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Human Colorectal Cancer and its Genetic Basis

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Description

Colorectal cancer emerges in the colon (colon is the longest part of large intestine) and rectum (lower gastrointestinal tract). Colon cancer evolves through various well characterized morphological stages, which are polyps, benign adenomas, and carcinomas. Various researches showed that colon cancer arises from a series of mutations that commonly occur in a well-defined order. Generally, tumors are of two types (Benign tumor and malignant tumor), If the tumor is cancerous it is called as Malignant Tumor; if tumor is not cancerous it is known as Benign tumor. The initial phase in colon carcinogenesis is loss of a useful APC gene, resulting in formation of on the inside of the colon wall. This is not seen in every case of colon cancer. Same phenotype is the result of different combination of mutations. Mutations may arise through loss of activity in Tumor Suppress genes or accelerated activity by Oncogenes. Polyp cells contain the same mutations in the APC gene, which is a tumor-suppressor gene, and both alleles of the APC gene must carry an inactivating mutation for polyps to form because cells with one wild-type APC gene express enough APC protein to function normally. Myc is produced in absence of APC gene. Cells homozygous for APC mutations proliferate at a rate higher than normal and form polyps.

DNA from different human colon carcinomas generally contains mutations in all these genes including APC gene which result in mutations in the tumor suppressors APC and p53, and an activating mutation in the dominant oncogene K-ras—establishing that multiple mutations in the same cell are needed for the cancer to form.

Some of the mutations have growth advantages at preliminary stage of tumor development, whereas some mutations develop in the final stages, which includes invasion and metastasis. Invasion and metstasis are necessary for the malignant phenotype. The number of mutations needed for colon cancer progression may at first seem surprising, seemingly an effective barrier to tumorigenesis. Colon carcinoma provides an excellent example of the multi-hit mode of cancer. The degree to which this model applies to cancer is only now being learned, but it is clear that multiple types of cancer involve multiple mutations. Cancer cells that split away from a primary tumor are moved around the body by the circulation system or the lymphatic framework. The cells that escape from a primary site and form a secondary site or sites are known as secondary cancers or metastases. The point of origin where genes result in uncontrollable growth to form a tumour, is known as the primary site and Metastasis is the term generally used to indicate spread of cancer. Primary tumors can be differentiated from metastatic tumors by the pattern of gene expression. Recent research studies found that subset of solid primary tumors has been found to have characteristics more typical of metastatic tumors, suggesting that it may be possible to identify primary tumors that have a greater probability of becoming metastatic. This also raises the possibility that, at least for some types of cancer, the initiating events of the primary tumor may set a course toward metastasis.

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