of individuals to environmental factors is influenced by their genetic polymorphism and kinds of mutations that control metabolic, hormonal and regulatory pathways.

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