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Significance of estrogens metabolites in the pathophysiology of PCOS

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The human ovary undergoes to cyclic changes of estradiol secretion during the menstrual cycle, which is associated with extended angiogenesis during follicular development and early Corpus luteum or declining angiogenesis components during Corpus Luteum regression. Ovarian estradiol exerts different pro angiogenic effects in the follicular development during the preovulatory phase of the normal menstrual cycles. Angiogenesis comprise proliferation of endothelial cells that gradually progress in developing follicle accomplishment high levels in antral follicle and subsequently at the time of ovulation. In contrast follicular atresia is associate with inadequate development and/ or regression of the theca vasculature. It is known that estradiol is metabolized by diverse metabolic pathways including hydroxylation, glucuronidation, sulfonation and methylation to form estrogen metabolites (EMs). Interestingly estradiol metabolites acting through different angiogenic pathways could effect negatively or completely follicular development. Based on those findings and the known action of catechol estrogens, 4-hydroxyestrone (4-OHE1) and 2-hydroxyestradiol (2-OHE2), as pro-angiogenic and methoxyestrogens, 2- methoxyestradiol (2-ME2) and 2-methoxyestrone (2-ME1), which are anti-angiogenic we determined the levels of these estradiol metabolites in human follicular fluid of normal fertile women as well in PCOS with follicular arrest. Interestingly the levels of pro-angiogenic estradiol metabolites were significantly reduced in follicular fluid of PCOS women supporting the hypothesis that these estrogens metabolites may play an important role in follicular arrest of PCOS women.

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