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Atrial natriuretic factor (ANF) effects on L-, N-, and P/Q-type voltage-operated calcium channels.

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Abstract

1. We have previously reported that atrial natriuretic factor (ANF) decreases neuronal norepinephrine (NE) release. The mechanism that mediates NE release from presynaptic membrane to synaptic cleft is a strongly calcium-dependent process. The modulator effect of ANF may be related to modifications in calcium influx at the presynaptic nerve ending by interaction with voltage-operated calcium channels (VOCCs). 2. On this basis we investigated the effects of ANF on K+-induced 45Ca2+ uptake and evoked neuronal NE release in the presence of specific L-, N-, and P/Q-type calcium channel blockers in the rat hypothalamus. 3. Results showed that ANF inhibited K+-induced 45Ca2+ uptake in a concentration-dependent fashion. Concentration-response curves to VOCC blockers nifedipine (NFD, L-type channel blocker), omega-conotoxin GVIA (CTX, N-type channel blocker), and omega-agatoxin IVA (AGA, P/Q-type channel blocker) showed that all the blockers decreased NE release. Incubation of ANF plus NFD showed an additive effect as compared to NFD or ANF alone. However, when the hypothalamic tissue was incubated in the presence of ANF plus CTX or AGA there were no differences in neuronal NE release as compared to calcium channel blockers or ANF alone. 4. These results suggest that ANF decreases NE release by an L-type calcium channel independent mechanism by inhibiting N- and/or P/Q-type calcium channels at the neuronal presynaptic level. Thus, ANF modulates neuronal NE release through different mechanisms involving presynaptic calcium channel inhibition.