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Colon cancer

The human genes mutated in some colon cancers are homologous to enzymes in the DNA mismatch repair pathway in the E. coli bacterium (above) as well as yeast and mice.

The American Cancer Society estimates that there will be 93,800 new cases of colon cancer diagnosed in the US in 2000, with 47,700 resulting deaths. All kinds of cancer occur when cell division, normally a very highly regulated process, gets out of control. While environmental factors can certainly contribute to a person's risk of cancer (e.g. smoking, diet, and exercise), most cancers have a genetic basis too. Literally hundreds of genes and proteins are involved in monitoring the process of cell division and DNA replication; a mutation in one or more of these genes or proteins can sometimes lead to uncontrolled cancerous growth.

Colon cancer is one of the most common inherited cancer syndromes known. Among the genes found to be involved in colorectal cancer are: *MSH2* and *MSH6* both on chromosome 2 and *MLH1*, on chromosome 3. Normally, the protein products of these genes help to repair mistakes made in DNA replication. If the MSH2, MSH6, and MLH1 proteins are mutated and therefore don't work properly, the replication mistakes are not repaired, leading to damaged DNA and, in this case, colon cancer.

It is not clear why mutations in genes that are essential in all tissues preferentially cause cancer in the colon. However, studies on the equivalent genes in mice and brewer's yeast are helping to further our understanding of the mechanisms of DNA repair and the role that environmental factors might play in colon cancer incidence.

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