



**19th International Conference on New Findings
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**The Impact of Oxidative Stress and Chronic Endometrial Inflammation on
Endometrial Receptivity and Recurrent Implantation Failure**

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Successful embryo implantation requires a precise molecular crosstalk between the blastocyst and a receptive endometrium. Recurrent implantation failure (RIF) is frequently driven by a compromised uterine environment, where oxidative stress and chronic endometritis act as primary disruptors. This review elucidates the mechanistic link between the overproduction of Reactive oxygen species (ROS) and the failure of endometrial receptivity. Oxidative stress induces lipid peroxidation and DNA damage within the endometrial epithelium, which directly impairs the expression of critical adhesion molecules, specifically integrins and L-selectin ligands, effectively "closing" the window of implantation. Furthermore, chronic inflammation triggers a persistent release of proinflammatory cytokines (such as TNF- α and IL-6), which shifts the uterine immune profile from a tolerant to a hostile state, hindering the apposition and adhesion of the embryo. The review also examines the downregulation of the homeobox gene HOXA10, a key regulator of decidualization, under oxidative conditions.



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By synthesizing these molecular insights, it is demonstrated that restoring the redox balance and resolving chronic inflammation are prerequisite steps for successful implantation. Moreover, a targeted therapeutic approach to mitigate oxidative stress can rejuvenate the endometrial environment, thereby improving live birth rates in patients suffering from unexplained RIF.

Keywords: Endometrial Receptivity, Oxidative Stress, RIF,
Proinflammatory Cytokines

References

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