

Fig. S1. (related to main Fig. 3)

Sequence conservation of the Dishevelled PDZ domain. Sequence alignments of the DVL PDZ domain across diverse animal species. Amino acids not similar between human (Hs) DVL2 and *Drosophila* (Dm) Dsh are indicated (*) although none are key peptide binding contacts (*red font*).

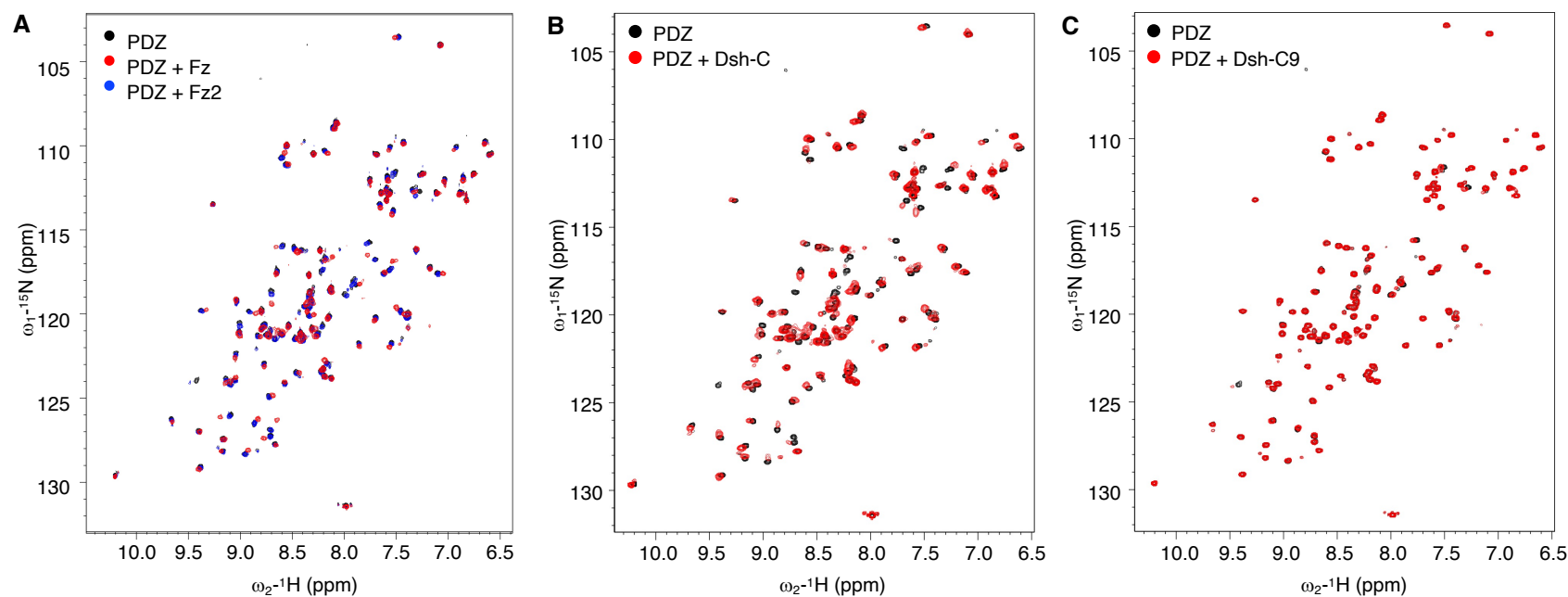


Fig. S2. (related to main Fig. 3 and 4)

Fz and Dsh-C but not Fz2 or Dsh-C9 peptide binding to PDZ. Overlay of HSQC spectra of 100 μ M 15 N-PDZ alone (*black*) or with 300 μ M Fz (*red*) or Fz2 (*blue*) (**A**) 150 μ M Dsh-C (*red*) (**B**) or with Dsh-C9 (*red*) (**C**).

