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# Local overexpression of Su(H)-MAPK variants affects Notch target gene expression and adult phenotypes in *Drosophila*

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ABSTRACT

In *Drosophila*, Notch and EGFR signalling pathways are closely intertwined. Their relationship is mostly antagonistic, and may in part be based on the phosphorylation of the Notch signal transducer Suppressor of Hairless [Su(H)] by MAPK. Su(H) is a transcription factor that together with several cofactors regulates the expression of Notch target genes.

Here we address the consequences of a local induction of three *Su(H)* variants on Notch target gene expression. To this end, wild-type *Su(H)*, a phospho-deficient $Su(H)^{MAPK\text{-}ko}$ and a phospho-mimetic $Su(H)^{MAPK\text{-}ac}$ isoform were overexpressed in the central domain of the wing anlagen. The expression of the Notch target genes *cut, wingless, E(spl)m8-HLH* and *vestigial*, was monitored. For the latter two, reporter genes were used (*E(spl)m8-lacZ*, $vg^{BE}$*-lacZ*). In general, $Su(H)^{MAPK\text{-}ko}$ induced a stronger response than wild-type *Su(H)*, whereas the response to $Su(H)^{MAPK\text{-}ac}$ was very weak. Notch target genes *cut, wingless* and $vg^{BE}$*-lacZ* were ectopically activated, whereas *E(spl)m8-lacZ* was repressed by overexpression of Su(H) proteins. In addition, in epistasis experiments an activated form of the EGF-receptor ($DER^{act}$) or the MAPK ($rl^{SEM}$) and individual Su(H) variants were co-overexpressed locally, to compare the resultant phenotypes in adult flies (thorax, wings and eyes) as well as to assay the response of the Notch target gene *cut* in cell clones.



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Specifications Table

| | |
|---|---|
| Subject area | *Biochemistry, Genetics and Molecular Biology* |
| More specific subject area | *Developmental Biology, Cellular signalling* |
| Type of data | *Figures and text* |
| How data was acquired | *Microscopy: Zeiss Axioskop linked to a Bio-Rad MRC1024 confocal microscope; Wild 5M stereomicroscope and Zeiss Axiophot coupled to an Optronics ES120 camera* |
| Data format | *Filtered data, analyzed* |
| Experimental factors | *Imaginal discs were dissected, fixed, washed and blocked before adding antibodies.* |
| Experimental features | *Tissue-specific expression of respective transgenes was induced with the Gal4:: UAS-system. Gene expression was monitored directly or from reporter genes by antibody staining of the protein products.* |
| Data source location | *n.a.* |
| Data accessibility | *The data is with this article* |

Value of the data

- This data shows the responses of several Notch target genes to modulations of Su(H) activity by the EGFR pathway.
- The data allow for a visual comparison of the spectrum of Notch target gene responses to Su (H) overexpression.
- Overexpression of activated components of the EGFR pathway and Su(H) variants, alone or in combination, can be compared in various *Drosophila* tissues.
- This data may be extended by analyses on $DER^{act}$ activity during *Drosophila* wing development.

## 1. Data

Suppressor of Hairless [Su(H)] is the transcription factor that regulates the expression of the target genes of the Notch signalling pathway [1,2]. Su(H) protein may be phosphorylated by MAPK as a result of Epidermal Growth Factor Receptor (EGFR) activation, providing a means of a direct cross-talk between these two pathways [3–5]. The response of several Notch target genes to the modulations of Su(H) by EGFR signalling activity was analysed by the local overexpression of either wild-type *Su(H)*, a phospho-deficient $Su(H)^{MAPK\text{-}ko}$ and a phospho-mimetic $Su(H)^{MAPK\text{-}ac}$ variant [3] using the Gal4::UAS system [6], and staining of the tissues with respective antibodies. Moreover, activated components of the EGFR pathway ($DER^{act}$, $rl^{SEM}$) were overexpressed alone or in combination with individual *Su(H)* variants. The response of the Notch target gene *cut* was observed in cell clones of wing imaginal discs, and the resultant phenotypes on thorax, wings and eyes were recorded in adult flies.

