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Examining Differential Gene Expression Between Luminal A and Basal-like Breast Cancer

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ABSTRACT

Breast cancer is a leading contributor to the global cancer burden as the most common form of cancer in women (Lukasiewicz et al., 2021). Scientists often examine breast cancer on a genetic level and use molecular subtypes to differentiate between symptoms and aggression levels. Overarching subtypes include luminal A, luminal B, HER2+, and triple-negative/basal-like breast cancers. Luminal A patients often have positive prognoses whereas basal-like cancers lead to largely negative patient outcomes, respectively. In order to examine what genes and alleles may cause the difference in symptoms and cancer severity between the two subtypes, data analyses were conducted to contrast and measure gene activity in each of the subtypes. Using The Cancer Genome Atlas's breast cancer database, key differentially expressed genes have been identified between the aggressive basal-like subtype and the more treatable luminal A subtype. This study has identified the top 3 differentially expressed genes for basal-like subtypes and the top 3 differentially expressed genes for luminal A through bioinformatics analysis using R. Literature reviews were conducted regarding FOXA1, FSIP1, NAT1, ROPN1, ROPN1B, and HORMAD1. Through examining these genes and biological pathways they impact, this paper highlights that genes highly expressed in luminal A subtypes may affect patient prognosis positively whilst genes with a significant presence in basal-like samples often encourage cancerous spread and tumor advancement.

INTRODUCTION

Breast cancer presents as the leading cause of cancer-related deaths in women worldwide (Jia et al., 2020). Each year, roughly 2.3 million new breast cancer cases arise worldwide and breast cancer incidents rates have been on an upward trend for the past thirty years (Lukasiewicz et al., 2021). Breast cancer comprises 29% of all new female cancer diagnoses in the United States (Lukasiewicz et al., 2021). As a result of medical and clinical advancements, treatments for breast cancer are evolving and changing the landscape of patient prognosis. Common treatments include chemotherapy, targeted drug therapy, hormone therapy, and radiation. Breast cancer occurs when the DNA of breast cells are altered, causing them to grow and divide uncontrollably. Causes and risk factors for cancer are multi-faceted and highly complex to identify, but environmental and genetic history have been identified as key factors for cancer

diagnosis (“Causes”, 2017). As a result of the irregular cell division, a mass of cells, called a tumor, may form on the breast and manifest in a lump (Jia et al., 2020). In certain cancer subtypes, cancer cells have receptors on them that bind to certain hormones, fueling cancerous growth (“What is Hormone Receptor-Positive Breast Cancer?”, 2026). Breast cancer can be divided into four main subtypes based on molecular and hormonal behaviors: luminal A, luminal B, HER2+, and triple-negative. Luminal A is characterized by being estrogen-receptor positive and/or progesterone-receptor positive. Because luminal A progression is dependent on estrogen and/or progesterone, hormone therapies that lower estrogen and progesterone levels or stop receptors from binding to these hormones are widely used to treat this subtype (Schlamadinger). The most aggressive of breast cancers, the triple-negative subtype, does not have estrogen, progesterone, and human epidermal growth factor receptors ("Breast Cancer- Symptoms and Causes”, 2025). This lack of receptors means it cannot be treated by hormone therapies and HER2 targeted therapies, making triple-negative the hardest subtype to treat. The majority of triple-negative breast cancers are basal-like (Schlamadinger, 2025). Basal-like cancers are distinguished based on gene expression patterns that resemble those of basal cells - a type of cell located on the outer layer of milk ducts (Schlamadinger, 2025). Basal-like cancers often result in poor prognosis and cancer progression (Schlamadinger, 2025). Note that this paper will use the terms triple-negative and basal interchangeably. This paper examines gene expression levels in luminal A and basal-like molecular subtypes in order to determine the top differentially expressed genes between the two and identify biological functions associated with the aforementioned genes. By contrasting the least aggressive and most aggressive breast cancer subtypes, this study hones in on potential genes that contribute to aggression or a lack of it and which genes should be used as clinical targets to dampen the severity of basal-like cancers. Widely used breast cancer treatments involving chemotherapy and radiation often pose significant side effects due to the harm they cause to healthy cells (Shokoohi et al., 2025). This paper investigates potential targets for gene therapy, an approach that only targets cancerous cells, offering a less debilitating solution. The families of these genes are involved with tumor suppression, gene regulation, recombination, cell division, inflammation, and metastasis. Future gene therapies may downregulate genes that contribute to the harmful natures of basal-like cancers and upregulate those which contribute to luminal A’s milder condition. The findings of this analysis stress the importance of isolating targets for gene therapy research, an underutilized technique in cancer care today.

