The evolution of breast cancer's molecular pathology.

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Abstract

The current hypothesis for the progression of human breast cancer proposes a multi-stage linear process starting with Flat Epithelial Atypia (FEA), moving on to Atypical Ductal Hyperplasia (ADH), developing into DCIS, and finally reaching the potentially fatal stage of invasive ductal carcinoma. Finding the molecular changes linked to the various stages of breast cancer growth has long been a difficult task for researchers studying human breast cancer. The inability to use cutting-edge molecular technology on the microscopic pre-invasive stages of breast carcinogenesis has hindered progress towards this goal up until recently.

Keywords: Molecular pathology, Flat epithelial atypia, Breast cancer.

Introduction

The most frequent carcinoma in women and the second-leading cause of cancer-related death in women is breast cancer. In the USA, new cases of in situ and invasive breast cancer are anticipated to be identified in 2010. An estimated 39,500 US women will pass away from breast cancer over this time period. Both the incidence and death of breast cancer have seen promising trends over the past 20 years. Since 1990, there has been a general levelling down of the incidence rates of breast cancer, with a decline of 3.5%/year from 2001 to 2004. Most significantly, mortality rates from breast cancer have decreased by 24% during the same period, with young women and those with oestrogen receptor positive illness seeing the greatest benefits [1].

The majority of breast cancers in the world are ductal and lobular subtypes, with the ductal subtype representing 40–75% of all instances that are diagnosed. The development of numerous linear models of breast cancer initiation, transformation, and progression, as shown, was influenced by epidemiological and morphological evidence. There have been two models developed for the ductal subtype. Flat epithelial atypia, Atypical Ductal Hyperplasia (ADH), and Ductal Carcinoma In Situ (DCIS) are recognised as the non-obligate antecedents of invasive and metastatic ductal carcinoma in the original "ductal" model proposed by Welling's and colleagues [2].

The cancer stem cell concept and the random clonal evolution model are the two most popular explanations for breast carcinogenesis. Any breast epithelial cell may be the target of random mutations, according to the sporadic clonal evolution theory. Over time, the cells that have favourable genetic and epigenetic modifications are chosen to advance tumours. According to the alternative cSC model, only stem and progenitor cells, which make up a relatively tiny percentage of the cancer's tumour cells, may start and maintain tumour growth. It has been proposed that stem cells may go through clonal evolution, creating a dynamic link between the two models, and that these three possibilities are not mutually exclusive [3].

The involvement of the non-neoplastic cells in the tumour microenvironment has largely gone unexplored because the primary focus of breast cancer research has been on the breast epithelial cell itself. In the pre-invasive stages of breast cancer progression, myoepithelial and inflammatory cells, as well as fibroblasts, myo-fibroblasts, adipocytes, inflammatory cells, and endothelial cells, are examples of non-neoplastic cells seen in the tumour microenvironment. Non-epithelial cells have typically been viewed as passive or receptive bystanders [4].

Several groups have carried out high-throughput genomic and transcriptomic comparative analysis of the various tissue and cellular compartments in the various inframammary stages of breast cancer progression to objectively determine the molecular alterations in cells composing the tumour microenvironment. The gene expression and genetic profiles of all significant cell types in the microenvironment of healthy breast tissue and in both in situ and invasive breast cancer using ex vivo purification techniques. The findings of this extensive investigation showed that gene expression changes happen in all cell types throughout development and that clonally restricted genetic modifications are only present in neoplastic epithelial cells [5].

Conclusion

Many unique insights into the intricate process of breast cancer progression have been produced as a result of the

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integration of numerous genomic and transcriptome analysis of the various stages of breast cancer. First, it appears that two separate molecular genetic pathways that are highly correlated with tumour grade drive the progression of human breast cancer. Second, the biggest molecular changes in the epithelium and non-epithelial components of the cancer microenvironment take place before local invasion. Finally, there are no significant further changes in gene expression in the epithelial compartment between the preinvasive and invasive phases of breast cancer. Fourth, as the disease progresses from a preinvasive to an invasive state, the non-epithelial compartment of the tumour micro milieu experiences significant changes in gene expression and epigenetics. Despite these noteworthy developments, scientists have only just begun to delve into this complex biological process. It is projected that the complexity of breast cancer progression will become more apparent in the next ten years of breast cancer research as a result of the introduction of new novel high-throughput genomic, epigenetic, and proteomic tools. We frequently hope that the knowledge obtained from such studies will lead to the development of more potent methods for both treating and preventing breast cancer.

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