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Integrative Transcriptomic Profiling Reveals Histone Variant–Driven Immune Escape in Breast Cancer

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Abstract

Immune evasion is a central and critical challenge in oncotherapy, often driven by complex transcriptional remodelling. This study presents a systems-level in-silico analysis of breast cancer transcriptomes to uncover a novel immune escape mechanism mimicking autoimmune stress. Using differential expression analysis (logFC > 2.0, FDR < 0.001), we discovered coordinated upregulation of histone variants (e.g., H2AC19, H3C11, H2AX, H4C8, H2BC21) with immune-related genes (MHC class II, complement, Fc receptors), patterning a robust SLE-like gene module (40 genes, FDR = 7.49 × 10^{-33}). Functional enrichment via Gene ontology and KEGG pathways revealed signatures of antigen processing, immune receptor activity, and chromatin remodeling. These findings propose chronic antigenic as important outcome of this mimicry, with complement and Fc receptor upregulation potentially recruiting immunosuppressive cells. Our results position chromatin remodeling, particularly via histone variants, as an upstream regulator of immune dysfunction, offering in-silico derived targets to overcome immunotherapy resistance. This study combines transcriptomic profiling and enrichment analysis to better understand tumor-immune interactions in breast cancer.

Keywords: Immune Evasion, Histone Variants, Chromatin Remodeling, Breast Cancer, SLE Mimicry, Immunotherapy Resistance.

Introduction

Immune evasion is beginning to gain recognition as one of the key hallmarks of cancer, facilitating the escape of tumors to circumvent destruction by the immune system [1, 2]. This evasion occurs through various mechanisms, which is often categorized into three main strategies: camouflage, coercion, and cytoprotection. A study showed that cancer cells leverage pathways typically used for maintaining physiological self-tolerance to avoid immune recognition and destruction [3]. The tumor microenvironment plays an important role in this process, with cancer cells modulating immune cell composition and activity. Cancer cells employ strategies such as: downregulating antigen presentation, expressing immune checkpoint molecules, and promoting the enrichment of immunosuppressive cells like Tregs and myeloid-derived suppressor cells [4].

Histone variants play important roles in the progression of cancer, particularly in solid tumors [5, 6] . They influence chromatin structure, gene regulation, and cellular plasticity, contributing to cancer initiation and development. Cancer cells hijack histone variants and their chaperones to disrupt homeostasis and contribute to tumor growth. Outstandingly, the dysfunctional regulation and presence of histone variants affect genes associated with immune evasion, influencing immunotherapy responsiveness. For example, another study showed that cancer stem cells (CSCs) utilize epigenetic reprogramming, including histone modifications, to regulate marker protein expression and tumor plasticity, enhancing survival and metastasis.

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Recent studies highlight the important role of histone variants in immune evasion and autoimmune diseases. Altered histone variants functions can be used by cancer cells, influencing immune evasion and therapy responsiveness. In virology, some viruses employ histone mimicry to evade host immune responses, demonstrating the vulnerability of epigenetic mechanisms to viral manipulation [7]. A study using massively parallel reporter

assays revealed that histone quantitative trait loci (hQTLs) are more likely to contribute to functional mechanisms than expression QTLs, with several variants identified as potentially causal for systemic lupus erythematosus (SLE) and other autoimmune diseases [8]. Cryo-EM studies have provided new insights into how histone variant-specific features influence chromatin structure and function, particularly in transcription regulation [9].

With our bioinformatics analysis in conjunction previously established insights, we propose that aberrant upregulation of histone variant genes in breast cancer may elicit autoimmune-like immune activation, reinforcing tumor escape from immune surveillance. This concept builds on new evidences that tumors may mimic autoimmune stress, particularly SLE-like epigenetic instability to reshape antigen presentation, thereby recruiting immunosuppressive cells.

