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# Salt-Sensing Mechanism of ENaC-Expressing Taste Bud Cells

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Summary

$Na^+$ is a major cation in the extracellular fluid of multicellular organisms and is essential for maintaining the body fluid volume. Animals have the ability to sense $Na^+$ in their food and drinks to regulate the amount of salt intake. Especially in developed countries full understanding of the salt taste mechanism is demanded for prevention of hypertention. Although epithalial sodium channel (ENaC) has been identified as the key player of $Na^+$-selective amiloride-sensitive salt taste reception from recent analysis of ENaCα knockout mice, the mechanism of salt taste transduction in ENaC-expressing taste bud cells is not known at all. In the present study, I aimed to establish a new technical basis to record responses to taste stimuli of taste cells in intact taste buds embedded in the lingual epithelium and, with this technique, to study the salt-sensing mechanism of ENaC-expressing taste cells. As a new research tool for recording taste cell responses, I generated mice that stably express a genetically-encoded calcium indicator, GCaMP3, selectively in *ENaCα*-expressing taste cells (*ENaCα-GCaMP3* mice) by crossing transgenic mice expressing Cre recombinase under the control of *ENaCα* promotor with Ai38 Cre reporter mice conditionally expressing GCaMP3. Generated *ENaCα-GCaMP3* mice displayed no abnormality in their appearance and development. By immunofluorescent staining, I confirmed that *ENaCα-GCaMP3* mice express GCaMP3 selectively in *ENaCα*-expressing taste cells. Those mice, therefore, will be a useful tool to study peripheral salt taste transduction mechanism in taste buds.