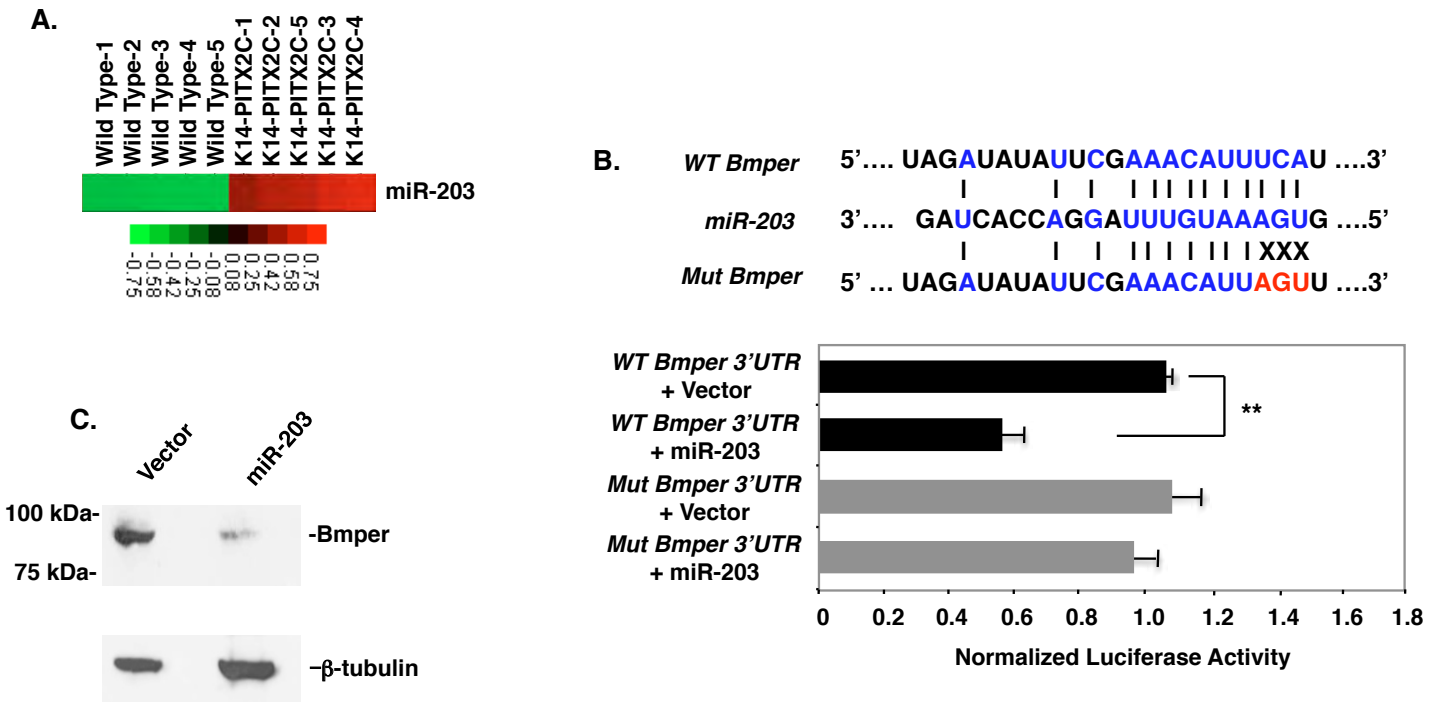

Fig. S1. Pitx2 endogenous activation of miR-203 and inhibition of Bmper by miR-203. (**A**) miR-203 expression in multiple samples from control (wild-type) and *Krt14-PITX2* overexpression mouse incisors. miR-203 is upregulated in the PITX2 overexpression mice. (**B**) The target sequence of miR-203 in the 3′-UTR of *Bmper* and the mutated *Bmper* 3′-UTR is shown. Normalized luciferase activity of the 3′-UTR *Bmper*-luciferase reporter (WT *Bmper 3′UTR*) with empty plasmid (Vector) or CMV-miR-203 (miR-203) shows loss of luciferase activity with expression of miR-203. There is no loss of luciferase activity when the miR-203 seed sequence is mutated (Mut *Bmper 3′UTR*). Error bars indicate ± s.e.; five independent experiments (n=5); $^{**}P$<0.01. (**C**) Western blot analysis shows a decrease in Bmper levels when miR-203 is overexpressed in LS-8 cells.

