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EGCG and the Modulation of Angiogenesis in Polycystic Ovary Syndrome: Mechanisms and Therapeutic Implications

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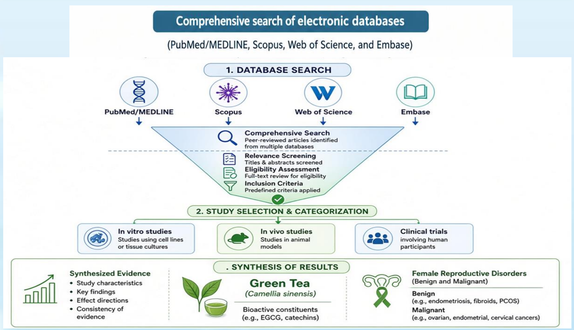
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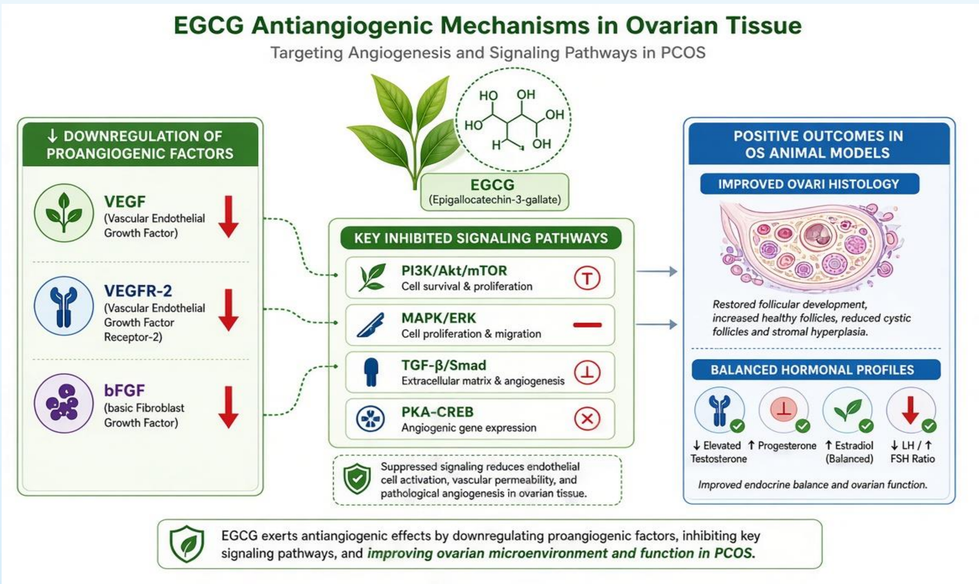
Backgrounds

Polycystic ovary syndrome (PCOS) is a common endocrine-metabolic disorder and a major cause of anovulatory infertility, often associated with hyperandrogenism and abnormal ovarian angiogenesis. Dysregulated angiogenesis, driven by increased vascular endothelial growth factor (VEGF), plays an important role in the pathophysiology of PCOS. Epigallocatechin-3-gallate (EGCG), the major bioactive catechin in green tea, has been shown to possess antiangiogenic, anti-inflammatory, and antioxidant properties. This review evaluates the therapeutic potential and molecular mechanisms of EGCG in modulating abnormal ovarian angiogenesis in PCOS.

Methods

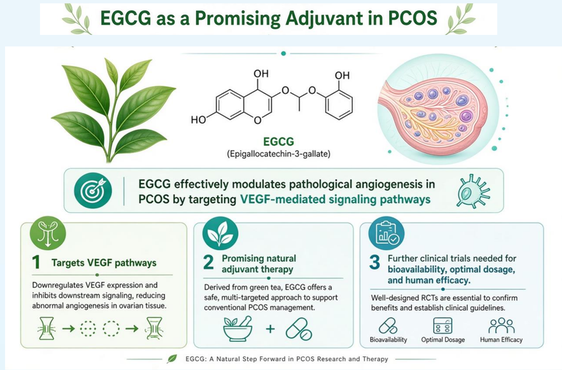


Findings



Evidence suggests that EGCG exerts its antiangiogenic effects by reducing the expression and secretion of proangiogenic mediators, particularly VEGF, VEGFR-2, and bFGF, through specific signaling pathways such as PI3K/Akt/mTOR, MAPK/ERK, TGF-β/Smad, and PKA-CREB. Systematic evaluations have shown improvements in ovarian histology and hormonal profiles in PCOS animals with EGCG.

Conclusion



References

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3. Wang S, Fang L, et al. The therapeutic potential of EGCG and pro-EGCG in mitigating ovarian hyperstimulation syndrome: unraveling the modulatory mechanism through the VEGF pathway. International Journal of Biological Sciences. 2025;21(7):3045.