### 1.1. Overexpression of Su(H) variants during wing development

UAS-*Su(H)*, UAS-$Su(H)^{MAPK\text{-}ko}$ and UAS-$Su(H)^{MAPK\text{-}ac}$ were overexpressed with *omb*-Gal4 [7] in wing imaginal discs of third instar *Drosophila* larvae. A total of four Notch target genes was analysed, *wingless* (Fig. 1) [8], *cut* (Fig. 2) [9], *E(spl)m8-HLH* [10] (using *E(spl)m8-lacZ* [11], Fig. 3) and *vestigial* [12] (using $vg^{BE}$*-lacZ* [13], Fig. 4). Overall, overexpression of $Su(H)^{MAPK\text{-}ko}$ caused a stronger response of the Notch target genes than that of wild-type *Su(H)*, whereas $Su(H)^{MAPK\text{-}ac}$ elicited the weakest effects, in agreement with a downregulation of *Su(H)* activity by MAPK-mediated phosphorylation [3].

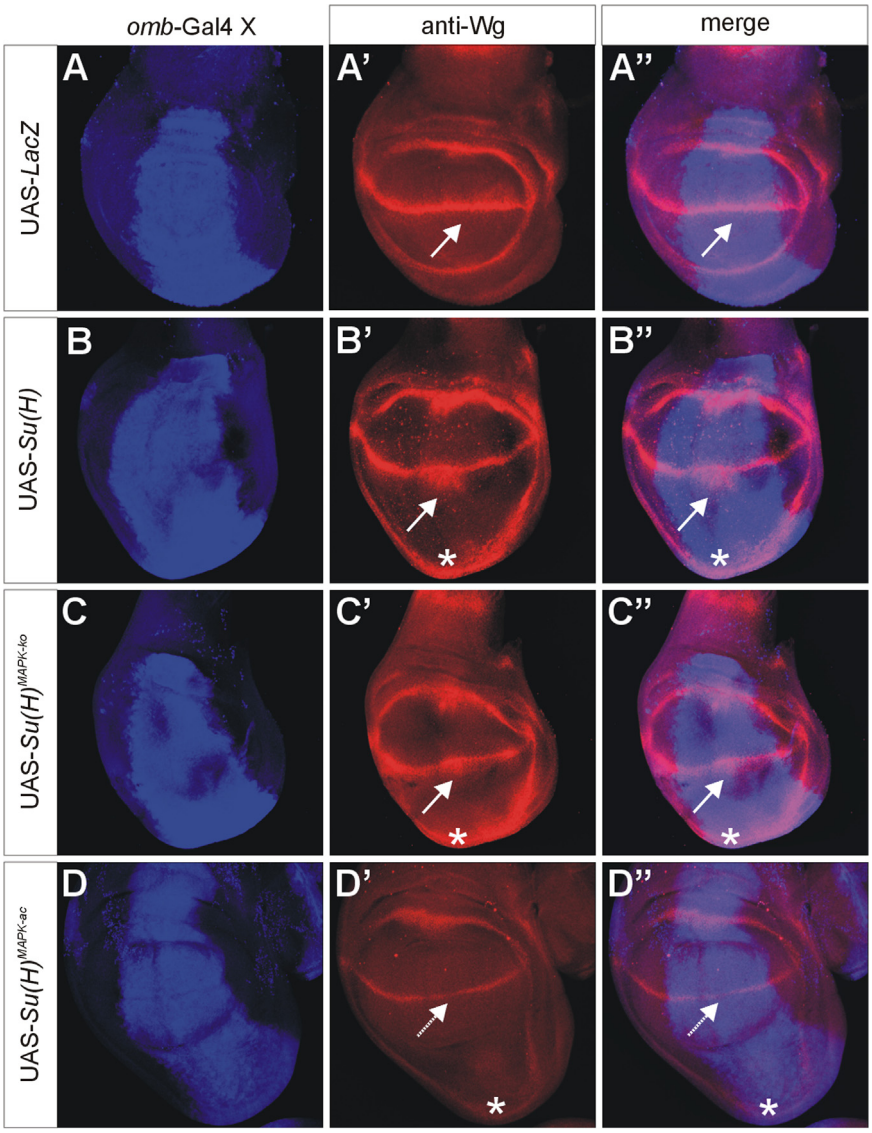


**Fig. 1.** Response of the Notch target gene *wingless*. Overexpression of the UAS-*Su*(*H*) variants as indicated; the *omb*-expression domain is highlighted in blue in A–D and A″–D″ (A,A′ anti-beta galactosidase staining; B–D and B′–D′, anti-Su(H) staining). Expression of *wingless* (Wg) is shown in red (A′–D″). UAS-*lacZ* served as control. Note expansion of *wingless* expression along the dorso-ventral boundary (arrows) upon overexpression of *Su*(*H*) and $Su(H)^{MAPK\text{-}ko}$, but not $Su(H)^{MAPK\text{-}ac}$. Overgrowth of the ventral disc is marked by asterisks and is a consequence of the overexpression of Su(H) protein (B′–D″).

### *1.2. Response of cut expression to the combined induction of Su(H) variants and activated components of the EGFR pathway during wing development*

The expression of the Notch target gene *cut* was analysed in cell clones overexpressing either of the three Su(H) isoforms alone or in combination with the activated EGF-receptor ($DER^{act}$) or the activated MAPK ($rl^{SEM}$) [14,15] (Fig. 5). Overexpression clones were induced in wing imaginal discs [16]. *Su*(*H*) overexpression induced *cut* expression, whereas it repressed it when simultaneously