Materials and Methods

Data Acquisition and Pre-processing

The study utilized publicly available gene expression data from the GEO database (Accession: GSE134359), comprising transcriptomic profiles of normal and cancerous human tissues. Raw data were extracted from the series matrix file using line-based indexing to isolate the expression matrix. Probe-level counts were parsed and filtered to include samples from 12 normal tissues and 30 cancer tissues, as specified by GSM identifiers. The resulting dataset was subjected to sanity checks for data type consistency.

Normalization and Differential Expression Analysis

Quantile normalization was performed using the normalizeBetweenArrays() function from the limma package to correct for distributional differences across arrays. A design matrix was constructed to distinguish between "Normal_tissue" and "Cancer" conditions, followed by linear modeling (lmFit) and empirical Bayes moderation (eBayes). Contrast matrices were defined to compute differential expression between cancer and normal groups. Adjusted p-values (FDR < 0.05) and log2 fold changes were used to identify significantly deregulated genes, which were visualized using a volcano plot.

Gene Annotation and Classification

Probe identifiers were annotated using the hta20transcriptcluster.db package to map probes to gene symbols. Significantly upregulated (logFC > 1) and downregulated (logFC < -1) genes were stratified and visualized using a Venn diagram.

Functional Enrichment Analysis

To assess biological relevance, gene ontology (GO) enrichment was conducted using the clusterProfiler and org.Hs.eg.db packages. Gene symbols were converted to Entrez IDs via bitr() and analyzed across Biological Process (BP), Cellular Compartment (CC) and Molecular Function (MF) categories. Enrichment was quantified using adjusted p-values (FDR < 0.05), and top categories were visualized with bar plots to illustrate overrepresented functional terms.

Network Clustering

Network construction and clustering were performed in Cytoscape v3.10.3 using the MCODE plugin to identify densely connected molecular modules from the upregulated gene set. Clusters were ranked by MCODE score, and Cluster 2 (score = 26.846%; 27 nodes; 349 edges) was selected as a representative module for functional inference. GO enrichment across Biological Process, Molecular Function, and Cellular Component ontologies was carried out using clusterProfiler with an FDR < 0.05. Immune-related categories, including antigen processing and presentation via MHC pathways were noted as hypothesis-generating indicators of coordinated biological activity.

Pathway Enrichment Analysis

Pathway enrichment analysis was performed using the WebGestalt (WEB-based Gene SeT AnaLysis Toolkit) platform, selecting the KEGG (Kyoto Encyclopedia of Genes and Genomes) pathway database as the functional reference. Significance was determined using the hypergeometric test with Benjamini–Hochberg FDR correction. The top enriched pathway is summarised in Table 1.

Results

Upregulation of histone variant genes in breast cancer

Transcriptomic analysis revealed significant upregulation of a broad panel of histone variant, immune-related, and complement genes in breast cancer tissues. Notably, histone variants including H2AC19, H3C11, H2AC13, H4C8, H2BC21, H3C15, H3C10, H2AC8, H2BC8, H4C15, H4C12, H2BC7, H2BC12, H2BC14, H2BC4, H3C7, H2BC5, H2BC17, H2AC6, H2BC11, H4C11, H2AC21, H3C2, H2AC16, H2AC17, H2AX, H2AC11, together with antigen-presentation genes HLA-DQA1, HLA-DRB5, HLA-DQA2, HLA-DRB3, HLA-DQB1, HLA-DRB1, complement components C1QC, C4B, C4A, C2, Fc receptor genes FCGR3A, FCGR1A, and the co-stimulatory molecule CD86, all displayed fold changes exceeding 2.0 with adjusted p-values < 0.001. These genes are collectively associated with chromatin remodeling, antigen processing and presentation, innate immune activation, and immune checkpoint regulation, highlighting their potential integrated role in tumor progression and immune modulation.

