Inducible nitric oxide synthase in paraventricular nucleus: features of expression during the essential hypertension development

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Introduction

Recent data prove that NO originated from magnocellular neurons of paraventricular nucleus (PVN) is involved in sympathetic outflow modulation. One of the enzymes which are mediate NO production is the inducible nitric oxide synthase (iNOS). It was showed the involvement of iNOS mediated NO in modulation of activity of the brain sympathetic centres with the development of cardiovascular effects.

Aim

Therefore, the **purpose** of our study was the defining of the features of iNOS expression in magnocellular part of PVN in spontaneously hypertensive rats (SHR).

Materials and methods

The study was carried out on 2 groups of mature animals. 1st group was used as a control group and consisted of 10 Wistar male rats with systolic pressure of 125±5 mm Hg. 2nd group consisted of 10 SHR male rats with systolic pressure of 155±5 mm Hg. With aim of iNOS identification we used immunofluorescence assay. The obtained microphotos were processed with ImageJ. We defined the contain, concentration and specific area of the immunoreactive material (IRM) to iNOS.

Results

After the statistical analysis we have found IRM to iNOS in magnocellular part of PVN was diffusely allocated. In both groups we also have found the neurons, in which IRM was located in cytoplasmic granules, wherein the amount of granules in SHR was markedly lower compared with the control group.

It was found the pattern of iNOS expression in SHR was characterized with the significant increase of IRM contain by 120,7% (p_{st}<0,0005), its concentration by 68% (p_{st}<0,0005) and its specific area by 26,3% (p_{st}<0,0005) compared with respective figures of control group.

Conclusion

1. The iNOS constitutively expresses in magnocellular part of PVN in Wistar rats. 2. The obtained data show the nitrergic system of hypothalamus takes an active part in the primary hypertension development which is accompanied by significant increase of iNOS expression.