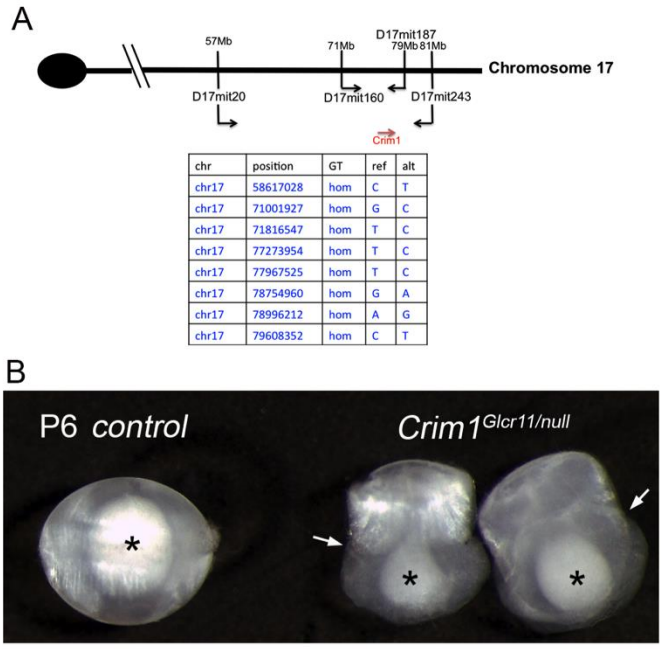

chr	position	GT	ref	alt
chr17	58617028	hom	C	T
chr17	71001927	hom	G	C
chr17	71816547	hom	T	C
chr17	77273954	hom	T	C
chr17	77967525	hom	T	C
chr17	78754960	hom	G	A
chr17	78996212	hom	A	G
chr17	79608352	hom	C	T

Fig. S1. **Genetic mapping of *Crim1*^{glcr11} mutant.**

(A) Genetic map of the *glcr11* interval on mouse chromosome 17. Polymorphic markers are shown. 8 homozygous mutations within the 26 Mb region are listed in the lower panel. (**B**) P6 compound heterozygous *Crim1*$^{Glcr11/null}$ mice exhibit posterior lens rupture (arrows) and other lens phenotypes observed in the individual homozygous lines, establishing allelism via non-complementation. (*) shows cataract fixation artifact.

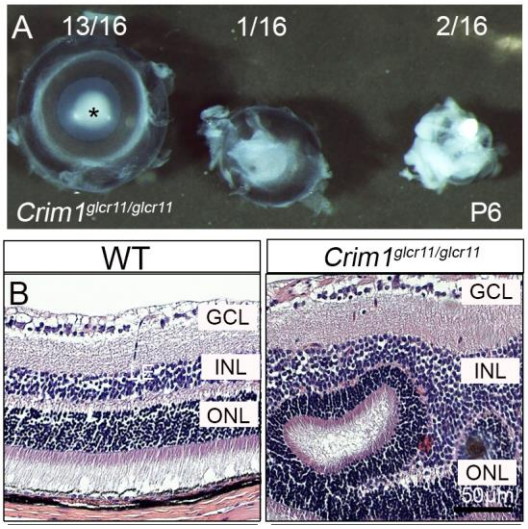

Fig. S2. *Crim1*^{glcr11} mutant mice develop microphthalmia and retina rosette.

(**A**) P6 *Crim1*^{glcr11} mutant mice develop variable lens phenotypes with 3/16 (~19%) developing overt microphthalmia. (*) shows cataract fixation artifact. (**B**) At 2 month, *Crim1*^{glcr11} mutant mice develop retina dysplasia with rosettes formation in the retinal tissue.

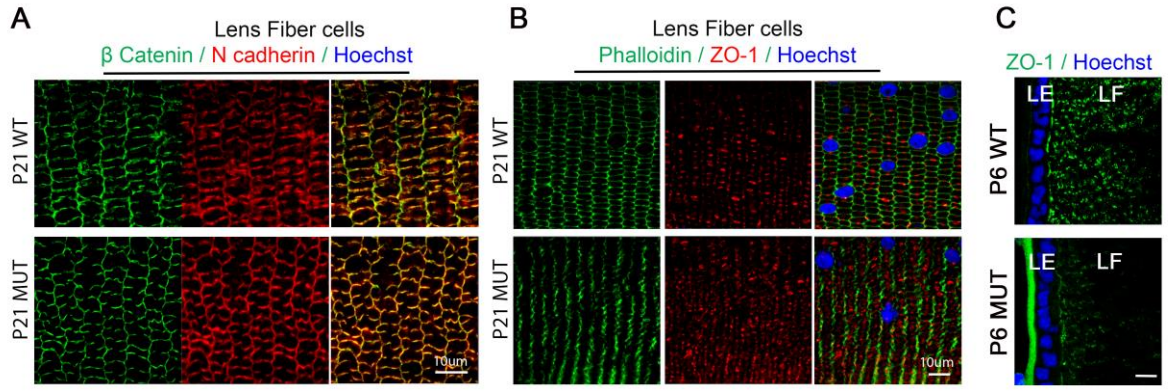

Fig. S3. *Crim1*^{glcr11} mutants display defects in lens cell adhesion and cell polarity. (A-B) Altered expression patterns of adhesion proteins (β catenin and N-cadherin) (**A**) and the polarity protein ZO-1 in P21 *Crim1*^{glcr11} mutant central fiber cells (**B**). (**C**) Decreased ZO-1 expression is observed in regions where epithelium has close contact with fiber cell.

