Commentary
Volume 07:04, 2024

Journal of Biomedical and Pharmaceutical Sciences

ISSN: 2952-8100 Open Access

Implications of Drug on Cytoskeletal Proteins in Cancer Cell Mechanics

Aamiro Cortez*

Department of Otolaryngology and Maxillofacial Surgery, St. Vincent De Paul Hospital, 1 Wójta Radtkego St., 81-348 Gdynia, Poland

Introduction

Cytoskeletal proteins play a pivotal role in cancer cell mechanics, influencing cellular shape, movement, and interactions with the extracellular matrix. These proteins, including actin, tubulin, and intermediate filaments, are critical for processes such as cell migration, invasion, and metastasis. In cancer, aberrant regulation of cytoskeletal dynamics often leads to enhanced migratory capabilities and altered cell adhesion properties, which contribute to tumour progression and resistance to therapies. Understanding the mechanisms by which cytoskeletal proteins affect cancer cell behaviour provides valuable insights for drug development. By targeting specific cytoskeletal components or their regulatory pathways, researchers can develop novel therapeutic strategies aimed at disrupting cancer cell movement and metastasis. 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

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 pectin and flagging. These interactions are crucial for In cancer cells, the interaction between KRT6 and actin is often deregulated, leading to altered cell morphology and increased invasive potential maintaining the structural integrity of the cell and facilitating dynamic changes in cell shape and movement.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 tumour types, including breast, lung, and head and neck cancers, suggesting a role in tumorigenesis [1]. 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 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. 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].

In cancer cells, 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 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].

KRT6 can also interact with signalling 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 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 [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

For instance, drugs that inhibit actin polymerization or stabilize

***Address for Correspondence:** Aamiro Cortez, Department of Otolaryngology and Maxillofacial Surgery, St. Vincent De Paul Hospital, 1 Wójta Radtkego St., 81-348 Gdynia, Poland; E-mail: cortez@Aamiro234.eu*

Received: 02 July, 2024, Manuscript No. jbps-24-146749; **Editor Assigned**: 04 July, 2024, PreQC No. P-146749; **Reviewed**: 16 July, 2024, QC No. Q-146749; **Revised**: 22 July, 2024, Manuscript No. R-146749; **Published**: 29 July, 2024, DOI: 10.37421/2952-8100.2024.7.474

microtubules have shown promise in preclinical studies and clinical trials. These approaches not only aim to impede cancer cell spread but also to enhance the efficacy of existing treatments. As our knowledge of cytoskeletal proteins and their roles in cancer continues to evolve, targeted therapies that address these molecular targets hold the potential to improve patient outcomes and revolutionize cancer treatment paradigms. 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. KRT6 plays a critical role in maintaining the structural integrity of epithelial cells and is involved in various physiological processes. Future research should focus on elucidating the detailed mechanisms of these interactions and developing novel therapies that target KRT6 and its associated cytoskeletal proteins to improve cancer treatment outcomes.

Acknowledgement

None.

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.

How to cite this article: Cortez, Aamiro. "Implications of Drug on Cytoskeletal Proteins in Cancer Cell Mechanics." *J Biomed Pharm Sci* 7 (2024): 474.