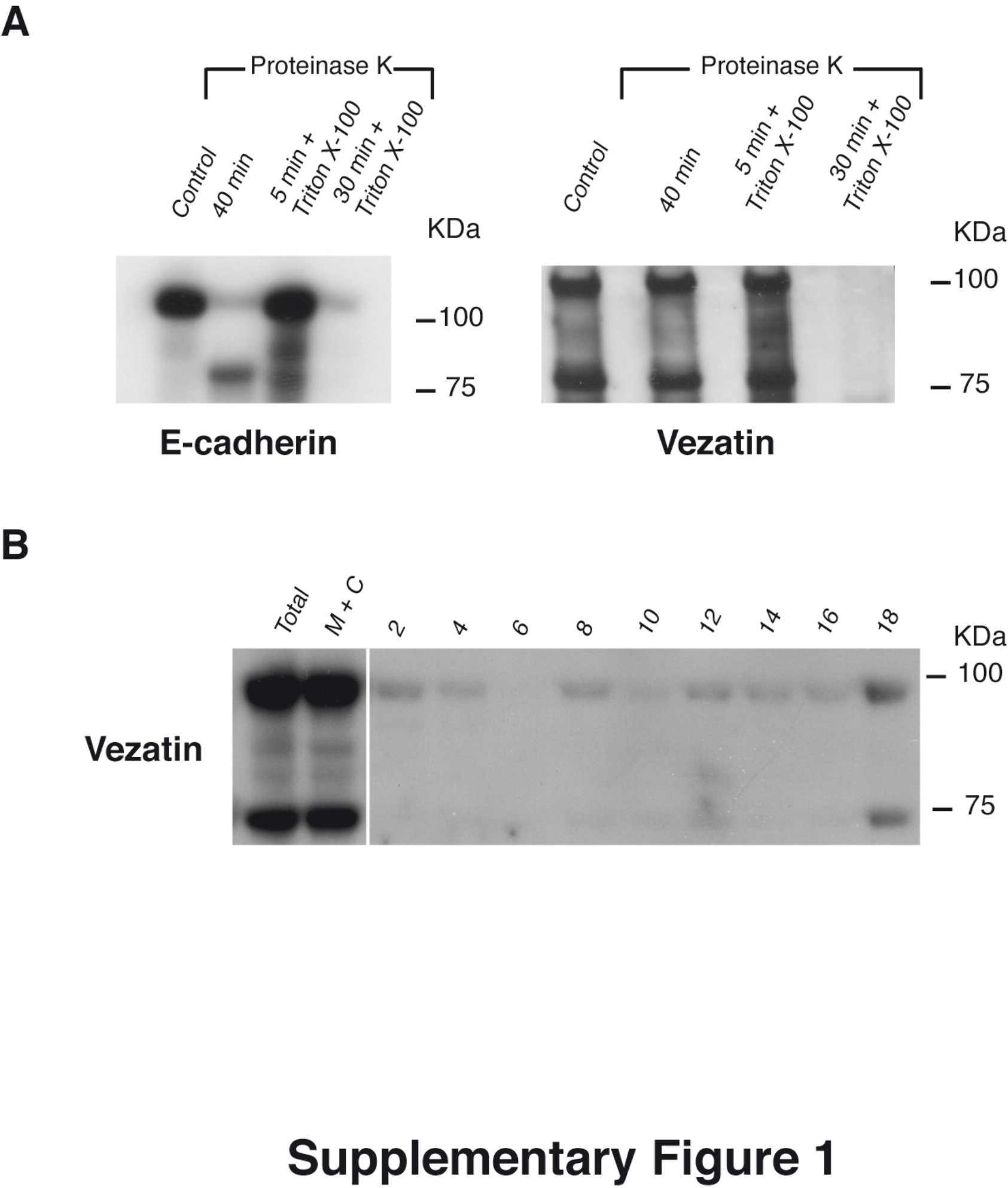
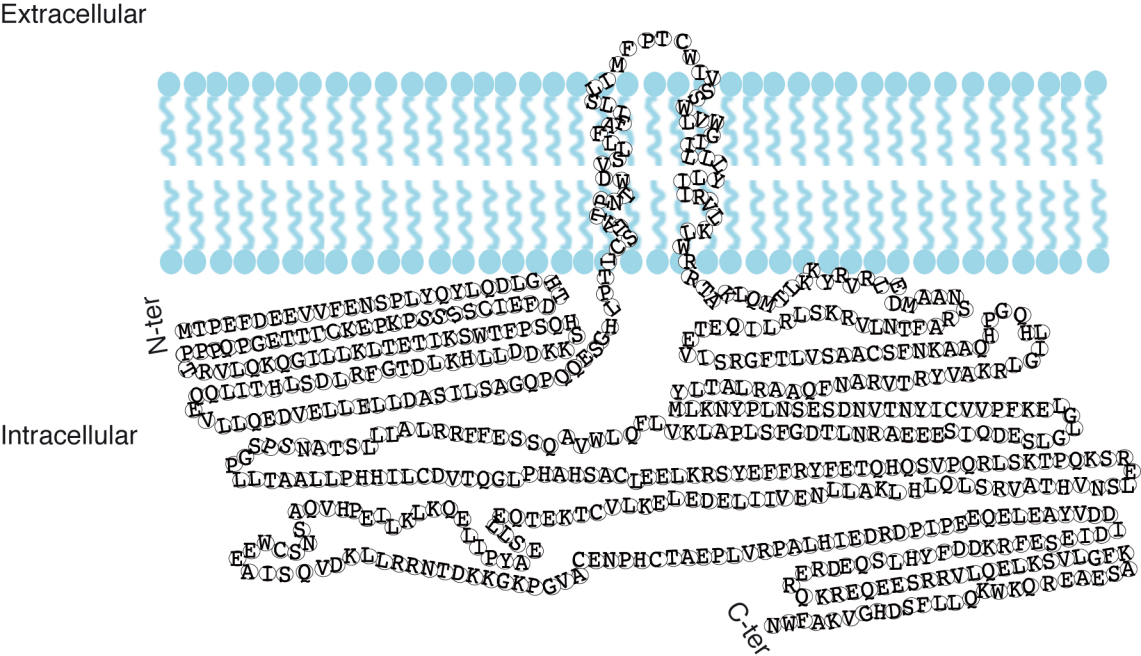
