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|  | Right now there are limited treatments for cancer patients. High energy radiation targets the tumor with a laser, and chemotherapy uses drugs that kill rapidly dividing cells, including the cancer, but also stomach lining cells and hair follicle cells, leaving patients with stomach pains and loss of hair.    The need for new cancer treatments is being concentrated on the study of the genome. Cancer runs in some families because their genomes are similar to each other’s and may be more likely to develop certain cancer causing genes.    Breast cancer is second most common type of cancer in U.S. and studies show that breast cancer is more likely to affect women who have a family history of breast cancer. BRCA 1 and 2 are important genes involved in breast cancer. Mutations in either gene increase the risk for developing breast cancer, because both are tumor suppressor genes, and their wild-type alleles suppress breast cancer (Biology, Mitchell, Reece, and Campbell).    The study of these and other genes associated with inherited cancer may lead to new methods for early diagnosis and treatment of all cancers. One example, the Ras oncogene, has a point mutation that leads to the hyperactive version of the Ras protein, which leads to excessive cell division. The normally functioning Ras protein relays a growth signal from a growth factor receptor on the plasma membrane of a cell, directing the synthesis of other proteins that stimulate the cell cycle. Ras proto-oncogene is critical to the regulation of cell division, so if it is amplified, the excessive cell division will lead to a tumor. Ras is found in 30% of human cancers, showing that this gene indeed plays an important role.    The p53 protein is a tumor suppressor gene found in 50% of human cancers. p53 gene is normally expressed when there is damage to the cell’s DNA. p53 activates a signal to halt the cell cycle to allow time for the cell to repair the damaged DNA. If the DNA is irreparable, p53 activates suicide genes to cause cell death by apoptosis. Therefore, if p53 is missing, damaged DNA will go uncorrected, and cancer may ensue.    It takes, however, more than one mutation to cause cancer. In fact, multiple mutations underlie the development of cancer. Since cancer results from an accumulation of mutations, the older an organism is, the more likely it is to get cancer. For a cell to be cancerous, there must be at least one oncogene, and the mutation or loss of several tumor-suppressor genes. Loss of tumor suppressor genes is recessive, so both alleles must block tumor suppression, but oncogenes behave as dominant alleles.    Cancers due mainly to dominant oncogenes are the most likely targets for drug therapy. Overexpression can be attacked by antibodies. Tumors caused by mutations in tumor-suppressor genes are harder to treat because these result from the loss of a normal protein. Among the approaches now being attempted is a reintroduction of the p53 gene into a tumor. With p53, the damaged DNA will be repaired and abnormal cells will die by apoptosis. (Molecular Cell Biology Ch.24)  <[Back](http://docs.google.com/intro2.html)><[Next](http://docs.google.com/intro4.html)> |
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