Opinion
Volume 09:06, 2024

Journal of Molecular Histology and Medical Physiology

ISSN: 2684-494X

Open Access

How Cancer Cells Spread: The Mechanism of Metastasis

Evans Gad*

Department of Oncology and Metabolism, University of Sheffield, Sheffield S10 2RX, UK

Introduction

Cancer metastasis is the process by which cancer cells spread from their original (primary) location to other parts of the body, forming secondary tumors. This ability of cancer cells to invade distant organs and tissues is one of the hallmarks of cancer, making it far more challenging to treat. The spread of cancer involves a complex series of events that enable cancer cells to move through the body, evade the immune system, and establish secondary growths. Understanding how cancer cells spread has been a critical focus in cancer research because this process is responsible for the majority of cancer-related deaths. The first step in the metastatic process is the ability of cancer cells to invade the tissue surrounding the primary tumor. Cancer cells can acquire the ability to degrade the Extracellular Matrix (ECM), which is the structural framework that supports tissue architecture.

Description

This degradation is facilitated by the secretion of enzymes such as Matrix Metalloproteinases (MMPs) that break down the ECM and allow cancer cells to penetrate surrounding tissues. As these cells detach from the primary tumor, they gain access to local blood vessels or lymphatic vessels, both of which serve as routes for cancer cells to travel to distant sites. This transition from a non-invasive state to an invasive one is often mediated by changes in cell adhesion properties. Cancer cells lose their normal cell-to-cell adhesion by down regulating the expression of adhesion molecules like E-cadherin, which helps maintain the integrity of normal tissue structures. Once in the bloodstream or lymphatic system, cancer cells encounter new challenges. In the circulatory system, cancer cells are subjected to mechanical shear stress, which can damage or even kill them. However, many cancer cells are able to survive this stress by forming clusters or emboli with platelets or other blood cells, which help shield them from the immune system and improve their chances of survival. Platelets can also promote cancer cell survival by secreting growth factors that protect the cancer cells from apoptosis, the programmed cell death that normally occurs in the face of such stress [1,2].

Additionally, cancer cells have to overcome immune surveillance mechanisms. The immune system is constantly monitoring the body for abnormal cells, including cancer cells, and attempts to eliminate them. However, many cancer cells develop mechanisms to evade the immune system. For example, some cancer cells may express proteins that suppress the immune response, such as PD-L1, which binds to PD-1 receptors on immune cells and prevents them from attacking the cancer cells. Other mechanisms include the secretion of immunosuppressive cytokines or the recruitment of immune cells that actually support tumor growth rather than combat it, such as regulatory T cells or myeloid-derived suppressor cells [3]. Once cancer cells have successfully navigated the bloodstream or lymphatic system, they must then extravagate, or exit, from the vasculature and invade the target organ. Extravasation is facilitated by further changes in the adhesion properties of cancer cells, as they adhere to the endothelial cells lining blood vessels.

They can then penetrate the blood vessel wall through the action of proteases, similar to the mechanisms that allow them to invade surrounding tissue at the primary site. Once outside the blood vessel, the cancer cells must once again interact with the ECM of the new tissue. In this step, the cancer cells can either remain in a dormant state or begin to proliferate and form new tumors, depending on the microenvironment of the secondary site. The microenvironment of the secondary tissue plays a crucial role in determining whether cancer cells will successfully establish metastatic growth. In some organs, the conditions may not be conducive to tumor growth, and cancer cells may die before they can form a secondary tumor. However, in other organs, the microenvironment may be more favourable. For example, the liver and lungs are common sites of metastasis for many cancers, including breast, colon, and lung cancer.

These organs provide a rich blood supply, as well as growth factors and other components in the ECM that support the survival and proliferation of metastatic cancer cells. The ability of cancer cells to adapt to and manipulate these microenvironments is one of the reasons metastasis is so difficult to treat. In the secondary tumor, cancer cells often undergo a process of reprogramming, where they alter their metabolic processes to adapt to the new conditions. Many metastatic cells shift to a more glycolytic form of metabolism, known as the Warburg effect, even in the presence of oxygen. This shift enables cancer cells to generate the energy and biosynthetic materials needed for rapid growth and proliferation. Furthermore, cancer cells in secondary tumors can influence the surrounding stromal cells, including fibroblasts, endothelial cells, and immune cells, to create a more tumor-friendly environment. For example, cancer cells can stimulate fibroblasts to secrete growth factors, collagen, and other extracellular matrix proteins, which in turn support the expansion of the tumor [4].

