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Transcriptomic Patterns in Endometriosis: From Molecular Mechanisms to Biomarker Potential-A Mini-Review

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Abstract

Endometriosis is a chronic, estrogen-dependent gynecological disorder characterized by the presence of endometrial-like tissue outside the uterine cavity, frequently associated with pelvic pain and infertility. Advances in high-throughput transcriptomic technologies have enabled comprehensive profiling of both coding and non-coding RNAs, yielding novel insights into the molecular mechanisms underlying this complex condition. Differential expression analyses consistently reveal aberrant regulation of mRNAs involved in inflammation, angiogenesis, extracellular matrix (ECM) remodeling, and hormone receptor signaling. In parallel, microRNAs (miRNAs) are increasingly recognized as crucial post-transcriptional regulators that contribute to epithelial-mesenchymal transition (EMT), cell survival, and immune dysregulation. Long non-coding RNAs (lncRNAs) further complicate this landscape by functioning as competing endogenous RNAs (ceRNAs), sequestering miRNAs and indirectly modulating gene expression networks. The integration of these transcriptomic patterns suggests the existence of intricate, interconnected regulatory networks that drive lesion establishment and progression. Furthermore, circulating RNAs in serum and plasma show considerable promise as non-invasive biomarkers, while RNA-targeted interventions may represent novel therapeutic avenues. This mini-review synthesizes current knowledge on transcriptomic alterations in endometriosis, highlighting their diagnostic potential, mechanistic significance, and future implications for precision medicine.

Keywords: endometriosis, transcriptomics, mRNA, microRNA, long non-coding RNA, biomarkers

1. Introduction

Endometriosis is a chronic gynecological disorder characterized by the ectopic implantation and growth of endometrial-like tissue outside the uterine cavity, primarily affecting the ovaries, pelvic peritoneum, and, in severe cases, distant organs. It affects approximately 10–15% of women of reproductive age, contributing significantly to infertility and pelvic pain [1-4]. Despite its prevalence, the disease often remains underdiagnosed for many years, with a median diagnostic delay of 7–10 years, largely due to nonspecific symptoms and the reliance on invasive laparoscopy as the gold standard for diagnosis [5].

The etiology of endometriosis is multifactorial, involving genetic predisposition, epigenetic regulation, hormonal imbalances, immune dysregulation, and environmental influences [6-8]. In recent years, transcriptomic research has emerged as a powerful approach to unravel the molecular complexity of this disorder. High-throughput technologies such as microarray profiling and RNA sequencing (RNA-seq) have enabled the systematic exploration of both coding and non-coding RNAs, offering new insights into the cellular pathways that underpin endometriotic lesion establishment and persistence [9,10].

Among coding RNAs, dysregulation of genes associated with inflammation, angiogenesis, extracellular matrix remodeling, and hormone signaling have been recurrently observed in endometriotic tissues [11]. Beyond protein-coding genes, non-coding RNAs, including microRNAs (miRNAs), long non-coding RNAs (lncRNAs), and circular RNAs (circRNAs), have gained attention as key post-transcriptional regulators of gene expression in endometriosis [12, 13]. These molecules modulate cellular proliferation, apoptosis, invasion, and immune interactions, and increasingly serve as potential biomarkers detectable in body fluids [14].

Collectively, transcriptomic research has greatly advanced our understanding of endometriosis by uncovering both coding and non-coding RNA networks that contribute to its development and persistence. This mini-review synthesizes current evidence on mRNA, miRNA, and lncRNA expression patterns in endometriosis, emphasizing their mechanistic relevance and exploring their potential as diagnostic biomarkers and therapeutic targets.

Transcriptomic analyses consistently report altered mRNA expression in endometriotic lesions compared to eutopic endometrium. Dysregulated pathways include estrogen and progesterone signaling, immune modulation, and extracellular matrix remodeling [15-17].

For instance, overexpression of VEGFA and IL6 contributes to angiogenesis and chronic inflammation,

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whereas aberrant expression of MMP2 and MMP9 promotes tissue invasion [18]. Additionally, downregulation of progesterone receptor (PGR) and associated target genes underscore progesterone resistance, a hallmark of endometriosis-related infertility [19]. Key dysregulated mRNAs and their associated pathways are summarized in Table 1.

