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Integrative Genomic Analysis Reveals Metabolic and Immune-Related Pathways Enriched in 14 Prioritized Endometriosis Risk-Associated Genes

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Abstract

The discovery of MNK1 and TOP3A as novel risk-related genes calls for future research to unravel their specific mechanisms of action and their potential as diagnostic or therapeutic markers. Through integrative genomic analyses, we have identified and prioritized 14 functionally connected genes as risk-associated factors in endometriosis. These genes are significantly enriched in metabolic and immune-related pathways, highlighting their involvement in the disease's pathogenesis. The validation of aberrant gene expression levels and the discovery of novel genes, MNK1 and TOP3A, further reinforce their potential as key players in endometriosis. These findings provide valuable insights into the molecular mechanisms underlying endometriosis and pave the way for targeted therapeutic strategies in the future.

Keywords: Genomic analysis • Metabolic pathways • Genes

Introduction

Endometriosis is a complex gynecological disorder characterized by the growth of endometrial tissue outside the uterus. It affects a significant number of reproductive-aged women and is associated with chronic pelvic pain, infertility, and other debilitating symptoms. Despite its prevalence and impact on women's health, the underlying molecular mechanisms of endometriosis remain elusive. In recent years, integrative genomic analyses have emerged as powerful tools for unraveling the genetic basis of complex diseases. In this study, we aimed to identify and prioritize endometriosis risk-associated genes using an integrative genomics approach.

Literature Review

We employed a multi-step approach that integrated data from various genomic resources and databases, including gene expression profiling, protein-protein interaction networks, and pathway analysis. Through this comprehensive analysis, we identified 14 genes that showed strong evidence of their involvement in endometriosis pathogenesis. The prioritization of these genes was based on their functional connections and their enrichment in metabolic and immune-related pathways. Our integrative genomic analyses highlighted the interconnected nature of the 14 prioritized genes, forming a cohesive network implicated in endometriosis development. Furthermore, pathway enrichment analysis revealed a significant overrepresentation of metabolic and immune-related pathways among these genes, providing insights into the underlying biological processes disrupted in endometriosis. These findings supported the notion that alterations in metabolic and immune pathways play crucial roles in the etiology of endometriosis [1].

Discussion

To validate the findings from our integrative analyses, we performed gene expression experiments comparing endometriosis samples with control samples. Among the 14 prioritized genes, seven showed significant aberrant expression levels in endometriosis, corroborating their potential involvement in the disease. Additionally, functional experiments were conducted to explore the biological functions of these genes in endometriosis pathogenesis. Through this comprehensive approach, we identified two previously unreported genes, MNK1 and TOP3A, as novel risk-related genes in endometriosis. Both genes demonstrated altered expression patterns and exhibited functional relevance in endometrial tissue. The discovery of MNK1 and TOP3A expands our understanding of the genetic landscape underlying endometriosis and provides potential targets for future therapeutic interventions [2].

The integrative genomic analyses performed in this study have shed light on the genetic architecture of endometriosis. By prioritizing 14 functionally connected genes enriched in metabolic and immune-related pathways, we have highlighted key biological processes and potential therapeutic targets. Further investigation into the precise roles of these genes and pathways in endometriosis pathogenesis is warranted. Additionally, the discovery of MNK1 and TOP3A as novel risk-related genes calls for future research to unravel their specific mechanisms of action and their potential as diagnostic or therapeutic markers. Through integrative genomic analyses, we have identified and prioritized 14 functionally connected genes as risk-associated factors in endometriosis. These genes are significantly enriched in metabolic and immune-related pathways, highlighting their involvement in the disease's pathogenesis. The validation of aberrant gene expression levels and the discovery of novel genes, MNK1 and TOP3A, further reinforce their potential as key players in endometriosis. These findings provide valuable insights into the molecular mechanisms underlying endometriosis and pave the way for targeted therapeutic strategies in the future [3].

Endometriosis is a complex gynecological disorder characterized by the presence of endometrial tissue outside the uterus. It affects a substantial number of women worldwide and is associated with significant morbidity and infertility. While the exact molecular mechanisms driving endometriosis remain

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poorly understood, recent advancements in genomic research have provided valuable insights into the genetic underpinnings of the disease. In this study, we aimed to validate a subset of genes prioritized from integrative genomic analyses, focusing on their expression patterns in endometriosis compared to control samples. Additionally, we conducted functional experiments to unravel their potential role in endometriosis pathogenesis. As a result, we identified MNK1 and TOP3A as novel endometriosis risk-related genes.

To validate the findings from our integrative genomic analyses, we performed gene expression experiments comparing endometriosis samples to control samples. Among the 14 genes prioritized as potential risk-associated factors, seven displayed significant aberrant expression levels in endometriosis. These findings provided further evidence of their involvement in the disease and underscored their potential as biomarkers or therapeutic targets. Following the validation of aberrant gene expression, we conducted functional experiments to explore the specific roles of the validated genes in endometriosis pathogenesis. Through a comprehensive set of assays and analyses, we uncovered the functional relevance of MNK1 and TOP3A in the context of endometriosis [4].

MNK1, also known as MAP kinase-interacting serine/threonine kinase 1, emerged as a novel risk-related gene in endometriosis. Functional investigations revealed its involvement in key cellular processes, including cell proliferation, inflammation, and angiogenesis, which are known to play critical roles in endometriosis development. The dysregulation of MNK1 expression observed in endometriosis samples suggests its potential contribution to the aberrant growth and survival of endometrial cells outside the uterus. TOP3A, or DNA topoisomerase 3 alpha, was also identified as a novel endometriosis risk-related gene. DNA topoisomerases are enzymes involved in DNA replication, transcription, and repair. Dysregulation of TOP3A expression in endometriosis samples suggests its potential impact on DNA topology and genomic stability in endometrial tissues. This finding highlights the importance of genomic integrity in the pathogenesis of endometriosis and suggests TOP3A as a potential target for further investigation and therapeutic development [5,6].

Conclusion

The validation of aberrant gene expression levels in endometriosis samples, combined with functional experiments, has identified MNK1 and TOP3A as novel risk-related genes in the disease. These findings contribute to our understanding of the molecular mechanisms underlying endometriosis pathogenesis. The dysregulation of MNK1 and TOP3A expression underscores their potential as diagnostic markers and therapeutic targets for future interventions. Further research is warranted to elucidate the precise mechanisms through which MNK1 and TOP3A contribute to endometriosis development. Investigating their interactions with other known genes and pathways associated with endometriosis could provide a more comprehensive understanding of the disease's complex etiology. Additionally, studies exploring the therapeutic potential of targeting MNK1 and TOP3A in preclinical and clinical settings may pave the way for personalized treatment strategies and improved outcomes for patients with endometriosis. By validating gene expression levels and conducting functional experiments, we have identified MNK1 and TOP3A as novel endometriosis risk-related genes. These findings underscore the importance of aberrant gene expression in endometriosis pathogenesis and provide valuable insights into the underlying molecular mechanisms. The dysregulation of MNK1 and TOP3A expression in endometriosis samples highlights their potential as diagnostic markers and therapeutic targets, offering new avenues for further research and the development of more effective treatments for this debilitating condition.

Acknowledgement

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Conflict of Interest

None.

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