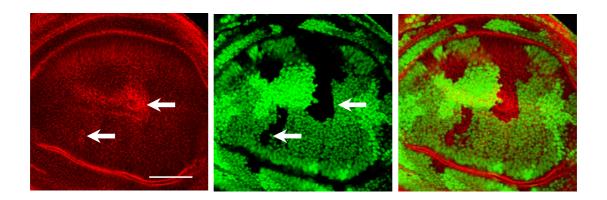
Supplementary information list

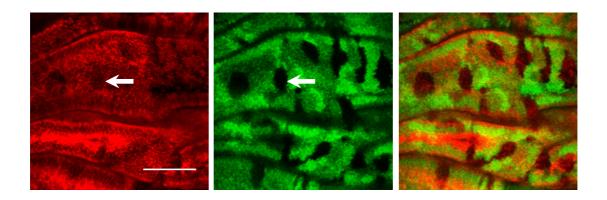
Supplementary Figure S1 Arm protein levels are increased in *CSN5*^{null} mutant clones generated in wing discs.

Supplementary Figure S2 CSN regulates Ci¹⁵⁵ level by a post-transcriptional mechanism.

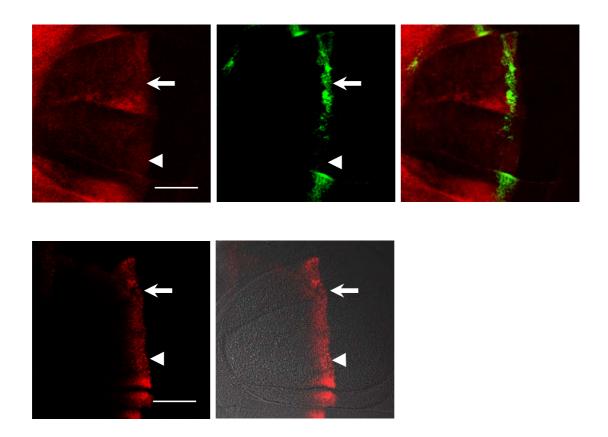
Supplementary Figure S3 Effects of partial Ci depletion on Hh signaling responses.



Supplementary Figure S1 Arm protein levels are increased in $CSN5^{null}$ mutant clones generated in wing discs. $CSN5^{null}$ clones marked by the absence of GFP (green) are generated in wing discs. Arm protein levels (red) are higher in the $CSN5^{null}$ cells (arrows), as compared to wild-type cells. Scale bar represents 50 μ m.



Supplementary Figure S2 CSN regulates Ci^{155} levels by a post-transcriptional mechanism. $CSN5^{null}$ clones marked by the absence of GFP (green) are generated in wing discs expressing the wild-type HA-ci transgene under the control of ms1096-GAL4. The levels of Ci revealed by 2A1 staining (red) are reduced in $CSN5^{null}$ clones (arrows). Scale bar represents 50 μ m.



Supplementary Figure S3 Effects of partial Ci depletion on Hh signaling responses.

(Upper row of panels) By using *ms1096-GAL4*, which has higher expression in dorsal and lower expression in ventral wing pouches, to express *ci-dsRNAi*, the expression levels of *dpp-lacZ* (green) are abolished in dorsal (arrowhead) but remain detectable in ventral region (arrow). The corresponding Ci levels are also revealed by Ci staining (red). (Lower row of panels) *ms1096-GAL4* driven *ci-dsRNAi* has no significant effect on *ptc-lacZ* expression (red) in both dorsal (arrowhead) and ventral (arrow) wing pouch. Scale bars represent 50 μm.