

Supplementary Figure 1

Otk binds Wnt4 but not Axin. (A) Wild-type denticle belt. (B) Loss of denticles observed when Otk RNAi was expressed. (C) HA tagged Wnt5 and Wnt4 were expressed in embryos with the UAS/GAL4 system, precipitated with the HA antibody along with a no-HA control embryo extract, and probed for endogenous Otk protein. Otk precipitated with Wnt4 robustly but not with Wnt5 or the no-HA control. Otk was present in all extracts as the input control shows. (D) HA tagged Otk and FLAG tagged APC2 were expressed in embryos, precipitated with the respective tag antibodies, and probed for endogenous Axin protein.

Supplementary Figure 2

Wnt3a and Wnt8 co-precipitate a deletion construct of PTK7 consisting of only its extracellular domain (exPTK7). The co-precipitated exPTK7 is shown in the upper panels and the *Xenopus* lysates used for immunoprecipitation in the lower panels. Injected constructs are indicated at the top, antibodies used for Western blotting on the right. (A) Myc-tagged Wnt3a co-precipitates HA-tagged exPTK7. (B) Myc-tagged Wnt8 co-precipitates HA-tagged exPTK7.

Supplementary Figure 3

PTK7 expression in *Xenopus* development. (A) Temporal PTK7 expression pattern analyzed by RT-PCR. (B) In situ hybridization detects PTK7 expression in the animal hemisphere (a,b), the neural tube (nt) and the cranial neural crest cells (nc) (c). Upper panel shows PTK7 expression, lower panel the sense control. The animal (A) and vegetal (V) hemisphere are shown. (C) PTK7 expression does not show a dorsal-ventral bias, which would be suggestive of a function in dorsal-ventral patterning. PTK7

