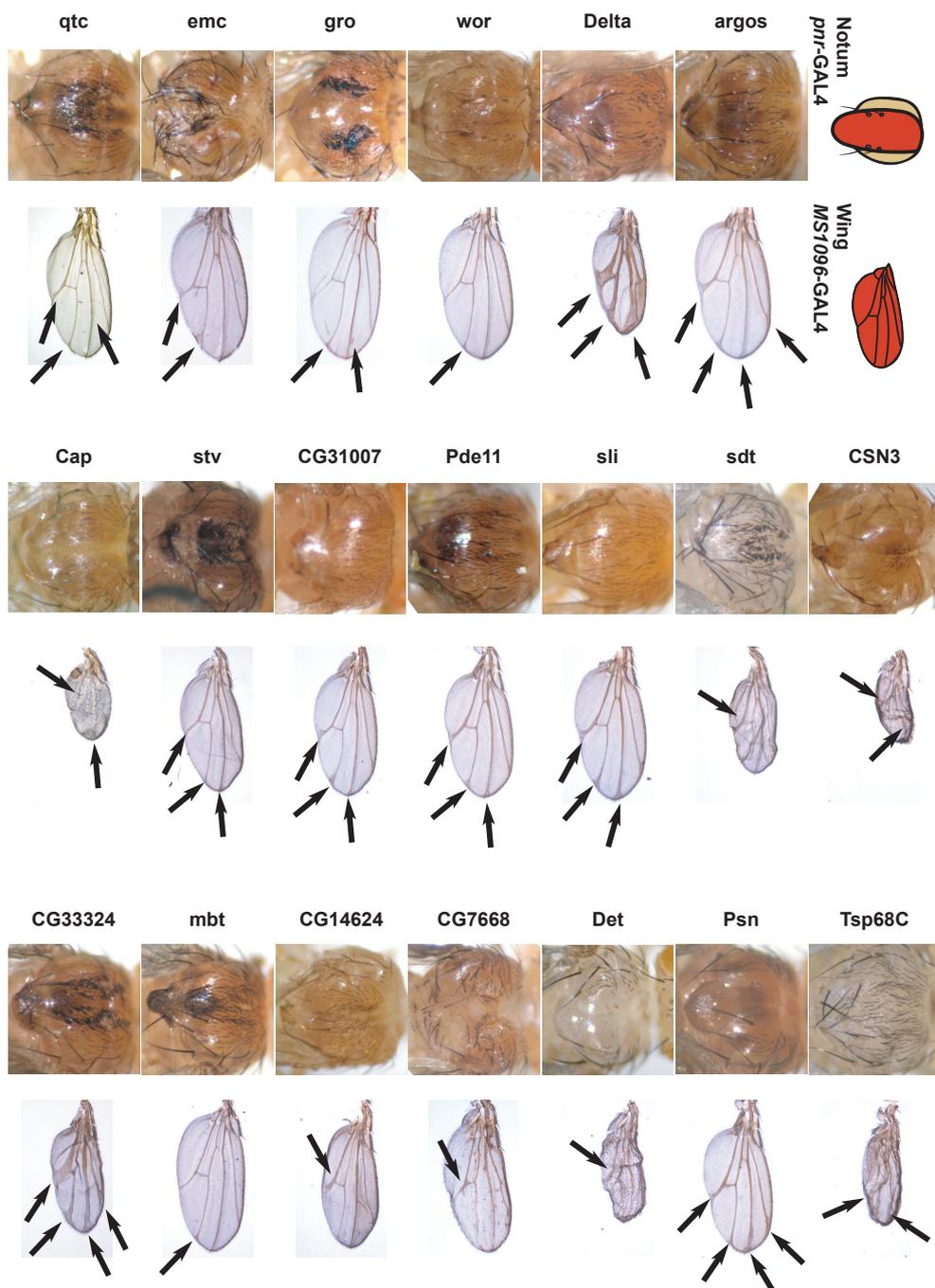


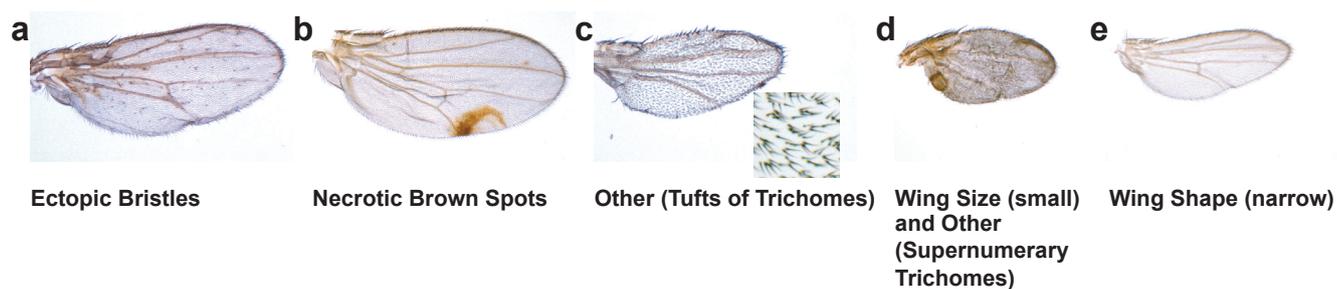
## SUPPLEMENTARY INFORMATION



**Supplementary Figure 1.**

**Notum and wing phenotypes of general Notch regulators**

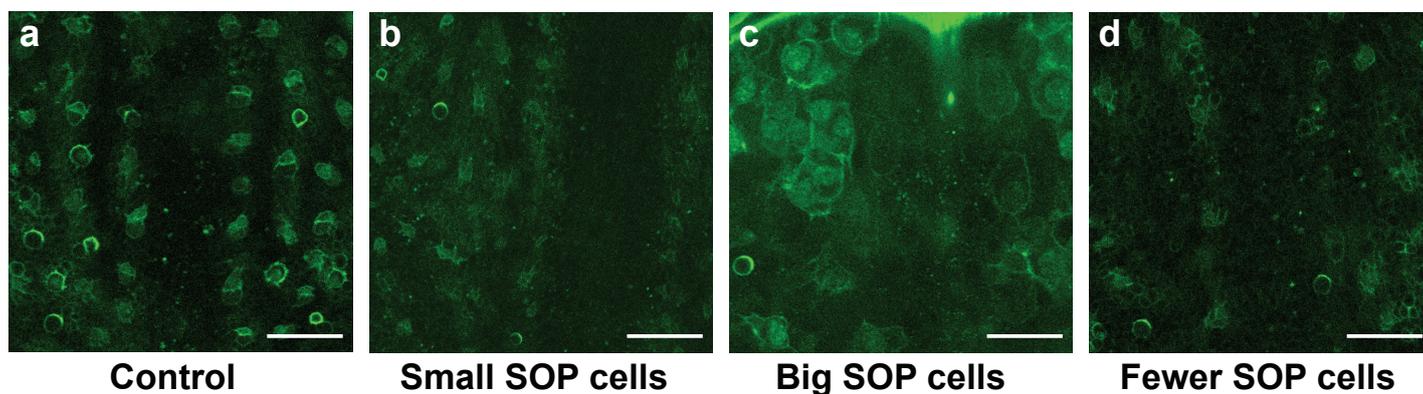
Representative images for 20 genes whose knockdown resulted in a lateral inhibition phenotype on the notum, and broader delta-like vein phenotypes (arrows) in the wing. TIDs for examples are as follows: 3238 (*argos*), 3720 (*Delta*), 6248 (*wor*), 6316 (*gro*), 11257 (*emc*), 17349 (*qtc*), 12821 (*CSN3*), 19428 (*sdt/Smr*), 20210 (*sli*), 25197 (*Pde11*), 33739 (*CG31007*), 34408 (*stv*), 39205 (*Cap*), 37441 (*Tsp68C*), 43082 (*Psn*), 43113 (*Det*), 43649 (*CG7668*), 44064 (*CG14624*), 46043 (*mbt/Smr*), and 47835 (*CG33324*).



