Supporting Information

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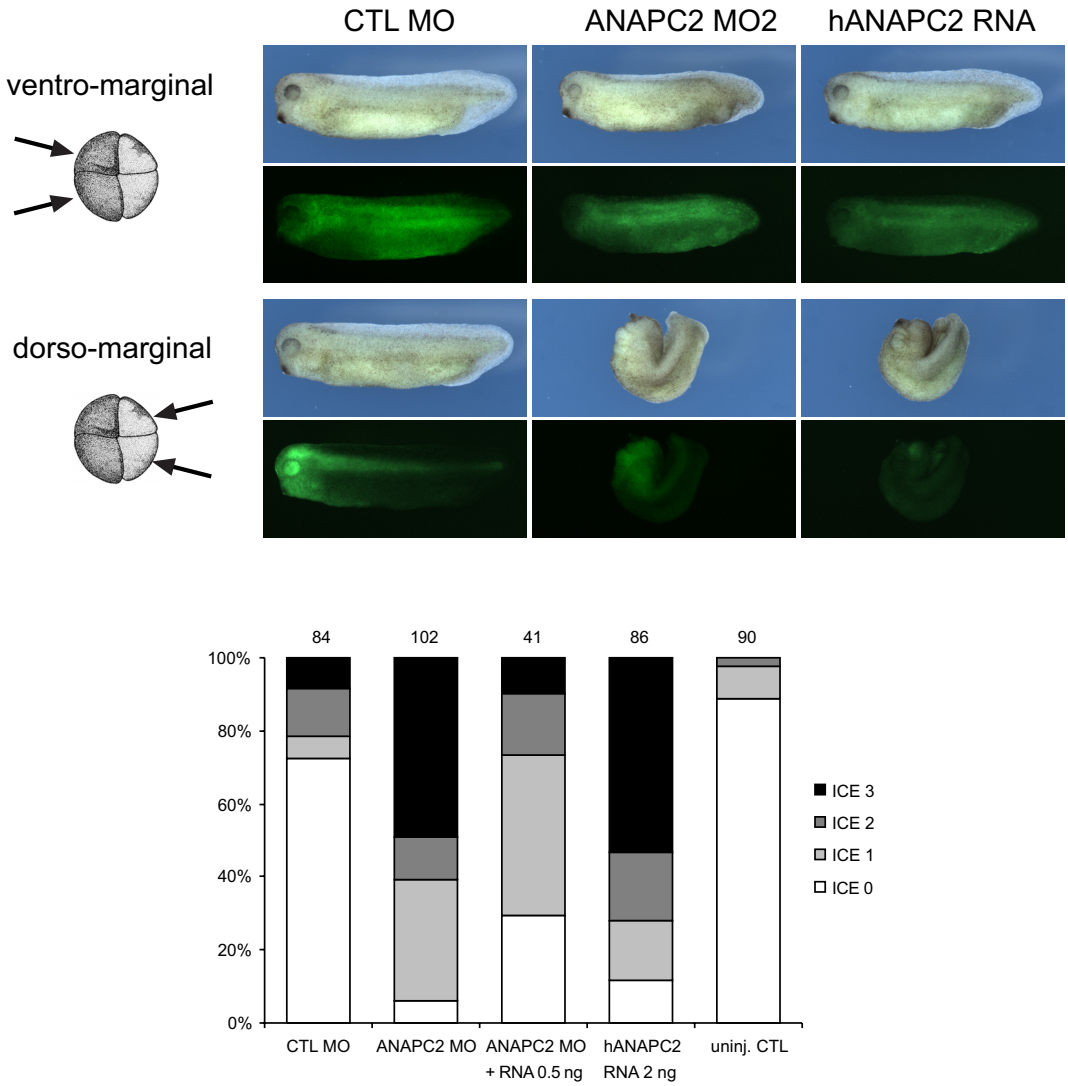

Fig. S1. ANAPC2 knockdown causes axis elongation defects. *Xenopus* embryos were injected at the four-cell stage with control (CTL) MO, ANAPC2 MO, or human ANAPC2 RNA together with membrane-targeted GFP into blastomeres as indicated. (*Upper*) Bright-field images. (*Lower*) The GFP fluorescence of the same embryo. Note that ventral injection leads to GFP expression in the epidermis of the trunk, whereas dorsal injection leads to expression in neural tissue, notochord, and somites. ANAPC2 depletion and overexpression in the ventral embryo do not lead to gross malformations. The dorsal injection leads to severe axis elongation defects and dorsal bending. The changes were quantified according to the impaired convergent extension (ICE) score, ranging from 1 (mild) to 3 (severe). Numbers above the bars represent the total number of injected embryos.