METHODS

A bioinformatics approach was utilized in order to analyze luminal A and basal subtypes in breast cancer patients. Specifically, a 2011 data freeze was extracted from The Cancer Genome Atlas Breast Invasive Carcinoma (TCGA-BRCA) database from the National Institute of Health’s “Comprehensive molecular portraits of human breast tumors.” R Studio was leveraged for parsing through two data tables, one containing patient subtype labels (Final Full BRCA Sample Summary) and the other containing RNA-seq data from luminal A and basal-like tissue (BRCA.exp.547.med.txt). RNA sequencing data quantifies the expression of each gene from the tissue, helping us identify potential drivers of the cancer. Using limma, R functions were employed in order to establish a system that would overlay the table containing PAM50 molecular subtype labels for each patient barcode with the RNAseq data. The algorithm identified which

patient barcodes had a match in both sets, meaning both elements (subtype and RNAseq) could be accessed for each patient. The RNAseq data was already normalized and ready for analysis. The following statistical analysis involving these samples extracted and compared gene expression across the patient data. Using mathematical comparisons such as logFC and P-value, a table of the top differentially expressed genes between subtypes was compiled. Log FC demonstrates how much of a change or difference is present whereas P-values represent the surety and significance of each result. Log2FC was calculated by subtracting the luminal A expression levels from the basal-like expression levels.

For the literature review component of this paper, PubMed, the NIH, and Google Scholar were used to explore relevant research. Research was conducted regarding the functions of the top three differentially expressed genes that were highly expressed in luminal A subtypes and the top three differentially expressed genes which were highly expressed in basal subtypes. Specifically, the roles of FOXA1, FSIP1, and NAT1 in luminal A breast cancer were investigated as well as ROPN1, ROPN1B, and HORMAD1 functions critical to triple-negative and basal breast cancer.

RESULTS

Through RNA-sequencing bioinformatics analysis, the top ten differentially expressed genes amongst the luminal A and basal-like patients cataloged in the TCGA-BRCA database were determined. FOXA1, FSIP1, and NAT1 were the top three genes that were more highly expressed in luminal A patients than in basal patients. ROPN1, ROPN1B, and HORMAD1 were the top genes that were most significantly expressed in basal-like subtypes.

Gene	**logFC**	**AveExpr**	**t-value**	**P.Value**	**adj.P.Val**	**b-value**
ROPN1	4.987981	0.6324067	26.01176	1.016069e-92	6.942007e-90	200.8511
ROPN1B	4.942758	0.5505150	24.11583	6.233706e-84	2.737933e-81	180.7017
HORMAD1	4.935530	0.8114783	26.99537	3.108988e-97	3.186194e-94	211.2003
Gene	**logFC**	**AveExpr**	**t-value**	**P.Value**	**adj.P.Val**	**b-value**
NAT1	-4.622563	-0.2396054	-22.99880	1.028698e-78	3.012124e-76	168.7371
FSIP1	-4.996159	-0.6087167	-23.94901	3.736330e-83	1.531646e-80	178.9184
FOXA1	-6.028796	-0.8785346	-46.73092	9.612256e-178	5.910576e-174	395.3544

Table 1: six differentially expressed genes between basal and luminal A patients from the TCGA-BRCA database using R limma analysis. The first half of the table shows the top three differentially expressed genes which are upregulated in basal-like cancers. The bottom half of the table shows the top three differentially expressed genes which are upregulated in luminal A cancers. Genes with positive log fold changes (logFC) are upregulated in basal-like cancers and those with

negative logFC are upregulated in Luminal A cancers. The table is ordered in accordance with logFC values.

