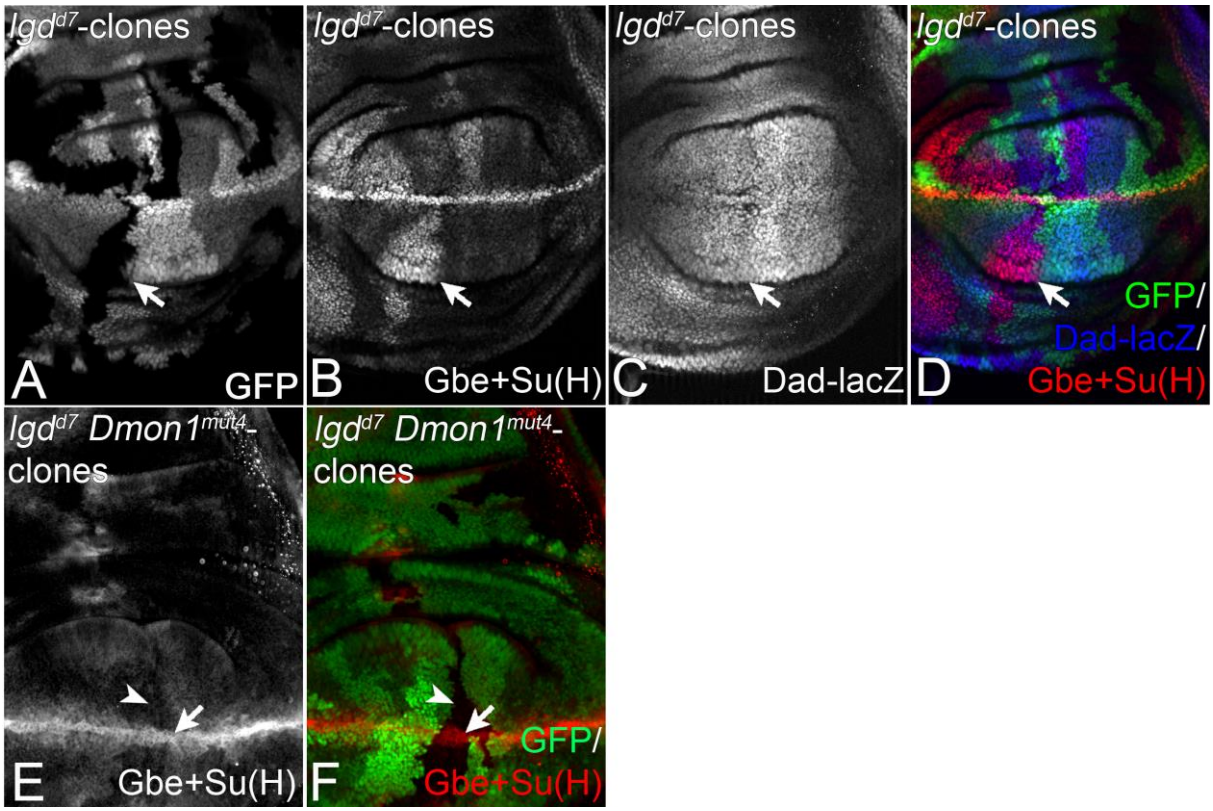


Fig. S1. Analysis of the activity of the Dpp and Notch pathways in *lgd* mutant and *lgd Dmon1* double mutant cell clones of the wing imaginal disc. (A-D) Loss of *lgd* function in the wing imaginal discs results in activation of the Notch but not the Dpp pathway. The clones are labelled by absence of GFP. While the Notch activity marker is upregulated in *lgd* mutant clones, the expression of dad-lacZ is not affected. The arrow highlights one of the clones. (E, F) the loss of function of *Dmon1* in *lgd* cells results in a suppression of the ectopic activation of the Notch pathway. The arrow point to a *lgd* mutant clone that cross the dorsoventral (D/V) boundary of the wing anlage. While the ligand dependent activation of Gbe+Su(H) along the D/V boundary is not affected (arrow) no ectopic activation of the Notch activity reporter can be observed in the area of the *lgd Dmon1* clone, away from the boundary (arrowhead). This is in contrast to the loss of function of *lgd* shown in (B, arrow).

