

Roles of neurohypophyseal hormones in the control of stress responses following salt intake

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Summary

Salt loading reduces neuroendocrine responses to stressful stimuli. The neural mechanisms underlying this reducing effects are unclear. Noxious stimuli facilitate noradrenaline release in the hypothalamus and, as a result, activate oxytocin neurones. We examined effects of salt loading upon plasma oxytocin concentrations and noradrenaline release in the hypothalamus after footshocks. Male rats were allowed to drink 2 % NaCl for 7 days. Salt loading reduced footshock-induced increase in plasma oxytocin concentrations and noradrenaline release in the supraoptic nucleus. On the other hand, salt loading did not significantly change activation of A1 catecholaminergic neurones in the medulla oblongata, as measured by expression of Fos protein. Local application of an oxytocin receptor antagonist reduced noradrenaline release in the supraoptic nucleus following footshocks. An icv administration of an oxytocin receptor antagonist also reduced the increase in plasma oxytocin concentrations following footshocks but not that following conditioned fear stimuli. These data suggest that salt loading presynaptically suppresses noradrenaline release in the hypothalamus and that oxytocin released during salt loading facilitates the noradrenaline release.