A search for neurotransmitters involved in salt appetite: enhancement of acetylcholine release in the gustatory cortex of salt-deprived rats in response to concentrated NaCl.

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Summary

When animals are deficient in body sodium, they consume considerable quantities of concentrated salty solutions that are normally avoided at such high concentrations. Compared with lesion and electrophysiological studies on salt appetite, neurochemical information about this phenomenon is quite limited. Since we recently reported that aversive taste stimuli facilitate extracellular acetylcholine (ACh) release in the gustatory cortex (CGA), in the present experiment we measured ACh levels in the CGA in response to normally aversive concentrated (0.5 M) NaCl in freely moving rats using the microdialysis technique. Salt appetite was evoked by two injections of a natriuretic drug, furosemide (7 mg/rat) 24 h previously. At least 4 h before each experiment, a microdialysis probe (2.0 mm in membrane length and 0.5 mm in diameter) was implanted in the CGA via the guide cannula. The probe was perfused at a flow rate of 2 μl/min with artificial cerebrospinal fluid. Four hours after the probe implantation, each animal received a 5 ml intraoral infusion of 0.5 M NaCl for 5 min. Samples were collected at 20-min intervals and dialysates were analyzed by high-performance liquid chromatography with electrochemical detection. The mean basal release of ACh before stimulation was 160 ± 29 fmol/ $10 \mu l$ (mean \pm SE, n=13). ACh levels in response to concentrated NaCl in salt-deprived rats were significantly higher than those in control rats. Behavioral analyses revealed highly positive correlation between ACh levels and overt activity of animals after taste stimulation in salt-deprived rats. Although the magnitude and duration of ACh release were larger in animals that received an aversive taste stimulus in the previous study, it is suggested that ACh in the CGA is involved in the normal behavioral expression of salt appetite.