Gene expression of estrogen receptors subtypes (ER-α and ER-β) and inflammatory markers (IL-6 and TNF-α) in the uterine tissue from primary dysmenorrhea rat model

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ABSTRACT

Introduction: Primary Dysmenorrhea (PD) is a common gynaecological disorder affecting women worldwide. The pathogenesis of PD is influenced by estrogen metabolism and chronic inflammation, which is modulated by several factors including the estrobolome. Therefore, this study aims to assess the mRNA expression levels of Estrogen Receptors (ER-α, ER-β) and inflammatory markers IL-6 and TNF-α in uterine tissue samples obtained from PD rat model via quantitative real-time PCR (qPCR). **Materials and Methods:** 28 female Sprague-Dawley rats were divided into four groups: control group (Group 1), PD-induced control (Group 2), PD treated with mefenamic acid (Group 3), and PD treated with probiotics (Group 4). The mRNA gene expressions of ER-α, ER-β, IL-6, and TNF-α in uterine samples were analyzed using qPCR, with GADPH and HRPT1 as housekeeping genes. **Results:** The ER-α expression was significantly higher (1.35-fold, p=0.0018) in PD-induced group compared to the control. ER-β expression was significantly lower in the PD-induced group (0.08-fold, p=0.0003). Meanwhile, inflammatory markers IL-6 and TNF-α were notably lower in PD groups treated with mefenamic acid and probiotics, with values of (0.88-fold, p=0.0283) and (0.35-fold, $p < 0.0001$), respectively, compared to PD-induced control group. **Conclusions:** These results indicated disruptions in estrogen metabolism may leads to the pathogenesis of PD. Additionally, probiotics was suggested to have promising potential as therapeutics option, comparable to mefenamic acid, in reducing PD-associated inflammation. Ultimately, these findings provided preliminary data for the development of more effective therapeutic strategies in managing PD symptoms, with further research needed in the future.