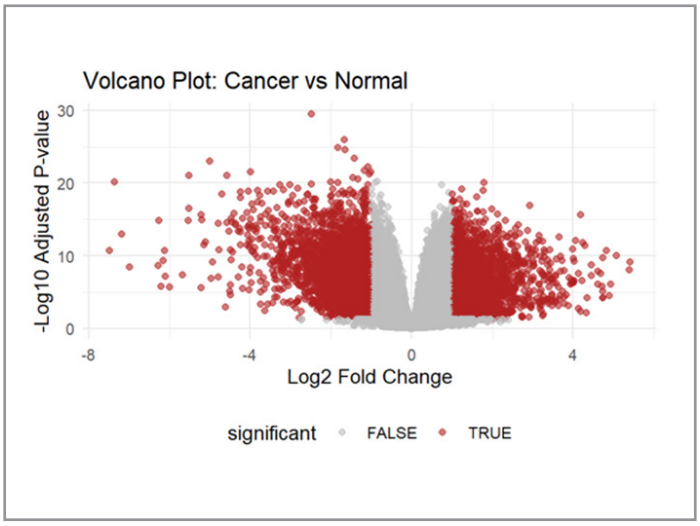

Figure 1: Volcano Plot of Differentially Expressed Genes. Each point represents a gene, plotted by its $\log_2$ fold change versus $-\log_{10}$ adjusted p-value. Genes significantly upregulated or downregulated in cancer tissues compared to normal controls are highlighted in red, while non-significant genes are marked in grey. The broad distribution illustrates transcriptional deregulation between the two conditions, with the greatest changes observed in genes at the plot's far left and right extremities.

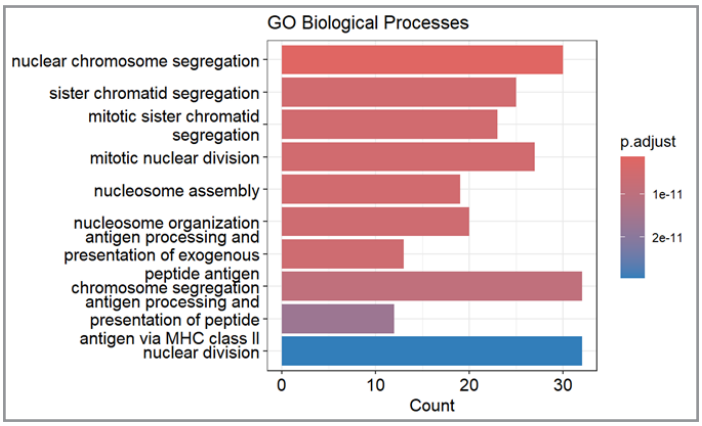

Figure 2: Immune-related Biological Processes in Cluster 2. GO enrichment analysis of Cluster 2 genes (FDR < 0.05) reveals over-representation of immune-associated processes, including antigen processing and presentation via MHC class I/II pathways. These terms underscore the module's potential role in modulating tumor–immune interactions.

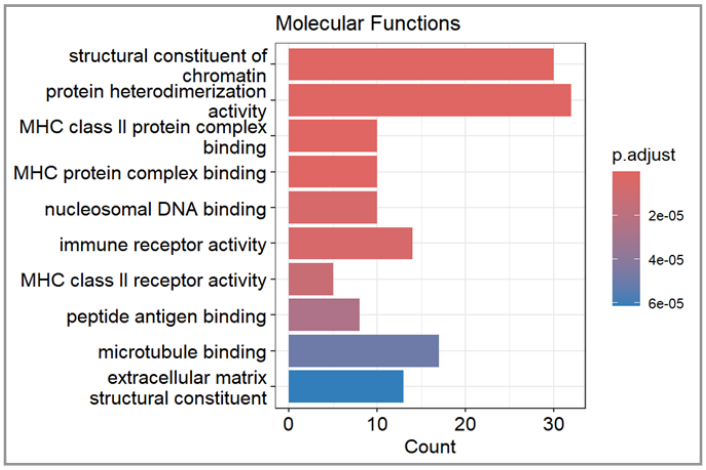

Figure 3: Immune-related Molecular Functions in Cluster 2. GO molecular-function enrichment of Cluster 2 genes (FDR < 0.05) highlights immune-specific activities, including MHC class II protein complex binding, MHC protein complex binding, MHC class II receptor activity, peptide antigen binding, and immune receptor activity. These terms emphasize the module's potential involvement in antigen recognition and presentation pathways central to tumor–immune system interplay.

