Commentary

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Immune Cells Particularly Natural Killer Cells in the Treatment of Solid Tumors

Hang Johan*

Department of Surgery, Hanyang University, Seoul, South Korea

***Corresponding author:** Hang Johan, Department of Surgery, Hanyang University, Seoul, South Korea,E-mail: Hang@gmail.com

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Description

Importantly, it sheds new light on the possibility of using TRIM proteins as therapeutic targets for breast cancer. When endoplasmic reticulum calcium stores are depleted, Store-Operated Calcium Entry (SOCE) is a crucial calcium signaling pathway that regulates calcium influx across the plasma membrane. While SOCE dysregulation has been linked to pathophysiological conditions like inflammation and cancer, SOCE plays a role in regulating a number of physiological processes like cell migration and proliferation. This study, taken as a whole, demonstrated that SOCE plays a crucial role in the cisplatin resistance of breast cancer cells and that targeting this pathway is essential for improving breast cancer treatment. The use of immune cells, particularly Natural Killer cells (NK), in the treatment of solid tumors, including breast cancer, has not been satisfactory due to the immunosuppressive microenvironment of the tumor and the immunogenicity of breast cancer cells. As a result, the treatment of breast cancer requires the discovery of novel therapies. Exogenous glucocorticoids, as well as endogenous acute or chronic exposure to glucocorticoid stress hormones, have not yet been evaluated for their impact on methylation patterns in breast cancer tissues of varying etiologist.

Genomics Methods

Gathering proof recommends that the TRIM protein family act as disease silencer proteins or oncoproteins in bosom malignant growth. The TRIM protein's roles and molecular mechanisms in breast cancer were the primary focus of this review. Inflammation plays a role in the pathophysiology of cancer as well as the response of cancer cells to chemotherapeutic agents like cisplatin, establishing the well-established link between the two. In fact, inflammation reduces the effectiveness of cisplatin against cancer cells. In order to investigate the epigenetic modifications and their role in the progression and aetiology of breast cancer, in vitro and in vivo models were developed. Cortisol, a glucocorticoid, was used to treat a group of triple-negative breast cancer cell lines. This caused epigenetic changes like the loss of methylation on the promoter regions of tumor suppressor genes like ESR1 and on the LINE-1 repetitive element, which is a surrogate marker for global methylation. Our research demonstrates that stress and glucocorticoid treatment can alter the DNA methylation landscape in breast cancer. Individual health can be harmed when a habitat's microbial balance is disturbed and polymorphic micro biomes have recently been proposed as emerging cancer hallmarks. The composition of the Intratumorally micro biome can now be characterized even in tissues like the breast thanks to modern sequencing and met genomics methods. A comprehensive literature review on various aspects of the microbial landscape of breast tissue and breast tumors and its connection to systemic therapy was conducted by us. New research indicates that the micro biome composition Intratumorally differs from that of normal breast tissue and other types of tumors. In addition, the existing body of knowledge as well as the studies that have been reported on gut bacteria that are capable of metabolizing estrogen is discussed because of their significance in the context of breast cancer. Another topic of interest is the connection between cancer treatment and gut micro biome, and the data that are currently available are discussed. Nevertheless, the gut-breast-cancer therapy axis is at the center of the emerging micro biome field in the context of breast cancer, which raises a number of questions. The kind of stressor and how long it lasts can affect how people react to psychological stress. We are only now beginning to comprehend how this stress hormone response affects crucial breast cancer processes like DNA repair and cell proliferation. However, the epigenetic changes that stress hormones cause in breast cancer are unknown. The world's most common cancer, breast cancer still kills most people through recurrence, metastasis, and drug resistance despite significant advancements in treatment methods. The cell proliferation, migration, invasion, and metastasis of the tumor are all aided by the proteins of the tripartite motif family. DNA and histone modifications within chromatin are examples of epigenetic mechanisms that may play a role in directing the transcriptional processes in cancer cells in response to changes caused by endogenous stress hormones.

Gene Expression

According to our findings, the SOCE inhibitor (BTP2) increased cisplatin cytotoxicity against resistant breast cancer cells by promoting apoptosis and inhibiting cell migration and proliferation. In addition, in comparison to cisplatin-sensitive breast cancer cells, we observed an increase in the gene

expression of STIM1 and ORAI1, two major SOCE components. The hypoxic microenvironment of bosom malignant growth significantly diminishes oxygen-subordinate free extreme age. The generation of free radicals is lessened in tumor cells when Glutathione is overexpressed. With the help of photothermal conversion, we encased the alkyl radical initiator, AIPH, in hollow mesoporous CuS nanoparticles and BSA. Under near-infrared laser irradiation, AIPH was released and decomposed to produce alkyl radicals in hypoxic breast cancer. This was done through the photothermal conversion effect of CuS. Because it could form a complex with GSH, CuS consumed high levels of GSH in tumor cells and enhanced free radical treatment. The rationally designed free-radical Nano generator's anti-tumor efficacy in the hypoxic breast cancer microenvironment was demonstrated *in vivo* and *in vitro* without systemic toxicity. Through i) prevention, ii) detection, iii) diagnosis, iv) treatment, and v) the capacity of our health systems, the Breast Cancer Revealed initiative was designed and implemented to know the status of breast cancer at each point of breast cancer care. Lastly, this study demonstrated that cisplatin therapy increased the gene expression of the inflammatory mediators COX2, IL-8, and TNF- as well as COX2 protein. However, the effect of cisplatin on the inflammatory mediators was reversed when SOCE inhibition with BTP2 was used. In addition, combining different cancer treatments is a good way to make treatment work better. Using BIBR1532, we stimulated NK cell cytotoxicity against breast cancer cells by inhibiting telomerase. After being cured for 24 hours with an IC50 level of BIBR1532, the MDA-MB-231 cell line was washed with PBS and co-cultured with NK cells from the peripheral blood for 5 hours. Finally, we looked at how telomerase inhibition affected NK cell cytotoxicity and breast cancer apoptosis. The expression of apoptotic-related genes and hTERT were also evaluated. The results showed that NK cell cytotoxicity against breast cancer is increased when telomerase is inhibited. Furthermore, by suppressing hTERT, increasing bax expression, and enhancing bad expression, telomerase inhibition and NK cell synergy increased cell death in breast cancer cells. In conclusion, NK cell therapy is more effective against breast cancer cells when telomerase suppression is suppressed. As a result, treating breast cancer cells with NK cells and telomerase inhibition can be beneficial.