**Supplementary Figure 2.**

**Additional *MS1096*-GAL4 wing phenotypes**

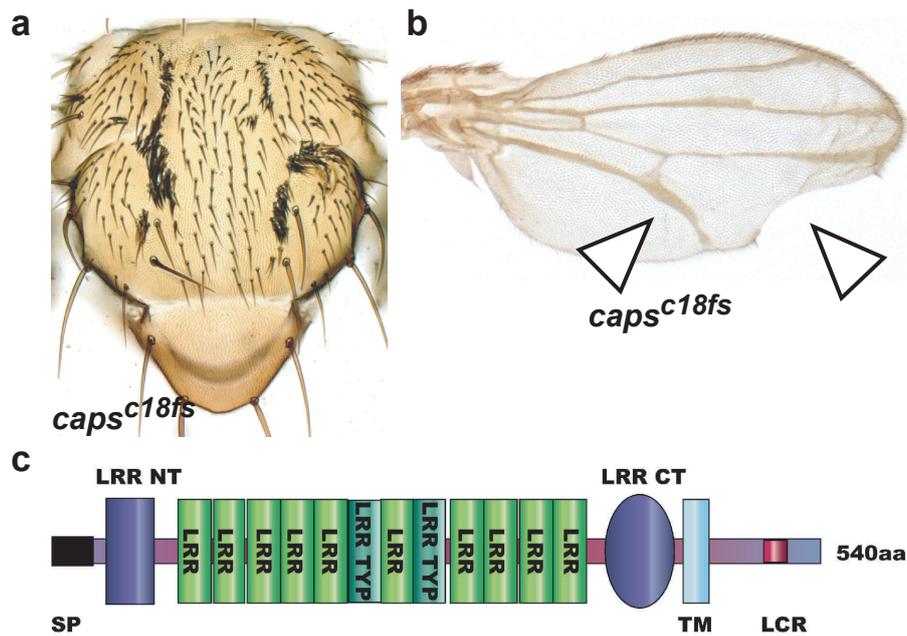
Ectopic bristles on the wing blade (a), necrotic brown spots (b) and diverse “other” (example shown for tufts of trichomes, c) phenotypes were observed. Changes in wing size (d) and shape (e) were also scored. TIDs for examples are as follows: 43649 (ectopic bristles), 33510 (necrotic spots), 32421 (other), 39205 (wing size), and 22496 (wing shape).