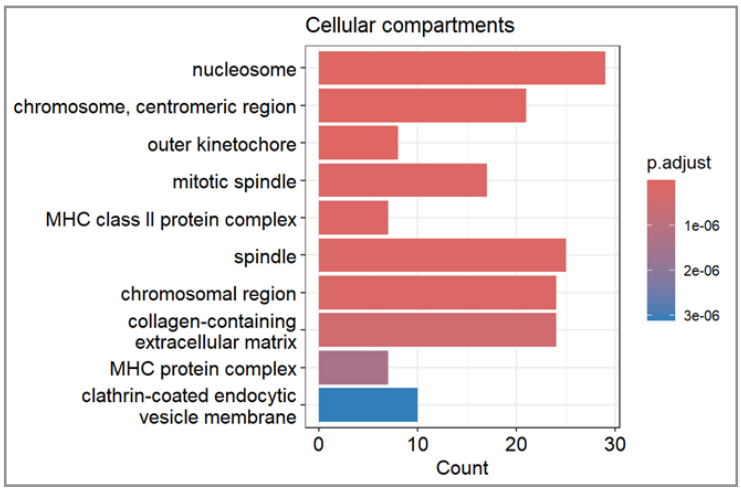

Figure 4: Immune-related Cellular Components in Cluster 2. GO cellular-component enrichment of Cluster 2 genes (FDR < 0.05) identifies immune-associated localizations, including the MHC class II protein complex and broader MHC protein complex, alongside compartments such as the clathrin-coated endocytic vesicle membrane that facilitate antigen uptake and presentation. These enriched components point to the module's potential role in orchestrating antigen processing and display within the tumor–immune microenvironment.

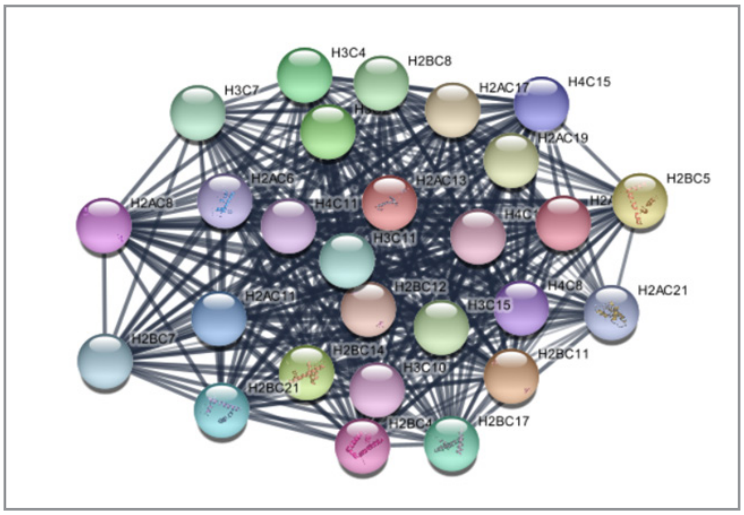

Figure 5: Immune-regulatory network module in Cluster 2. Network construction in Cytoscape and clustering via MCODE identified nine molecular modules. Cluster 2 (score = 26.846%; 27 nodes; 349 edges) represents a densely interconnected subnetwork enriched for immune regulation, including antigen processing and presentation through MHC pathways. The tight connectivity of this module suggests coordinated control points within the tumor–immune interface.