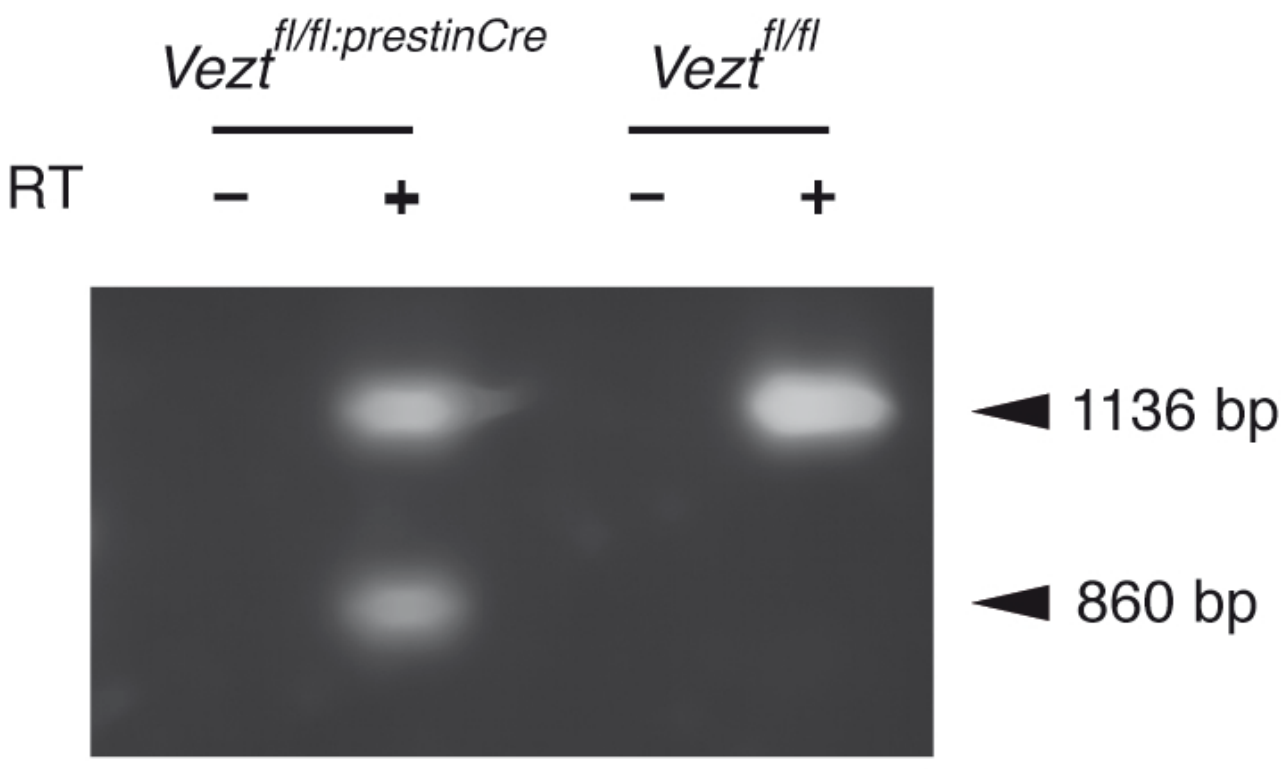