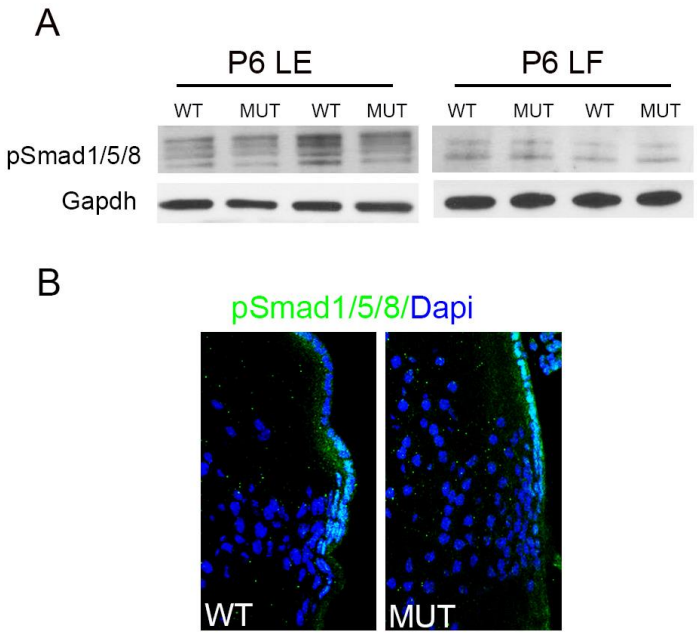

Fig. S4. phospho-Smad 1/5/8 levels are unchanged in *Crim1*^{glcr11} mutants.

(**A**) Western blot analyses of phosphor-Smad1/5/8 in P6 wild type and mutant LE cells and fiber cells. (**B**) Immunostaining of phosphor-Smad1/5/8 showed no difference in *Crim1*^{glcr11} mutants, compared to wild type.

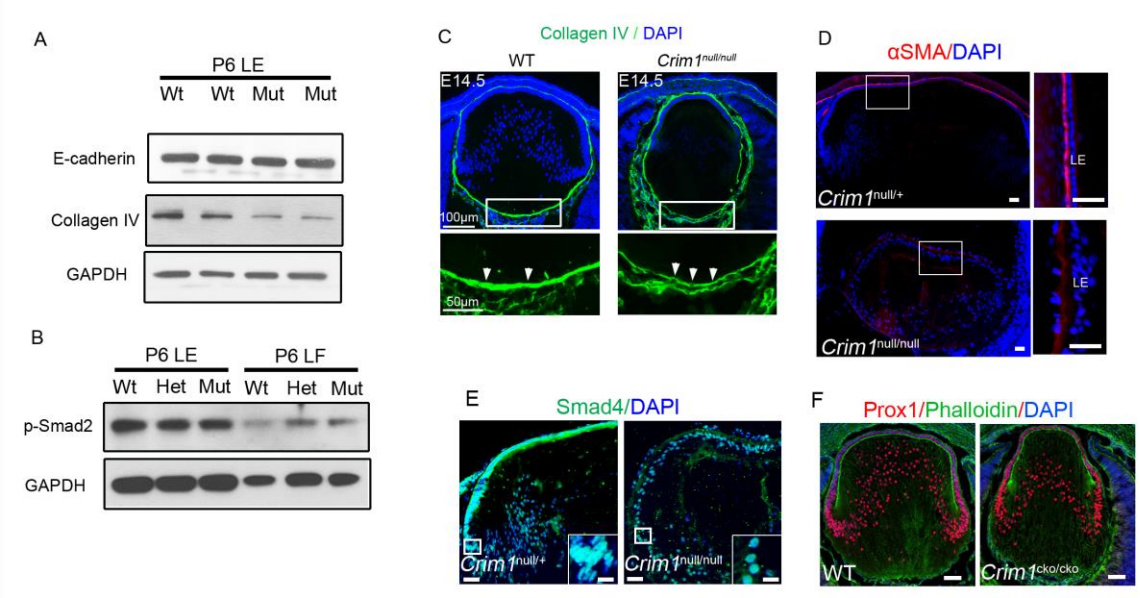

Fig. S5. *Crim1*-deficient lens epithelium is disorganized in a way that does not involve pathologic EMT.

(**A**) Western blot analyses of P6 wild type and *Crim1*^{glcr11} mutant LE cells with the indicated antibodies. (**B**) Western blot analyses of phospho-Smad2 in P6 wild type and *Crim1*^{glcr11} mutant LE cells and fiber cells. (**C**) Immunostaining for Collagen IV shows diffuse decreased expression in the posterior capsule of *Crim1*^{null} mutant lens compared to wild type control lens. (**D-E**) Immunostaining of E18.5 lenses for alpha-smooth muscle actin (**D**) and Smad4 (**E**) shows no difference between control and *Crim1*^{null} mutant. Inserts show higher magnification of nuclear localization of Smad4. (**F**) Immunostaining of E14.5 lenses for Prox1 and Phalloidin shows no difference between control and *Crim1*^{cko} mutant.