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Effect of Salt Intake on Satiety and its Mechanism

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Summary

Salt is an essential mineral for homeostasis and is an important ingredient to induce salt taste. Furthermore, salt may induce satiety and fulfilment, however, the underlying mechanisms remain unproved. Salt intake increases the extracellular fluid osmolarity, thereby promoting the release of the posterior pituitary hormones oxytocin and arginine-vasopressin (AVP). Peripheral oxytocin and AVP reportedly reduce food intake, and oxytocin-induced anorexigenic effect involves vagal afferent nerves. On the other hand, the mechanism of AVP-induced suppressing food intake is unclear. In the present study, we examined whether AVP directly acts on vagal afferent neurons through AVP receptor, and whether this action on vagal afferents suppress food intake.

We examined effects of AVP on cytosolic Ca^{2+} concentration ($[Ca^{2+}]_i$) using ratiometric fura-2 microfluorometry in the neurons isolated from the vagal afferent nodose ganglion of C57BL/6J mice. AVP increased $[Ca^{2+}]_i$ in single nodose ganglion neurons (NGNs). The incidence of $[Ca^{2+}]_i$ responses to AVP showed a concentration-dependency, with a maximal value around 15% at 10^{-8} and 10^{-7} M. AVP failed to increase $[Ca^{2+}]_i$ in NGNs from vasopressin V1a receptor knockout (V1aR KO) mice. Intraperitoneal administration of AVP decreased food intake in wild-type mice, but not V1aR KO mice. AVP-induced anorexigenic effect was blunted by subdiaphragmatic vagotomy. In contrast, when intracerebroventricular injected V1aR antagonist (SR49059) did not affect the AVP-induced anorexigenic effects. In conclusion, AVP directly activates vagal afferents via V1aR, thereby suppressing food intake. This vagal afferent-mediated effect of AVP might be involved in salt-induced satiety.