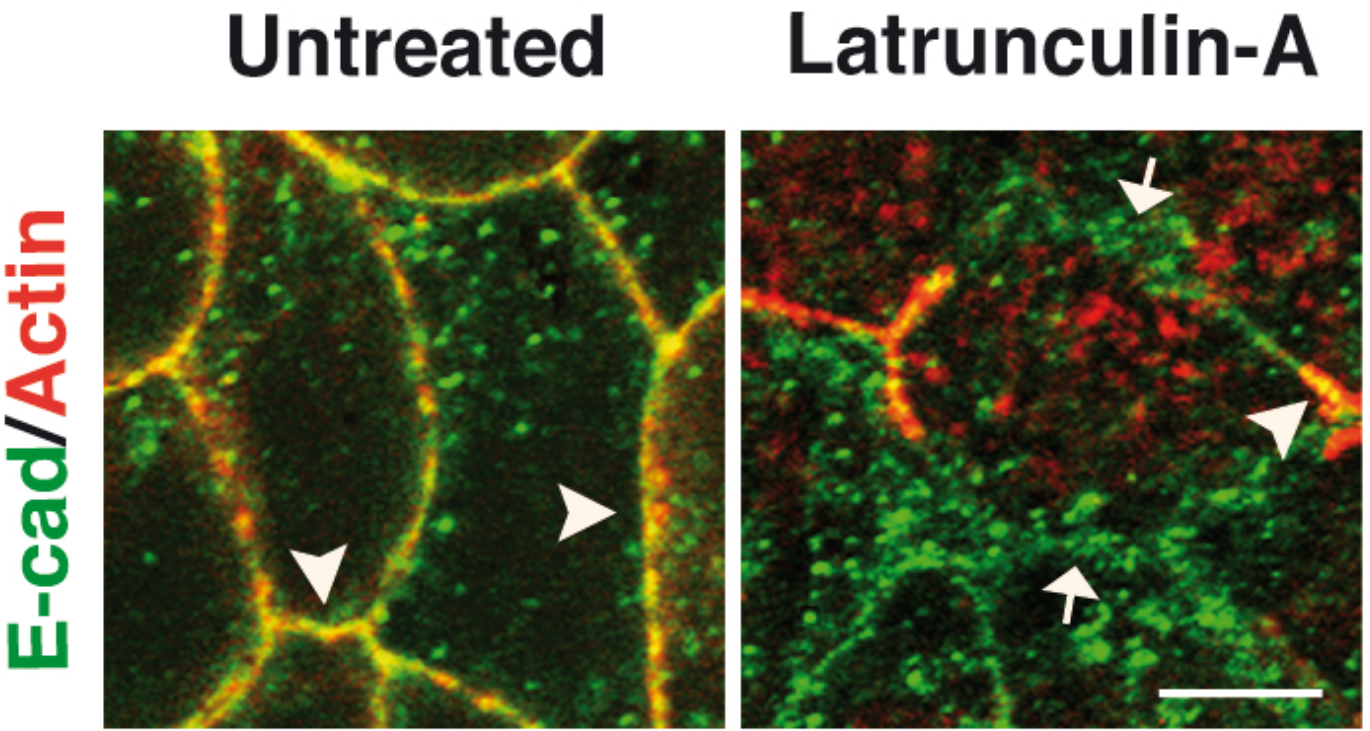
Supplementary Figure 1



Supplementary Figure 2



Supplementary Figure 3



Supplementary Figure 4

LEGENDS TO SUPPLEMENTARY FIGURES

Figure S1: Vezatin topology. **(A) Limited proteolysis experiments on confluent MDCKII cells.** Time lengths for enzyme digestion are indicated on top of each lane. E-cadherin, but not vezatin, undergoes proteolysis by proteinase K after 40 min of digestion in the absence of Triton X-100. In contrast, both proteins undergo proteolysis by proteinase K in the presence of Triton X-100. **(B) OptiPrep™ density gradient ultracentrifugation.** Confluent MDCKII cells were lysed in a iodixanol solution and subjected to ultracentrifugation. "Total" refers to MDCKII homogenate. Lanes: M + C, membrane and cytosol fraction; Equal volumes from each gradient fraction (*2, 4, 6, 8, 10, 12, 14, 16, 18*) were loaded onto the gel. The two vezatin isoforms detected on western blot using the antibody directed against the C-terminal region of the protein (see Materials and methods) have apparent molecular weights of approximately 90 kDa and 70 kDa. They are more abundant in the gradient top fraction (fraction *18*). Dividing line has been used to separate lanes that were not contiguous in the original gel.

