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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 of microRNAs that control the expression of 100-500 genes can occur at many levels. Genes affected by these changes can be broadly divided into two categories. Oncogenes can be normal genes that are improperly expressed at high levels, or altered genes that have new characteristics. In any case, expression of these genes promotes the malignant phenotype of cancer cells. Usually, when tissue is damaged or infected, the damaged cells cause inflammation by stimulating specific patterns of enzymatic activity and cytokine gene expression in surrounding cells. Individual clusters of molecules (cytokine clusters) are secreted, acting as mediators and inducing the activity of a cascade of subsequent biochemical changes. Each cytokine binds to a specific receptor of a different cell type, and each cell type changes the activity of the intracellular signal transduction pathway depending on the receptor expressed by the cell and the signal transduction molecule present in the cell.

Overall, this reprogramming process induces gradual changes in cell phenotype, ultimately leading to restoration of tissue function and restoration of essential structural integrity. This allows tissue to heal in response to productive communication between the cells present at the site of injury and the immune system. An important element of healing is the regulation of cytokine gene expression. This allows a complementary group of cells to respond to inflammatory mediators in a way that causes gradual and significant changes in tissue physiology. Cancer cells have permanent or reversible changes in their genome that partially interfere with communication with surrounding cells and the immune system. Cancer cells do not communicate with the tissue microenvironment in a way that protects tissue integrity. Instead, cancer cells can move and survive where they can interfere with tissue function. Cancer cells usually survive by "rebuilding" signaling pathways that protect tissues from the immune system. This change in immune response is also evident in the early stages of malignancy.