FOXA1 is highly expressed in luminal A subtypes

Forkhead box transcription factor (FOXA1) is a gene highly expressed in luminal subtypes of breast cancer, and its functions are integral to disease progression. FOXA1's logFC places it as the most differentially expressed gene in the examined dataset and its adjusted P-value shows it is significant (Table 1). As a transcription factor, the FOXA1 protein binds to DNA to maintain and control several gene expression levels (Zhu et al., 2024) While FOXA1's role in breast cancer has yet to be fully explored, this gene has already proven to have significant impacts on the disease because it regulates a large amount of gene expression in each cell. High expression levels of FOXA1 are correlated with more positive prognosis in breast cancer, as is typical of luminal subtypes (Fonesca-Benitez et al., 2025). FOXA1 works with biological pathways to suppress genes and biomarkers linked to basal and triple-negative subtypes (Fonesca-Benitez et al., 2025). Current research suggests that this gene is upregulated in luminal subtypes in breast cancer but is downregulated in basal-like subtypes (Fonesca-Benitez et al., 2025). This study corroborates these results, as FOXA1 was the top differentially expressed gene between basal-like and luminal A subtypes. Expression of FOXA1 may be integral to the less severe symptoms, higher survival rates, and better patient outcomes. The difference in expression levels between the two subtypes pertinent to this study indicates that therapies involving FOXA1 could limit fatalities and poor survival in breast cancer patients.

FSIP1 is highly expressed in luminal A subtypes

Fibrous Sheath Interacting Protein (FSIP1) is a gene highly associated with cancer proliferation and disease progression. Its logFC marks its expression as highly differentially expressed between subtypes in the TCGA-BRCA database (Table 1). FSIP1, in both triple-negative and hormone-receptor-positive patients, is a key indicator of poor prognosis (Yan et al., 2019). FSIP1 is overexpressed in all breast cancer cases (Yan et al., 2019). FSIP1's functioning in breast cancer also involves the multidrug resistance protein 1 (MRP1) (Yan et al., 2019). Their interactions reduce patient receptiveness to chemotherapy treatments (Yan et al., 2019). Downregulating FSIP1 results in reduced proliferation and migration. Due to FSIP1's contributions to malignant transformation, overexpression of this gene results in poor patient outcomes across subtypes.

NAT1 is highly expressed in luminal A subtypes

N-acetyltransferase 1 (NAT1) encodes an enzyme crucial for carcinogen metabolism. NAT1's adjusted P-value and logFC illustrate that it is the third top significantly differentially expressed gene which is upregulated in luminal A subtypes (Table 1). NAT1 is affiliated with luminal subtypes as, amongst breast cancer tumors, its expression was highest in samples with significant estrogen-receptor expression (Zhang et al., 2018). NAT1 has also been linked to positive prognosis in luminal subtype patients. High expression levels of this gene had a positive correlation to reduced relapse rates (Zhang et al., 2018).

However, NAT1 has been linked to the promotion of bone metastasis (Zhao et al., 2020). NAT1's role in breast cancer is multi-faceted and its connection to estrogen-receptor expression has not been fully investigated yet.

ROPN1 and ROPN1B are highly expressed in basal-like subtypes

Ropporin-1 (ROPN1), a gene involved in sperm motility, is significantly overexpressed in basal-like breast cancers (Cintra et al, 2025). ROPN1 and ROPN1B have positive logFCs, marking them as upregulated in basal-like cancers, and their P-values enforce their significance (Table 1). ROPN1 is associated with post-chemotherapy resistance, thus diminishing the effectiveness of cancer treatments for basal-like patients (Cintra et al, 2025). ROPN1 has also been linked to poor prognosis and negative patient outcomes (Cintra et al, 2025). Additionally, this gene plays a key role in hypomethylation, a process connected to cancer progression (Cintra et al, 2025). Both ROPN1B and ROPN1 are related to cell motility, a process that can promote cancerous progression, and invasion (Mehta, 2010). Downregulating these genes has been shown to adversely affect motility, a process that can promote cancerous progression (Mehta, 2010). ROPN1B is particularly effective in diminishing motility (Mehta, 2010). ROPN1 and ROPN1B each play a crucial role in cancer aggressiveness.

