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The Role of KERATIN 6 in in Cancer Cell Mechanics and Migration

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Introduction

Keratin 6 (KERATIN 6) 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. KERATIN 6 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 KERATIN 6 and other cytoskeletal proteins in cancer cells, highlighting the implications for cancer progression and potential therapeutic strategies. KERATIN 6 exists in multiple isoforms (KERATIN 6A, KERATIN 6B, and KERATIN 6C), 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 KERATIN 6 is to provide mechanical stability to epithelial cells, allowing them to withstand various forms of stress.

Description

KERATIN 6 expressions 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 KERATIN 6 has been observed in various tumour types, including breast, lung, and head and neck cancers, suggesting a role in tumour genesis [1]. The interaction between KERATIN 6 and actin is often deregulated, leading to altered cell morphology and increased invasive potential. Studies have shown that KERATIN 6 overexpression can enhance the migratory and invasive capabilities of cancer cells, partly through its interaction with the actin cytoskeleton.

Interaction with cytoskeletal proteins

Actin is a major component of the cytoskeleton, involved in various cellular processes, including cell shape, motility, and division. KERATIN 6 interacts with the actin cytoskeleton through linker proteins such as pectin and flagging. 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 KERATIN 6 and actin is often deregulated, leading to altered cell morphology and increased invasive potential. Studies have shown that KERATIN 6 overexpression can enhance the migratory and invasive capabilities of cancer cells, partly through its interaction with the actin cytoskeleton. This suggests that targeting the KERATIN 6-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. KERATIN 6 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 KERATIN 6-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 KERATIN 6-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 KERATIN 6 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 KERATIN 6 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 KERATIN 6 and other cytoskeletal proteins play a critical role in cancer progression. Aberrant expression and deregulation of these interactions can contribute to various aspects of tumour genesis, including increased cell proliferation, enhanced migratory and invasive capabilities, and resistance to apoptosis. The interaction between KERATIN 6 and the actin cytoskeleton is particularly important for cancer cell migration and invasion. Overexpression of KERATIN 6 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.

KERATIN 6 can also interact with signalling pathways that regulate apoptosis, the programmed cell death process. In cancer cells, overexpression of KERATIN 6 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 KERATIN 6 and microtubules is crucial for proper cell division. Deregulation of this interaction can lead to abnormal cell division and increased proliferation, contributing to tumour growth. Targeting the KERATIN 6-microtubule interaction could help to inhibit cancer cell proliferation and reduce tumour size.

Therapeutic implications

Given the crucial role of KERATIN 6 and its interactions with other cytoskeletal proteins in cancer progression, targeting these interactions represents a promising therapeutic strategy. Potential approaches include. Inhibition of KERATIN 6 expression: Small molecules or RNA-based therapies could be used to reduce KERATIN 6 expression in cancer cells, thereby inhibiting its pro-tumorigenic effects. Disruption of KERATIN 6-actin interaction: Targeting the proteins that mediate the interaction between KERATIN 6 and the actin cytoskeleton could help to inhibit cancer cell migration and invasion. Targeting KERATIN 6-microtubule interaction: Drugs that disrupt the interaction between KERATIN 6 and microtubules could inhibit cancer cell division and reduce tumour growth. Modulation of intermediate filament networks: Therapies aimed at altering the organization of intermediate filaments could influence the mechanical properties of cancer cells and reduce their invasive potential. KRT6, or Keratin 6, is a type of intermediate filament protein that is a member of the keratin family. It plays a crucial role in the structural integrity and mechanical resilience of epithelial cells, including those found in the skin, hair, and nails. KRT6 is often expressed in tissues that

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experience high levels of mechanical stress, such as the palms of the hands and soles of the feet. In the context of cancer cells, KRT6 can have significant implications. Its expression levels and interactions with other cytoskeletal proteins can be altered in various cancers, influencing tumour.

Conclusion

KERATIN 6 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 KERATIN 6 and other cytoskeletal proteins are often deregulated, contributing to cancer progression. Understanding these interactions provides insights into the mechanisms of tumour genesis 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 KERATIN 6 and its associated cytoskeletal proteins to improve cancer treatment outcomes.

Acknowledgement

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Conflict of Interest

None.

References

1. Quintanilla, Miguel, Ken Brown, Martin Ramsden and Allan Balmain, et al. "Carcinogen-specific mutation and amplification of Ha-ras during mouse skin carcinogenesis." *Nature* 322 (1986): 78-80.
2. Yuspa, Stuart H. "The pathogenesis of squamous cell cancer: Lessons learned from studies of skin carcinogenesis." *J Dermatol Sci* 17 (1998): 1-7.
3. Derka, S., E. Vairaktaris, V. Papakosta and S. Vassiliou, et al. "Cell proliferation and apoptosis culminate in early stages of oral oncogenesis." *Oral Oncol* 42 (2006): 540-550.
4. Goutzanis, L., E. Vairaktaris, C. Yapijakis and N. Kavantzas, et al. "Diabetes may increase risk for oral cancer through the insulin receptor substrate-1 and focal adhesion kinase pathway." *Oral Oncol* 43 (2007): 165-173.
5. Vairaktaris, E., S. Spyridonidou, V. Papakosta and A. Vylliotis, et al. "The hamster model of sequential oral oncogenesis." *Oral Oncol* 44 (2008): 315-324.

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