Figure S2: Topological model of vezatin. Vezatin is an integral membrane protein with two transmembrane domains, a short extracellular interdomain (7 aa), and cytoplasmic N- and C-terminal ends. Only the short isoform (617 aa) is represented here.

Figure S3: RT-PCR detection of vezatin transcripts in the cochlear sensory epithelium of *Vezt*$^{fl/fl}$ and *Vezt*$^{fl/fl:PrestinCre}$ mice. Only one 1136 bp PCR product is detected in *Vezt*$^{fl/fl}$ mice. In *Vezt*$^{fl/fl:PrestinCre}$ mice, both 1136 bp and 860 bp PCR products (see Materials and methods) are detected, as expected from a conditional knockout that is restricted to the sensory cells of the inner ear.

Figure S4: Effects of F-actin disrupting drugs on E-cadherin distribution in MDCKII cells.

In untreated MDCKII cells (left panel), E-cadherin (E-cad) and actin are colocalised at cell-cell contacts (arrowheads). Treatment of the cells by latrunculin-A (right panel) causes disruption of F-actin bundles at AJs. The E-cadherin junctional labelling becomes diffuse (arrows). The residual F-actin and E-cadherin junctional labellings at the plasma membrane, however, still colocalise (arrowhead). Scale bar = 5 μm.