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Articles Highlighted

Basis of variability in smelling cut grass

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Smell blindness or specific anosmias describe the inability of some individuals to detect certain odorants. Besides other causes specific anosmias as well as impaired sensitivity to certain odorants may have a genetic basis. However, in light of the huge number of olfactory receptor (OR) genes with numerous polymorphisms it surprises that only in very few cases smell deficits have been associated with variations in OR genes. In a previous study, a genome-wide association approach linked a region on chromosome 6 that overlapped with a cluster of 25 OR genes with detection threshold for cis-3-hexen-1-ol. This compound smells like cut grass and is an abundantly used aroma compound added to numerous foods and beverages. The authors, McRae *et al*, now extended their recent work by identifying 147 variants of those 25 OR genes in an ethnically mixed study population. Three of them were tightly linked to cis-3-hexen-1-ol thresholds out of which one occurred in the functional *OR2J3*. In heterologous expression assay OR2J3 was activated by the "grassy" odorant. Of the 5 identified haplotypes, one that changes the receptor's amino acid sequence in positions, 113 and 226, abolished responsiveness to cis-3-hexen-1-ol and explains ~26% of the variation in smelling the "grassy" odorant in the study population.

Toward an identity code in the mammalian olfactory system

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Olfactory receptors respond to the structural properties of volatiles in a combinatorial fashion such that one compound can activate several ORs and *vice versa* that one OR is sensitive to several compounds. Each olfactory sensory neuron expresses a single OR and axons of all olfactory sensory neurons expressing the same OR project to the same few glomeruli in the olfactory bulb. This logic suggests that chemical structures are represented by a pattern of active glomeruli in the olfactory bulb, often referred to as chemotopic map. Falasconi *et al* now aimed at identifying specific glomerular regions that are activated by related odorants. They collected and analyzed by novel computational methods numerous odorant-evoked activity maps across the rat olfactory bulb. The data confirm the existence of glomerular response clusters to similar odorants and reveal quite specific sub-clusters for odorants sharing chemical groups. This organization appears to hierarchical, i.e., the more similar odorants are the more pronounced can the responses clusters be subdivided. Thus, these data reinforce the concept of an identity code in the mammalian olfactory system.

Allergic rhinitis affects nasal epithelia

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Allergic rhinitis is the most common atopic disease. Although it is often associated with severe olfactory loss only little is known about its effects on olfactory and nasal respiratory tissue *per se*. To address this issue Carr *et al* use a mouse model of this disease. After infusion of ovalbumin as nasal allergen for sensitization and challenge they observed massive infiltration by eosinophils of the *lamina propria* subjacent to the respiratory epithelium. Moreover, the respiratory epithelium was altered by hyperplasia and hypertrophy. The olfactory mucosa was more variably affected. It showed a 20% decrease in the density of olfactory sensory neurons and globose basal cells whereas the number of supporting cells did not change. Damage of the olfactory epithelium occurred more often in areas proximal to the respiratory epithelium and was more severe after longer exposure to the allergen. It involved subepithelial swellings of the Bowman's glands which crowded the olfactory epithelium and nerve bundles. The septal organ appeared most sensitive and was replaced in all examined sections by hypertrophied respiratory epithelium with remaining Bowman's glands indicating its original location. The authors conclude that allergic rhinitis exerts complex effects on olfactory function involving interaction of respiratory and olfactory epithelium.

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