Enrichment of Systemic lupus Erythematosus Pathway

To explore the functional relevance of these genes, we performed pathway enrichment analysis using WebGestalt, focusing on KEGG pathways. The analysis revealed a highly significant enrichment of the systemic lupus erythematosus (SLE) pathway (hsa05322), with 40 genes overlapping from our dataset (FDR = 7.49×10^{-33}; enrichment ratio = 13.57). These genes were initially clustered using Cytoscape, which identified tightly connected modules enriched in immune and epigenetic regulators.

Table 1: KEGG pathway enrichment result for Systemic lupus erythematosus. Summary of the top enriched pathway identified using WebGestalt with the KEGG database. The table shows pathway size, number of overlapping genes from the input list, enrichment ratio, statistical significance (p-value, FDR), and the list of overlapping genes.

Pathway	Size	Overlap	Enrichment Ratio	Signifcane(p-value, FDR)	Genes
Systemic lupus erythematosus	131	40	13.5669	2.1266e 35, 7.4856e 33	HLA DQA1, H2AC19, H3C11, H2AC13, H4C8, H2BC21, H3C15, H3C10, H2AC8, H2BC8, H4C15, HLA DRB5, H4C12, H2BC7, H2BC12, C1QC, H2BC14, H2BC4, H3C7, H2BC5, H2BC17, H2AC6, HLA DQA2, H2BC11, HLA DRB3, H4C11, C4B, H2AC21, FCGR3A, C4A, H3C2, HLA DQB1, C2, H2AC16, HLA DRB1, FCGR1A, H2AC17, H2AX, H2AC11, CD86

These genes are central to autoimmune signaling, antigen presentation, and immune cell regulation, supporting the hypothesis that breast cancer cells may exploit autoimmune-like mechanisms, particularly SLE-associated epigenetic stress to evade immune surveillance.

Discussion

Our aim in this study is to explore the complex molecular signals that define breast cancer phenotypes at the intersection of chromatin architecture and immune modulation. By combiing transcriptomic profiling with systems-level network analysis, we discovered a coordinated upregulation of histone variant genes, immune-regulatory transcripts, and complement components that together form a complex tumour–immune. Such changes which encompass alterations in nucleosome composition, heightened antigen presentation machinery, and activation of innate immune effectors represents a potential "early warning" signature of tumours capable of evading immune surveillance.

Histone variant upregulation may remodel chromatin accessibility to favour transcriptional programmes that reinforce antigen presentation and chronic immune activation, creating a paradoxical setting in which tumours present abundant antigen yet co-opt regulatory checkpoints to evade cytotoxic clearance. The co-occurrence of elevated MHC class II transcripts, Fcγ receptor genes, and complement components suggests a rewired interface in which antigen display is coupled to complement-mediated inflammation, potentially driving an immunological milieu reminiscent of SLE. In autoimmune disease, such sustained activation leads to tissue damage; in the neoplastic context, our data point toward its exploitation for tumour progression, tolerance induction, and metastatic competence.

Recent research has highlighted the critical role of histone variants in cancer pathobiology, particularly in breast cancer. H2A.J, a mammalian-specific histone variant, shows cell-type specific expression with striking enrichment in luminal epithelial cells of multiple glands, including breast tissue . This variant is highly expressed in carcinoma cell lines, especially those derived from luminal breast and prostate cancers, and appears to function as a novel marker for luminal epithelial cancers [10]. The broader histone variant network is increasingly recognized as being hijacked by solid tumors, with variants and their chaperones serving as either tumor-promoting or tumor-suppressive players in cancer pathogenesis. Macro H2A isoforms, whose expression is disrupted across multiple breast cancer subtypes, have been shown to remodel transcriptional landscapes in ways that promote tumour adaptation and progression . More broadly, H2A variants participate directly in DNA damage sensing and repair, transcriptional regulation, and chromosomal segregation; core processes whose dysregulation can both drive tumour evolution and create therapeutic vulnerabilities [11, 12].

