Commentary

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Oncogenes: The Molecular Warriors of Cancer's Battlefield

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Description

Oncogenes are genes capable of triggering cancer development by promoting cell growth and division, unlike tumor suppressor genes that typically inhibit these processes. They represent a significant focus in cancer research and therapy, as deciphering their roles and regulation can yield valuable insights into cancer development and progression, as well as potential therapeutic targets. The discovery of oncogenes began in the early 20th century through research on retroviruses, which can transform normal cells into cancerous ones. Scientists identified viral oncogenes (v-onc) carried by these viruses, which induce uncontrolled cell growth and contribute to tumor formation. Further investigations revealed that these viral oncogenes are derived from normal cellular genes, known as cellular oncogenes (c-onc), vital for various cell signaling pathways. Among the earliest cellular oncogenes identified was Src, initially discovered in the Rous sarcoma virus, a retrovirus causing cancer in chickens. The src gene encodes a protein kinase that regulates cell growth and proliferation. Mutations in src can result in its continuous activation, promoting unchecked cell division and the formation of tumors.

Role of oncogenes in cancer development

Since the identification of the Src gene, numerous other oncogenes have been discovered across various cancer types. These genes encode proteins involved in diverse cellular processes such as cell cycle regulation, apoptosis, DNA repair, and cell adhesion. Mutations or amplifications in these oncogenes can drive abnormal cell growth and survival, contributing significantly to cancer development and progression. Oncogenes exert their oncogenic effects through multiple mechanisms. They may produce proteins that are constitutively active, promoting excessive cell proliferation or blocking apoptosis. Additionally, oncogenes can activate downstream signaling pathways like Ras-MAPK or PI3K-Akt, which are crucial for cell growth and survival. Furthermore, they may interfere with cellular mechanisms responsible for DNA repair or cell cycle control, leading to genomic instability and facilitating tumor progression. Beyond their role in cancer initiation and advancement, oncogenes are pivotal targets in cancer therapy. Targeted treatments designed to inhibit oncogene activity have demonstrated substantial success in treating specific cancer types. For instance, therapies targeting the BCR-ABL fusion protein in Chronic Myeloid Leukemia (CML) or BRAF kinase in melanoma have significantly improved patient outcomes. These therapies work by selectively blocking the function of oncogenic proteins, thereby curbing tumor growth and proliferation.

Cancer therapy

Targeting oncogenes in cancer therapy poses significant challenges despite their therapeutic potential. These challenges include the development of drug resistance and the heterogeneous nature of tumors. Cancer cells often acquire mutations or activate alternative signaling pathways that enable them to evade the effects of targeted therapies. Additionally, tumors frequently harbor multiple oncogenic alterations, necessitating the use of combination therapies to effectively suppress tumor growth. Targeted therapies, such as small molecule inhibitors and monoclonal antibodies, provide precise mechanisms to block the activity of oncogenic proteins. Examples include Imatinib, which targets the BCR-ABL fusion protein in Chronic Myeloid Leukemia (CML), Trastuzumab targeting *HER2* in *HER2*-positive breast cancers, and BRAF inhibitors addressing mutated BRAF in melanoma. These therapies have revolutionized cancer treatment by specifically inhibiting oncogene-driven pathways, leading to improved outcomes for patients with certain types of cancer. However, ongoing research is essential to overcome the complexities associated with targeting oncogenes and to develop more effective and durable treatments for cancer in the future.