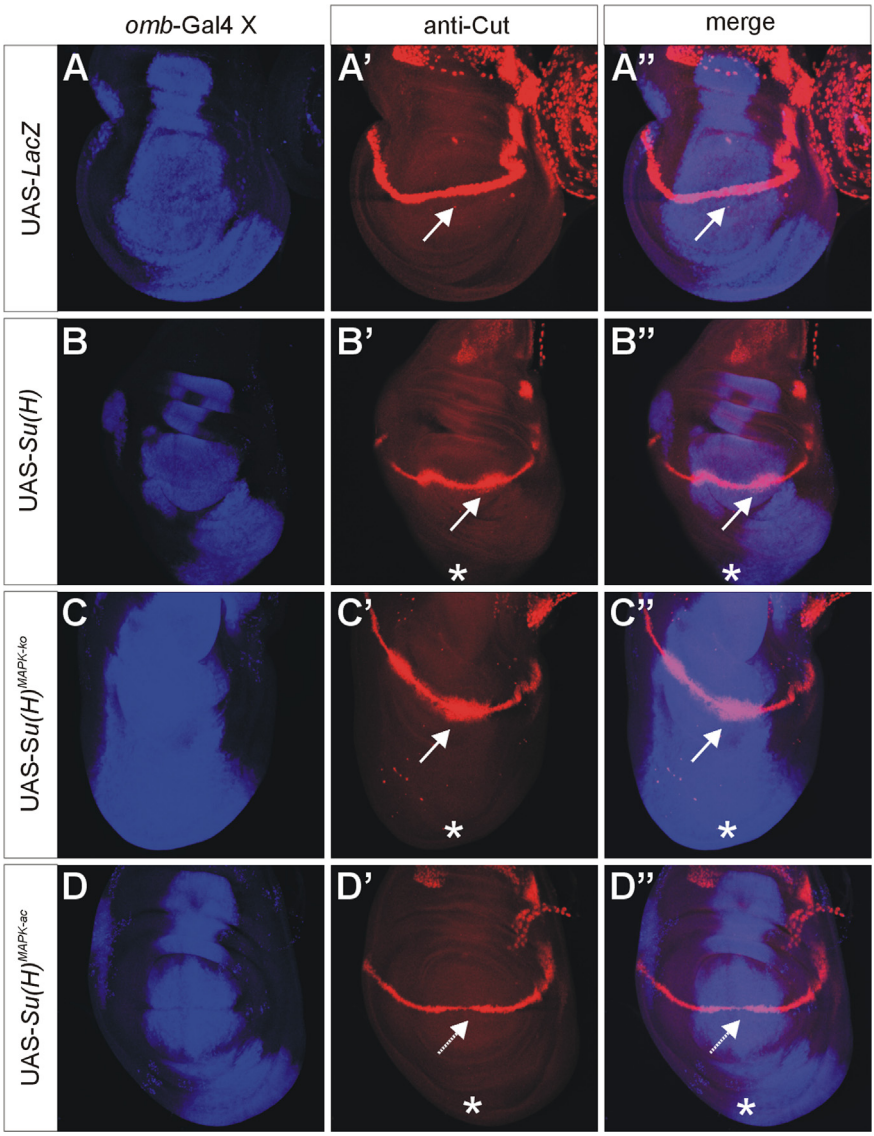


**Fig. 2.** Response of the Notch target gene *cut*. Overexpression of the UAS-*Su*(*H*) variants as indicated; the *omb*-expression domain is highlighted in blue in A–D and A″–D″ (A,A′ anti-beta galactosidase staining; B–D and B′–D′, anti-Su(H) staining). Expression of *cut* is shown in red (A′–D″). UAS-*lacZ* served as control. Note expansion of *cut* expression along the dorso-ventral boundary (arrows) upon overexpression of *Su*(*H*) and $Su(H)^{MAPK\text{-}ko}$, but not $Su(H)^{MAPK\text{-}ac}$. Overgrowth of the ventral disc is marked by asterisks and is a consequence of the overexpression of Su(H) protein (B′–D″).

overexpressed with $rl^{SEM}$ (Fig. 5A–A‴ and C–C‴) [3]. Likewise repression was observed with *Su*(*H*)$^{MAPK\text{-}ko}$, but less with $Su(H)^{MAPK\text{-}ac}$ (Fig. 5D and E‴). Cell clones overexpressing $DER^{act}$ were frequently distorted, and *cut* expression was induced at the boundary of $DER^{act}$ expressing and non-expressing cells independent of the overexpression of any *Su*(*H*) variant (arrowheads in Fig. 5F′–I‴).