Table 1: Selected dysregulated mRNAs implicated in endometriosis

Gene	Function	Expression Pattern	Pathway Involvement
VEGFA	Angiogenesis	Upregulated	Angiogenic signaling [20]
IL6	Cytokine, inflammation	Upregulated	Immune activation [21]
MMP2/MMP9	Matrix degradation	Upregulated	ECM remodeling [22]
PGR	Progesterone receptor	Downregulated	Hormone resistance [23]
ESR1	Estrogen receptor	Dysregulated	Estrogen signaling [24]

2. mRNA Expression Profiles in Endometriosis

2.1. Non-Coding RNAs in Endometriosis

2.1.1. microRNAs

miRNAs function as post-transcriptional repressors and are profoundly implicated in endometriosis. Dysregulated miRNAs modulate key processes such as proliferation, apoptosis, and invasion. Notably, the miR-200 family is downregulated in endometriotic tissue, facilitating epithelial–mesenchymal transition (EMT) by derepressing transcription factors such as ZEB1 and ZEB2 [25]. In contrast, miR-21 is frequently upregulated and promotes survival through inhibition of PTEN [26].

2.1.2. Long Non-Coding RNAs

lncRNAs exert multifaceted regulatory effects via mechanisms encompassing chromatin remodeling, transcriptional regulation, and ceRNA activity. Prominent examples include H19, which modulates estrogen signaling, and MALAT1, which promotes proliferation and migration through interaction with EMT-associated pathways [27]. NEAT1, often upregulated, has been reported to regulate inflammatory signaling and immune escape [28, 29]. Selected miRNAs and lncRNAs with validated roles in endometriosis are detailed in Table 2.

Table 2. Representative non-coding RNAs implicated in endometriosis

ncRNA	Type	Function	Expression Pattern	Mechanism
miR-200 family	miRNA	EMT regulation	Downregulated	Targets *ZEB1/ZEB2* [30]
miR-21	miRNA	Cell survival	Upregulated	Inhibits *PTEN* [31]
H19	lncRNA	Hormone signaling	Upregulated	Estrogen modulation [32]
MALAT1	lncRNA	Proliferation, migration	Upregulated	EMT promotion [33]
NEAT1	lncRNA	Inflammation, immune modulation	Upregulated	ceRNA regulation [34]

3. Integrative Transcriptomic Analyses and Biomarker Potential

High-throughput integrative studies combining mRNA, miRNA, and lncRNA profiles have revealed ceRNA networks that govern gene regulation in endometriosis. For example, H19 and MALAT1 act as miRNA sponges, indirectly influencing mRNAs implicated in inflammation and EMT. Similarly, dysregulated miRNAs in circulation, such as miR-200 family members, have been proposed as non-invasive biomarkers [35, 36].

Clinical translation of transcriptomic signatures remains a challenge, as reproducibility across cohorts is limited. However, progress in serum-based transcriptomic assays and bioinformatic classifiers is promising [37].

An integrative model illustrating how coding and non-coding RNAs collectively influence the molecular pathways that drive endometriotic lesion persistence is presented in Figure 1.

4. Discussion

The integration of transcriptomic data in endometriosis research has deepened our mechanistic understanding of the disease. Dysregulated mRNA signatures highlight processes such as angiogenesis, immune modulation, and extracellular matrix remodeling, which are crucial for lesion survival [38-40]. Non-coding RNAs further refine these regulatory landscapes, functioning through complex competing endogenous RNA (ceRNA) networks that govern gene–gene and RNA–RNA interactions [41].

Emerging evidence indicates that epigenetic regulation of transcriptomes contributes substantially to the persistence of endometriotic lesions. Aberrant DNA methylation and histone modifications alter transcription factor accessibility, thereby shaping gene expression profiles [42]. For instance, hypermethylation of the HOXA10 promoter reduces its expression in the endometrium, impairing implantation and contributing to infertility [43]. Similarly, dysregulated expression of epigenetically controlled lncRNAs, such as H19 and NEAT1, links chromatin-level changes to altered cellular behavior [44].

From a systems biology perspective, network-based analyses have revealed that hub RNAs (e.g., MALAT1,

miR-200 family) occupy central positions in regulatory modules and may represent critical intervention points [45]. This observation supports the concept that targeting non-coding RNA hubs could provide therapeutic leverage beyond classical hormone modulation. However, given the context-dependent activity of ncRNAs, validation across heterogeneous patient cohorts remains essential [46].

