Role of intracellular Ca^{2+} on salt taste transduction mechanism

Yukio Okada, Takenori Miyamoto, Rie Fujiyama and Yoshinori Uchida

Deaprtment of Physiology, Nagasaki University School of Dentistry

Summary

Taste stimuli bind to specific receptor proteins, triggering a intracellular cascade that results in membrane depolarization and intracellular Ca^{2+} increase. The increase of Ca^{2+} level at the presynaptic terminal elicits a secretion of neurotransmitter, communicating taste information to the gustatory nerve. Amiloride-sensitive Na^+ channel and -insensitive cation channel have been proposed to be related to the salt taste transduction. In the present study, we examined the effect of intracellular Ca^{2+} level on the membrane properties of taste cells isolated from bullfrogs using a whole-cell patch clamp technique. Under holding the membrane potential to -50 mV, Ca^{2+}-ionophore, ionomycin (3 μM) induced a inward current accompanied with conductance increase, resulting in membrane depolarization. Elimination of external Na^+ hyperpolarized the resting potential, and decreased magnitude of the ionomycin-induced current. Intracellular application of 50 μM 1,4,5-IP_3 from the patch pipette and acid stimulus (2 mM acetic acid) also elicited similar response in frog taste cells. The Ca^{2+}-activated conductance may be related to salt and acid taste transduction mechanisms. We would like to examine the effect of taste stimuli on intracellular Ca^{2+} level measured using an optical method.