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MYC Oncogene in Cancer Progression and Therapy Resistance

Sarah Reterz*

Department of Pediatric, University of Lausanne, Lausanne, Switzerland

Introduction

The MYC oncogene occupies a central role in the biology of many human cancers, acting as a powerful driver of tumor initiation, progression, and therapeutic resistance. MYC encodes a transcription factor that regulates the expression of a wide array of genes involved in fundamental cellular processes such as proliferation, metabolism, differentiation, and apoptosis. Under normal physiological conditions, MYC activity is tightly controlled to ensure proper cell growth and homeostasis. However, in cancer cells, deregulation of MYC through gene amplification, translocation, increased transcription, or enhanced protein stability leads to persistent activation of MYC-driven transcriptional programs. This aberrant MYC activity fuels uncontrolled cell division, promotes metabolic reprogramming, and supports a malignant phenotype characterized by increased invasiveness and metastatic potential. The pervasive involvement of MYC in cancer biology has made it a focal point for understanding tumor progression and the mechanisms underlying resistance to various forms of therapy [1].

Description

MYC functions primarily as a transcription factor by forming heterodimers with its partner protein MAX, which then bind to specific DNA sequences known as E-boxes to regulate the transcription of target genes. These targets span a broad range of biological pathways, including those governing cell cycle progression, ribosome biogenesis, metabolism, DNA replication, and repair. By coordinating these processes, MYC controls cellular growth and division. In cancer, sustained MYC activation disrupts normal regulatory checkpoints, leading to unchecked proliferation. Moreover, MYC influences the tumor microenvironment by regulating angiogenesis, immune evasion, and interactions with stromal cells, thereby facilitating tumor expansion and progression. The oncogenic potential of MYC is further amplified by its ability to induce genomic instability and epigenetic alterations, which generate additional mutations and epigenetic states that drive tumor heterogeneity and evolution [2,3].

The role of MYC in cancer progression is well documented across a variety of tumor types. In hematological malignancies such as Burkitt lymphoma, chromosomal translocations lead to MYC overexpression, which is essential for tumor development and maintenance. Similarly, in solid tumors including breast cancer, lung cancer, colorectal cancer, and neuroblastoma, MYC amplification or overexpression correlates with aggressive disease, poor prognosis, and increased metastatic capacity. MYC-driven cancers often display hallmark features such as high proliferative indices, resistance to apoptosis, altered metabolism favoring aerobic glycolysis, and increased capacity for invasion and dissemination. These features underscore the multifaceted role of MYC as a master regulator of oncogenic processes that shape tumor biology [4].

In addition to driving tumor growth, MYC contributes significantly to therapy resistance, a major challenge in cancer treatment. Resistance can be intrinsic, present before therapy initiation, or acquired during treatment, often leading to disease relapse and poor clinical outcomes. MYC promotes resistance through multiple mechanisms. It upregulates the expression of genes involved in DNA damage repair and cell survival pathways, enabling cancer cells to withstand cytotoxic insults from chemotherapy and radiation. MYC also modulates cellular metabolism to support energy production and biosynthesis under stress conditions imposed by treatment. Furthermore, MYC-driven alterations in the tumor microenvironment, including suppression of immune responses and promotion of angiogenesis, create a protective niche that shields cancer cells from therapeutic effects. These resistance mechanisms complicate treatment and highlight the need for strategies that effectively target MYC or its downstream pathways [5].

Conclusion

In summary, the MYC oncogene is a pivotal driver of cancer progression and a key mediator of therapy resistance across diverse tumor types. Its ability to orchestrate multiple oncogenic processes, including proliferation, metabolism, genomic instability, and microenvironmental interactions, underpins its central role in tumor biology. While directly targeting MYC remains a formidable challenge, ongoing advances in understanding its regulation and downstream effects are yielding novel therapeutic strategies aimed at overcoming resistance and improving patient outcomes. Continued research and clinical development efforts focused on MYC promise to transform the management of aggressive cancers and offer new hope for patients facing therapy-refractory disease.

Acknowledgement

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Conflict of Interest

None.

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***Address for Correspondence:** Sarah Reterz, Department of Pediatric, University of Lausanne, Lausanne, Switzerland; E-mail: reterzsarah@gmail.com*

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