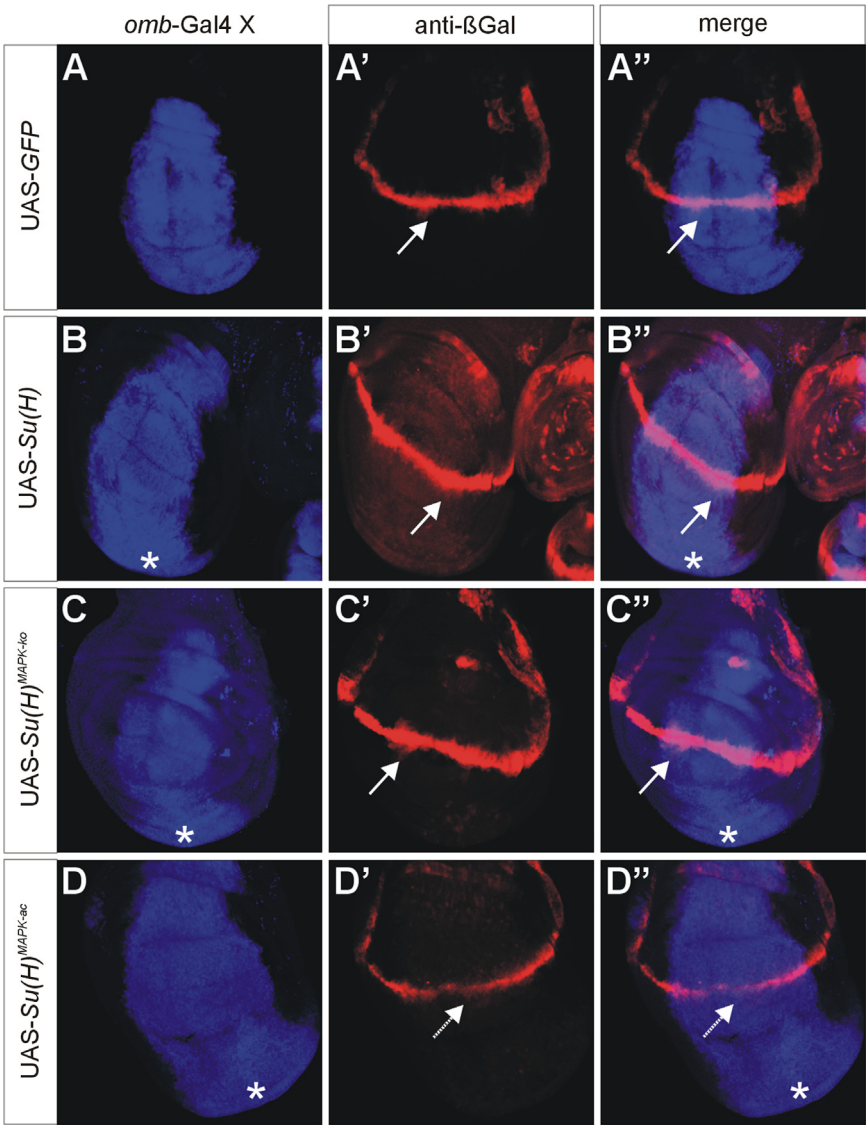


**Fig. 3.** Response of the Notch target gene *vestigial*. Overexpression of the UAS-*Su*(*H*) variants as indicated; the *omb*-expression domain is highlighted in blue in A–D and A″–D″ (A,A′ green fluorescent protein GFP; B–D and B′–D′, anti-Su(H) staining). Expression of the *vestigial* reporter $vg^{BE}$-*lacZ* is shown in red (A′–D″). UAS-*GFP* served as control. Note expansion of $vg^{BE}$-*lacZ* expression along the dorso-ventral boundary (arrows) upon overexpression of *Su*(*H*) and $Su(H)^{MAPK\text{-}ko}$, but not $Su(H)^{MAPK\text{-}ac}$. Overgrowth of the ventral disc is marked by asterisks and is a consequence of the overexpression of Su(H) protein (B–D, B″–D″).

### *1.3. Adult phenotypes resulting from the combined overexpression of Su(H) variants and activated components of the EGFR pathway*

Overexpression of UAS-$DER^{act}$ in the thorax (Fig. 6) or the wing anlagen (Fig. 7A) using *Bx*-Gal4 [17] was fully epistatic to the *Su*(*H*) gain of function phenotypes. This was in contrast to the simultaneous overexpression of UAS-$rl^{SEM}$ with the UAS-*Su*(*H*) isoforms: in these experiments the *Su*(*H*) gain of function phenotypes prevailed (Figs. 6 and 7B). It has been described before that the overexpression of *Su*(*H*) in the developing sensory organs using *sca*-Gal4 causes a shaft to socket transformation [18],

