9th Yazd International Congress and Student Award on Reproductive Medicine with 4th Congress of Reproductive Genetics

Oral Presentations

O-16

Beneficial effects of minocycline on the ovary of polycystic ovary syndrome mouse model: Molecular docking analysis and evaluation of *TNF-α, TNFR2, TLR-4* gene expression

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Background: Polycystic ovary syndrome (PCOS) is the most common cause of ovulatory infertility. Inflammation may be involved in the pathogenesis and development of PCOS.

Objective: We investigated the anti-inflammatory effect of minocycline on tumor necrosis factor-α (TNF-α), tumor necrosis factor receptor 2 (TNFR2), and toll-like receptor 4 (TLR4) expression levels and the key features of PCOS in a mouse model.

Materials and Methods: Molecular docking was performed by Molecular Operating Environment software. PCOS was induced by estradiol valerate injection (2 mg/kg/day) in 40 mice. After 28 days, the mice were divided into five groups, including control, PCOS, minocycline control, minocycline PCOS model (50 mg/kg), and letrozole PCOS (0.5 mg/kg). The Levels of follicle-stimulating hormone,luteinizing hormone, estradiol (E2), and testosterone were determined by ELISA. H&E staining was used for histological analysis in the ovarian tissues.

Results: Docking scores were -10.35, -10.57, and -12.45 kcal/mol for TNFα, TLR-4, and TNFR2, respectively. The expression levels of TNF-α, TNFR2, and TLR4 were detected by Real-Time PCR. PCOS models exhibited acyclicity, a significant increase in E2 levels ($p < 0.01$), and no difference in follicle-stimulating hormone, luteinizing hormone, and testosterone. The expression levels of TNF-α, TNFR2, and TLR-4 significantly increased in PCOS (2.70, 7.90, and 14.83-fold, respectively). Estradiol valerate treatment significantly increased graafian follicles ($p < 0.001$) and decreased corpus luteum (CL) ($p < 0.01$). Minocycline treatment in PCOS led to a significant decrease in E2 ($p < 0.01$) and graafian follicles ($p < 0.001$) and a significant increase in the CL numbers ($p < 0.05$).

Conclusion: Our findings showed the positive effects of minocycline on E2 level, CL and graafian follicles counts, suggesting that minocycline might inhibit these proteins and improve ovulation in our mouse model of PCOS.

Key words: *PCOS, Minocycline, TNF-α, TNFR2, TLR-4.*

The original full text of this abstract has been published in Journal of Reproductive Immunology 2021; 144: 103289. https://doi.org/ https://doi.org/10.1016/j.jri.2021.103289.

How to cite to this article: *Khajouei A, Hosseini E, Abdizadeh T, Kian M, Ghasemi S. Beneficial effects of minocycline on the ovary of polycystic ovary syndrome mouse model: Molecular docking analysis and evaluation of TNF-α, TNFR2, TLR-4 gene expression. Journal of Reproductive Immunology 2021; 144: 103289.*