

Fig. S1. Variability in β -catenin recombination in *cβcat* mutants. Immunostaining for β -catenin in (A) control, (B) Class A or (C) Class C embryos shows complete, absent or partial staining (arrows), respectively, in the canal pouch epithelium at E12.0 (tamoxifen administration at E10.0).

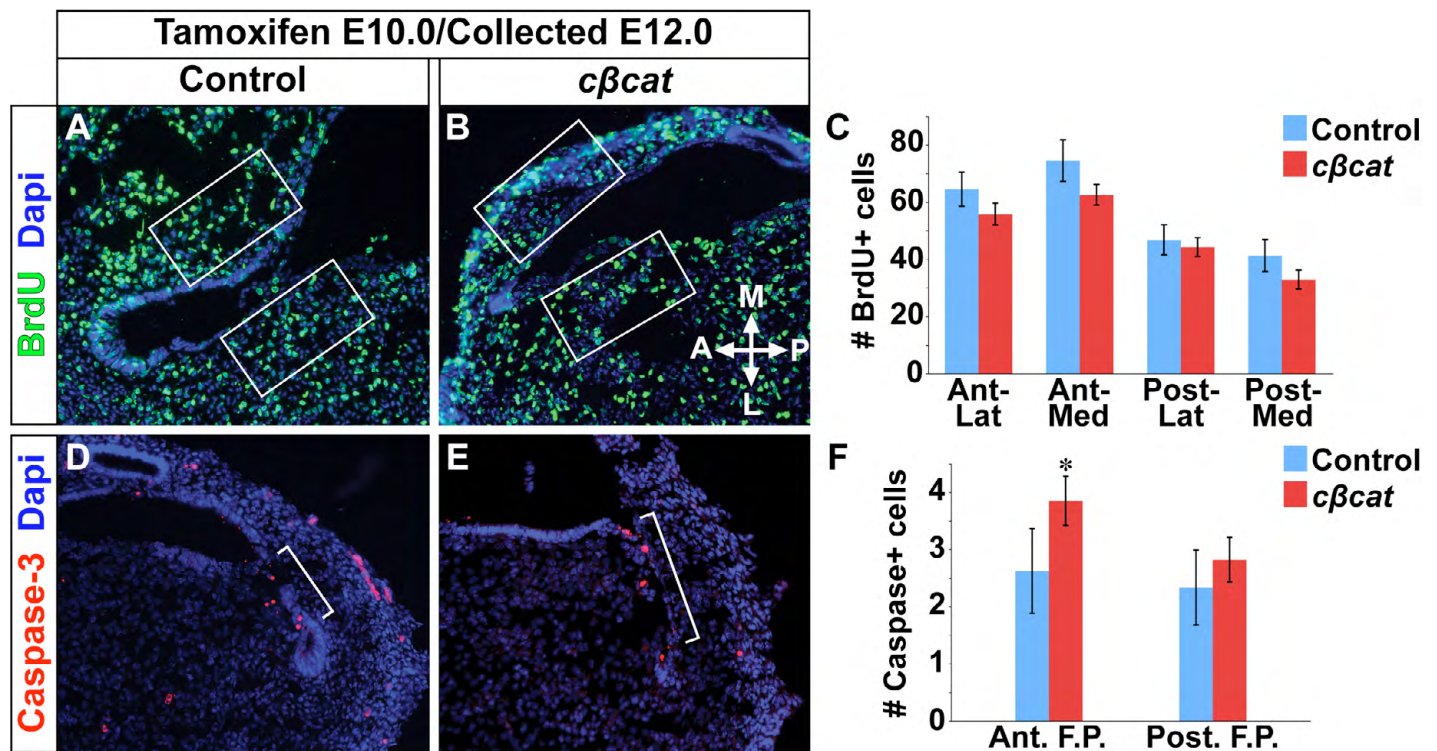


Fig. S2. Resorption defects in Class A *cβcat* mutants are not attributable to impairments in cellular proliferation or apoptosis. (A,B) BrdU-positive cells were counted in four different regions of the vertical canal pouch in control and *cβcat* mutants within a fixed-sized box adjacent to the lateral and medial sides of the anterior (shown) and posterior fusion plate. (C) The average number of BrdU-positive cells per section was not significantly different between control and *cβcat* embryos when recombination was induced at E10.0 and tissue was collected at E12.0. (D,E) Activated caspase 3 staining was evaluated at the same time point in the anterior and posterior (shown) fusion plate (bracket) of control and *cβcat* embryos. (F) The average number of cells per section undergoing apoptosis was slightly elevated in the anterior, but not posterior, fusion plate region of *cβcat* mutants compared with controls (* $P=0.095$). Error bars represent the s.e.m. A or Ant., anterior; L or Lat., lateral; M or Med., medial; P or Post., posterior; F.P., fusion plate.

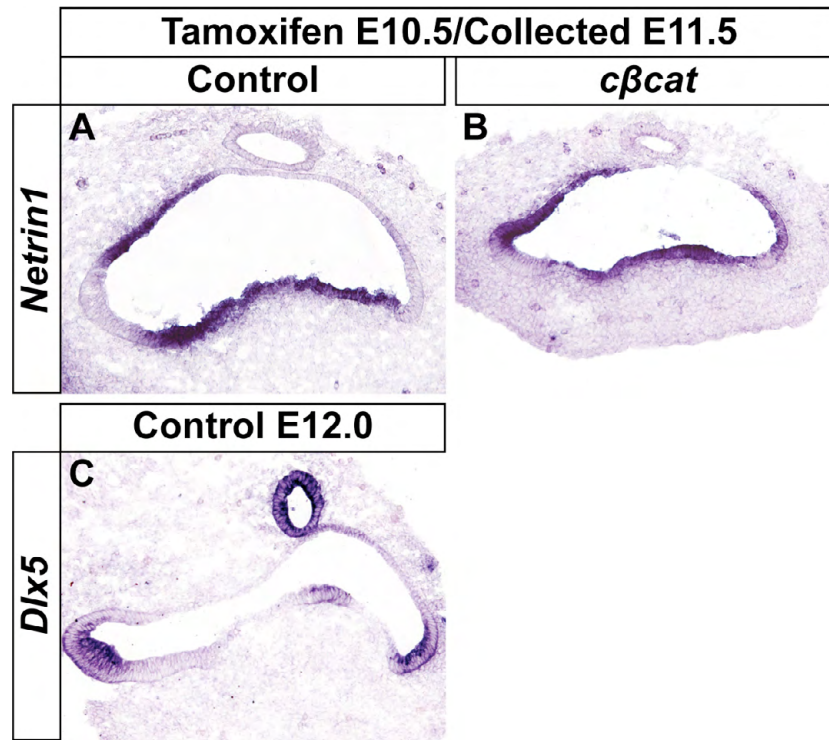


Fig. S3. The onset of *Ntn1* expression occurs correctly in *cβcat* mutants. (A,B) The initiation of *Ntn1* expression is not delayed in *cβcat* compared with control embryos at E11.5 (tamoxifen administered at E10.5). (C) *Dlx5* expression in the vertical canal pouch rims of control embryos at E12.0.