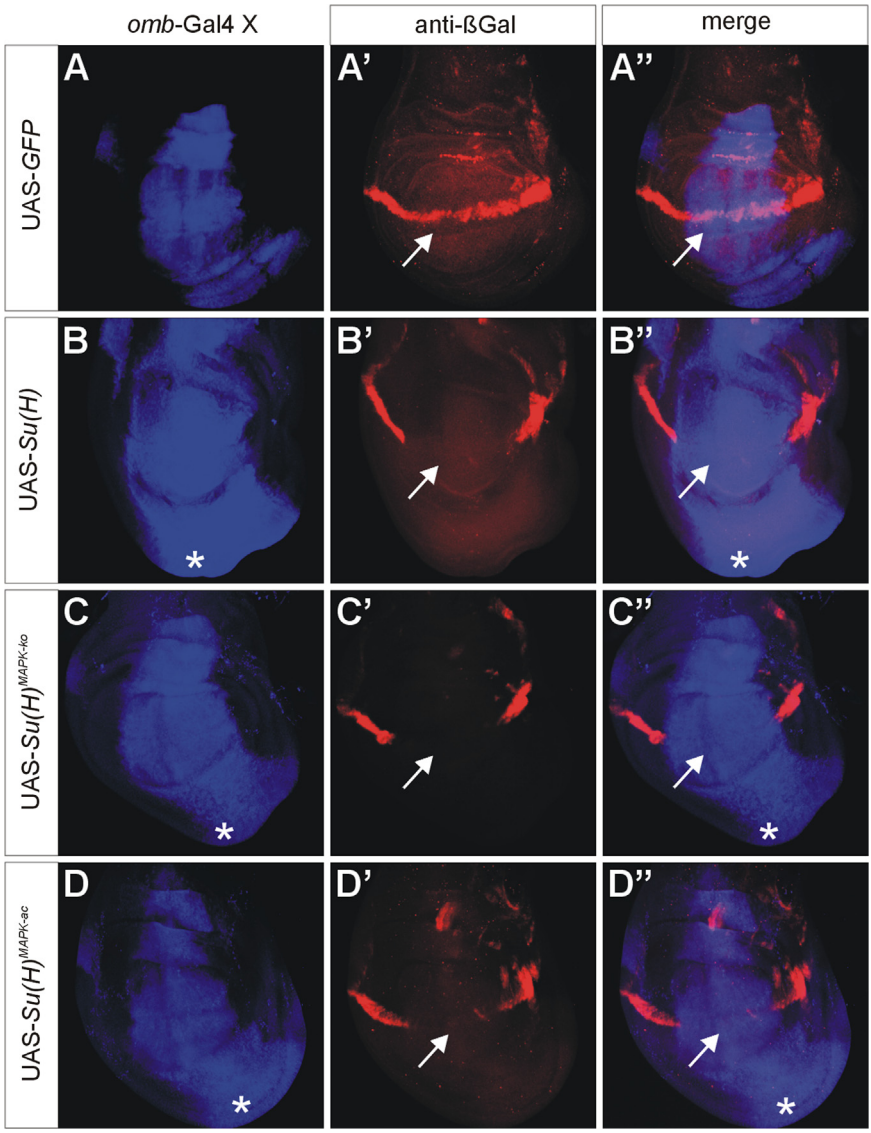


**Fig. 4.** Response of the Notch target gene *E(spl)m8-HLH*. Overexpression of the UAS-*Su(H)* variants as indicated; the *omb*-expression domain is highlighted in blue in A–D and A″–D″ (A,A′ green fluorescent protein GFP; B–D and B′–D′, anti-Su (H) staining). Expression of the *E(spl)m8-HLH* reporter *E(spl)m8-lacZ* is shown in red (A′–D″). UAS-*GFP* served as control. Note repression of *E(spl)m8-lacZ* along the dorso-ventral boundary (arrows) upon overexpression of the three *Su(H)* variants (B′–D″); overgrowth of the ventral disc is marked by asterisks (B–D, B″–D″).

which we also observed upon overexpression of $Su(H)^{MAPK\text{-}ko}$ or $Su(H)^{MAPK\text{-}ac}$ (Fig. 8). Whereas *sca*:: $rl^{SEM}$ was similar to the control, *sca*:: $DER^{act}$ animals developed tufts of macrochaetae on the posterior thorax (Fig. 8). Interestingly, in combination with any of the *Su(H)* variants, the double socket phenotype resulting from *Su(H)* overexpression prevailed (Fig. 8). Finally, consequences of *Su(H)* overexpression in the developing eye using *gmr*-Gal4 were addressed (Fig. 9). As the Gal4::UAS system is temperature sensitive, phenotypes were strong at 29 °C, revealing defects in the control as well [19]. At this temperature, *Su(H)* overexpression caused overgrowth of the eye, irregular facets and necrosis. At 25 °C the phenotypes were much weaker resembling the control. A combination with $rl^{SEM}$

