



P154 Effect of Vitamin D Deficiency on Insulin Induced Vasodilatation and Receptor Expression in Rat Model of Polycystic Ovary Syndrome

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ABSTRACT

In polycystic ovary syndrome (PCOS) hyperandrogenism and metabolic dysfunction increase cardiovascular risk. Vitamin D3 deficiency is a common comorbidity in PCOS. Our aim was to examine the alterations of insulin-induced vasodilation and receptor expression in rat aorta in a PCOS model.

Methods: Female Wistar rats were treated as follows: 1. vitamin D supplemented group (D+T-); 2. vitamin D deficient (D-T-), 3. vitamin D supplemented with transdermal testosterone application (D+T+) and 4. vitamin D deficient with transdermal testosterone (D-T+). Wire myograph was used for testing insulin relaxation of aorta rings in physiological salt solution and under NOS inhibition. Insulin (IR) and vitamin D receptor (VDR) density was examined by immunohistochemistry.

Results: Insulin-induced vasodilatation of the aorta rings were significantly lower in both vitamin deficient compared to the vitamin supplemented groups ($p < 0.05$). NOS inhibition significantly reduce the relaxation. Aorta endothelial IR expression was significantly higher in the vitamin D deficient group, meanwhile in the testosterone-treated groups (D+T+, D-T+) the expression was significantly lower (Area%: D+: 0.830 ± 0.10 ; D+T+: 0.298 ± 0.06 ; D-: 1.364 ± 0.12 ; D-T+: 0.354 ± 0.15 , $p < 0.05$ in D- & D+T+ & D-T+ vs D+. $p < 0.01$ D+T+ & D-T+ vs D-). VDR density was significantly higher in the vitamin D deficient groups in comparison to the supplemented groups (Area% VDR: D+: 41.56 ± 5.58 vs D-: 60.63 ± 5.23) Testosterone treatment have not any effect on VDR expression.

Conclusion: Vitamin-D deficiency causes impaired insulin induced vasodilation. Increased IR density could not compensate altered insulin-induced relaxation.

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