Notably, dysregulated expression is not confined to the H2A family: variants from the H2B, H3, and H4 lineages also show altered abundance and functional engagement in breast cancer, influencing tumour plasticity, prognosis, and potentially, response to therapy [13, 14]. Altered histone variant deposition emerges as a potent regulator of nucleosome dynamics and chromatin accessibility, with direct implications for transcriptional plasticity in cancer. Incorporation of the H2A.Z variant lowers the energetic threshold for DNA end-unwrapping, triggering spontaneous release of ~40 bp from both nucleosomal termini and thereby enhancing DNA accessibility and nucleosome mobility [15, 16]. This destabilising effect is mediated predominantly through the N- and C-terminal tails of H2A.Z, which modulate histone–DNA contacts; by contrast, the H3.3 variant exerts minimal influence on unwrapping kinetics.

Such structural re-programming is not random: deposition of histone H3 variants is tightly choreographed by cell-cycle phase, cellular potency, stress responses, and fate transitions, with measurable consequences for nucleosome stability and the maintenance or re-specification of cell identity [17]. Variant-driven plasticity intersects with the function of the SWI/SNF chromatin-remodeling complex, a central regulator of gene expression that is altered in approximately 20–25% of all human cancers [18, 19]. Its key subunits such as ARID1A, which is involved with complex targeting, and SMARCA4, responsible for ATPase activity, are central to maintaining enhancer fidelity and directing lineage-specific transcription [20]. Mutations in these components disturb enhancer architecture, inhibit differentiation, and promote stem-like cellular states, effects that is consistent with those seen with dysfunctional histone variants upregulation. ARID1A mutations are particularly frequent, occurring in 40–50% of ovarian clear cell carcinomas and 15–30% of cholangiocarcinomas, highlighting their clinical significance [21].

Therapeutically, cancers with SWI/SNF mutations often exhibit synthetic-lethal vulnerabilities. Importantly, ARID1A loss has been associated with enhanced responses to immune checkpoint blockade. These findings are consistent with our study, which demonstrates that disruption of chromatin remodelers and altered histone variants expression can generate chromatin states resembling that of viral infection. This leads to the exposure of cryptic regulatory elements, amplification of interferon signaling, and increased tumor immunogenicity.

Chromatin remodeling helps control how immune response genes, antigen presentation machinery (APM), and immune checkpoints are expressed. Drugs that affect epigenetics, like histone deacetylase inhibitors and DNA methyltransferase inhibitors, can increase the expression and function of HLA class I APM components in different cancers, which helps explain why they work well with immune checkpoint inhibitors (ICIs) [22]. The SWI/SNF complex also plays a role in shaping tumor immunogenicity by influencing interferon signaling and DNA repair [23].

This link between chromatin remodeling and immune activity isn't limited to our model. Other studies have shown that immune-related genes are often upregulated in various cancers. For example, in muscle-invasive bladder cancer, there's strong activation of chemokines, antigen-processing genes, and IFN-γ–responsive transcripts [24]. Similarly, in a non-cancer setting, inflammatory bowel disease, the colon's epithelial cells show increased expression of antigen-presentation machinery, including MHC class II molecules and key transcription factors [25]. Taken together with our findings, these observations support the idea that abnormal changes in antigen-processing and presentation whether caused by histone variants, SWI/SNF disruption, or other epigenetic shifts, can send the body into "autoimmune

mimicry" state. From analysis, our breast cancer results show a proliferative–immunogenic program controlled by two major axes:

(1) Antigen-presentation machinery and immune effectors, with strong upregulation of multiple MHC-II genes (HLA-DQA1, HLA-DQA2, HLA-DQB1, HLA-DRB1, HLA-DRB3, HLA-DRB5) and co-stimulatory and immune-complex receptors (CD86, FCGR1A, FCGR3A), alongside complement components (C1QC, C2, C4A, C4B) that can modulate immune cell recruitment and activation; and
(2) Core histone and histone-variant genes (including a broad set of H2A, H2B, H3, and H4 family members such as H2AC19, H2BC21, H3C15, H4C8, H2BC8, H3C10, H2AX) that define chromatin structure and are central to mitotic progression and epigenetic regulation.

