Cell Types and Lineages in Taste Buds

Thomas E. Finger
Rocky Mountain Taste and Smell Center, Department of Cell and Developmental Biology, University of Colorado Health Sciences Center, Denver, CO 80262 USA

Correspondence should be sent to: Thomas E. Finger, e-mail: tom.finger@uchsc.edu

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Taste buds are the sensory endorgans for gustation. In mammals, taste buds comprise a collection of ~50–100 elongate epithelial cells and a small number of proliferative basal cells. Ultrastructural studies reveal three distinct anatomical types of elongate taste cells within each taste bud: Type I, Type II and Type III, first defined by Murray based on his studies of rabbit foliate taste buds (Murray, 1973). Figure 1 summarizes the key features of each cell type including differences in apical structure, cytoplasmic organelles and nuclear configuration as well as the overall shape of the taste cell.

Type I cells, sometimes called ‘dark cells’ (e.g. Delay et al., 1986; Nelson and Finger, 1993; Pumplin et al., 1997) extend lamellate processes around other types of taste cells and express GLAST, a glial glutamate transporter (Lawton et al., 2000). These features suggest a glial function for Type I cells, e.g. transmitter clearance and functional isolation of other taste cell types.

Type II taste cells have a characteristic large, round nucleus and express all of the elements of the taste transduction cascade for sweet, umami and bitter, including T1R or T2R families of taste receptors (for bitter, sweet and umami) (Hoon et al., 1999; Miyoshi et al., 2001), the downstream transduction components, PLCβ2 and IP3R3 (Clapp et al., 2001; Miyoshi et al., 2001), and gustducin (Boughter et al., 1997; Yang et al., 2000b). These taste cells thus are the transducing cells for these taste qualities.

Type III cells are characterized by morphologically identifiable synaptic contacts with the gustatory nerve fibers and expression of the synaptic membrane protein (Yang et al., 2000a) SNAP25 as well as the neural cell adhesion molecule (NCAM) (Nelson and Finger, 1993). The presence of a prominent synaptic contact implicates these cells in transmission of information to the nervous system.

A continuing question in the field is how the different types of elongate taste cells come to be replaced from the proliferative basal cell population. Two main hypotheses have been put forward: (i) that the different cell types arise from a common progenitor which generates a single type of cell which then morphs from one cell type to another as it matures; and (ii) that a basal cell gives rise to an immature taste cell which then differentiates into only one of the different morphological types of taste cell. The most recent postulation of the single lineage hypothesis suggested that Type I cells change into Type III cells which then mature into Type II cells (Delay et al., 1986). However, our recent studies entailing chimeric analysis in mice (Stone et al., 2002) demonstrate that multiple lineages must exist within a taste bud, i.e. that the three cell types are not merely different stages of development of a single taste cell type.

Recent data from Miura and co-workers (Kusakabe et al., 2002; Miura et al., 2003; see also Miura, these proceedings) suggest that Type II and Type III cells may originate from a common cell, one that expresses NCAM and Mash1. We re-evaluated our chimeric analysis data to test the possibility that Type I cells may originate from one basal cell population while Type II and III cells derive from a separate proliferative population. In order to perform this re-analysis, we made the assumption that Type I cells represent roughly 40% of the population of cells in any given taste bud. Accordingly we reduced by 40% the total number of cells for each counted taste bud in the previous study. Recognizing that this is a crude approximation at best, we still were able to test the question of whether serotonin-expressing taste cells (a subset of Type III cells) tend to carry the chimeric marker at a higher than a chance rate, i.e. whether Type III cells still represent a distinct lineage after mathematically eliminating the Type I cells. None of the taste buds examined in the previous study show a significant correlation of chimeric marker and serotoninergic phenotype after elimination of the presumed Type I cells (40% of the total population). Hence we have no evidence for a distinct serotonin lineage once we assume that Type I cells arise from a different proliferative population. It is important to note that this re-analysis does not prove that Type II and Type III cells arise from a common progenitor, rather the analysis says that we have insufficient data to disprove the hypothesis of common lineage. Thus the question of whether Type II and Type III cells are distinct lineages or merely different endpoints from a common intermediate cell type remains open to further investigation.

In summary, taking together the morphological, molecular and lineage studies, two likely hypotheses emerge as to lineage within taste buds. One possibility is that the three different cell types: Type I, Type II and Type III each arise from a unique proliferative population, whether it be a progenitor cell population or merely an intermediate transit amplifying population. Alternatively, there may exist two proliferative populations, one that generates only Type I cells.
and the other which generates an immature cell which can differentiate into either a Type II cell or a Type III cell. We consider it highly unlikely that a mature Type III cell morphs into a phenotypically mature Type II cell. Several features mitigate against this scheme. First, in transgenic mice wherein β-gal is driven from the BDNF locus (Yee et al., 2003), phenotypically mature Type III cells express the β-gal protein which is stable for several days even in the absence of protein synthesis. Based on BrDU birth-dating studies, we know that both Type II and Type III cells reach phenotypic maturity at 3–4 days post-mitosis (see Boughter et al., 1997; Yee et al., 2003). If Type III cells regularly transform into Type II cells over a 24 h period, then we would expect to find numerous Type II cells retaining high levels of β-gal protein. Only rare Type II cells show coincident β-gal label. Further, transformation of a Type III cell into a Type II cell would necessitate major re-organization of the apical cytoskeleton and loss of synaptic structures, all in the same 24 h period. While these considerations do not totally exclude the possibility of Type III cells transforming into Type II cells, we believe that other hypotheses are more parsimonious.

References