Antagonistic sensory cues generate gustatory plasticity in *Caenorhabditis elegans*

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Salt taste is essential for ion and water homeostasis, however our understanding of the molecular salt perception is relatively limited. The nematode *Caenorhabditis elegans* is a well-suited model for studying molecular mechanisms that govern behaviour. C. elegans shows strong chemo-attraction to salts, which diminishes upon prolonged exposure. This behaviour allows the molecular analysis of salt taste and its plasticity. We used cell-specific rescue and genetic cell inactivation to identify sensory neurons involved and we analysed 66 mutant strains to identify genes that modulate this behaviour. We show that inputs via the ASE taste neurons and three other sensory cell types generate gustatory plasticity. Our results suggest that salt detection in the ASE neurons uses two signalling pathways, one for low salt and one for high salt concentrations. The low salt detection involves cGMP and Ca^{2+} signalling. Furthermore, we identified 47 genes that modulate gustatory plasticity. This behaviour is modulated by G protein, cGMP, and Ca^{2+} signalling in the ASI sensory neurons, G proteins and TRP channels in the nociceptive neurons, and cGMP signalling in neurons exposed to the body fluid. Integration of these signals requires glutamate, dopamine, and serotonin. Our results suggest a model in which antagonistic inputs from multiple sensory neurons are integrated to from a behavioural response.

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Poster session Neuroscience 1 on Wednesday 2 June