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Authors
Hollenbeck, Piper LW
Beites, Crestina
Kim, Joon
et al.

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Follistatin Directs Patterning and Development of Sox2-Expressing Taste Bud Progenitors

Piper L.W. Hollenbeck[1,2], Crestina Beites[1,2], Joon Kim[1,3], Robin Lovell-Badge[4] and Anne L. Calof[1,2]

[1]Department of Anatomy & Neurobiology, University of California, Irvine, Irvine, USA, [2]Center for Complex Biological Systems, University of California, Irvine, Irvine, USA, [3]Department of Neurosciences, University of California, San Diego, La Jolla, USA and [4]Division of Stem Cell Biology and Developmental Genetics, Medical Research Council, National Institute of Medical Research, The Ridgeway, Mill Hill, London, United Kingdom

Signaling from subjacent mesenchymal tissues is known to direct the morphogenesis of many epithelium-derived organs, including hair follicles, teeth, and the ductal elements of mammary glands. Although mesenchyme-derived molecular signals that direct taste bud morphogenesis have been postulated to exist, none have yet been described. Using mouse genetics and molecular analysis of gene expression, we identified the secreted TGF-β antagonist, follistatin (Fst), as such a factor. Follistatin is expressed diffusely throughout the tongue in early development and is restricted to the mesenchyme around embryonic stage 14.5, which coincides with taste papilla induction and patterning. Tongues from mice null for *Fst* (*Fst*-/-) have morphological defects including changes in papilla spacing, dysplasia of the epithelial-mesenchymal border, and loss of barrier formation in the intermolar eminence (IE). In the anterior tongue, an absence of *Fst* results in significantly decreased *Shh* expression in fungiform papillae, whereas expression of *Sox2*, while decreased in the apex of the papillae, is expanded basally along the epithelial-mesenchymal border. Interestingly in the IE, a region normally devoid of gustatory character, loss of *Fst* results in the expansion of molecules important for patterning gustatory papillae (Sox2, β-catenin and Shh). Additionally we observed de novo localization of gustducin, and innervation of the IE in regions where *Sox2* is expanded, suggesting an expansion of functional taste buds in a non-gustatory region. Altogether, these findings demonstrate a critical role for Fst in directing morphogenesis and patterning of taste papillae, and suggest that Fst acts upstream of multiple signaling pathways involved in taste bud development.

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