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Role of Cytoskeletal Proteins in Cancer Cell Mechanics and Migration

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Introduction

Cytoskeletal proteins (KRT6) is a type II cytokeratin, a member of the keratin family of intermediate filament proteins. It is primarily expressed in epithelial tissues and plays a crucial role in maintaining the structural integrity of epithelial cells. KRT6 is known for its involvement in various physiological processes, including wound healing and the response to stress. However, its role in cancer has garnered significant attention in recent years. This article explores the interactions between KRT6 and other cytoskeletal proteins in cancer cells, highlighting the implications for cancer progression and potential therapeutic strategies. KRT6 exists in multiple isoforms (KRT6A, KRT6B, and KRT6C), each encoded by different genes. These isoforms share a high degree of homology and are co-expressed with their type I keratin partners (KRT16 and KRT17) to form intermediate filaments. The primary function of KRT6 is to provide mechanical stability to epithelial cells, allowing them to withstand various forms of stress.

Description

KRT6 expression is typically induced in response to cellular stress, such as injury or inflammation. This induction is regulated by several signalling pathways, including those involving cytokines and growth factors. In cancer, aberrant expression of KRT6 has been observed in various tumor types, including breast, lung, and head and neck cancers, suggesting a role in tumour genesis [1]. Actin is a major component of the cytoskeleton, involved in various cellular processes, including cell shape, motility, and division. KRT6 interacts with the actin cytoskeleton through linker proteins such as plectin and filaggrin. These interactions are crucial for maintaining the structural integrity of the cell and facilitating dynamic changes in cell shape and movement. In cancer cells, the interaction between KRT6 and actin is often deregulated, leading to altered cell morphology and increased invasive potential. Studies have shown that KRT6 overexpression can enhance the migratory and invasive capabilities of cancer cells, partly through its interaction with the actin cytoskeleton. This suggests that targeting the KRT6-actin interaction could be a potential therapeutic strategy to inhibit cancer cell invasion and metastasis [2].

Microtubules are another key component of the cytoskeleton, involved in intracellular transport, cell division, and maintenance of cell shape. KRT6 interacts with microtubules through microtubule-associated proteins (MAPs) and other linker proteins. This interaction is essential for the proper organization of the cytoskeleton and coordination of cellular processes. In cancer cells, the KRT6-microtubule interaction is often altered, contributing to changes in cell division and migration. Deregulation of this interaction can lead to abnormal mitotic spindle formation and chromosomal instability, both of which are hallmarks of cancer. Targeting the KRT6-microtubule interaction could therefore represent a novel approach to disrupt cancer cell division and reduce tumour growth [3]. Intermediate filaments, including other keratins, vimentin, and desmin, interact with KRT6 to form a complex network that provides structural support to the cell. These interactions are mediated by various linker proteins and are essential for the mechanical resilience of epithelial cells.

In cancer cells, the expression and organization of intermediate filaments are often altered, leading to changes in cell stiffness and mechanical properties. The interaction between KRT6 and other intermediate filaments can influence cell behaviour, including migration, invasion, and resistance to mechanical stress. Understanding these interactions could provide insights into the mechanisms of cancer progression and identify potential targets for therapeutic intervention [4]. The interactions between KRT6 and other cytoskeletal proteins play a critical role in cancer progression. Aberrant expression and deregulation of these interactions can contribute to various aspects of tumorigenesis, including increased cell proliferation, enhanced migratory and invasive capabilities, and resistance to apoptosis. The interaction between KRT6 and the actin cytoskeleton is particularly important for cancer cell migration and invasion. Overexpression of KRT6 can lead to enhanced cell motility, facilitating the spread of cancer cells to distant sites. Targeting this interaction could help to inhibit metastasis and improve patient outcomes.

KRT6 can also interact with signaling pathways that regulate apoptosis, the programmed cell death process. In cancer cells, overexpression of KRT6 can contribute to resistance to apoptosis, allowing cancer cells to survive and proliferate despite treatment. Understanding the mechanisms of this interaction could provide new targets for therapies aimed at inducing apoptosis in cancer cells [5]. The interaction between KRT6 and microtubules is crucial for proper cell division. Dysregulation of this interaction can lead to abnormal cell division and increased proliferation, contributing to tumour growth. Targeting the KRT6-microtubule interaction could help to inhibit cancer cell proliferation and reduce tumour size.

Given the crucial role of KRT6 and its interactions with other cytoskeletal proteins in cancer progression, targeting these interactions represents a promising therapeutic strategy. Potential approaches include. Small molecules or RNA-based therapies could be used to reduce KRT6 expression in cancer cells, thereby inhibiting its pro-tumorigenic effects. Targeting the proteins that mediate the interaction between KRT6 and the actin cytoskeleton could help to inhibit cancer cell migration and invasion. Drugs that disrupt the interaction between KRT6 and microtubules could inhibit cancer cell division and reduce tumour growth. Therapies aimed at altering the organization of intermediate filaments could influence the mechanical properties of cancer cells and reduce their invasive potential.

Conclusion

KRT6 plays a critical role in maintaining the structural integrity of epithelial cells and is involved in various physiological processes. In cancer cells, the interactions between KRT6 and other cytoskeletal proteins are often dysregulated, contributing to cancer progression. Understanding these interactions provides insights into the mechanisms of tumorigenesis and identifies potential targets for therapeutic intervention. Future research should focus on elucidating the detailed mechanisms of these interactions and developing novel therapies that target KRT6 and its associated cytoskeletal

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proteins to improve cancer treatment outcomes.

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Conflict of Interest

None.

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