Mechanisms underlying anti-stress effects of salt loading

Tatsushi Onaka[1] and Kazufumi Honda[2]

Department of Physiology[1], Department of Internal Medicine[2],
Jichi Medical School

Summary

Salt loading has been shown to reduce ACTH release from the pituitary after stressful stimuli. The mechanisms underlying this anti-stress effect are not clear at present. Oxytocin release from the pituitary is also facilitated under stressful condition. Oxytocin release after stressful stimuli is mediated by noradrenergic neurones. Noxious stimuli activate oxytocin neurones in the hypothalamic paraventricular and supraoptic nuclei, and as a result, facilitate oxytocin release into the circulation. This oxytocin response has been shown to be mediated by activation of A1 noradrenergic neurones in the ventrolateral medulla oblongata. On the other hand, conditioned fear stimuli or CCK facilitate oxytocin release via A2 noradrenergic neurones in the dorsomedial medulla oblongata.

We firstly examined whether salt loading also reduces oxytocin responses to stressful stimuli in Wistar male rats. Noxious stimuli facilitated noradrenaline release within the supraoptic nucleus and increased plasma oxytocin concentrations. Salt loading reduced noradrenaline release within the hypothalamus in response to noxious stimuli and decreased plasma oxytocin concentrations after noxious stimuli. On the other hand, acute salt loading did not reduce plasma oxytocin response to CCK administration.

Osmotic stimulation has been shown to facilitate glutamic acid and oxytocin release within the supraoptic nucleus. We then examined role of NMDA receptor and oxytocin receptor activation upon oxytocin release after stressful stimuli. Local infusion of an NMDA receptor antagonist, MK-801, augmented hypothalamic noradrenaline release and potentiated plasma oxytocin response to noxious stimuli. On the other hand, an oxytocin receptor antagonist reduced hypothalamic noradrenaline release and decreased plasma oxytocin response to noxious stimuli.

All theses data suggest that effects of salt loading upon oxytocin activation under stressful conditions are dependent upon stressful stimuli used, and that activation of NMDA receptors and oxytocin receptors is involved in oxytocin release in response to stressful stimuli via presynaptic modulation of noradrenaline release within the hypothalamus.