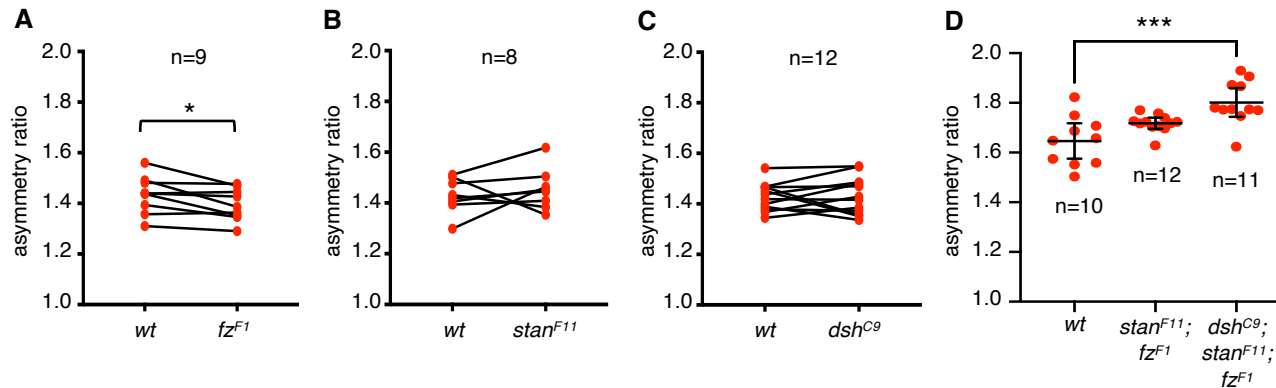


Fig. S3. (related to main Fig. 4)

PBM truncations modulate the polar distribution of Fmi. Quantitation of mean Fmi polarity in single, double and triple mutant wings compared to *wt* tissue, as indicated underneath graphs; paired T-tests were used to compare values in the same wing, * $p < 0.05$ (A-C), one-way ANOVA with Dunnetts multiple comparisons test was used to compare mutant wings to *wt* wings immunolabelled in parallel, *** $p < 0.001$; Error bars indicate 95% confidence intervals; N-numbers are indicated on graphs. The *fz^{F1}* truncation slightly reduces the polarised distribution of Fmi (A), whereas double and triple truncations increase polarity of Fmi compared to *wt* tissue (D), suggesting a modulatory and partially redundant role of these PBM in generating or maintaining proximodistal polarity. This could be somewhat indirect as the levels of *Stbm* and Fmi are also slightly reduced in these mutants.

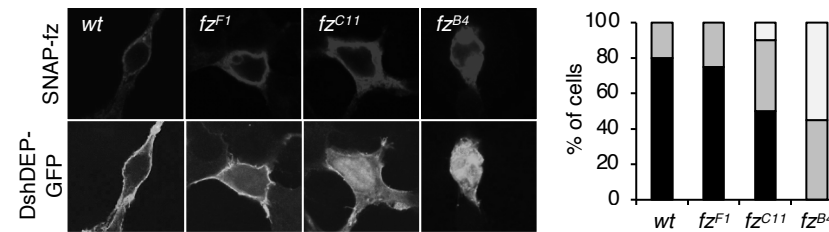


Fig. S4. (related to main **Fig. 4**)

DshDEP-GFP not recruited to SNAP-Fz^{B4}. Left, representative images of cells. Graph shows quantitation of *wt* or mutant Fz-dependent recruitment of Dsh DEP–GFP to the plasma membrane as strong (*black*), weak (*grey*); or none (*white*); 100 cells were scored in each case.

