Colorectal Cancer Pathway

Colorectal cancer (CRC) is one of the leading causes of cancer lethality. In the United States, 160,000 new cases of CRC are diagnosed each year, and 57,000 patients die of this disease. The molecular origins of CRC are relatively well-characterized and strongly related to accumulation of genetic mutations. In general, the genetic alterations in CRC progression are determined by one of two separate and distinct underlying pathways of genomic instability: microsatellite instability (MSI) or chromosome instability (CIN).

**CIN Pathway:** CIN occurs in most colon cancers and leads to a distinct pattern of gene alterations that contribute to tumor formation. Genes involved in the CIN pathway are those coding for APC, K-Ras, Smad4 and p53.

**MSI Pathway:** MSI occurs in a smaller number of colon cancers than CIN. It results from defects in the DNA mismatch repair (MMR) system or hypermethylation of the MLH1 promoter. Genes that encode microsatellite repeats, such as β-Catenin, TGFβR2 and Bax, are often found with mutations due to MMR defects.

The CIN and MIS mutations mainly alter the Wnt, EGFR, TGFβR and DCC signaling routes to facilitate proliferation of colorectal carcinoma.

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