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Type I receptor ALK3-mediated BMP signaling in the tongue mesenchyme is required to activate Wnt-ß catenin for the formation of taste papillae.

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The development of taste papillae requires mesenchymal-epithelial interactions via multiple molecular pathways, including bone morphogenetic protein (BMP) signaling. In the BMP signaling cascade, type I receptors (ALK2, ALK3, ALK6) are the main determinants of downstream signaling specificity. Our studies have demonstrated that ALK2 mediatedBMP signaling in tongue mesenchyme plays an important role in regulating tongue shape and size. Here we report that ALK3mediated BMP signaling in tongue mesenchyme exerts important roles in the development of taste papillae. We used transgenic mouse models to constitutively activate (*ca*) or conditionally knock out (*cKO*) the *Alk3* receptor in a mesenchyme-specific manner using *Wnt1-Cre.* At E12.5, when Shh^{+} taste papilla placodes normally emerge, taste papilla placodes were absent in the *Wnt1-Cre/Alk3 cKO* tongue. In contrast to *Wnt1-Cre/Alk3 cKO*, *Wnt1-Cre/caAlk3* mutants did not possess obvious changes in papilla pattern. Our data indicate that high levels of ALK3-BMP signaling is needed for the formation of taste papillae. Tongue organ cultures with Wnt-β catenin signaling activator LiCl showed development of taste papillae in the *Wnt1-Cre/Alk3 cKO* tongue. Our data suggests that ALK3-BMP signaling in tongue mesenchyme activates Wnt-β catenin signaling for taste papilla formation. Further studies are ongoing to explore the mechanism by which ALK3-BMP signaling plays its role in the formation of taste papillae.