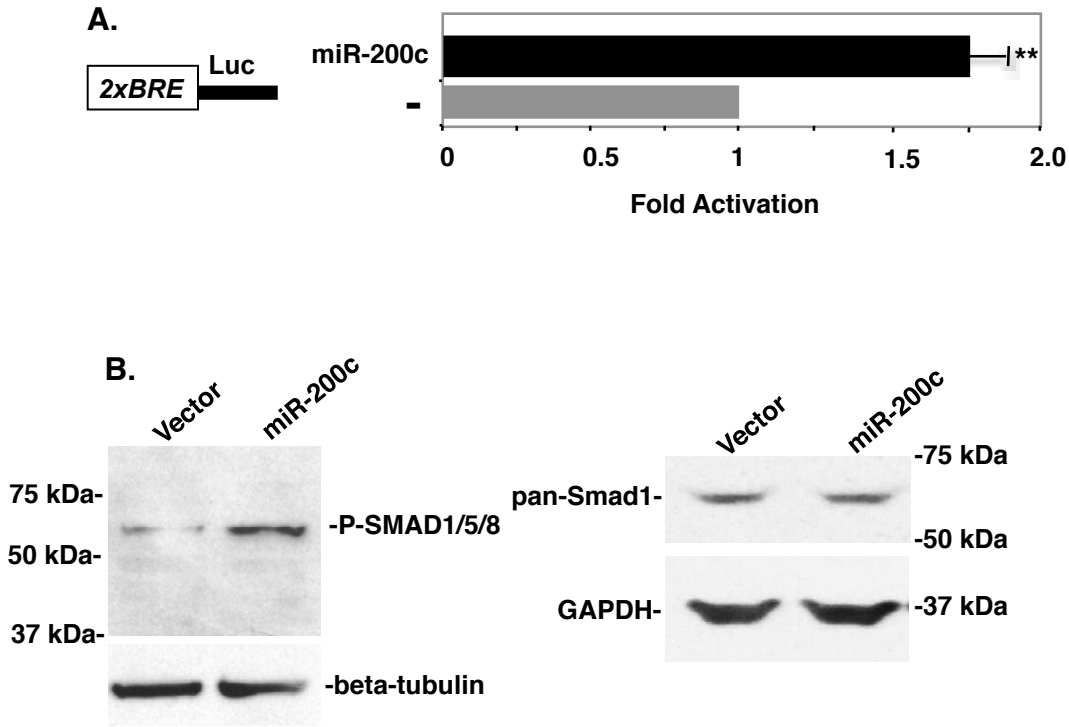

Fig. S2. miR-200c activates Bmp signaling and p-Smad1/5/8. (**A**) Luciferase activity of the Bmp reporter (2×BRE-Luc) co-transfected with empty vector (Vector) or CMV-miR-200c (miR-200c) in LS-8 cells. Error bars indicate ± s.e.; five independent experiments (n=5); $^{**}P$<0.005. (**B**) Western blot analysis showing levels of phospho (p)-Smad1/5/8 upon 48-hour expression of miR-200c and pan-Smad1 in LS-8 cells.

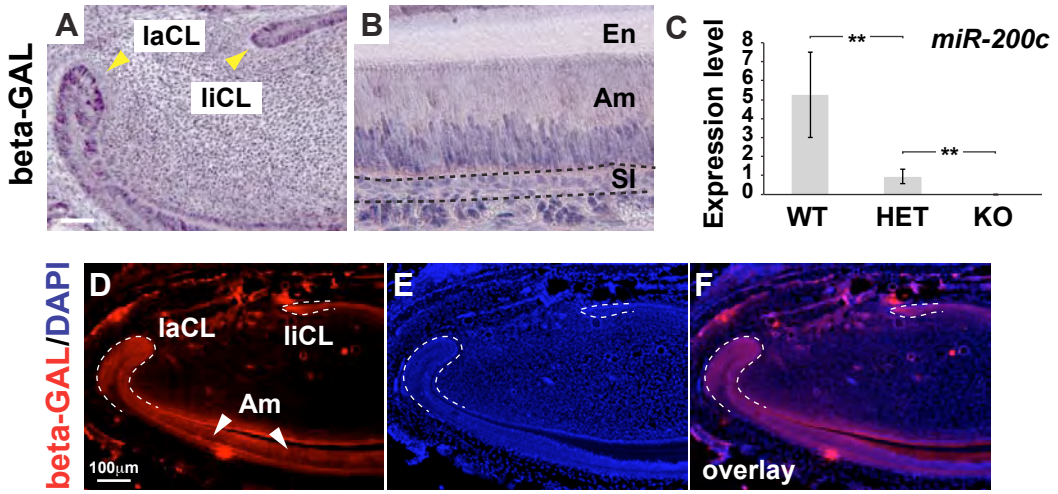

Fig. S3. Expression of miR-200c and deletion of *miR-200c/141* in mutant mice. (**A**,**B**) Localization of miR-200c by beta-galactosidase (beta-Gal) staining in the labial and lingual CL (laCL and liCL) in the ameloblasts (Am) and stratum intermedium (SI). (**C**) Confirmation of the loss of miR-200c expression in knockout mice by PCR. (**D-F**) Lower magnification of beta-Gal staining confirms specific expression in the LaCL, LiCL and ameloblasts. Error bars indicate ±s.e., three independent experiments (n=5); ***P*<0.01.

Table S1. RT-PCR primers

Gene	Forward	Reverse
Nog	CGGCCAGCACTATCTACACA	GCGTCTCGTTCAGATCCTTC
Bmper	ATCAAAGTGCACTGGGAACC	AGGACAGAGGACTGGCTTGA
Htra1	CATCTCCTTCGCAATTCCAT	GACGGTCCTTCAGCTCTTTG
Chrdl2	CAGGTGTACACGTTGGCATC	TCTGGAGTCTGGGCTAGGAA
Amel	TTTTGCTATGCCCCTACCAC	GTGATGAGGCTGAAGGGTGT