Virtual Screening of Surface Receptors of Breast Cancer Cells with Acquired Endocrine Resistance

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From National Conference on Interdisciplinary Research and Innovations in Biosciences, NATCON -2018. Post Graduate & Research Department of Biochemistry, Mohamed Sathak College of Arts & Science, Sholinganallur, Chennai-600119, India. 24th & 25th January 2018.

American J of Bio-pharm Biochem and Life Sci 2018 January, Vol. 4 (Suppl 1): **PP27**

ABSTRACT

Three-fourth of the total breast cancer cases are observed to be estrogen- and/or progesterone-receptor responsive, where hormonal estrogen and progesterone act as the principal extracellular stimulants, constitutionally promoting the growth and proliferation of these tumours. Therefore, targeted suppression of hormone-stimulated signalling pathways through appropriate endocrine therapy has emerged to be the mainstream of treatment for hormone-responsive breast cancers. Regardless of its appreciable advantages, endocrine therapy has frequently been associated with acquired resistance against hormone-antagonists in several clinical cases, and urges for designing effective methods for prevention and treatment. During the past few years, molecular and clinical researchers have laid significant emphasis on delineating the molecular mechanisms that govern the development of endocrine resistance in breast cancers, and proposing prospective strategies for their prevention, as well as developing appropriate approaches for predicting the likelihood of developing resistance to endocrine therapy. The fundamental molecular mechanisms underlying the development of endocrine resistance will include: somatic mutations successively resulting in conformational modifications and differential responsiveness of the hormone-receptors, acquired hypersensitivity to suboptimal levels and/or residual reserves of natural hormones, activation of hormone-independent signalling pathways that stimulate the growth and proliferation of these tumours, molecular cross-talk between innate growth-factor signalling pathways that supplement these tumours with alternate survival signals. The current study attempted to screen and identify using appropriate computational techniques, the polymorphic surface receptors on breast cancer cells that have acquired endocrine resistance to common hormone-antagonists. The results shall be helpful in predicting the responsiveness of breast cancers to common endocrine therapeutics.

Published: February 2018.