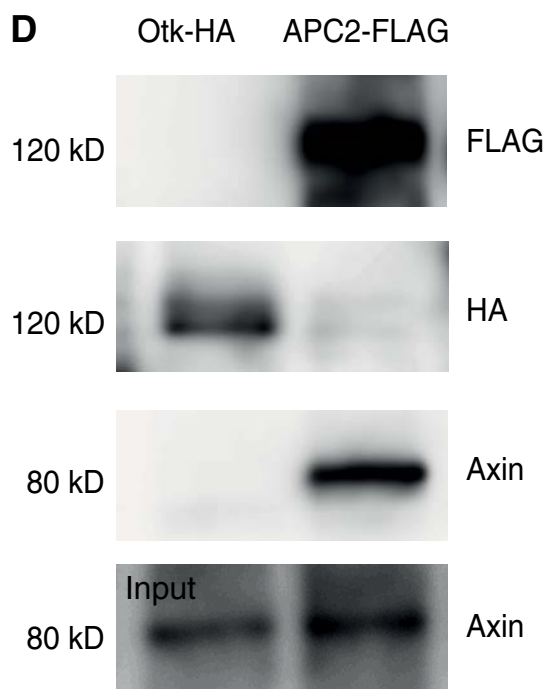
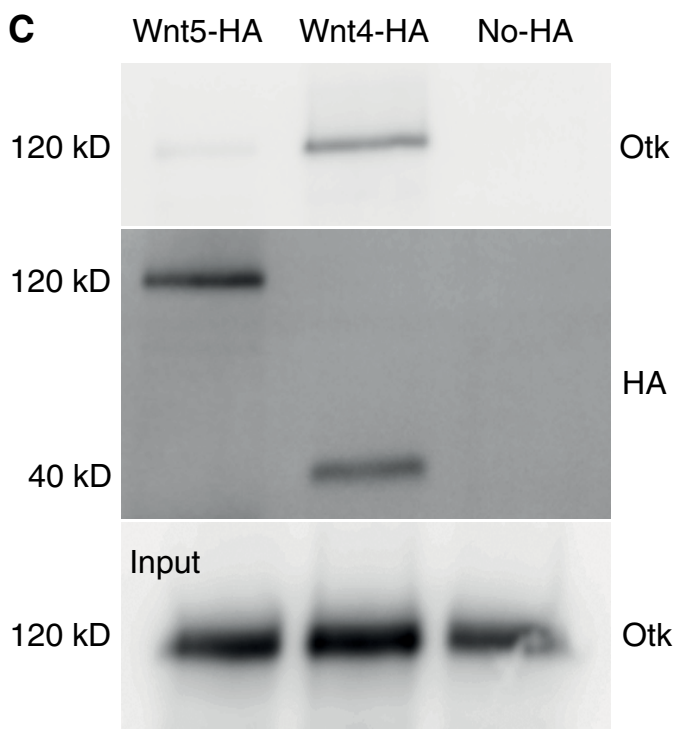
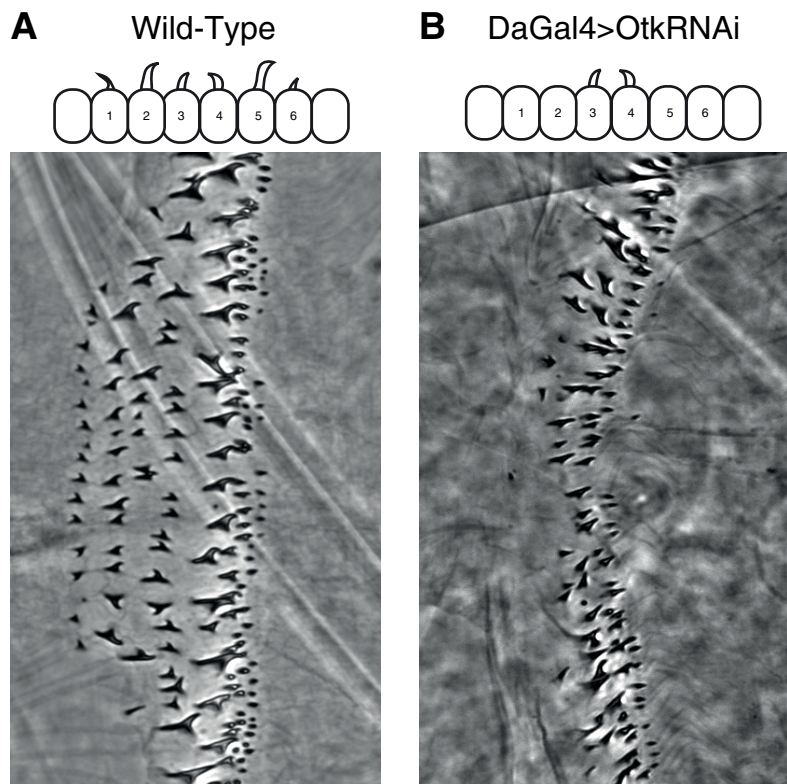
expression is compared to the expression of chordin (d-f). (a,d) dorsal view, (b,e) lateral view, (c,f) transverse section, white errors indicate the dorsal side.

Supplementary Figure 4

Dominant negative Wnt8 (dnXwnt8) partially rescues the PTK7 loss of function

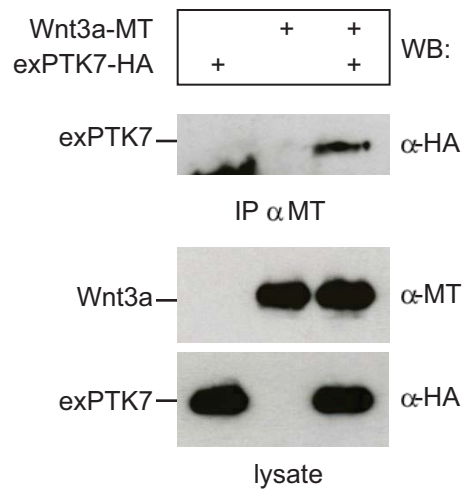
neural tube closure defect. (A) Embryos were injected at the one cell-stage with 20 ng PTK7 MO or its mismatch control (mmMO) in combination with 200 pg *dnXwnt8* RNA as indicated. (B) Graph showing three independent experiments, s.e.m. are shown. As injection of dnXwnt8 leads to a delayed neural tube closure phenotype where the anterior remains wider, these phenotypes were categorized as "slow", while the severe neural tube closure defects caused by the PTK7 MO are referred to as "nt defect". Stars indicate experimental conditions that are significantly different ($p < 0.05$ in a Student t-test).

