OPEN ACCESS Freely available online

ISSN: 2157-7633
Journal of Stem Cell Research & Therapy

Short Communication

The Therapeutic Potential of Retinoids in Inhibiting the Potential Targets in Breast Cancer Stem Cells

Antonia Medrano*

Department of Regenerative Medicine, University of Granada, Armilla, Spain

DESCRIPTION

Breast cancer remains a significant health concern worldwide, with a variety of treatment modalities targeting different aspects of the disease. One emerging area of interest in breast cancer research is the role of Cancer Stem Cells (CSCs) in tumor initiation, progression, and treatment resistance. Among these CSCs, Breast Cancer Stem Cells (BCSCs) have gained attention due to their ability to self-renew, differentiate, and drive tumor growth. Targeting BCSCs presents a potential strategy for improving breast cancer treatment outcomes. In recent years, retinoids, a class of compounds derived from vitamin A, have shown potential in inhibiting BCSCs and disrupting their main pathways, offering a novel therapeutic approach. This article explores the therapeutic potential of retinoids in targeting BCSCs and inhibiting their potential molecular targets.

Understanding Breast Cancer Stem Cells (BCSCs)

BCSCs are a subpopulation of cancer cells within breast tumors that possess stem cell-like properties. These cells are characterized by their ability to self-renew, differentiate into various cell types within the tumor, and resist conventional cancer therapies. BCSCs have been implicated in tumor initiation, metastasis, and recurrence, making them a critical target for effective breast cancer treatment [1,2].

The role of retinoids in BCSC inhibition

Retinoids, including retinoic acid and its derivatives, exert their effects by binding to specific nuclear receptors, such as Retinoic Acid Receptors (RARs) and Retinoid X Receptors (RXRs). These receptors regulate gene expression involved in various cellular processes, including proliferation, differentiation, and apoptosis. Emerging evidence suggests that retinoids can target BCSCs through multiple mechanisms:

Differentiation induction: Retinoids promote the differentiation of BCSCs into more mature, non-tumorigenic cell types, thereby reducing the pool of self-renewing stem-like cells within the tumor [3].

Apoptosis induction: Retinoids have been shown to induce apoptosis, or programmed cell death, in BCSCs, leading to the elimination of these therapy-resistant cells [4].

Inhibition of signaling pathways: Retinoids can interfere with main signaling pathways implicated in BCSC maintenance and survival, such as Notch, Wnt/β-catenin, and Hedgehog pathways, thereby disrupting BCSC self-renewal and proliferation.

Regulation of Epithelial-Mesenchymal Transition (EMT): Retinoids inhibit the process of EMT, which is associated with cancer stem cell-like properties and metastatic potential, thereby reducing the aggressiveness of BCSCs [5].

Potential molecular targets of retinoids in BCSCs

Several molecular targets have been identified through which retinoids exert their inhibitory effects on BCSCs:

Notch signaling pathway: Retinoids downregulate Notch signaling, a pathway involved in maintaining stem cell characteristics and promoting tumor progression. Inhibition of Notch signaling by retinoids leads to decreased self-renewal and expansion of BCSCs [6].

Wnt/β-catenin signaling pathway: Retinoids interfere with Wnt/β-catenin signaling, which plays a crucial role in regulating stem cell fate and tumor initiation. By inhibiting this pathway, retinoids suppress the self-renewal capacity of BCSCs and inhibit tumor growth [7].

Hedgehog signaling pathway: Retinoids modulate Hedgehog signaling, which is implicated in regulating stem cell maintenance and promoting cancer progression. Inhibition of Hedgehog signaling by retinoids attenuates BCSC self-renewal and inhibits tumor growth [8].

