

THE EFFECT OF URIDINE ON FERROPTOSIS PATHWAYS IN RATS WITH POLYCYSTIC OVARY SYNDROME INDUCED BY DEHYDROEPIANDROSTERONE

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THESIS ABSTRACT

In this study, the effects of uridine on ovarian folliculogenesis and ferroptosis-related pathways were investigated in a dehydroepiandrosterone (DHEA) induced polycystic ovary syndrome (PCOS) rat model. Uridine was administered at doses of 250 or 500 mg/kg to rats with experimentally induced PCOS. Serum LH, FSH, testosterone, and ferroptosis related markers in ovarian tissue or serum were evaluated using ELISA, while estrous cyclicity and folliculogenesis were assessed by histological methods. The findings demonstrated that the PCOS group exhibited increased serum LH/FSH ratio, testosterone, and Fe^{2+} levels, as well as elevated ovarian SLC7A11, MDA, ACSL4, and ALOX15 levels, whereas GSH, GPX4, and HO-1 levels were decreased. In addition, an increase in atretic and cystic follicle numbers and disrupted estrous cyclicity were observed. Uridine treatment increased GSH, GPX4, and HO-1 levels while reducing MDA, SLC7A11, and Fe^{2+} levels. Histologically, uridine administration decreased the numbers of atretic and cystic follicles and improved primordial follicle survival. In conclusion, uridine may exert beneficial effects on ferroptosis related mechanisms and ovarian morphology in PCOS.

APPLICATION AREAS OF THE THESIS RESULTS

The findings of this study highlight the importance of ferroptosis in the pathogenesis of polycystic ovary syndrome (PCOS) and suggest potential protective effects of uridine against ferroptosis-related damage and impaired folliculogenesis. These results may contribute to the identification of novel therapeutic targets for PCOS and provide a basis for future experimental studies in this field.

ACADEMIC ACTIVITIES

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KEY WORDS

- ✓ Polycystic Ovary Syndrome
- ✓ Dehydroepiandrosterone
- ✓ Ferroptosis
- ✓ Uridine
- ✓ Folliculogenesis

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