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Molecular Basis of Colorectal Cancer: Insights into the Disease Mechanisms

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Description

Colorectal cancer is a complex and multifactorial disease that arises from the accumulation of genetic and epigenetic alterations in the colonic epithelium. This disease is the third most common cancer and the second leading cause of cancer-related deaths worldwide [1]. Despite significant advances in treatment and early detection, the overall survival rate for patients with advanced colorectal cancer remains poor. The exact cause of colorectal cancer is unknown, but there are several risk factors that can increase the likelihood of developing the disease. Age is a major risk factor, as most cases of colorectal cancer occur in people over the age of 50 [2]. Other risk factors include a family history of colorectal cancer, a personal history of inflammatory bowel disease, a diet high in red and processed meats, physical inactivity, obesity, and smoking. The majority of colorectal cancers arise from non-inherited genetic mutations that accumulate over time. Mutations in tumour suppressor genes, such as APC (Antigen cells), Tumor Protein 53 (*TP53*), and Mothers Against Decapentaplegic Homolog 4 (*SMAD4*), and oncogenes, such as Kirsten Rat Sarcoma Viral Oncogene Homolog (KRAS), V-Raf Murine Sarcoma Viral Oncogene Homolog B1 (*BRAF*), and Phosphatidylinositol-4,5-Bisphosphate 3-Kinase Catalytic Subunit Alpha (PIK3CA), are commonly observed in colorectal cancer [3,4]. These genes play important roles in regulating cell proliferation, differentiation, and survival. Mutations in these genes can lead to uncontrolled cell growth and contribute to the development of cancer. The APC gene is mutated in more than 80% of sporadic colorectal cancers [5]. APC encodes a protein that regulates the Wnt (Wingless-related Integration Site) signaling pathway which is important for cell proliferation and differentiation. Mutations in APC lead to the activation of the Wnt pathway and contribute to the development of colorectal cancer. The *TP53* gene is mutated in approximately 50% of colorectal cancers [6,7]. *TP53* encodes a protein that plays a vital role in DNA repair and cell cycle regulation. Mutations in *TP53* can lead to genomic instability and contribute to the development of cancer. The *KRAS* gene (Kirsten Rat Sarcoma Viral Oncogene Homolog) is mutated in approximately 40% of colorectal cancers [8]. *KRAS* encodes a protein that regulates cell growth and differentiation. Mutations in *KRAS* can lead to uncontrolled cell growth and contribute to the development of cancer.

Epigenetic alterations in colorectal cancer

Epigenetic alterations, including DNA methylation, histone modification, and non-coding RNA regulation, also play an important role in the development and progression of colorectal cancer. These alterations can affect gene expression and contribute to the formation of cancer [9,10]. DNA methylation is a common epigenetic alteration observed in colorectal cancer. Aberrant DNA methylation can lead to the silencing of tumour suppressor genes and contribute to the development of cancer. For example, methylation of the promoter region of the MutL Homolog 1 (*MLH1*) gene, which is involved in DNA repair, is commonly observed in colorectal cancer. Histone modifications, such as acetylation and methylation, can also affect gene expression and contribute to the development of colorectal cancer [11]. These modifications can alter the chromatin structure and affect the accessibility of DNA to transcription factors and other regulatory proteins.

Conclusion

Colorectal cancer is a complex disease that arises from the accumulation of genetic and epigenetic alterations in the colonic epithelium. These alterations can affect the expression of genes involved in cell proliferation, differentiation, and survival, leading to the development and progression of cancer. Understanding the molecular basis of colorectal cancer is vital for the development of effective treatments and the identification of new therapeutic targets. Ongoing studies in this field continue to shed light on the mechanisms underlying colorectal cancer and help improve patient outcomes.

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