A key feature of metastasis is its selective nature. Not all cancer cells that enter the bloodstream or lymphatic system are capable of forming secondary tumors. Many cancer cells fail to survive the journey, either due to immune system attack, mechanical damage, or insufficient support from the microenvironment in the distant tissue. However, those that do survive and manage to establish secondary growths often display a heightened ability to adapt to different tissue environments. This ability is facilitated by a process known as Epithelial-to-Mesenchymal Transition (EMT), where epithelial cancer cells lose their characteristic cell polarity and adhesion properties, becoming more migratory and invasive in the process. This allows them to infiltrate distant tissues and form secondary tumors [5].

Metastasis is not only a result of the inherent characteristics of the cancer cells but is also influenced by the interplay between the tumor and its microenvironment. Tumors secrete various signalling molecules that recruit stromal cells, such as macrophages, fibroblasts, and endothelial cells, which play roles in supporting tumor growth and facilitating metastatic spread. For example, Cancer-Associated Fibroblasts (CAFs) can secrete cytokines and growth factors that enhance tumor cell proliferation and angiogenesis (the formation of new blood vessels), providing the tumor with a blood supply that is critical for its growth. Additionally, these stromal cells can create a favourable environment for metastasis by remodelling the extracellular matrix and producing factors that promote tumor cell migration and invasion.

***Address for Correspondence:** Evans Gad, Department of Oncology and Metabolism, University of Sheffield, Sheffield S10 2RX, UK; E-mail: evansgad@gmail.com*

Received: 01 November, 2024, Manuscript No. jmhmp-25-157355; **Editor Assigned:** 04 November, 2024, PreQC No. P-jmhmp-25-157355; **Reviewed:** 15 November, 2024, QC No. Q-jmhmp-25-157355; **Revised:** 21 November, 2024, Manuscript No. R-jmhmp-25-157355; **Published:** 28 November, 2024, DOI: 10.37421/2684-494X.2024.9.256

Conclusion

In conclusion, the process of metastasis is a multifaceted and highly orchestrated series of events that enable cancer cells to spread throughout the body and establish secondary tumors. From the initial invasion of surrounding tissues to the colonization of distant organs, metastasis involves a range of cellular behaviours, including changes in cell adhesion, migration, and survival. Understanding the molecular and cellular mechanisms that underlie metastasis is essential for developing targeted therapies aimed at preventing or treating metastatic cancer. Despite the challenges, advances in cancer research continue to shed light on the complex biology of metastasis, offering hope for improved treatments and better outcomes for patients with metastatic cancer.

Acknowledgement

None.

Conflict of Interest

None.

References

1. Danielsson, Frida, McKenzie Kirsten Peterson, Helena Caldeira Araújo and Franziska Lautenschläger, et al. "Vimentin diversity in health and disease." *Cells* 7 (2018): 147.
2. Baldovini, Chiara, Giulio Rossi and Alessia Ciarrocchi. "Approaches to tumor classification in pulmonary sarcomatoid carcinoma." *Lung Cancer* (2019): 131-149.
3. Kidd, Martha E., Dale K. Shumaker and Karen M. Ridge. "The role of vimentin intermediate filaments in the progression of lung cancer." *Am J Respir Cell Mol Biol* 50 (2014): 1-6.
4. Zhang, Yujie and Branko Stefanovic. "LARP6 meets collagen mRNA: specific regulation of type I collagen expression." *Int J Mol Sci* 17 (2016): 419.
5. Wu, Jianping, Qian Xie, Yanjun Liu and Yanan Gao, et al. "A small vimentin-binding molecule blocks cancer exosome release and reduces cancer cell mobility." *Front Pharmacol* 12 (2021): 627394.

How to cite this article: Gad, Evans. "How Cancer Cells Spread: The Mechanism of Metastasis." *J Mol Hist Med Phys* 9 (2024): 256.