The adrenergic system plays an important role in the regulation of blood pressure. In the spontaneously hypertensive rat, the most studied model of essential hypertension, many components of the adrenergic system are altered. Changes in expression level of any catecholamine biosynthetic enzymes or any adrenergic receptor subtypes could be one of the causes of hypertension development. In this work, the expression of adrenergic system genes was measured in adrenal gland, renal cortex and renal medulla of the spontaneously hypertensive (SHR), Wistar-Kyoto and Brown Norway rats at the age of thirteen weeks.

In adrenal gland of SHR, all four catecholamine biosynthetic enzymes (tyrosine hydroxylase, DOPA decarboxylase, dopamine β-hydroxylase and phenylethanolamine-N-methyltransferase) and almost all subtypes of adrenergic receptors (with the exception of *Adra1a* and *Adra1d*) were underexpressed. This generally decreased expression in adrenal gland of SHR suggests that at least a part of regulation of adrenergic system gene expression is common. The mechanism of this downregulation in SHR could be a negative feedback through adrenergic receptors stimulated by high plasma noradrenaline concentration.

In the kidney of SHR, there were no differences in the expression of most of adrenergic receptor subtypes with the exception of Adrb1 and Adra2c, which were overexpressed. This difference in expression is rather a consequence or concomitant phenomenon than a cause of hypertension development.