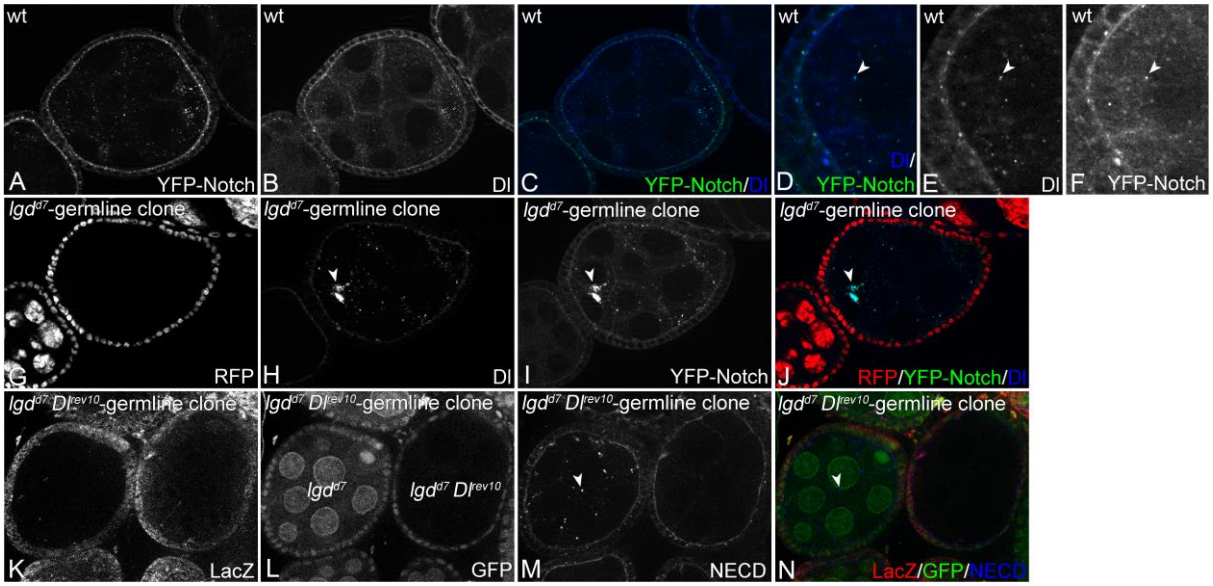


Fig. S2. Dl signalling from GCs to FCs causes the accumulation of the NECD in GCs. (A-F) Wildtype egg chamber. YFP-Notch and Dl co-localise in endosomes of the GCs. The arrowhead points to one of the Dl and YFP-Notch. (G-J) Colocalisation of YFP-N and Dl in the enlarged endosomes of *lgd* mutant GCs (arrowhead). (K-N) Generation of *Dl lgd* double mutant GCs. The clones are generated in hsFlp; lgd^{d7} FRT 40A/ arm. lacZ FRT40A; Dl^{rev10} FRT82B/ Ub.*GFP* FRT 82B flies. (K) Two egg chambers where *lgd* is lost, indicated by the loss of lacZ staining. (L) The absence of GFP in the right chamber indicate that this chamber has also lost the function of *Dl* in its GCs. (M) The left *lgd* mutant GCs contain the NECD (arrowhead). In contrast, NECD is absent in the right *Dl lgd* double mutant GCs. This indicates that the accumulation of NECD in GCs is dependent of the presence of Dl. (N) merge of the single channels shown in (K-M).

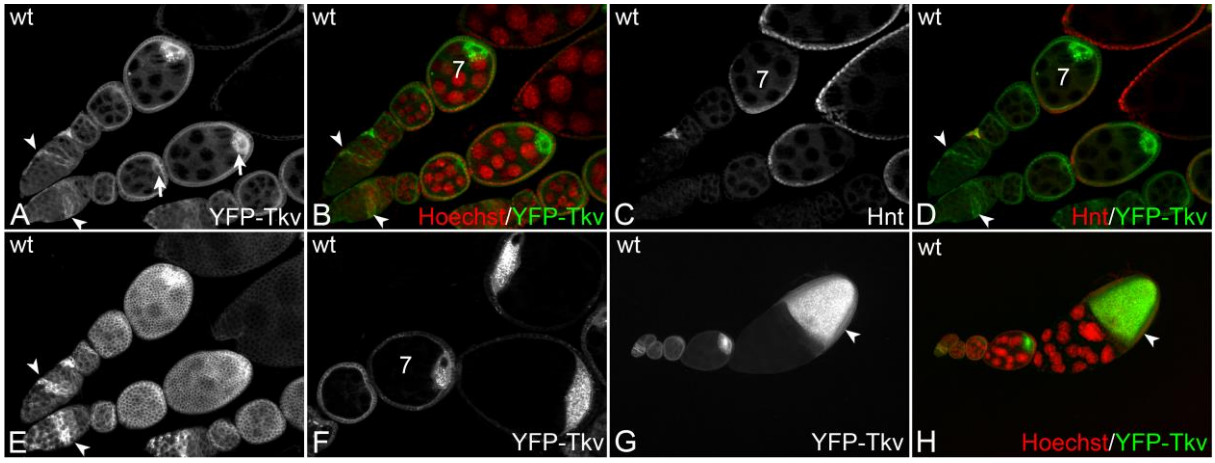


Fig. S3. Expression of YFP-Tkv during oogenesis. (A-E) Comparison of expression of YFP-Tkv with that of Hnt. (E) The ovarioles shown in (A-D) at the plane of the follicle epithelium. YFP-Tkv is expressed in all cells of the germarium at different levels during normal oogenesis. Surprisingly, the expression in the GSCs and cystoblasts is low compared to the surrounding cells. Expression peaks in the mitotic cyst stage 2B (arrowheads in A, B, D and E). Tkv-GFP continues to be expressed at high levels in early egg chambers up to stage 6/7 in FCs and GCs. During stage 6/7 it overlaps with the expression of Hnt. Moreover, is accumulates in the oocyte from stage 3 onwards (arrows in A). (F-H) After stage 7, expression declines to low levels in in FCs and GCs with the exception of the oocyte, which maintains high levels of Tkv-GFP throughout the rest of development. From stage 10 onwards the levels of Tkv-YFP increases again in FCs that surround the oocyte (G, H, arrowhead).