Correspondence to: Antonia Medrano, Department of Regenerative Medicine, University of Granada, Armilla, Spain, E-mail: medranoantonibio3@gmail.com

Received: 22-Feb-2024; Manuscript No. JSCRT-24-25285; **Editor assigned:** 26-Feb-2024; PreQC. No. JSCRT-24-25285 (PQ); **Reviewed:** 11-Mar-2024; QC. No. JSCRT-24-25285; **Revised:** 18-Mar-2024; Manuscript No. JSCRT-24-25285 (R); **Published:** 25-Mar-2024, DOI: 10.35248/2157-7633.24.14.628

Citation: Medrano A (2024) The Therapeutic Potential of Retinoids in Inhibiting the Potential Targets in Breast Cancer Stem Cells. J Stem Cell Res Ther. 14:628.

PI3K/Akt/mTOR pathway: Retinoids inhibit the PI3K/Akt/mTOR pathway, which is frequently dysregulated in breast cancer and associated with BCSC survival and resistance to therapy. By targeting this pathway, retinoids sensitize BCSCs to conventional treatments and suppress tumor growth [9,10].

Clinical implications and future directions

The therapeutic potential of retinoids in targeting BCSCs holds significant potential for improving breast cancer treatment outcomes. However, several challenges need to be addressed to translate these findings into clinical applications. These include identifying optimal retinoid formulations, determining effective dosing regimens, and minimizing potential side effects associated with retinoid therapy.

Future research efforts should focus on elucidating the precise mechanisms underlying the inhibitory effects of retinoids on BCSCs and identifying biomarkers predictive of patient response to retinoid-based therapies. Moreover, combination approaches incorporating retinoids with existing standard-of-care treatments, such as chemotherapy and targeted therapies, warrant investigation to enhance therapeutic efficacy and overcome treatment resistance.

CONCLUSION

In conclusion, retinoids represent a potential class of compounds with therapeutic potential in targeting BCSCs and inhibiting their important molecular pathways. By inducing differentiation, promoting apoptosis, and interfering with signaling pathways involved in BCSC maintenance, retinoids offer a novel approach for combating breast cancer progression and recurrence. Further research and clinical trials are needed to fully exploit the therapeutic benefits of retinoids in breast cancer treatment and improve patient outcomes.

REFERENCES

1. Velasco-Velázquez MA, Homsi N, De La Fuente M, Pestell RG. Breast cancer stem cells. Int J Biochem Cell Biol. 2012;44(4): 573-537.
2. Liu S, Wicha MS. Targeting breast cancer stem cells. J Clin Oncol. 2010;28(25):4006-4012.
3. Pham PV, Phan NL, Nguyen NT, Truong NH, Duong TT, Le DV, et al. Differentiation of breast cancer stem cells by knockdown of CD44: Promising differentiation therapy. J Transl Med. 2011;9:209.
4. Aztopal N, Erkisa M, Erturk E, Ulukaya E, Tokullugil AH, Ari F. Valproic acid, a histone deacetylase inhibitor, induces apoptosis in breast cancer stem cells. Chem Biol Interact. 2018;280:51-58.
5. May CD, Sphyris N, Evans KW, Werden SJ, Guo W, Mani SA. Epithelial-mesenchymal transition and cancer stem cells: A dangerously dynamic duo in breast cancer progression. Breast Cancer Res. 2011;13:202.
6. Suman S, Das TP, Damodaran C. Silencing NOTCH signaling causes growth arrest in both breast cancer stem cells and breast cancer cells. Br J cancer. 2013;109(10):2587-2596.
7. Tang T, Guo C, Xia T, Zhang R, Zen K, Pan Y, et al. LncCCAT1 promotes breast cancer stem cell function through activating WNT/β-catenin signaling. Theranostics. 2019;9(24):7384.
8. Bhateja P, Cherian M, Majumder S, Ramaswamy B. The Hedgehog signaling pathway: A viable target in breast cancer?. Cancers (Basel). 2019;11(8):1126.
9. Xia P, Xu XY. PI3K/Akt/mTOR signaling pathway in cancer stem cells: From basic research to clinical application. Am J Cancer Res. 2015;5(5):1602.
10. Ginestier C, Wicinski J, Cervera N, Monville F, Finetti P, Bertucci F, et al. Retinoid signaling regulates breast cancer stem cell differentiation. Cell Cycle. 2009;8(20):3297-3302.