

Fig. S1. Baz is absent from the leading edge in live embryos. (A) UAS-E-cadherin-GFP (green) and UAS-mCherry-Baz (magenta) expressed using *en*-Gal4 reveals absence of Baz from leading edge cell-cell junctions (arrows). (B) UAS-mCherry-Moesin (green) and UAS-GFP-Baz (magenta) expressed using *en*-Gal4 reveals absence of Baz from actin-rich region of the leading edge (arrows). Scale bar: 5 μ m.

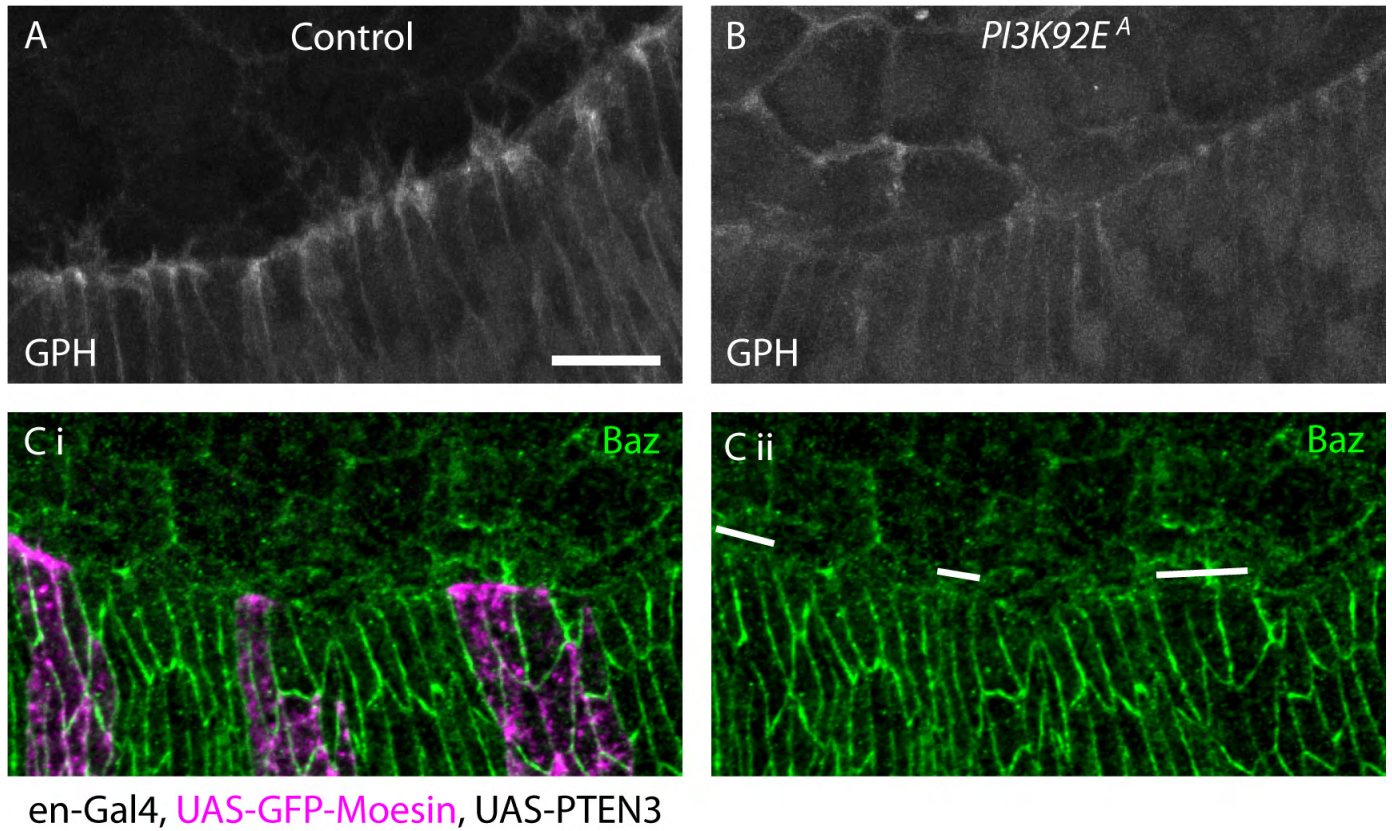


Fig. S2. PIP3 distribution in zygotic *PI3K92E* mutant embryo and Baz distribution in Pten3-expressing embryo. (A,B) GPH expressed ubiquitously in wild-type (A) or *PI3K92E^A* zygotic mutant (B) embryos reveals that leading edge PIP3 is severely depleted in the absence of zygotic PI3K expression. (C) Staining of endogenous Baz (green) in an embryo expressing UAS-Pten3 in stripes using *en*-Gal4 reveals that cellular distribution of Baz is unaffected by depletion of PIP3. UAS-GFP-Moesin (magenta) is also expressed under control of *en*-Gal4 to allow Pten3-expressing cells to be identified. Magenta channel has been removed from Cii and Pten3-expressing cells are instead indicated by white bars. Scale bar: 10 μ m.

