

Fig. S1. nub>brk induces apoptosis and defective cell/tissue morphogenesis. (C,E-G,I,J) Transverse sections (*x-z* images). Arrows indicate extruded cells. (A-C') Expressing *brk* by nub-Gal4 induced decreased proliferation (A), cell death (B) and changes in tissue morphogenesis as well as cell extrusion (C). Inset in A shows the nub-Gal4 expression pattern. (**D-G**') Repressing cell death rescued the defect in proliferation (D,G) but not in tissue morphogenesis (E). Wild-type control (F). (**H-J**) *dpp* mutant wing discs showed medially enhanced proliferation (H) along with extrusion of cells and disruption of normal tissue morphology (I,J).

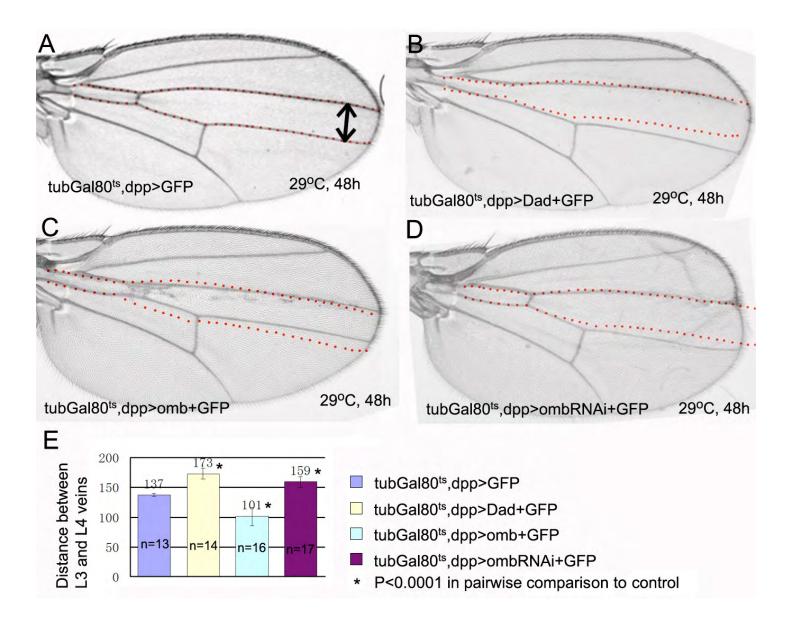


Fig. S2. Attenuated Dpp-Omb signaling increases the distance between L3 and L4 in the adult wing. Red dotted lines outline the normal position of L3 and L4 as observed in the control wing. (A) Control wing. (B) Repression of Dpp signaling by expressing Dad in the dpp-Gal4 domain increased the distance between L3 and L4. (C) Expressing *omb* in the dpp-Gal4 domain reduced the distance between L3 and L4. (D) Repression of *omb* in the dpp-Gal4 domain increased the distance between L3 and L4. (E) Statistics of the L3-L4 distance measurements (in arbitrary units). *t*-tests of pairwise comparisons showed significant differences between control and the three experimental genotypes (P<0.0001). Error bars indicate s.d.

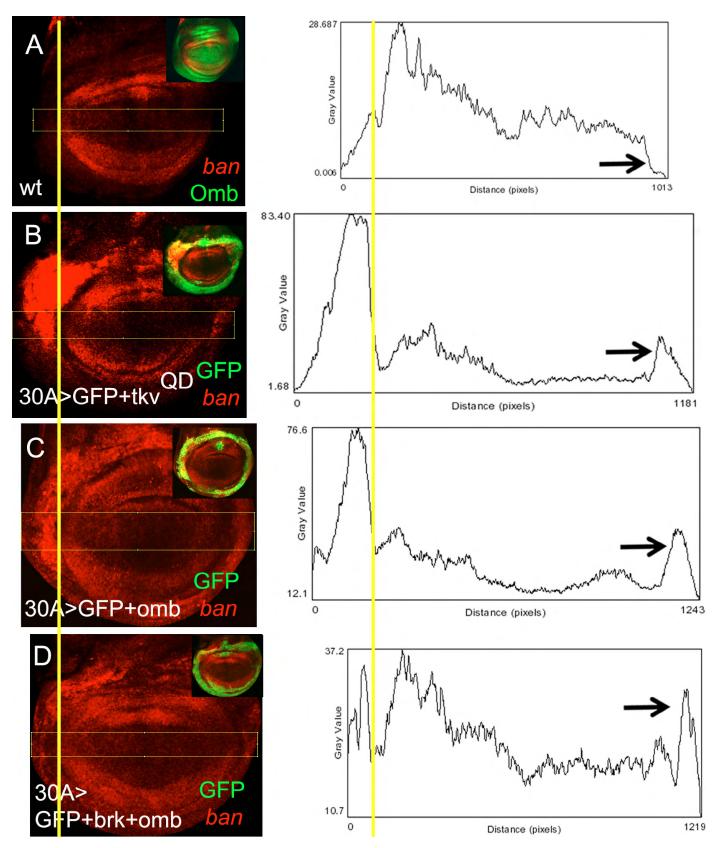


Fig. S3. Quantification of *ban* **expression.** Omb or Dpp signaling was manipulated in the 30A-Gal4 domain as indicated. A to D correspond to A, C, D and E of Fig. 3. Fluorescence intensities were measured (by ImageJ) in the boxed regions. Panels are oriented such that the anterior hinge-pouch fold is in vertical register (yellow line). (**A**) Low *ban* expression in lateral wing disc. (**B**) Lateral Tkv^{QD} induced *ban* expression in the lateral wing disc. Upregulated *ban* only occurred in the lateral regions where *omb* was elevated, but not in pleura and medial hinge where *omb* was not induced. (**C**) 30A>omb induced *ban* expression in the entire 30A-Gal4 domain. (**D**) When *brk* and *omb* were co-expressed, *ban* was still upregulated laterally. Note that the 30A-Gal4 activity in D was weaker than in B and C. *brk* overexpression apparently attenuated 30A-Gal4 activity. In all three 30A-Gal4-driven experiments, lateral upregulation of *ban* by Tkv^{QD} or Omb was stronger anteriorly than posteriorly, which reflects lateral differences in Gal4 activity (see insets in B-D). This also implies that *ban* is sensitive to the level of Omb in the lateral wing.

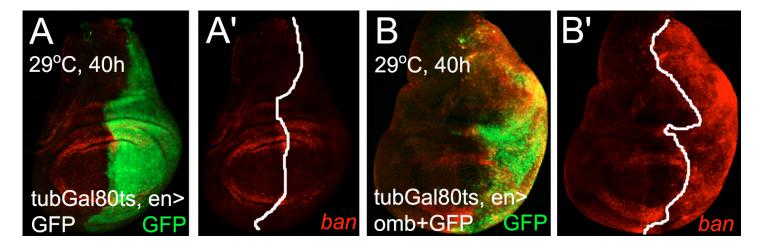


Fig. S4. Omb induces *ban* expression in the lateral wing. (A,A') Control experiment. (B,B') Expressing *omb* by en-Gal4 under temporal Gal80^{ts} control for 40 hours induced the elimination of posterior pouch and enhanced lateral *ban* expression.