Supplementary Fig. 1

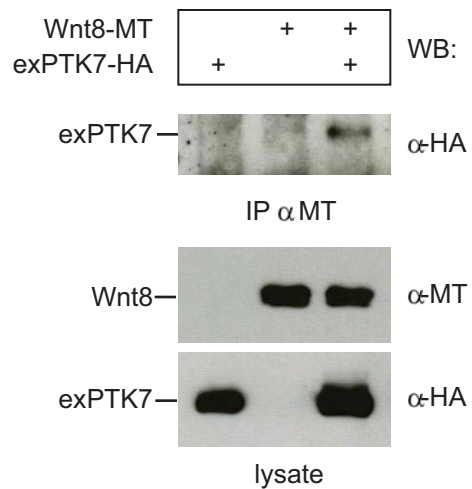


Supplementary Fig. 2

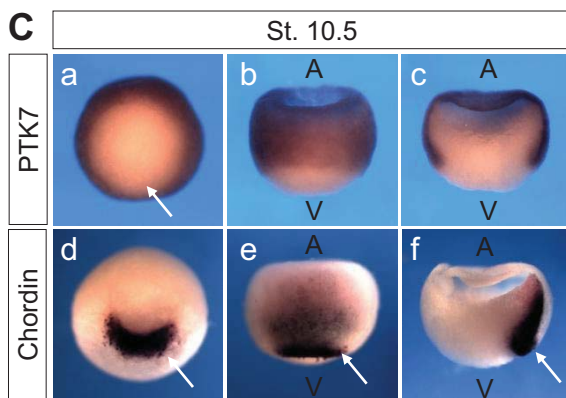
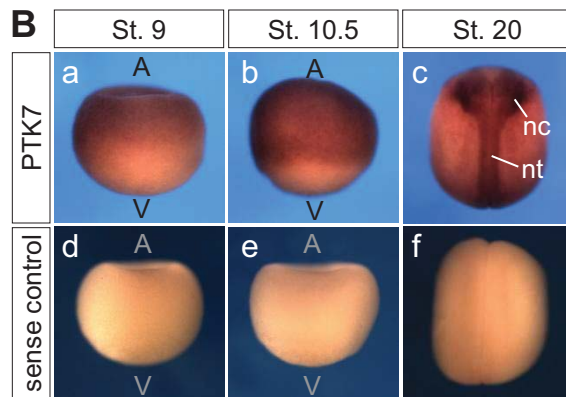
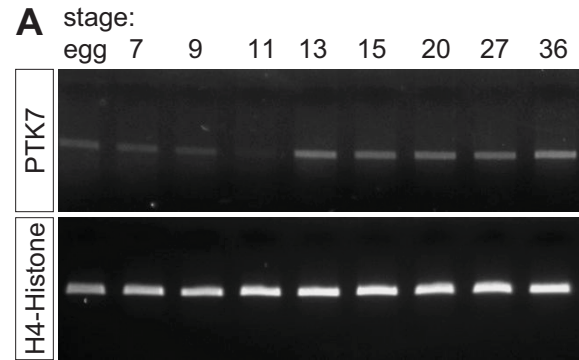
A



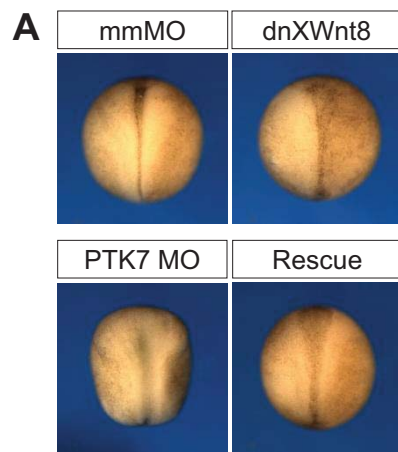
B



Supplementary Fig. 3



Supplementary Fig. 4



B % neural tube closure defects