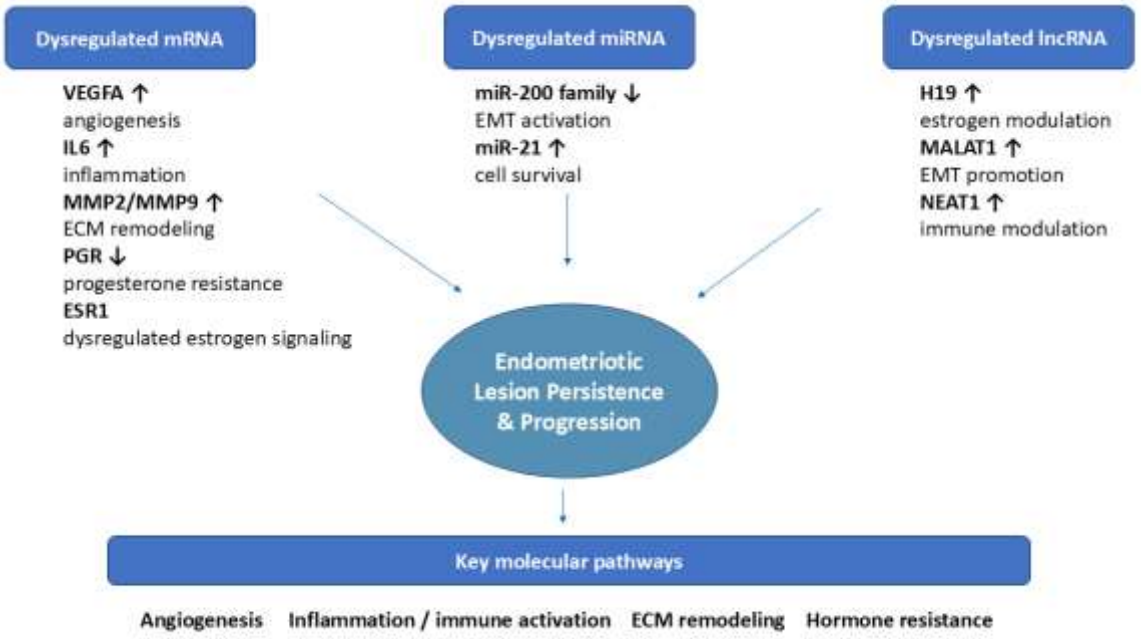

Figure 1: Integrated transcriptomic model of dysregulated coding and non-coding RNAs in endometriosis

Another critical translational avenue is the development of non-invasive biomarkers. Circulating miRNAs such as miR-200 family members and miR-21 show promise as diagnostic tools, yet their clinical applicability is limited by inter-individual variability and the lack of standardized assays [47]. Integrating multiple transcriptomic signals into composite signatures, possibly via machine learning models, has the potential to improve diagnostic precision [48]. Moreover, saliva, menstrual effluent, and extracellular vesicles have emerged as alternative biofluid sources for transcriptomic biomarker discovery, widening the scope of accessible non-invasive samples [49]. While transcriptomic studies have vastly expanded our molecular understanding of endometriosis, several challenges persist. The heterogeneity of transcriptomic profiles across different studies and patient populations hampers reproducibility and generalizability.

Future research must prioritize larger, well-controlled, multi-center studies that incorporate multi-omics integration (transcriptomic, epigenomic, proteomic) to capture the full complexity of the disease. The promising field of RNA-based therapeutics (e.g., miRNA mimics or antagomirs, lncRNA targeting) remains in its infancy for endometriosis but represents a frontier for intervention. Overcoming delivery challenges and ensuring target specificity will be crucial steps forward. Furthermore, the prospective validation of transcriptomic biomarkers in large, independent cohorts is indispensable for their eventual adoption into clinical practice.

5. Conclusion

Transcriptomic research has provided unprecedented insight into the molecular underpinnings of endometriosis. Dysregulated mRNAs delineate pathways of hormone resistance, invasion, and inflammation, while non-coding RNAs—particularly miRNAs and lncRNAs—modulate these pathways through fine regulatory networks. The crosstalk within ceRNA networks underscores the remarkable complexity of transcriptomic regulation in endometriotic lesions. Overall, these studies not only expand our mechanistic knowledge but also open tangible avenues for precision diagnostics and therapeutic innovation. As multi-omics integration progresses, the future lies in systematically translating these molecular insights into clinically actionable strategies, ultimately improving outcomes for patients afflicted by this debilitating condition.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

Author Contributions

S.S designed the research theme and executed the research process. S.S. and A.S. wrote and edited the manuscript. Both authors have accepted responsibility for the entire content of this manuscript and approved its submission.

Ethical Consideration

Not applicable.

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Declaration of AI Use

The authors employed the AI tool ChatGPT during the writing process exclusively to assist with grammar, syntax, and readability. Following this assistance, the authors thoroughly reviewed, substantively edited, and validated all scholarly content. The authors are ultimately accountable for all ideas, conclusions, and the integrity of the published work.

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