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GLUCOCORTICOID PRERECEPTOR METABOLISM IN THE LIVER OF 5a-DIHYDROTESTOSTERONE-TREATED RATS AS ANIMAL MODEL OF POLYCYSTIC OVARY SYNDROME

Sanja Jelača[1], Jelena Nestorov[1], Nataša Veličković[1], Ana Teofilović[1], Ana Đorđević[1], Marina Nikolić[1], Đuro Macut[2], Ivana Božić-Antić[2], Gordana Matić[1], Danijela Vojnović Milutinović[1]

[1]*Department of Biochemistry, Institute for Biological Research "Siniša Stanković", University of* Belgrade, Belgrade, Serbia; [2]*Clinic of Endocrinology, Diabetes and Metabolic Diseases, Clinical Center of Serbia and School of Medicine, University of Belgrade, Belgrade, Serbia.*

Introduction: Polycystic ovary syndrome (PCOS) is a reproductive and metabolic disorder characterized by hyperandrogenism, ovulatory dysfunction, visceral obesity and insulin resistance. PCOS is also associated with enhanced cortisol metabolite excretion, as well as with altered peripheral glucocorticoid metabolism, which is inevitably linked to insulin resistance characteristic for women with PCOS. The main enzymes involved in glucocorticoid prereceptor metabolism are 11β-hydroxysteroid dehydrogenase type 1 (11βHSD1) that regenerates corticosterone from its inactive precursor, and 5a and 5β reductases (5aR and 5βR) that inactivate corticosterone. In this study, female rats treated with nonaromatizable 5a-dihydrotestosterone (DHT) were used as an animal model of PCOS and the aim was to examine whether this treatment affects hepatic glucocorticoid prereceptor metabolism.
Methods: We analyzed the effects of prolonged treatment of prepubertal rats with DHT on body and liver masses, and plasma and liver corticosterone levels. The expression of hepatic 11βHSD1, hexose-6-phosphate dehydrogenase (H6PDH), 5aR, 5βR and glucocorticoid receptor (GR) were analyzed by real-time PCR and Western blot methods.
Results: DHT treatment induced an increase in body and liver masses, an elevation of hepatic 11βHSD1 expression and a reduction of 5aR mRNA level, leading to tissue corticosterone rise and GR nuclear accumulation. In addition, H6PDH and 5βR mRNA levels remained unchanged.
Conclusion: DHT treatment affected hepatic glucocorticoid prereceptor metabolism through enhanced corticosterone availability and its decreased inactivation, which led to enhanced GR activation. Further studies should reveal possible link between enhanced hepatic glucocorticoid signaling and metabolic disturbances observed in PCOS.

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