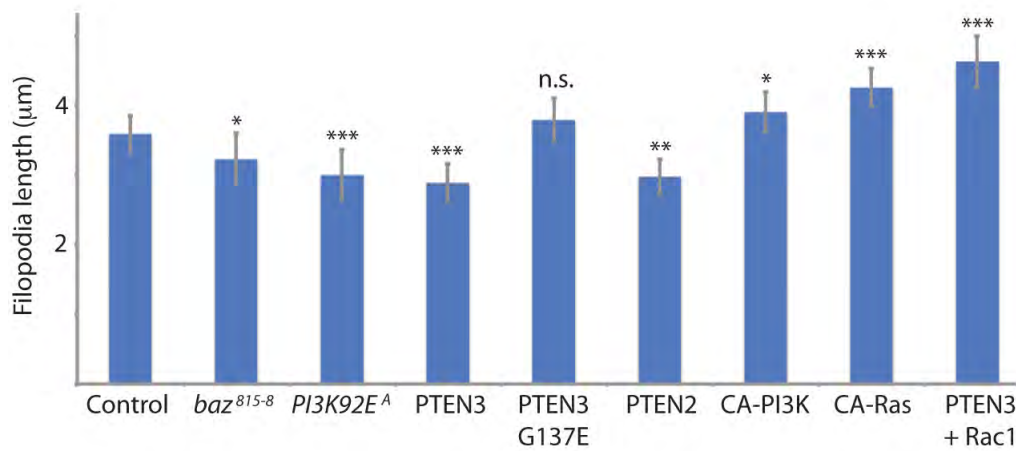
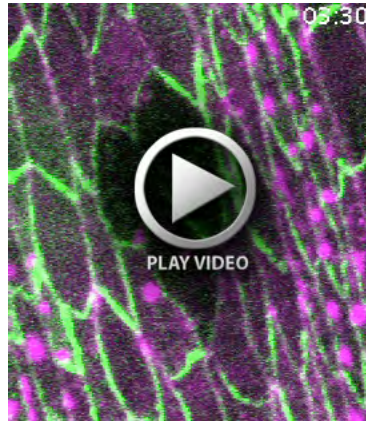


Fig. S3. Effect of altering PIP3 on filopodia length. Length of leading edge filopodia measured in embryos expressing UAS-GFP-Moesin using *en*-Gal4 alongside the indicated transgenes or in indicated mutant. $n \geq 47$ stripes from at least 11 embryos for each genotype. *** $P < 0.001$, filopodia length significantly different from control. ** $P < 0.01$, * $P < 0.05$, n.s. indicates $P > 0.05$. Error bars indicate s.e.m.



Movie 1. Dynamics of actin and Baz during epithelial wound healing. Live imaging of laser wound healing in an embryo expressing GFP-Baz (green) and mCherry-Moesin (magenta) reveals that accumulation of actin at the wound edge coincides with the loss of Baz. Images are recorded at 90-second intervals. Stills are shown in Fig. 1C.



Movie 2. Zippering during DC occurs at a consistent rate in a control embryo. Live imaging of the last stage of DC in an embryo expressing GFP-Moesin ubiquitously (green) and UAS-mCherry-Moesin (magenta) using *en*-Gal4 shows that zippering proceeds at a similar rate in *en*-Gal4-expressing stripes and in the intervening non-*en*-Gal4 stripes. Images are recorded at 120-second intervals. Stills are shown in Fig. 5D.



Movie 3. Zippering speed is reduced in cells overexpressing PTEN3. Live imaging of the last stage of DC in an embryo expressing GFP-Moesin ubiquitously (green) with UAS-mCherry-Moesin (magenta) and UAS-Pten3 expressed using *en-Gal4*. Zippering is slower in the *en-Gal4* stripes expressing Pten3, such that stripes are skipped by the zippering canthus. Images are recorded at 120-second intervals. Stills are shown in Fig. 5E.



Movie 4. Filopodia form at locations on the leading edge at which PIP3 levels are elevated. Live imaging of DC in an embryo expressing UAS-GPH (green) and UAS-mCherry-Moesin (magenta) using *en-Gal4* reveals spatial and temporal correlation between accumulation of PIP3 at the leading edge and initiation of filopodia. Images are recorded at 15-second intervals. Stills are shown in Fig. 6C.



Movie 5. PIP3 accumulates at cell-cell junctions along epithelial wound edges. Live imaging of laser wound healing in embryo expressing GPH (green) and mCherry-Baz (magenta) in the epidermis reveals that PIP3 accumulates at wound edge cell-cell junctions. Junctions at which PIP3 accumulates are devoid of Baz. Images are recorded at 150-second intervals. Stills shown in Fig. 7A.