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|  | A proto-oncogene is normal cellular gene that will become an oncogene when it gains a dominant function mutation. There are several ways in which a proto-oncogene can be mutated to become an oncogene: either by rearrangement of DNA within the genome, by amplification of a proto-oncogene, or by point mutation. Rearrangement of DNA is what occurs when malignant cells are frequently found to contain chromosomes that have broken and rejoined incorrectly, translocating fragments from one chromosome to another. This may cause important genes to be placed in the wrong location, so the cell has no regulation and can become cancerous. The second way a proto-oncogene can be mutated into an oncogene is amplification. Increases in the number of copies of an oncogene will cause too many signals to be made, too many proteins to be built, and so the cell may become cancerous. The third way is a point mutation. A point mutation changes the gene’s protein product to one that is more active or more resistant to degradation than the normal protein.  The tumor-suppressor gene is a normal cellular gene that will become an oncogene when there is a recessive loss-of-function mutation. Some tumor suppressor genes make protein products that inhibit cell division, and its protein products normally help prevent uncontrolled cell growth. If these genes are not expressed, then excessive growth will lead to cancer. Some tumor suppressor proteins normally repair damaged DNA, which prevents cancer causing mutations. To have a tumor suppressor gene that repairs damaged DNA become mutated is traumatic for the cell because now it has no protection against common mutations. Another tumor suppressor gene controls adhesion of cells to each other or to the extra cellular matrix. If one of these genes is mutated, the cancer cells will not have proper cell anchorage, and are likely to pile loosely on top of each other.    Cancer cells do not respond normally to the body’s control mechanisms. By dividing excessively they can kill the organism. Normal cells exhibit density dependent inhibition, which means they will stop dividing and the tissue will stop growing after a certain point. But cancer cells do not stop dividing. Culture cancer cells will divide indefinitely if they are given proper nutrients. The breast cancer cells from Henrietta Lacks are still dividing today in culture since 1951 (Biology, 221). The immortality of many cancer cells is caused by a gene that regulates telomerase on the ends of chromosomes. Normally, a telomere functions as a tandem array of a short DNA sequence, TTAGGG. Telomeres provide the solution of the inability of DNA polymerases to completely replicate the end of a double stranded DNA molecule. In cancer, the enzyme telomerase prevents erosion of the ends of the chromosomes, thus removing a natural limit on the number of times the cells can divide. Specific inhibitors of telomerase have been suggested as cancer therapeutic agents.  Rapidly dividing cells form a tumor, a mass of abnormal cells within normal tissue. The problem begins when a single cell undergoes transformation, and if it escapes body’s immune system, it will continue to divide rapidly to form a tumor. The tumor is benign if it remains in one site, and the lump can be removed by surgery. The tumor is malignant if it can impair the functions of organs. This is defined as cancer. Cancer cells may separate from the original tumor, enter blood and lymph vessels, and proliferate to form more tumors, which is a process called metastasis.  <[Back](http://docs.google.com/intro.html)><[Next](http://docs.google.com/intro3.html)> |
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