

Estradiol Down-Regulates Gene Expression of Angiotensin-II Receptor Type One in Ovariectomized Rats

عنوان البحث باللغة العربية:التعبير الجيني للنوع الأول من مستقبلات الأنجيوتينسن 2 في الفئران مستأصلة المبايض عند علاجها بالأستراديول مع مغلفات مستقبلات أى من بيتا- 1 أو أنجيوتينسن 2 المشتركون فى البحث:

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Abstract: Both estrogen deficiency and activation of renin-angiotensin- aldosterone system (RAAS) are implicated in the development of cardiovascular diseases in postmenopausal women, therefore this study aimed to clarify the influence of estradiol (E2) on expression of angiotensin receptor type one (AT1R) in target tissues of ovariectomized rats and whether it can add to the antihypertensive effects of angiotensin receptor blockers (ARBs). Fifty adult female rats were equally divided into 5 groups: Control group, ovariectomized (OVX) group not receiving any treatment, OVX group treated with conjugated estrogen (premarin), OVX group treated with valsartan and OVX group treated with premarin and valsartan. Treatments started 4 weeks after ovariectomy and continued for 8 weeks. By the end of experiment serum levels of renin, angiotensin-II, aldosterone, Na⁺ and K⁺ were measured. Hearts, adrenal glands, and kidneys were removed to quantify the gene expression of AT1R. Ovariectomy resulted in a significant increase in serum concentrations of renin, angiotensin-II, aldosterone, Na⁺ and AT1R gene expression, as well as a significant decrease in K⁺ concentration compared to control group. These findings were improved in all groups received either estrogen and/or valsartan. The best results were achieved when both drugs were used together. **In conclusion** concomitant use of estrogen with ARBs may provide a more effective form of RAAS blockade than the monotherapy of either of them.