Fig. 1D

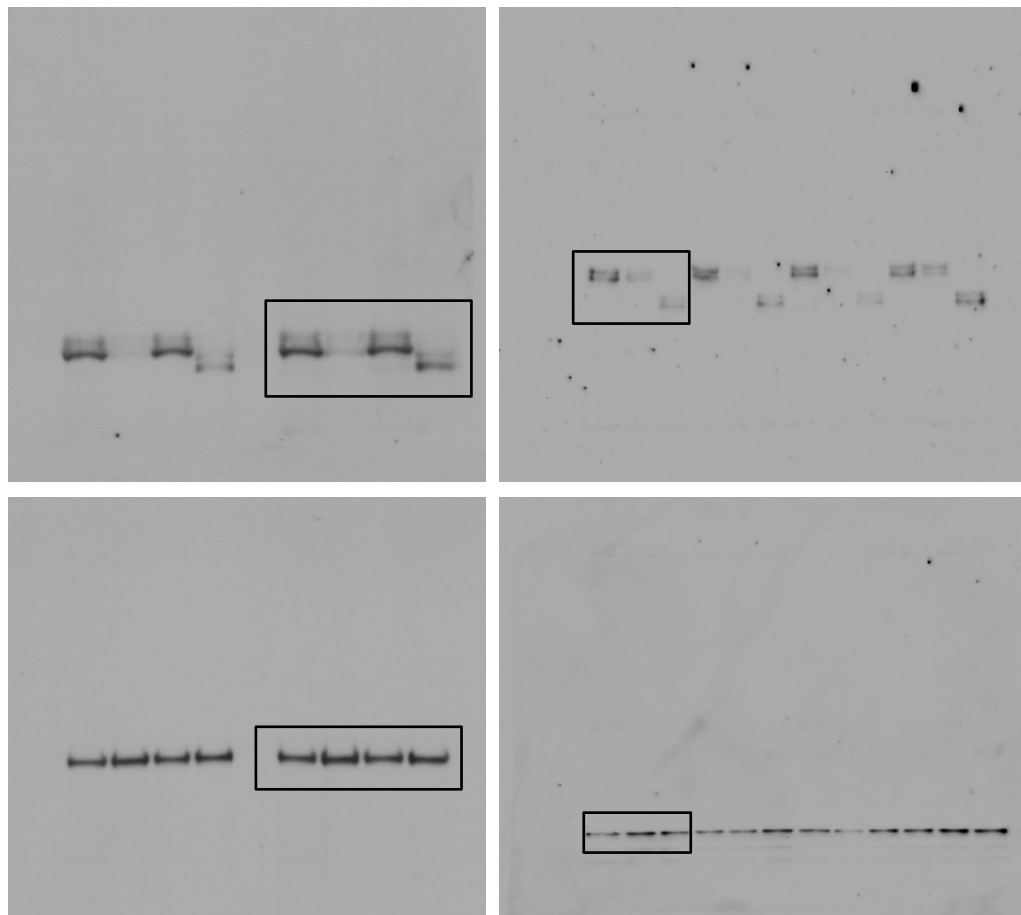


Fig. 4B

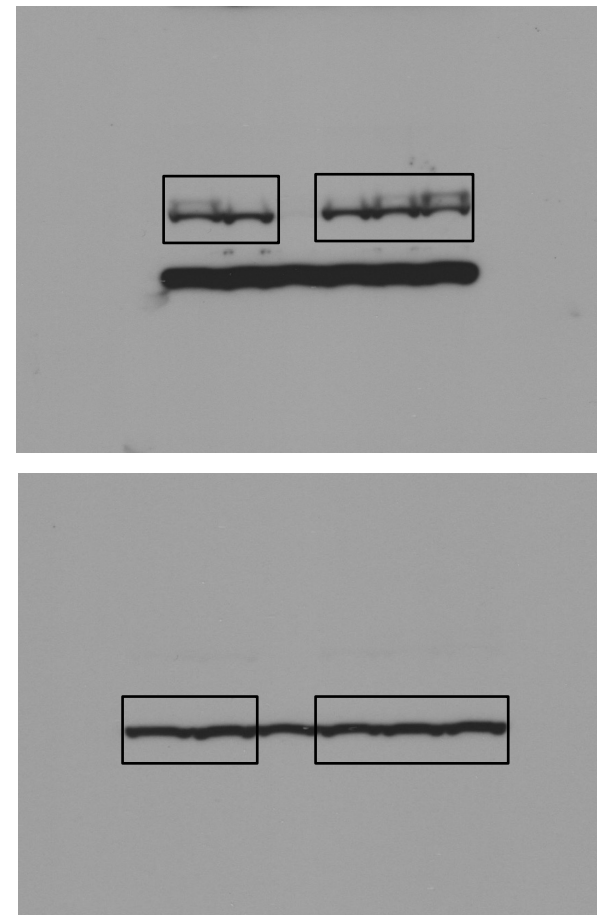


Fig. S5. Blot transparency (related to main **Fig. 1D** and **Fig. 4B**)