Opinion
Volume 12:3, 2023

Journal of Integrative Oncology

ISSN: 2329-6771 Open Access

Cellular Oncology: Understanding the Molecular Mechanisms of Cancer

Persian Brat*

Department of Oncology, University of Dudley, Dudley, UK

Introduction

Cancer and its treatment can have a profound impact on a patient's life and their family members. The diagnosis of cancer can cause significant emotional distress, anxiety, and depression in patients and their loved ones. In addition, cancer treatment can be physically demanding and may lead to side effects such as fatigue, pain, nausea, and difficulty sleeping. This can negatively impact a patient's quality of life, making it difficult for them to carry out daily activities and maintain social relationships. Cancer pain is a complex phenomenon involving various mechanisms. It can result from the direct pressure or invasion of tumors on nerves or tissues, the release of inflammatory mediators, and neuropathic changes in the nervous system. While the study did not collect information on cancer type, disease history, or treatment within the past year, it provides valuable insights into the safety and effectiveness of COVID-19 vaccines in cancer patients. By including a diverse population of cancer patients, the study can help identify potential subgroups that may have lower immune responses to the vaccine and develop strategies to optimize vaccine safety and efficacy in this population.

Description

Selenium is an essential trace element that has been studied for its potential role in cancer prevention. Some selenium compounds have demonstrated chemopreventive properties, meaning they can help prevent the initiation, promotion, and progression of cancer. The selenium compounds in question have been found to inhibit the growth of cancer cells and induce apoptosis when tested in vitro (in a controlled laboratory environment). Apoptosis is a crucial process that eliminates damaged or abnormal cells. The research indicates that primary cultures of oral carcinoma biopsies (samples of cancerous tissue) are significantly more sensitive to the induction of apoptosis by SDG compared to normal oral mucosa cultures (healthy tissue). This heightened sensitivity suggests that SDG may selectively target cancer cells while sparing normal cells. The study associates the induction of apoptosis in cancer cells with the activation of Fas ligand, a well-known mediator of apoptosis in various contexts. Fas ligand triggers the cell death process in response to specific signals.

The activation of stress kinase signaling pathways, particularly the Jun NH2-terminal Kinase (JNK), is also observed. These pathways are involved in cellular responses to stress and can regulate cell survival and death decisions. The research notes the induction of heme oxygenise, another marker of stress responses, in response to selenium compounds like selenite and SDG. Heme oxygenise is an enzyme involved in heme metabolism and can play a role in protecting cells from oxidative stress. Cancer patients who are undergoing immunosuppressive treatments may have impaired immune responses to the vaccine, while older patients and those with comorbidities may experience altered reactogenicity or adverse reactions. Additionally, cancer patients may already have abnormal immune responses due to the presence of the tumor, which can further complicate the effects of the vaccine.

Cancer is caused by mutations in genes that control cell growth and division, and DNA damage plays a crucial role in the development of these mutations. Mutations can accumulate in a cell's DNA due to exposure to various environmental factors, including tobacco smoke, radiation, and certain chemicals. However, mutations can also arise spontaneously during DNA replication, even in the absence of external factors. When the proteins that normally repair DNA damage are not working properly due to gene mutations, these mutations can accumulate and spread throughout the cell and its daughter cells, leading to additional abnormalities. Some of these mutated cells die, while others acquire a selective advantage that allows them to multiply much more rapidly than normal cells. However, more recent studies have shed some light on this issue. Some studies have suggested that cancer patients who have received B-cell exhausting agents may have lower seroconversion rates and antibody titers following COVID-19 vaccination. However, these studies have been limited in size and may not fully represent the entire cancer patient population. To address these concerns, the CANVAX study was initiated to investigate the safety and effectiveness of COVID-19 vaccines in cancer patients [1-5].

Conclusion

Overall, these findings suggest that selenium compounds, particularly Selenodiglutathione (SDG), may have a potential role in cancer prevention and treatment by selectively inducing apoptosis in cancer cells and activating stress response pathways. Further research is needed to better understand the mechanisms involved and to assess the safety and efficacy of these compounds in clinical settings. This has led to some uncertainty regarding the safety and efficacy of the vaccines in this population. The effective vaccination strategies aim to induce long-lasting immune responses by stimulating the desired antigen(s) and promoting the development of antigen-specific memory B and T cells. However, additional factors such as co-stimulatory molecules and stimulatory cytokines may be required for productive T cell priming. To enhance the immunogenicity of vaccines and promote long-lasting immunity, adjuvants and/or a "prime-boost" strategy of multiple doses may be necessary. Ultimately, the goal of vaccination is to provide protection against specific pathogens while maintaining a tolerable safety profile.

Acknowledgement

We thank the anonymous reviewers for their constructive criticisms of the manuscript. The support from ROMA (Research Optimization and recovery in the Manufacturing industry), of the Research Council of Norway is highly appreciated by the authors.

Conflict of Interest

The Author declares there is no conflict of interest associated with this manuscript.

References

1. Khandekar, Melin J., Paul Cohen and Bruce M. Spiegelman. "Molecular mechanisms of cancer development in obesity." *Nat Rev Cancer* 11 (2011): 886-895.
2. Wolf, Katarina and Peter Friedl. "Molecular mechanisms of cancer cell invasion and plasticity." *Br J Dermatol* 154 (2006): 11-15.

***Address for Correspondence:** Persian Brat, Department of Oncology, University of Dudley, Dudley, UK, E-mail: Hing 851@edu.in*

Received: 27 April, 2023, Manuscript No. jio-23-112993; **Editor assigned**: 29 April, 2023, Pre QC No. P-112993; **Reviewed:** 12 May, 2023, QC No. Q-112993; **Revised:** 19 May, 2023, Manuscript No. R-112993; **Published:** 24 May, 2023, DOI: 10.37421/2329-6771.2023.12.434

3. Martin, D. and J. S. Gutkind. "Human tumor-associated viruses and new insights into the molecular mechanisms of cancer." *Oncogene* 27 (2008): S31-S42.

4. Di Masi, Alessandra, Loris Leboffe, Elisabetta De Marinis and Francesca Pagano, et al. "Retinoic acid receptors: From molecular mechanisms to cancer therapy." *Mol Aspects Med* 41 (2015): 1-115.

5. Fleming, Janis, Aurnab Ghose and Paul R. Harrison. "Molecular mechanisms of cancer prevention by selenium compounds." *Nutrition and Cancer* 40 (2001): 42-49.

How to cite this article: Brat, Persian. "Cellular Oncology: Understanding the Molecular Mechanisms of Cancer." *J Integr Oncol* 12 (2023): 434.