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Breast Cancer Cell Line Morphologies

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Description

In vivo and *in vitro* investigations with breast cancer cell lines have contributed significantly to our understanding of the disease. These provide an infinite source of homogeneous self-replicating material that is devoid of contaminating stromal cells and can frequently be cultivated in simple standard medium. The first described line, BT-20, was created in 1958. Since then, despite ongoing efforts, the number of permanent lines acquired has been startlingly low. Attempts to cultivate BCC from primary tumours have usually failed. For example, just 18 of 177 primaries yielded cell lines, despite the claimed success rate [1].

This low effectiveness was frequently related to technical challenges in extracting live tumour cells from their surrounding stroma. The majority of accessible BCC lines are from metastatic tumours, primarily pleural effusions. Effusions yielded a substantial number of dissociated, viable tumour cells with little or no contamination from fibroblasts or other tumour stroma cells. However, even with metastatic samples, long-term propagation success has been limited. For example, and successfully proliferated tumour cells in just 10%, 2%, and 25% of instances, respectively.

Many of today's BCC lines were created in the late 1970s. According to a Medline-based survey, a handful of them, particularly MCF-7, T-47D, and MDA-MB-231, account for more than two-thirds of all abstracts reporting investigations on the indicated BCC lines. The applicability of data acquired with such a small number of cell lines to tumours is dubious. To address the issue of representativeness, we gathered and examined diverse data acquired, especially in the recent decade, on both tumours and BCC lines.

Deregulation of many cellular processes, including genome stability, proliferation, apoptosis, motility, and angiogenesis, is required for the transformation of a normal, finite-life-span somatic epithelial cell into an immortalised, metastatic cell. Changes in genomic copy number and/or structure are particularly essential as deregulating events in cancer development, and the identification of recurring aberrations has revealed a plethora of critical oncogenes and tumour suppressors. In fact, recurrent genomic abnormalities have been documented to deactivate over a thousand genes in breast cancer alone.

The functional analysis of some of these genes in cell lines and xenografts has offered essential insights into their involvement in cellular physiology (. However, interpreting these findings in the context of breast cancer pathogenesis necessitates knowledge of how well the cell lines mimic abnormalities found in original tumours. To that purpose, we present here a thorough comparison of the molecular and biological characteristics of 51 breast cancer cell lines with those measured in actual breast cancers.

They have the same copy quantity and expression abnormalities as main tumours and carry nearly all of the recurring genomic abnormalities related with poor outcome in primary cancers. Furthermore, breast cancer cell lines, like main tumours, cluster into basal-like and luminal expression subgroups, however the partitioning of genomic aberrations between these subsets differs slightly from that of basal-like and luminal primary tumours. The collection displays diverse reactions to targeted medicines, mirroring clinical observations.

We conclude from these studies that the cell line collection reflects the majority of the important genomic and resulting transcriptional abnormalities found in primary breast tumours, and that analysing the functions of these genes in the ensemble of cell lines will accurately reflect how they contribute to breast cancer pathophysiologies. We also show how correlative investigations of the diverse responses to treatment with therapeutic drugs that target these genes might lead to the identification of genetic markers that predict response in particular patients [2].

Breast cancer is a very heterogeneous illness with a variety of clinical characteristics that are genetically and epigenetically unique. *In vivo* and *in vitro* investigations employing breast cancer cell lines have provided an inexhaustible source of homogeneous self-replicating materials using simple but standard conditions and techniques, accounting for a major amount of existing information on breast carcinomas. Thus, whether these cell lines accurately capture the molecular characteristics and represent the heterogeneity of the underlying malignancies is a crucial problem to address before generating therapeutically useful outcomes.

Though it has been concluded that breast cancer cell lines are, to a large extent, representative of breast carcinoma, with ER and HER2 being important stratifiers for their classification, continuous evidence has suggested dramatic genetic and epigenetic changes during the initial cell line establishment and subsequent serial passaging, implying that the resultant cell lines may have evolved significantly from the primary tumors [3]. Furthermore, various studies classify breast cancer cell lines into distinct categories, confounding our knowledge of cell line categorization and its relationship to malignancies.

We are overloaded with cell lines that lack systematic feature recording and consistent subtyping due to uneven nomenclatures, categorization, and even contradicting molecular characterization in different literatures. On the other hand, the number of cell lines often employed in breast cancer research is quite modest, with MCF7, T47D, and MDAMB231 accounting for more than two-thirds of the cell lines utilised in the associated studies. This begs the question of how representative these few cell lines are of the large array of breast cancers with varying clinical consequences. We are thus driven to identify the molecular traits and tumour subtypes that each cell line represents in order to facilitate breast cancer modelling using appropriate cell lines. Due to technical challenges in collecting viable tumour cells from the surrounding stroma and the bottleneck of long-term growth during culturing, very few cell lines have been established since the development of the first breast cancer cell line [4]. The majority of cell lines were developed in the late 1970s.

In general, cell line nomenclature does not indicate phenotypic relationship, but rather how they are produced, such as whether they are obtained from the same laboratory, the same patient, isolated by serial subculture from the same original population, or cultivated using the same method. For example, 'HCC series' cell lines were isolated at Hamon Cancer Centre; 'MDA series' cell lines were developed at M. D. Anderson Hospital and Tumor Institute; and '21 series' cell lines were established at M. D. Anderson Hospital and Tumor Institute. During their creation, the 'HMT series' experienced sequential subcultivation under diverse circumstances, including P53 mutation, MYC amplification, EGF-independence accompanied by tumorigenicity in nude mice, EGFR and

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HER2 overexpression [5]. 'SUM series' were created using the same selective medium while being isolated from various tumour tissues. Because cell lines are often called by the scientist who developed them, there are no rules governing how each cell line is named, particularly those that do not belong to any series.

Future Perspective

Though the majority of studies do not further stratify luminal cell lines into luminal A and B subtypes based on HER2 status, we support such differentiation not only for the sake of achieving consistent categorization with tumour subtyping to facilitate easy tumour modeling, but also to meet the requirement of drug response assays based on ER and HER2 status. A research utilising BT474 indicated the synergistic benefit of tamoxifen and Herceptin in the treatment of breast cancers, while MCF7 has traditionally been utilised for assessing tamoxifen-induced cell responsiveness.

Conflict of Interest

None.

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