HYPERTENSION

New non-RAS peptide modulates the vasoregulatory effects of angiotensin II

Researchers have identified a new peptide that can attenuate the vasoconstrictive effects of angiotensin II (Ang II). Joachim Jankowski and colleagues suggest that “vasoconstriction inhibiting factor” (VIF) could be a therapeutic target for renal and cardiovascular diseases.

Although the renin–angiotensin system (RAS) is involved in the regulation of blood pressure and in the pathophysiology of chronic kidney disease and heart failure, plasma concentrations of Ang II are not increased in patients with these diseases. Jankowski and colleagues, therefore, hypothesized that an unknown endogenous cofactor might modulate the actions of Ang II.

Using a chromatographic screen for vasoregulatory peptides, the researchers first identified VIF in bovine adrenal glands and then isolated the peptide from human plasma. They report that VIF acts on the Ang II type 2 receptor and inhibits Ang II-mediated phosphorylation

of p38 MAP kinase. In rats, VIF administration reduced Ang II-induced increases in blood pressure. Moreover, patients with chronic kidney disease or heart failure had significantly higher plasma concentrations of VIF than did healthy controls. The researchers suggest that VIF might counteract the harmful effects of Ang II in these patients.

“Our knowledge of the RAS system is substantially modified by these results, which might have an impact on the use of RAS inhibitors,” says Jankowski. “Further studies are required to analyse the interactions of VIF with Ang II metabolism and with other angiotensin peptides.”

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Original article Salem, S. *et al.* Identification of the “vasoconstriction inhibiting factor” (VIF), a potent endogenous cofactor of angiotensin II acting on the AT2 receptor. *Circulation* doi:10.1161/CIRCULATION.114.013168