Cancer cells consume themselves to stay alive.

Yuguang Pei*

Department of Anesthesiology, Peking Union Medical College Hospital, Beijing, China

Abstract

The current study focuses on the cancer cell membrane and reveals a brand-new method by which cancer cells can mend the harm that would otherwise kill them. The cell membrane serves as the exterior of all cells, including healthy cells and malignant cells. Additionally, membrane damage poses a risk to life. Since cells have a fluid inside, if a hole is formed in the cell membrane, the cell simply floats out and perishes, similar to a hole in a water balloon.

Keywords: Cancer, Cancer Cells, Macro Pinocytosis, Cell Injury

Introduction

In order to fast repair cell membrane damage, a team of Danish researchers has discovered that cancer cells employ a process known as macro pinocytosis. The method entails the cancer cells dragging the intact cell membrane in over the damaged area and sealing the hole in a couple of minutes. It is already a known tool for cells in other circumstances. The injured portion of the cell membrane is then divided into tiny spheres and sent to the so-called lysosomes, the cells' "stomach," where they are digested [1].

The cancer cells' membrane was damaged in the lab by the researchers using a laser, which causes macro pinocytosis by creating tiny holes in the membrane. Here they can observe that the cancer cell can no longer heal the damage and dies if the process is hindered by substances preventing the creation of the little membrane spheres [2].

When cancer spreads throughout the body, it is one of the most hazardous characteristics of the illness. The condition becomes harder to treat and often necessitates more intensive forms of treatment if tumours develop in new areas of the body. Additionally, cancer cells are particularly vulnerable to membrane damage when they spread across the body's tissues. Previous studies conducted by scientists at the Danish Cancer Society have demonstrated how cancer cells can mend the membrane in another way by tying off the injured area, much like when a lizard tosses its tail [3].

However, laboratory tests may show that aggressive cancer cells in particular exploit macro pinocytosis. This might be because, as the damaged membrane is broken down in the lysosomes, the cancer cell has the chance to recycle it. Cancer cells will benefit from this kind of recycling since they often divide and need a lot of energy and material to make new cells [4].

Uncontrolled division occurs in cancer cells. They consume a lot of energy and raw resources making new copies of themselves all the time. Scientists have demonstrated that one way they meet their needs is by digesting and recycling their own parts. The procedure is known as autophagy, and chaperone-mediated autophagy appears to be particularly important for cancer cells. In this state, chaperones, which are other proteins, direct the breakdown of proteins for component parts. The cancer cells were unable to obtain the components they need for reproduction when researchers stopped the chaperones' action, and many of them instead perished. The researchers intend to target CMA because it is uncommon in normal cells [5].

Conclusion

Since glucose, some amino acids, hormones, growth factors, and other known pathways leading to cell death are deficient or altered during fasting or FMD, these alternate metabolic states are far more challenging to maintain. Second, acquiring resistance can be avoided or minimized by fasting or FMDs. Thirdly, fasting or FMDs shield healthy cells and organs from the negative effects of a range of cancer medications. FMDs appear to be a workable dietary strategy to be researched in cancer prevention on the basis of preclinical and clinical evidence of practicality, tolerability, and efficacy (at lowering IGF1, visceral fat, and cardiovascular risk factors).

References

1. Lanier AP, Bender TR, Blot WJ, et al. Cancer incidence in Alaska natives. Int. J. Cancer. 1976;18(4):409-12.
2. Ziegler RG, Hoover RN, Pike MC, et al. Migration patterns and breast cancer risk in Asian-American women. JNCI: J. Natl Cancer Inst. 1993;85(22):1819-27.
3. Le GM, Gomez SL, Clarke CA, et al. Cancer incidence patterns among Vietnamese in the United States and Ha Noi, Vietnam. Int. J. Cancer. 2002;102(4):412-7.

***Correspondence to:** Yuguang Pei. Department of Anesthesiology, Peking Union Medical College Hospital, Beijing, China, E-mail: peiyuguang@163.com

***Received:** 24-Jan-2023, Manuscript No. AAMOR-23-89309; **Editor assigned:** 26-Jan-2023, PreQC No. AAMOR-23-89309(PQ); **Reviewed:** 09-Feb-2023, QC No AAMOR-23-89309; **Revised:** 14-Feb-2023, Manuscript No. AAMOR-23-89309(R); **Published:** 21-Feb-2023, DOI:10.35841/aamor-7.2.167*

***Citation:** Pei Y. Cancer cells consume themselves to stay alive. J Mol Oncol Res. 2023;7(2):167*

4. Hemminki K, Li X. Cancer risks in second-generation immigrants to Sweden. Int. J. Cancer. 2002;99(2):229-37.
5. Hanahan D, Weinberg RA. Hallmarks of cancer: the next generation. cell. 2011;144(5):646-74.

Citation: *Pei Y. Cancer cells consume themselves to stay alive. J Mol Oncol Res. 2023;7(2):167*