

Supporting Information

Maurya et al. 10.1073/pnas.1708321114

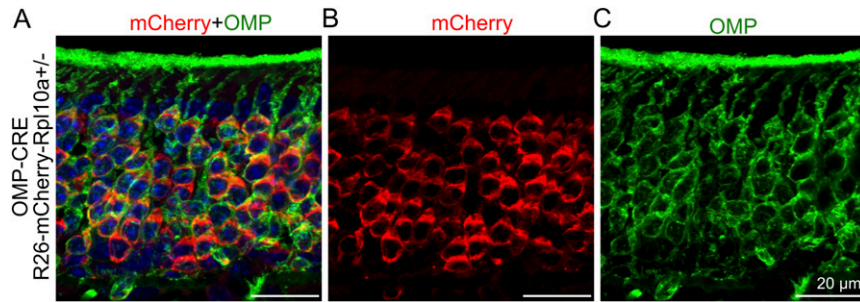


Fig. S1. CRE-mediated recombination in OSNs of transgenic OMP-CRE mice. OMP-CRE mice were crossed to a reporter mouse line (R26-mCherry-Rpl10a) that carry a mCherry reporter gene under the transcriptional control of a floxed (ROSA)26Sor locus (38). (A–C) A double mCherry and OMP immunohistochemical analysis showing that mCherry expression, activated by CRE-mediated recombination, is specific to OMP-positive OSNs.

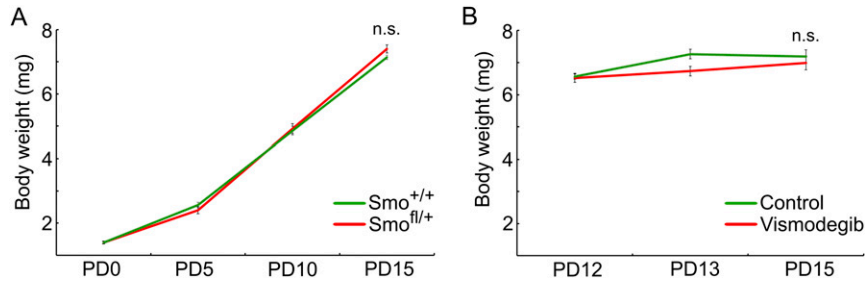


Fig. S2. Genetic and pharmacological inhibition of Smo does not affect body weight. (A) Body weight change of PD0–PD15 Smo^{+/+} and Smo^{fl/+} mice. Values represent mean \pm SEM, $n = 7$, n.s., not significant. (B) Body weight of vismodegib-treated and vehicle (DMSO)-treated control mice during the treatment period (PD12–PD15). Values represent mean \pm SEM, $n = 12$, n.s., not significant.

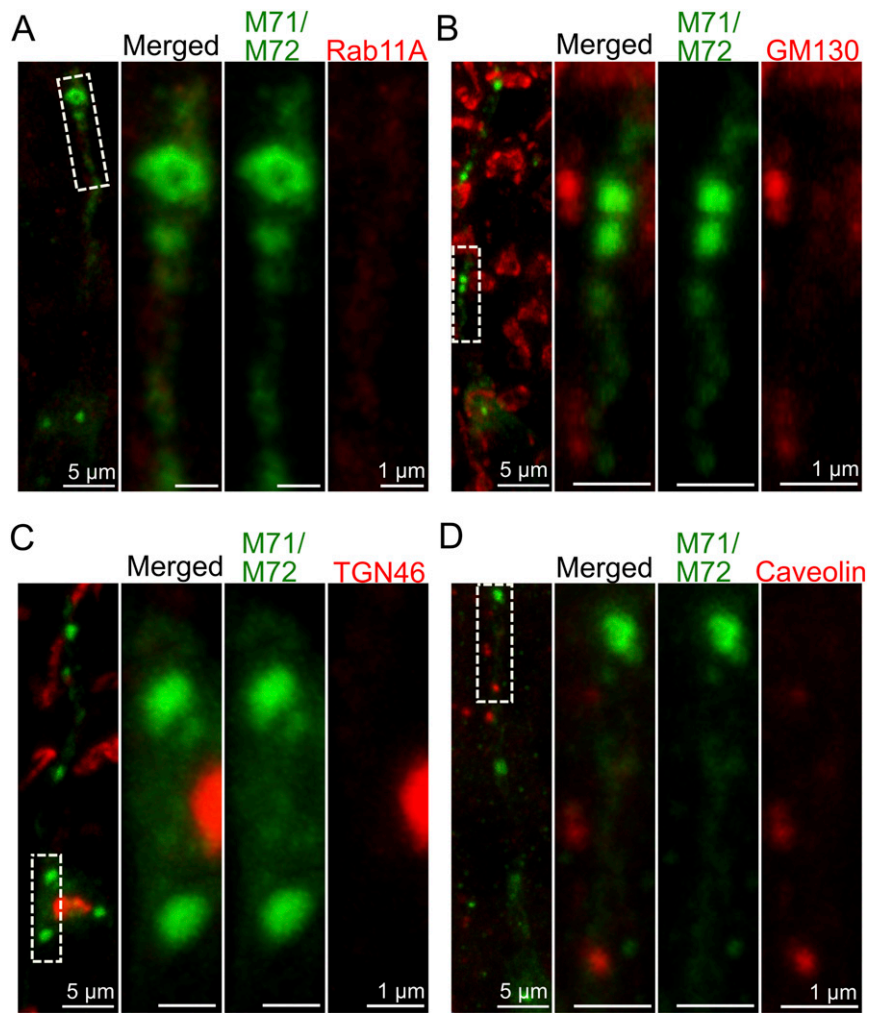


Fig. S4. OR-positive puncta do not colocalize with Rab11A, Golgi markers, and Caveolin. Double immunohistochemical analyses of OSNs, in vismodegib-treated mice, with anti-M71/M72 antibody and antibodies for (A) Rab11A, (B) GM130 (*cis*-Golgi marker), (C) TGN46 (*trans*-Golgi marker), and (D) Caveolin. In each panel, the three images to the *Right* are a close-up of the area that is boxed in the image to the *Left*.