This tumor phenotype closely resembles the lupus-like environment described earlier, characterized by sustained interferon signaling, persistent antigen presentation, and elevated cell proliferation. The increased expression of histone variants suggests active nucleosome turnover and chromatin remodeling, which help maintain transcriptional accessibility at key antigen-processing loci, including MHC genes, complement components, and Fc receptors. As a result, cancer cells remain immunologically visible even during mitosis, mirroring features of chronic autoimmune inflammation.

Recent studies have shown that cancer cells can adopt immune-like transcriptional profiles, contributing to an inflamed or autoimmune-like tumor state, which are recognized hallmark that shapes tumor–immune interactions and influences clinical outcomes [26]. In our transcriptomic data, we observed upregulation of MHC class II genes (HLA-DQA1, DQA2, DQB1, DRB1, DRB3, DRB5) alongside CD86, a pattern consistent with immune-driven selection pressures. Such pressures may favor the expansion of tumor clones with inactivated tumor suppressor genes [27].

These dynamics can significantly affect responses to immune checkpoint blockade. Tumors that have evaded immune detection yet retain high antigen presentation often respond better to immunotherapy [28]. However, in immunosuppressive microenvironments rich in tolerogenic cytokines, the same antigen presentation profile especially when accompanied by CD86 costimulation may instead promote the expansion of $FOXP3^+$ regulatory T cells. This is consistent with findings by Greilach et al. in non-cancer contexts [29].

Further supporting this model, our signature includes complement components (C1QC, C2, C4A, C4B) and Fcγ receptors (FCGR1A, FCGR3A), which suggest recruitment and activation of immunoregulatory myeloid cells. These populations can reinforce Treg-mediated suppression. Altogether, these observations point to a state where high antigen visibility does not guarantee tumor elimination. Instead, it may be exploited by the tumor to alter $CD4^+$ T-cell responses toward regulatory pathways, suppressing effective immunity and enabling immune escape.

Translational and Therapeutic Implications
Epigenetic and Immunological Biomarker Development

The coordinated upregulation of histone variant genes and chromatin-remodelling components in our dataset, together with elevated complement transcripts and systemic-lupus-erythematosus-like (SLE-like) immune signatures, positions these features as promising biomarkers for clinical application.

Limitations and Future Perspectives

While our bulk transcriptomic analysis has provided valuable insights into the molecular and immunological landscape of the tumours studied, several limitations should be acknowledged. The complexity of the tumour microenvironment (TME) introduces unavoidable admixture of tumour-intrinsic and non-tumour cell signals, and our interpretations are constrained by the inherent difficulty of cell-type deconvolution in bulk datasets.

There is a pressing need for functional validation of transcriptomic signatures. Linking specific molecular alterations to phenotypic outputs such as measurable immune-evasion mechanisms or therapy-resistance phenotypes will be essential to move from correlation to causation. These combined strategies will not only refine the biological interpretation of our current findings but also accelerate the translation of transcriptomic biomarkers into clinically actionable tools.

Declarations

Ethics Approval and Consent to Participate

Not applicable.

Consent for Publication

Not applicable.

Availability of Data and Materials

The dataset supporting the conclusions of this article is available in the (NCBI) database, https://www.ncbi.nlm.nih.gov/search/all/?term=GSE134359

Competing Interests

The authors declare that they have no competing interests.

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Authors' Contributions

The authors conceived the study, performed the analysis, and drafted the manuscript. All authors read and approved the final manuscript.

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Authors' Information

Not applicable.

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