



## **Implications of Mitochondria Transfer for Cancer Therapy and Metastatic Potential**

**Wei Yoon\***

*Department of Radiation Oncology, China Medical University, Taichung, Taiwan*

## **DESCRIPTION**

Mitochondria, often referred to as the "powerhouses" of the cell, play a pivotal role in cellular energy production, signaling and metabolism. In recent years, research has revealed that mitochondria are not only limited to functioning within individual cells but can also be transferred between cells, influencing various biological processes. This phenomenon of mitochondria transfer, in which mitochondria or their components move from one cell to another, has drawn significant attention in cancer research.

Understanding how mitochondria transfer affects cancer behavior may provide insights into how cancer cells evade the immune system, resist treatment and metastasize. Mitochondria transfer in cancer biology is an area of intense research due to its implications in altering the energy metabolism, genetic stability and overall functionality of cancer cells.

Mitochondria transfer can occur through different mechanisms, including tunneling nanotubes, extracellular vesicles and direct cell-to-cell contact. Tunneling nanotubes are thin, tubular structures that form between cells, allowing the direct transfer of cellular components, including mitochondria, from donor to recipient cells. This form of intercellular communication has been observed in various types of cells and appears to be an efficient means by which cancer cells can acquire functional mitochondria from surrounding cells [1-3].

Extracellular vesicles, such as exosomes and microvesicles, also facilitate mitochondria transfer. These vesicles, which are small particles released from cells, can contain whole mitochondria or mitochondrial DNA. When these vesicles fuse with a target cell, they release their contents, potentially altering the cellular behavior and metabolism of the recipient cell.

Direct cell-to-cell contact is another mechanism whereby mitochondria transfer occurs. This process involves a physical connection between cells, allowing the transfer of mitochondria or their components. This mechanism is less common than tunneling nanotubes and extracellular vesicles, yet it still plays a role in specific cell types and environments, especially in the cancer microenvironment [4-6].

One of the most significant impacts of mitochondria transfer in cancer biology is its effect on cellular metabolism. Cancer cells often exhibit altered metabolism, known as the Warburg effect, which is characterized by increased glycolysis even in the presence of oxygen. While this shift in energy production favors rapid cell proliferation, it also limits the efficiency of energy production. Mitochondria transfer can alter this metabolic profile, allowing cancer cells to benefit from oxidative phosphorylation and improved energy production.

In cases where cancer cells acquire healthy mitochondria from neighboring cells, their metabolic flexibility can be enhanced. These new mitochondria can increase the energy supply, promoting cell survival and resistance to metabolic stress. This metabolic adaptation is advantageous for cancer cells, especially in tumor microenvironments where nutrient availability is limited. Furthermore, mitochondria transfer enables cancer cells to switch between glycolysis and oxidative phosphorylation based on environmental conditions, enhancing their adaptability and survival potential.

Mitochondria transfer has been implicated in the development of drug resistance in cancer cells. Cancer treatment often targets cellular pathways involved in proliferation and metabolism, aiming to disrupt the growth and survival of cancer cells. However, mitochondria transfer can enable cancer cells to withstand these treatments, contributing to therapy resistance [7-10].

When cancer cells receive functional mitochondria from surrounding cells, they often gain an enhanced ability to resist drugs that target mitochondrial function or oxidative phosphorylation. These new mitochondria can compensate for damage inflicted by chemotherapy or radiation, helping cancer cells to survive treatment. Additionally, mitochondria transfer can lead to the repair of damaged mitochondrial DNA within

**Correspondence to:** Wei Yoon, Department of Radiation Oncology, China Medical University, Taichung, Taiwan, E-mail: we@yoon.cn

**Received:** 27-Aug-2024, Manuscript No. BEG-24-27541; **Editor assigned:** 29-Aug-2024, PreQC No. BEG-24-27541 (PQ); **Reviewed:** 12-Sep-2024, QC No. BEG-24-27541; **Revised:** 19-Sep-2024, Manuscript No. BEG-24-27541 (R); **Published:** 26-Sep-2024, DOI: 10.35248/2167-7662.24.12.269

**Citation:** Yoon W (2024). Implications of Mitochondria Transfer for Cancer Therapy and Metastatic Potential. J Bio Energetics. 12:269

**Copyright:** © 2024 Yoon W. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

cancer cells, restoring cellular functions that may have been impaired by drug treatment.