HORMAD1 is highly expressed in basal-like subtypes

HORMAD1 (Hormad domain containing protein 1) has a unique role in triple-negative breast cancer, for it regulates crucial cellular interactions. HORMAD1's logFC and small adjusted P-value communicate its importance (Table 1). High levels of HORMAD1 inhibit cell senescence, a state of interruption part of the cell aging cycle (Zong et al., 2026). HORMAD1 jeopardizes production of the p27 protein which can help induce cell senescence. When cell senescence is compromised, cells grow and divide uncontrollably. Additionally, HORMAD1 compromises homologous recombination, the process by which chromosomes exchange segments of DNA (Watkins et al., 2015). In triple-negative breast cancers, HORMAD1 promotes alternate DNA repair methods which cause genomic disruption (Watkins et al., 2015). However, HORMAD1 has also been associated with triple-negative cancers responding to platinum chemotherapy. Thus, downregulating HORMAD1 could result in complex results, for it has varying impacts upon breast cancer progression and treatment.

Summary Table

FOXA1	FOXA1's suppression of genes associated with basal-like cancers is significant to luminal A's relative passive progression in comparison to basal-like cancers.
FSIP1	Overexpression of FSIP1 is a predictor of poor prognosis, and it heavily contributes to cancer progression in breast cancer patients.
NAT1	NAT1 is overexpressed in luminal A cancers and has been generally linked to positive patient prognosis, although it has shown an association with bone metastasis and proliferation.
ROPN1 and ROPN1B	ROPN1 and ROPN1B contribute to cancer aggressiveness and poor patient prognosis.
HORMAD1	HORMAD1 disrupts homologous recombination and cell senescence, thus compromising protein production and encouraging cancerous growth.

CONCLUSION

This paper has established that FOXA1, FSIP1, NAT1, ROPN1, ROPN1B, and HORMAD1 each play a unique part in patient outcomes and cancer development regarding luminal A and basal subtypes. The results of this study have significant implications on future research involving the treatment of basal-like cancers. The scientific community can further benefit from these findings by applying therapies and treatments that downregulate genes with a large presence in basal-like cancers that have been associated with poor prognosis. In current studies, breast cancer experts are utilizing gene silencing, the prevention of the expression of certain genes that contribute to cancer progression. MicroRNAs, RNA molecules which regulate translation of genes into proteins, and small inhibitory RNAs, molecules that degrade mRNA, can be altered to prevent the expression of these genes in breast cancer patients (Shen et al., 2013). Furthermore, gene editing tools like CRISPR have been used to correct mutations in certain genes to upregulate or downregulate gene expression (Samuels et al., 2025). Genes like ROPN1, ROPN1B, and HORMAD1 may be valuable therapeutic and clinical targets for downregulation. Many of the biological pathways they are involved with contribute to cancer aggression and advancement. On the other hand, upregulating and encouraging expression of genes that are downregulated in basal-like cancers but contribute to luminal A's nonaggressive nature may halt cancerous spread and reduce cancer severity in

basal-like subtypes. However, both downregulation and upregulation must be executed with caution so as not to cause cellular disorder. Each of the aforementioned genes has a role in regular cell functions in addition to cancer progression, meaning that silencing certain genes or significantly upregulating others could have unintended effects. Furthermore, not all genes that are highly expressed in luminal A breast cancer are purely beneficial to disease mitigation and can perhaps promote further cancerous spread. Notably, FSIP1 is overexpressed in all breast cancer subtypes and was upregulated in this study's data analysis, yet greatly contributes to cancer severity and progression. Moreover, this paper solely discusses the functions of these genes in breast cancer, however their functions expand far beyond this scope. Although gene therapy comes with risks, the need for slowing cancer metastasis and spread can outweigh certain side effects. Thus, gene therapy continues to remain relevant in cancer studies. Research which evaluates the differences in gene expression and cancer severity between breast cancer subtypes is a highly relevant course of study and acts as a powerful tool for developing new clinical treatments for breast cancer.

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