Neuropeptide Y and Sympathetic Nervous System in Blood Pressure Regulation

To the Editor:

We read with great interest the recent article by Dr Michalkiewicz and his colleagues1 dealing with a possible link between neuropeptide Y (NPY) and sympathetic nervous system in the NPY-transgenic rats. The results of their presented study demonstrated that overexpression of endogenous NPY in the transgenic rats was associated with lower blood pressure in baseline and during stress. Dr Michalkiewicz proposed that the antiadrenergic action of NPY within the sympathetic nervous system may protect the cardiovascular system from excessive adrenergic excitations.

Several studies have reported the influences of NPY on sympathetic neurotransmission in both the central and peripheral nervous systems.2–7 In a study we presented earlier, the change in sympathetic neurotransmission in both the central and peripheral nervous systems.2–7 In an in vitro study using rat brain slices, we showed that NPY inhibited the stimulation-evoked NE release in a dose-dependent manner. It was also demonstrated that NPY potentiated the inhibitory effect of the α2-adrenergic receptor agonists on NE release. In contrast, blockade of the α2-adrenergic receptors or pretreatment of pertussis toxin (a potent inhibitor of the Gi-proteins) diminished the inhibitory effects of NPY on NE release. It would be possible that NPY might reduce NE release in the central nervous system partially mediated by the α2-adrenergic receptors and the pertussis toxin–sensitive Gi-proteins. In addition, the inhibitory effect of NPY on NE release was impaired in spontaneously hypertensive rats (SHR).8 It was reported that NPY increased the number of the α2-adrenergic receptor binding sites in medulla oblongata of normotensive Wistar-Kyoto rats, whereas NPY failed to increase the α2-adrenergic receptor binding sites in medulla oblongata of SHR, indicating that the interactions between NPY and α2-adrenergic receptors might be disturbed in hypertension.

In the separate series of the experiments, Dr Michalkiewicz9 showed that the pressor responses to exogenous NE were significantly increased in the NPY-transgenic rats. The finding might be consistent with the hypothesis that genetic upregulation of NPY could enhance the α2-adrenergic receptor sensitivity. Therefore, we would like to know whether the α2-adrenergic receptor function might be altered in the NPY-transgenic rats. In this context, it can be speculated that, because central stimulation of the α2-adrenergic receptors might markedly reduce blood pressure,10 the changes in the α2-adrenergic receptors might partially explain the blood pressure–lowering effect of NPY in this type of transgenic rats. Further studies should be performed to assess more thoroughly the relationships between NPY and sympathetic nervous system and their role in the blood pressure regulation.

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