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ENDOMETRIAL ABNORMALITIES IN WOMMEN WITH POLY CYSTIC OVARIAN SYNDROME

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Polycystic Ovarian Syndrome (PCOS) is a complex, multifactorial disorder affecting 8%−13% of reproductive-age women, with variations depending on diagnostic criteria and population studied. PCOS is primarily characterized by reproductive, endocrine, and metabolic dysfunctions. The most widely accepted diagnostic criteria, defines PCOS as the presence of at least two of the following three features: oligoovulation and/or anovulation, biochemical or clinical hyperandrogenism, and polycystic ovarian morphology.

Many studies have been done on different changes of the endometrium to try understand the underlying causes for symptoms and disorders that come with the disease. Thus in this study we look into changes of the endometrium of women of reproductive age to try understand different molecular and pathological changes that happen. This abstract compromises of information found on many articles on scientific databases such as Google Scholar and Pubmed about endometrial abnormalities in women with polycystic ovarian syndrome. As the disease affects women of reproductive age we have gathered journals relating to fertility problems. We have excluded any articles that do not comply with the criteria of study.

In PCOS patients, proteomic profiling revealed increased expression of Legumain, IGFBP-7, HGF receptor, and Cytokeratin-7, and decreased expression of CD20, following weight loss. IGFBP-7 and HGF receptor, both essential for endometrial decidualization and trophoblast interaction, were elevated, suggesting improved receptivity. Cytokeratin-7 upregulation may reflect epithelial remodeling, while CD20 downregulation suggests reduced inflammatory B-cell activity. Endometrial hyperplasia in PCOS stems from chronic anovulation, unopposed estrogen exposure, insulin resistance, and altered IGF signaling. These conditions promote unchecked proliferation via IGF-1/IGF-1R pathways and suppression of apoptosis through the PI3K/AKT/mTOR axis. Hyperandrogenism exacerbates these effects by disrupting decidualization and increasing oxidative stress, further impairing endometrial receptivity. Chronic low-grade inflammation, characterized by altered uNK cell profiles and elevated cytokines (IL-6, TNF-α, CRP), contributes to an inhospitable endometrial environment. Gut microbiota dysbiosis may further propagate systemic inflammation and insulin resistance, forming a self-reinforcing cycle of endometrial dysfunction.

In conclusion, endometrial dysfunction is a major contributors to infertility associated with PCOS. Furthermore, women with PCOS can have a long-term impact on uterine health and result in endometrial hyperplasia, remodeling, and cancer. Recent research suggests that metformin and anti-androgens may be able to reverse harmful endometrial alterations. Unfortunately, there is currently no specific treatment plan that can be advised to patients in order to guarantee a cure.