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Articles Highlighted

Shared and Unique G Alpha Proteins in Mammalian and Fish Chemosensation

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Whereas the role of orthologous receptor families in smell and taste of both fish and mammals has been appreciated widely, the corresponding G protein alpha subunits are less well investigated. Oka and Korsching now describe the G alpha protein family in several lower vertebrates and identified those subunits potentially involved in fish chemosensation. They demonstrate that alpha-gustducin is missing in all analyzed teleost and amphibian species. However, zebra fish appear to express Gi1b and G14a alpha subunits in their taste buds and Go1, Go2, Gi1b, and Golf2 alpha subunits in the olfactory epithelium. Thus, despite a general similarity, the vertebrate G protein alpha gene family exhibits clear-cut differences based on gene birth and death that may relate to respecification events and acquisition of new functions.

Lewy Bodies, Olfactory Dysfunction, and Aging

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Substantial impairment of olfactory function in old age has repeatedly been described and explained by age-related degenerative neural processes, some of which appear to be associated with Lewy bodies. However, the contribution of Lewy bodies to impaired olfactory performance in subjects without manifest disorders is not well understood. Wilson et al. now investigated the association of Lewy bodies with olfactory impairment using data of a longitudinal clinical–pathologic study and used olfactory data to identify subjects with this neuropathologic hallmark. To this end, older persons who did not suffer from Parkinson's disease completed a standard odor identification test and underwent neuropathological examination after they died during the follow-up period. Lewy bodies were found in ~13% of the dead, who did not differ demographically from unaffected persons but had lower olfactory test scores. Moreover, decreased olfactory function increased with severity of Lewy body disease. The greatest effects were caused by Lewy bodies of the neocortical or diffuse stage. Thus, olfactory impairment appears to aid detection of underlying Lewy body diseases in otherwise asymptomatic individuals.

Sour Transduction Mechanisms in Mammals

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Though many candidate sour taste receptors have been proposed, including acid-sensing ion channels, hyperpolarization-activated channels, polycystic kidney disease-like proteins (PKD1L3 + PKD2L1), and two-pore domain K+ channels (K2Ps), potential contributions of these channels to sour taste sensation have not unambiguously been demonstrated. Recently, it has been shown that responses to acids are mediated by a proton conductance in PKD2L1 expressing sour-sensing taste cells (Chang et al. 2010, Proc. Natl. Acad. Sci. U S A, 107, 22320–22325). Proton entry across the apical membrane leads to depolarization and generation of action potentials in the sour sensors. However, the molecules facilitating the proton entry across the apical membrane remained unknown. DeSimone, Lyall, and their colleagues have addressed this question in the present issue. By using NADPH oxidase knockout mice and a set of pharmacological agents, they demonstrate the involvement of a NADPH oxidase-dependent proton channel and a cAMP-PKA-activated proton channel in the molecules mediating H^+ conductance in sour sensitive cells. In addition, by apical application of H^+ transporters, which is novel for physiological studies of taste, they provide evidence for the idea that acidification of the taste cell cytoplasm is an intermediate step in sour transduction. However, the molecules for proton entry are not necessarily required for sour transduction elicited by weak organic acids which can non-selectively permeate the apical membrane of any taste cells in their undissociated neutral forms. Thus, the question how weak acids selectively stimulate PKD2L1 expressing sour-sensing cells must be solved in future studies.

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