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# A Study on the Central Nervous System that Regulates Salt Taste Preference

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Summary

Taste is important for enjoying food and maintaining our quality of life. However, excess consumption of appetitive salty or sweet foods could be the cause of lifestyle-related diseases. Although cumulative studies have elucidated peripheral taste sensing mechanisms mediated by taste receptors, it has not been fully understood how taste preference is regulated in the central nervous systems. Here, we focused on the central nervous system that regulates salt taste preference, and we studied the effects of psychological stress on salt taste preference. Chronic social defeat stress (CSDS) model is one of the most commonly used animal models of depression. CSDS is comprised of 1) short-term physical stress induced by the resident-intruder paradigm and 2) long-term psychological stress. In the present study, we used a subchronic and mild social defeat stress (sCSDS) model, which was generated by milder physical stress compared to the standard CSDS paradigm.

We used the 7-week-old C57BL/6J (B6) mice and the more than 5-month-old ICR mice. The B6 mice were habituated for 1 week and divided into sCSDS group and control group. Then, the B6 mice were exposed to physical attacks by the ICR mice for 30 sec to 5 min/day. The attacking duration was set to 5 min on Day 1, and it was reduced to 30 sec/day. From Day 11 to 15, the B6 mice were exposed to physical attacks for 30 sec. Then, the mice were moved into the neighboring compartment of the ICR mice for 24 hr. The sCSDS was performed from Day 1 to 15. On Day 11, social interaction test was performed. From Day 12 to 14, the two-bottle choice test using 300 mM NaCl and water was performed for 48 hr. On Day 14, the B6 mice were intraperitoneally injected furosemide and fed low sodium diet for 21 hr. Then, from Day 15 to 16, the two-bottle choice test using 300 mM NaCl and water were performed for 2 hr and 24 hr.

We demonstrated that sCSDS significantly increased body weight, food intake, and water intake, as shown previously. However, social interaction behavior was not significantly changed in the sCSDS mice. While the control mice severely avoided 300 mM NaCl, the preference for 300 mM NaCl dramatically upregulated in the sCSDS mice compared to the control mice ($p$-value $< 0.001$). After sodium depletion, both sCSDS mice and control mice preferred 300 mM NaCl, suggesting that sCSDS upregulated salt taste preference, but not abolished salt taste sensitivity. The present study showed the possibility that upregulation of salt taste preference could be related to psychological stress-induced cardiovascular diseases. Future investigations of the sCSDS model, which shows a high preference for salt taste, could contribute to understanding the mechanisms of central nervous systems regulating salt taste preference, and the new approach to achieving a reduction of dietary salt intake.