**Supplementary Figure 3.**

**Additional live imaging assay phenotypes**

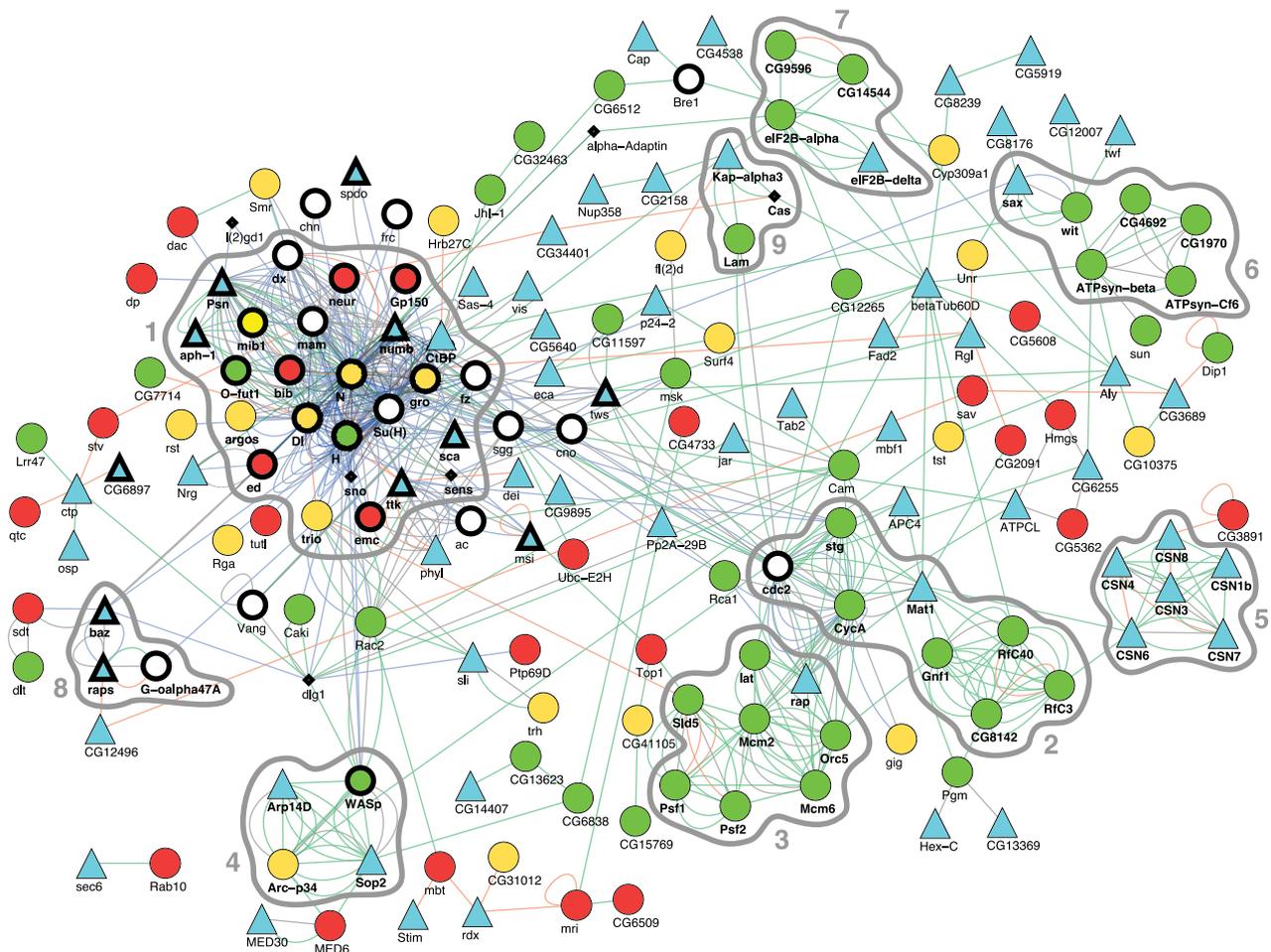
Confocal images of pupal nota show SOPs labeled by expression of *phyllopod*>>EGFP::Pon.LD (green) with simultaneous induction of RNAi. Relative to control pupae (a), changes in the size (b,c) and number (d) of SOP cells were observed. Scale bars: 10 $\mu$ m. TIDs for examples are as follows: 9660 (small SOP cells), 32421 (big SOP cells), and 21536 (fewer SOP cells).



