The Role of Signaling Pathways, microRNAs, and lncRNAs in the Pathophysiology of Endometriosis

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Abstract

Endometriosis is a chronic, estrogen-dependent gynecological disorder characterized by the growth of endometrial-like tissue outside the uterus, causing pelvic pain, infertility, and inflammation. Despite significant research, the exact etiology remains unclear. This review provides a comprehensive exploration of the molecular mechanisms underlying endometriosis, focusing on genetic factors, signaling pathways, microRNAs (miRNAs), and long non-coding RNAs (lncRNAs). Genetic predisposition plays a pivotal role, with polymorphisms and mutations in genes such as PTEN, TP53, KRAS, and hormone receptors linked to disease susceptibility. Dysregulated signaling pathways, including MAPK, PI3K/Akt, Wnt/β-catenin, and TGF-β, drive aberrant cellular proliferation, invasion, and resistance to apoptosis. miRNAs modulate inflammatory responses and cellular processes by targeting key genes involved in adhesion and migration, while lncRNAs regulate gene expression through competing endogenous RNA (ceRNA) interactions, chromatin remodeling, and protein binding, impacting pathways such as p38-MAPK and NF-κB. Specific lncRNAs like MEG3-210 and MALAT1 illustrate how genetic and epigenetic factors influence cellular behavior in endometriosis. Understanding these complex molecular networks enhances insights into pathogenesis and highlights potential therapeutic targets, offering hope for more effective treatment approaches.

Keywords: Endometriosis, Signaling pathways, micRNAs, LncRNAs.