## **CONCLUSION**

Mitochondria transfer is an emerging field of study in cancer biology, with significant implications for understanding and controlling cancer behavior. By transferring mitochondria from one cell to another, cancer cells gain enhanced metabolic capabilities, resistance to treatment and the ability to invade and metastasize. These advantages contribute to cancer progression, making mitochondria transfer a target of interest in developing new cancer therapies. Efforts to disrupt mitochondria transfer mechanisms or target transferred mitochondria could provide new avenues for improving cancer treatment outcomes. As research in this area continues to evolve, a deeper understanding of mitochondria transfer may lead to innovative strategies for combating cancer and improving patient prognosis.

## **REFERENCES**

- 1. Klein K, He K, Younes AI, Barsoumian HB, Chen D, Ozgen T, Mosaffa S, Patel RR, Gu M, Novaes J, Narayanan A. [Role of](https://www.frontiersin.org/journals/immunology/articles/10.3389/fimmu.2020.573326/full?redirect=false) [mitochondria in cancer immune evasion and potential therapeutic](https://www.frontiersin.org/journals/immunology/articles/10.3389/fimmu.2020.573326/full?redirect=false) [approaches](https://www.frontiersin.org/journals/immunology/articles/10.3389/fimmu.2020.573326/full?redirect=false). Front Immunol. 2020;11:573326.
- 2. Lee YG, Park DH, Chae YC. [Role of mitochondrial stress response](https://www.mdpi.com/2073-4409/11/5/771) [in cancer progression](https://www.mdpi.com/2073-4409/11/5/771). Cells. 2022;11(5):771.
- 3. Srinivasan S, Guha M, Kashina A, Avadhani NG. [Mitochondrial](https://www.sciencedirect.com/science/article/pii/S0005272817300051) [dysfunction and mitochondrial dynamics-The cancer connection](https://www.sciencedirect.com/science/article/pii/S0005272817300051). Biochim Biophys Acta Bioenerg. 2017;1858(8):602-614.
- 4. Oliveira GL, Coelho AR, Marques R, Oliveira PJ. [Cancer cell](https://www.sciencedirect.com/science/article/pii/S0925443920303641) [metabolism: Rewiring the mitochondrial hub.](https://www.sciencedirect.com/science/article/pii/S0925443920303641) Biochim Biophys Acta Mol Basis Dis. 2021;1867(2):166016.
- 5. Zhang L, Zhang W, Li Z, Lin S, Zheng T, Hao B, et al. [Mitochondria dysfunction in CD8+ T cells as an important](https://link.springer.com/article/10.1186/s13046-022-02439-6) [contributing factor for cancer development and a potential target](https://link.springer.com/article/10.1186/s13046-022-02439-6) [for cancer treatment: A review](https://link.springer.com/article/10.1186/s13046-022-02439-6). J Exp Clin Cancer Res. 2022;41(1): 227.
- 6. Chen F, Xue Y, Zhang W, Zhou H, Zhou Z, Chen T, et al. [The role](https://link.springer.com/article/10.1007/s10555-024-10211-9) [of mitochondria in tumor metastasis and advances in](https://link.springer.com/article/10.1007/s10555-024-10211-9) [mitochondria-targeted cancer therapy.](https://link.springer.com/article/10.1007/s10555-024-10211-9) Cancer Metastasis Rev. 2024;1-25.
- 7. Jin P, Jiang J, Zhou L, Huang Z, Nice EC, Huang C, et al. [Mitochondrial adaptation in cancer drug resistance: Prevalence,](https://link.springer.com/article/10.1186/s13045-022-01313-4) [mechanisms, and management](https://link.springer.com/article/10.1186/s13045-022-01313-4). J Hematol Oncol. 2022;15(1):97.
- 8. Bai R, Cui J. [Mitochondrial immune regulation and anti-tumor](https://www.sciencedirect.com/science/article/pii/S030438352300174X) [immunotherapy strategies targeting mitochondria](https://www.sciencedirect.com/science/article/pii/S030438352300174X). Cancer letters. 2023;564:216223.
- 9. Zong Y, Li H, Liao P, Chen L, Pan Y, Zheng Y, et al. [Mitochondrial](https://www.nature.com/articles/s41392-024-01839-8) [dysfunction: Mechanisms and advances in therapy.](https://www.nature.com/articles/s41392-024-01839-8) Signal Transduct Target Ther. 2024;9(1):124.
- 10. Filippi MD, Ghaffari S. [Mitochondria in the maintenance of](https://ashpublications.org/blood/article/133/18/1943/260448/Mitochondria-in-the-maintenance-of-hematopoietic) [hematopoietic stem cells: New perspectives and opportunities](https://ashpublications.org/blood/article/133/18/1943/260448/Mitochondria-in-the-maintenance-of-hematopoietic). Blood. 2019;133(18):1943-1952.