

# A Comprehensive Exploration of Breast Ductal Carcinoma and its Prognostic Implications

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## ABSTRACT

The occurrence of ductal carcinoma in situ (DCIS) as an early type of neoplasia has increased since the implementation of breast screening programmes. Despite the good prognosis, 20%-50% of DCIS patients will develop invasive ductal carcinoma (IDC) if not treated. It is critical to look for promising biomarkers for predicting the prognosis of DCIS. In this study, the Gene Expression Omnibus (GEO) database was used to investigate the expression of genes that differed between DCIS and normal tissue. To characterise the biological role and intrinsic process pathway, enrichment analysis was used. The hub genes were classified using the Cancer Genome Atlas Breast Cancer Dataset, and the results were confirmed using the Cytoscape plugins CytoHubba and MCODE. The core gene signature's prognostic ability was determined using time-dependent receiver operating characteristic (ROC), Kaplan-Meier survival curve, Oncomine databases, and UALCAN databases. Furthermore, the prognostic value of core genes was demonstrated in proliferation assays. In the current study, we discovered 217 common differentially expressed genes (DEGs), with 101 upregulated and 138 downregulated genes. The PPI network was used to obtain the top genes (protein-protein interaction).

**Keywords:** Breast cancer, Microbiome, Microbiota therapy, Microbiome diversity.

## INTRODUCTION

With over 60,000 cases diagnosed each year in the United States, ductal carcinoma *in situ* (DCIS) is the most common type of early-stage breast cancer. DCIS is characterised by the growth of neoplastic cells within the breast ducts and is considered the immediate precursor to invasive breast ductal carcinomas (IDC). DCIS is typically treated with surgery and radiotherapy, with or without adjuvant endocrine therapy [1]. Because many DCIS cases do not progress to IDC, overtreatment remains a significant issue, lowering patients' quality of life. However, up to 20% of patients experience disease recurrence, with half of cases presenting invasive disease, indicating that standard treatments are ineffective for a subset of patients with DCIS.

The molecular subtype of breast cancer influences prognosis in IDC but has less predictive value in DCIS. In some DCIS cases, increased expression of biomarkers such as COX-2, FOXA1, HER2, Ki-67, p16/INK4A, PR, and SIAH2 is associated with an increased risk of recurrence. Understanding the underlying mechanisms of DCIS progression allows us to identify new biomarkers associated with the development of IDC and develop treatment strategies

that are more patient-specific. Although mammary fat pad and subcutaneous injection of human cells in mice are common methods for modelling breast tumours, they do not accurately replicate the growth and progression of human DCIS [2].

Mammary intra-ductal (MIND) injection of breast cancer cells, on the other hand, results in the formation of DCIS lesions that eventually escape myoepithelial barriers and invade the surrounding stroma, resulting in IDC. The MIND model more closely resembles the progression of human DCIS than conventional transplant models [3]. Tissue homeostasis necessitates a balance of energy production and nutrient consumption, such as glucose. In normal mammalian cells, glucose is converted into pyruvate, which is then converted into acetyl COA, which fuels the TCA cycle and oxidative phosphorylation for ATP production. The TCA cycle and glycolysis provide the building blocks for the synthesis of lipids, amino acids, and nucleotides. Anaerobic glycolysis occurs when glucose metabolism in cancer cells is reprogrammed to generate lactate.

## Patient information

A 48-year-old female presented with bilateral breast masses that had been present for 5 months. She was G7P5A2 and had lactated

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for ten years. She used to have regular menstrual cycles. She did not have any chronic illness. She had a hysterectomy to treat heavy vaginal bleeding caused by a uterine fibroid [4].

### Clinical findings

There were bilateral palpable breast masses that were hard, mobile, and irregular on clinical examination. There was skin tethering and palpable axillary lymph nodes on the right side.

### Diagnostic assessment

On ultrasound, a heterogeneous irregular ill-defined mass-like lesion was seen in the upper outer quadrant of the right breast, and a hypoechoic irregular hypo-echoic mass 12\*13mm was seen in the upper outer quadrant of the left breast. (Bilateral U4a), and a mammogram revealed asymmetrical density in the upper outer quadrant of the right breast and an irregular ill-defined dense mass 10\*10mm in the upper outer quadrant of the left breast [5].

Breast carcinoma can develop from a benign tumour or coexist independently. Bilateral breast carcinomas are extremely rare, accounting for only 2-5% of all breast cancers, with only triple-negative breast cancers being rarer. In their lifetime, approximately 2-11% of breast cancer patients will develop cancer in the opposite breast, with an incidence rate ranging from 4 to 8 per 1000 people per year.

Bilateral tumours can be synchronous or metachronous. A family history of breast cancer is a risk factor for the development of unilateral breast carcinoma; it is not an unreasonable hypothesis to speculate that it may also be a risk factor for bilateral breast carcinoma. Some authors have provided data to support this hypothesis [6].

## CONCLUSION

According to the literature, the majority of patients (87%) were treated with mastectomy and axillary nodal evaluation, and the majority of them (73%) received locoregional radiotherapy after surgery. Only 8.7% of the patients had received adjuvant therapy, which typically included ovarian ablation (4.5%). There was no significant difference in treatment between patients with invasive ductal carcinoma and those with invasive lobular carcinoma. Invasive or in situ lobular carcinoma is thought to increase the risk of bilateral disease. Several studies have been conducted to assess the prognosis of patients with bilateral carcinoma.

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