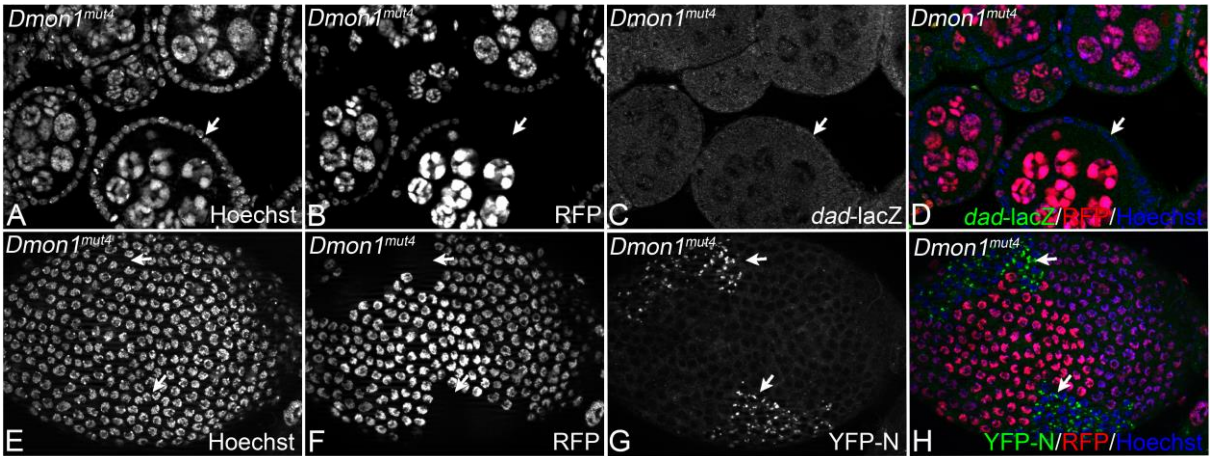
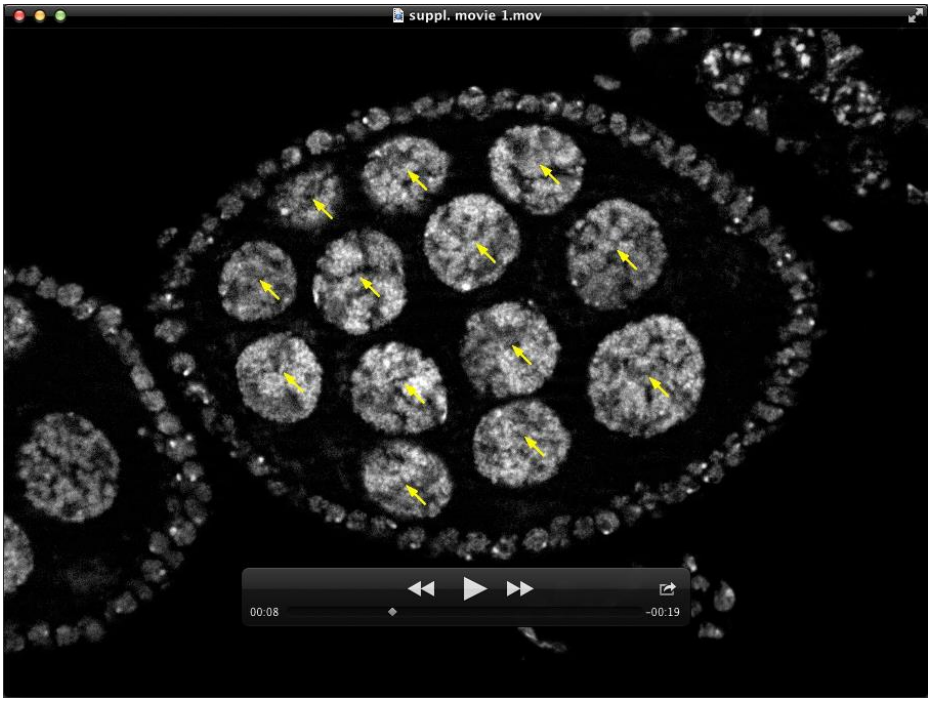


Fig. S4. *Dmon1* clones in FCs. The arrows point to the clones. (A-D) Expression of dad-lacZ is unaffected by loss of Dmon1 function. (E-H) The mutant cells accumulate Notch in enlarged endosomes.



Movie 1. Movie through an *lgd* mutant EC with 32 GCs. The GCs are highlighted by the yellow arrows.