#### Supplementary Figure 4.

##### *capricious* mutant clones cause Notch loss-of-function phenotypes

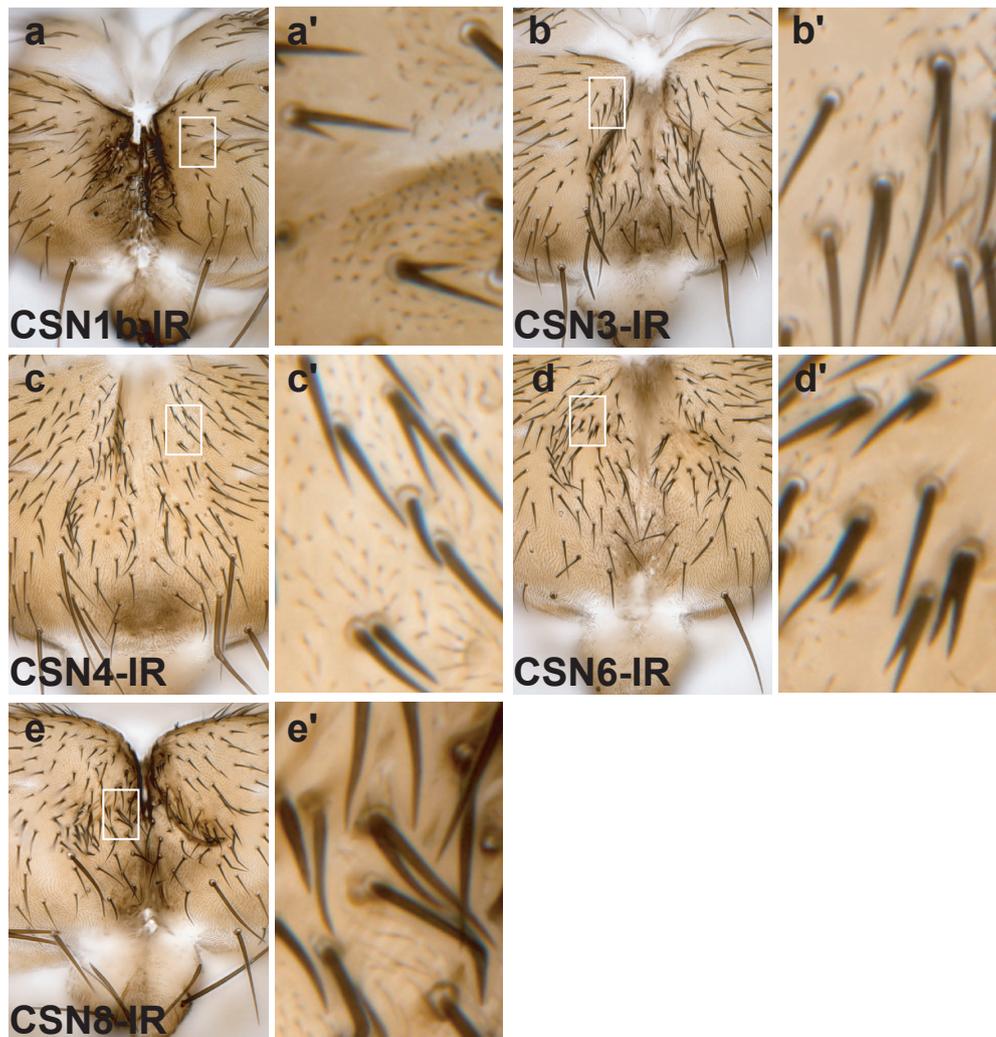
*a.* *caps* mutant clones on the notum cause a strong gain of bristles phenotype. *b.* Wing clones show both broad wing veins and wing margin notches (arrowheads). *c.* Caps is a 540 amino acid, single pass transmembrane (TM) protein with a putative signal peptide (SP), extracellular leucine-rich repeat (LRR) domains and an intracellular low complexity region (LCR).



**Supplementary Figure 5.**

### An interaction network for Notch signaling

Genes in the “lateral inhibition” and “asymmetric cell division” categories with physical or genetic interactions are displayed as a network map governing Notch signaling. Genes are shown as nodes, and relationships between nodes as edges. The genome-wide primary screen phenotype of each gene is indicated by the colour and shape of the node (gain, red circle; loss, green circle; both gain and loss, yellow circle; socket or hair duplication phenotypes, blue triangle; completely lethal, black diamond; no phenotype in gain, loss, socket or hair phenotypes (control genes only), white circle). Positive control gene nodes are outlined in bold. The type of interaction is indicated by edge colour (genetic (blue), interolog (green), yeast two-hybrid (orange) and other (gray)). MCODE-identified complexes are outlined in gray and rank is indicated.



**Supplementary Figure 6.**

**CSN knockdown causes cell fate transformations**

a-e. Notum phenotypes for knockdown of the indicated COP9 signalosome subunits show cell fate transformations (two hair and two socket), gain of bristles and notum malformation due to cell death. a'-e'. Enlargements of the areas boxed in white are shown. TIDs for examples are as follows: 34727 (CSN1b), 12822 (CSN3), 28942 (CSN4), 22307 (CSN6), and 50566 (CSN8).