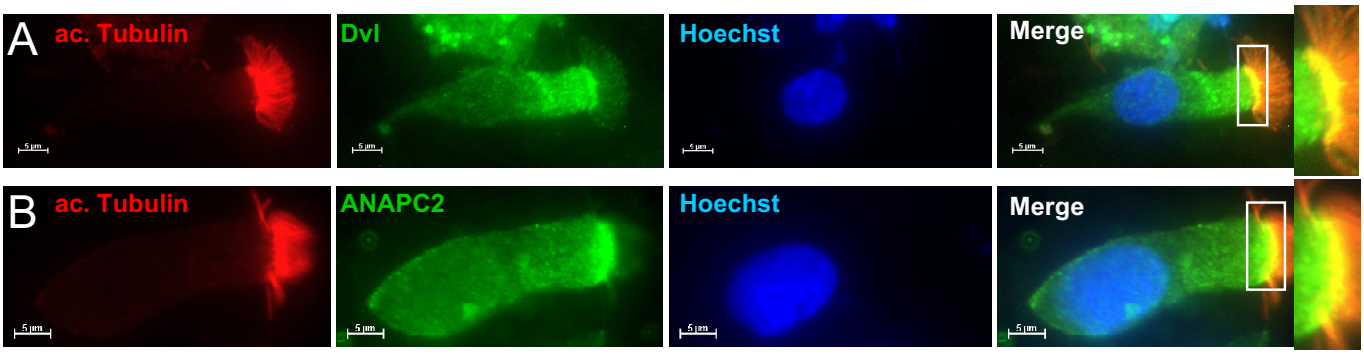

Fig. S2. ANAPC2 and Dvl localize to the subapical zone of respiratory epithelial cells. Human respiratory epithelial cells were incubated in bronchial epithelial growth medium for 2 h and subsequently stained with antibodies as indicated. Depicted are the single-channel fluorescent images and merged images. White boxes show the magnified apical region of the respiratory cells.

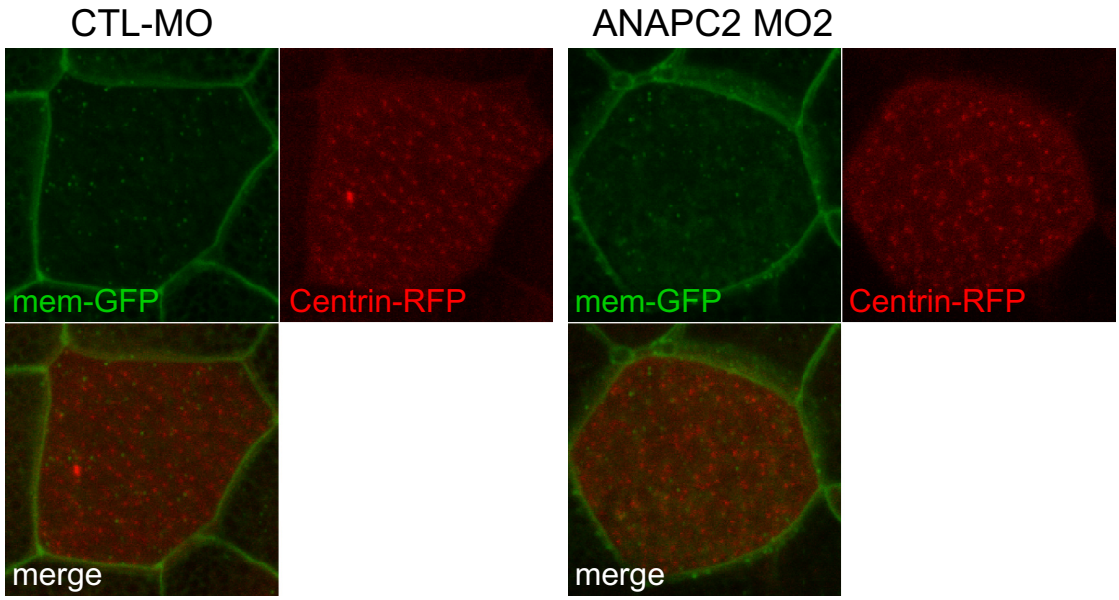

Fig. S3. ANAPC2 knockdown does not lead to aggregation of membrane-associated GFP. *Xenopus* ciliated epithelial cells expressing membrane-tagged GFP (mem-GFP) and Centrin-RFP to visualize basal bodies were scanned on a confocal microscope with identical settings. Stacked images of four optical sections at the apical membrane are depicted. ANAPC2 MO injections did not lead to changes in the localization or signal intensity of mem-GFP.