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Carcinogenesis as a Defect in the Cell Interactions

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Editorial Note

Carcinogenesis, also known as carcinogenesis or tumorigenesis, is the development of cancer in which normal cells are converted into cancer cells. This process is characterized by cell, heredity, epigenetic changes, and abnormal cell division. Cell division is a physiological process that occurs in almost all tissues under different circumstances. Normally, the balance between proliferation and programmed cell death is maintained in the form of apoptosis to ensure tissue or organ integrity. According to the generally accepted theory of carcinogenesis, somatic mutation theory, DNA mutations, and epimutations leading to cancer disrupt process programming and disrupt the normal balance between proliferation and cell death. By letting they confuse these orderly processes. This leads to uncontrolled cell division and the evolution of these cells through natural selection within the body. Only certain mutations cause cancer, but most mutations do not. Mutations in hereditary genes can make people more susceptible to cancer. In addition, environmental factors such as carcinogens and radiation cause mutations that can contribute to the development of cancer. Finally, random errors in normal DNA replication can cause carcinogenic mutations. Usually, a series of mutations in a particular class of genes are required before normal cells can turn into cancer cells. For example, on average, 15 "driver mutations" and 60 "passenger" mutations are found in colon cancer. Mutations in genes that regulate cell division, apoptosis (cell death), and DNA repair can cause uncontrolled cell proliferation and cancer.

Cancer is basically a disease that regulates tissue growth. In order for normal cells to become cancer cells, they need to modify genes that regulate cell growth and differentiation. Genetic and metamorphic changes range from the acquisition or loss of whole chromosomes to mutations that affect a single DNA nucleotide or activation