Commentary on: Using Omics to Investigate the Breast Cancer Landscape

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Description

Breast cancer is becoming more common across the world, with over 600,000 deaths recorded in 2018. Surgery, chemotherapy, radiotherapy, or targeting of classical breast cancer subtype markers such as estrogen receptor (ER) and HER2 are currently available treatment choices for breast cancer patients. These medications, however, are ineffective in preventing recurrence and metastasis. Improved knowledge of breast cancer and metastasis biology will aid in the discovery of new biomarkers and therapeutic opportunities to help patients be better stratified and treated. We'll start with an overview of current breast cancer biology methods and models, with a focus on 2D and 3D cell culture, including organoids, and in vivo models like the MMTV mouse model and xenografts originating from patients (PDX). Then, in the form of breast cancer susceptibility, breast cancer drivers, therapeutic response, and treatment resistance, genomic, transcriptomic, and proteomic methods, as well as their integration, will be considered. Finally, we'll go over how 'Omics datasets combined with conventional breast cancer models can be used to generate insights into breast cancer biology, recommend individual therapies in precision oncology, and create data repositories for meta-analysis. System biology has the ability to catalyze the next big advancement in breast cancer treatment options. Breast cancer is the most common cancer-related death of women around the world. It is a condition with a wide range of symptoms, Based on gene expression profiles, they are classified as Luminal A (LumA), Luminal B (LumB), epidermal growth factor receptor ERBB2/HER2-overexpressing (HER2+), and basal epithelial-like (BL). To kill viable cancer cells, breast cancer is usually treated with surgery, radiotherapy, cytotoxic chemotherapy, and/or targeted therapies. Both LumA and LumB breast cancers express estrogen receptors (ER). Cancer hallmarks are linked to dysregulated ER signaling. Cell proliferation, invasion, and the epithelial–mesenchymal transformation are all aided by ER target genes such as cyclin-dependent kinase (CDK) 1 and the kinase Src. The proliferation marker Ki67 is overexpressed in LumB cancers, which is linked to an increased risk of distant metastases and decreased progesterone receptor expression which causes more tumor-causing genes to be expressed. ER antagonists, aromatase inhibitors, and selective estrogen receptor degraders are used to treat LumA and LumB tumors. Loss of ER expression, ER mutations, or overexpression of alternative breast cancer-driving pathways including ERBB1/EGFR can all lead to therapeutic resistance. Targeted therapies against phosphoinositide 3-kinases (PI3K), mammalian target of rapamycin (mTOR), and CDK4/6 have recently been shown to be effective in the clinical setting in order to overcome resistance to conventional ER antagonists. ERBB2/HER2 is overexpressed in HER2+ breast cancers, which encourages proliferation by controlling CDKs and Cycling. HER2 dimerization with EGFR also activates mitogen-activated protein kinase (MAPKTargeted agents such as trastuzumab, pertuzumab, and neratinib are used to treat HER2+ breast cancers. Trastuzumab is an antibody that prevents HER2 dimerization, induces ubiquitin-dependent HER2 degradation, and promotes natural killer cell recruitment to tumors. Trastuzumab therapy resistance is caused by HER2 dimerization with other ERBB family members or by constitutive HER2 activation. Traditionally, immortalized cell lines derived from patient samples have been used to study breast cancer because they are simple and inexpensive to develop. These cell lines express biomarkers from various molecular subtypes of breast cancer and mimic some of the parent tumor's features, such as drug responses and transcriptomic profiles. Significant breakthroughs in breast cancer science, such as the discovery of oncogenes, have been made thanks to cell lines and the factors that influence metastatic tropism However, as opposed to primary tumors, breast cancer cell lines have more gene copy number differences, lack the in vivo microenvironment, and do not sustain primary tumor heterogeneity.

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