

MECHANISM FOR SALT APPETITE IN THE NUCLEUS OF THE SOLITARY TRACT OF BEHAVING RATS BY SPECIAL MOVABLE ELECTRODE

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Summary

Dietary sodium deprivation alters gustatory neural responses to the sodium stimuli both on the periphery and in the central nervous system. This change is thought to influence the compensatory behavior that restores sodium balance. Prior investigations used anesthetized preparations, across subjects designs, and in one instance, diets that differed in nutrient, as well as sodium, content. In the present experiment, we used the same awake, behaving animals before and during dietary sodium deprivation, isolated single neurons from the nucleus of the solitary tract (NST) and tested their response to a battery of sapid stimuli. Rats were fitted with a cranioplastic cap and two intraoral cannulae and trained to receive their water while restrained in the recording apparatus. During the first recording session, they were, maintained on a sodium replete diet (sodium deficient diet with 0.4% Na added) and 41 NST taste neurons were tested. Subsequently, the rats were switched to the sodium deficient diet for a minimum of 10 days and then an additional 58 NST cells were tested. Finally, the rats were returned to a sodium replete diet.

Under sodium deprivation, taste responses to the 4 standard stimuli were reduced. The mean response to NaCl decreased to 53 % of its pre-deprivation level; that for sucrose dropped to 41%; citric acid to 68%, and quinine HCl to 84%. The other stimuli exhibited similar decreases --, monosodium L-glutamate (MSG) dropped to 39 %, glycine to 35 %, and Polycose to 61 %. Based on the the best-response categories for the 4 standard stimuli, the response profiles of taste neurons were not changed by the dietary conditions. Compared with a prior study in an anesthetized animals, the change in diet conditions in the present experiment failed to produce a shift in NaCl responsiveness from NaCl-best neurons to sucrose-best cells. In the Na-replete state, 61% of the activity elicited by NaCl occurred in Na-best cells; 33% in sucrose-best neurons. In the depleted state, these figures were 60% and 26%, respectively. At higher concentrations, however, deprivation did alter the relative responsiveness of gustatory neurons to sucrose and NaCl. When the animals were sodium replete, in sucrose-best neurons, 1.0M NaCl elicited only 60% as much activity as that produced by 0.3M sucrose. When depleted, the response to strong salt was 101% that of sucrose. Similarly, for Na-best neurons, the response to 1.0M sucrose was only 38% of that to the 0.1M NaCl standard in the replete condition, but rose to 71% when the rats were sodium deprived.