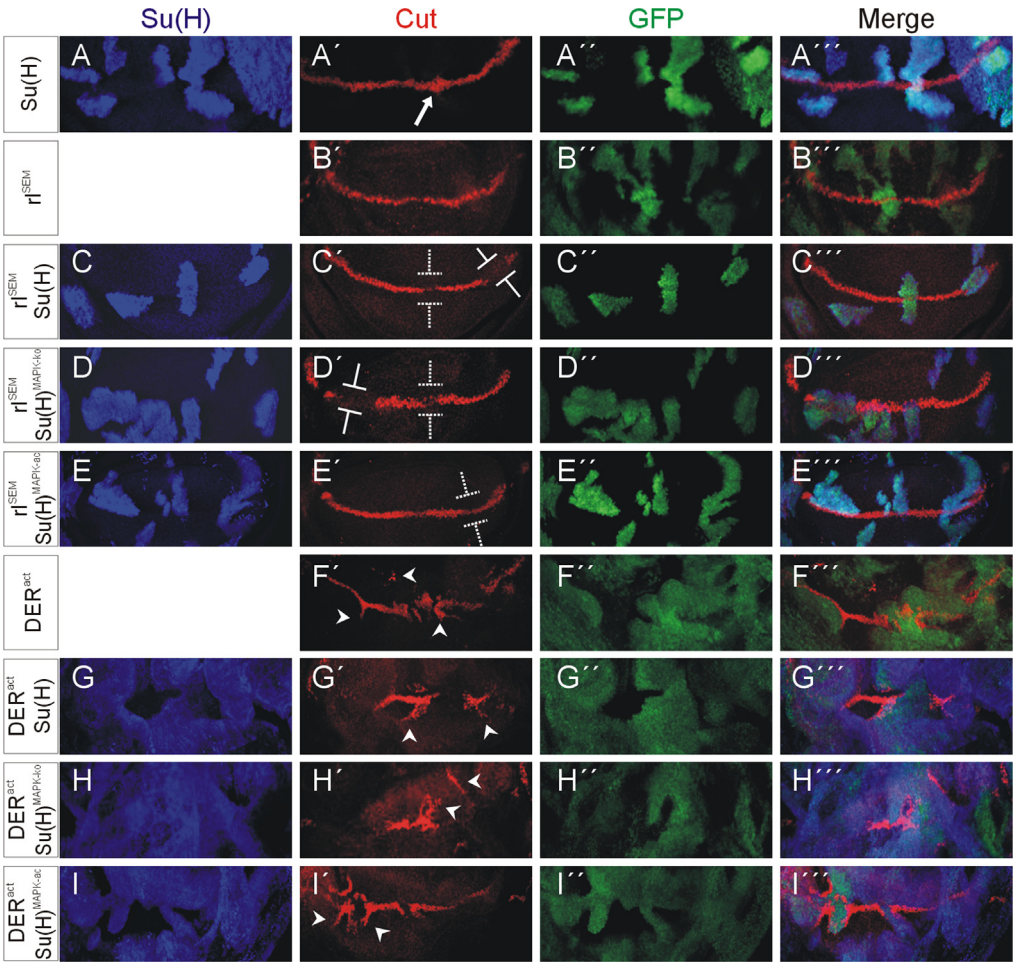


**Fig. 5.** Expression of *cut* in response to *Su(H)*, $DER^{act}$ and $rl^{SEM}$ overexpression. Overexpression clones were induced in wing imaginal discs. They are labelled by the presence of GFP (green in A″–I″). Ectopic Su(H) protein is labelled in blue (A–I, A‴–I‴), and *cut* expression is shown in red (A′–I′ and A‴–I‴). Constructs indicated at the left were under UAS-control. Note induction of *cut* upon overexpression of *Su(H)* (arrow in A′), but repression of *cut* by simultaneous overexpression of $rl^{SEM}$ (C′) labelled with blunt arrows. Likewise repression was seen in the combination with $Su(H)^{MAPK\text{-}ko}$ (D′) but not or weakly in combination with $Su(H)^{MAPK\text{-}ac}$ (E'). $DER^{act}$ overexpression clones were frequently distorted and induced *cut* expression along the boundary to the non-overexpressing cells (arrowheads in F′–I′).

enhanced the irregular facet and necrotic phenotype, whilst *gmr*::$rl^{SEM}$ flies were very similar to the control (Fig. 9).

## 2. Experimental design, materials and methods

### 2.1. Fly stocks, husbandry and analyses

Flies were obtained from the Bloomington stock collection if not noted otherwise. Fly husbandry was according to standard protocols at 29 °C, 25 °C or 18 °C as noted. $y^1$ $w^{1118}$, UAS-*lacZ* and UAS-*GFP* served as control. For information on fly stocks we refer to http://flybase.bio.indiana.edu. Adult wings of female flies were dehydrated in ethanol and mounted in Euparal (Roth, Karlsruhe, Germany) and

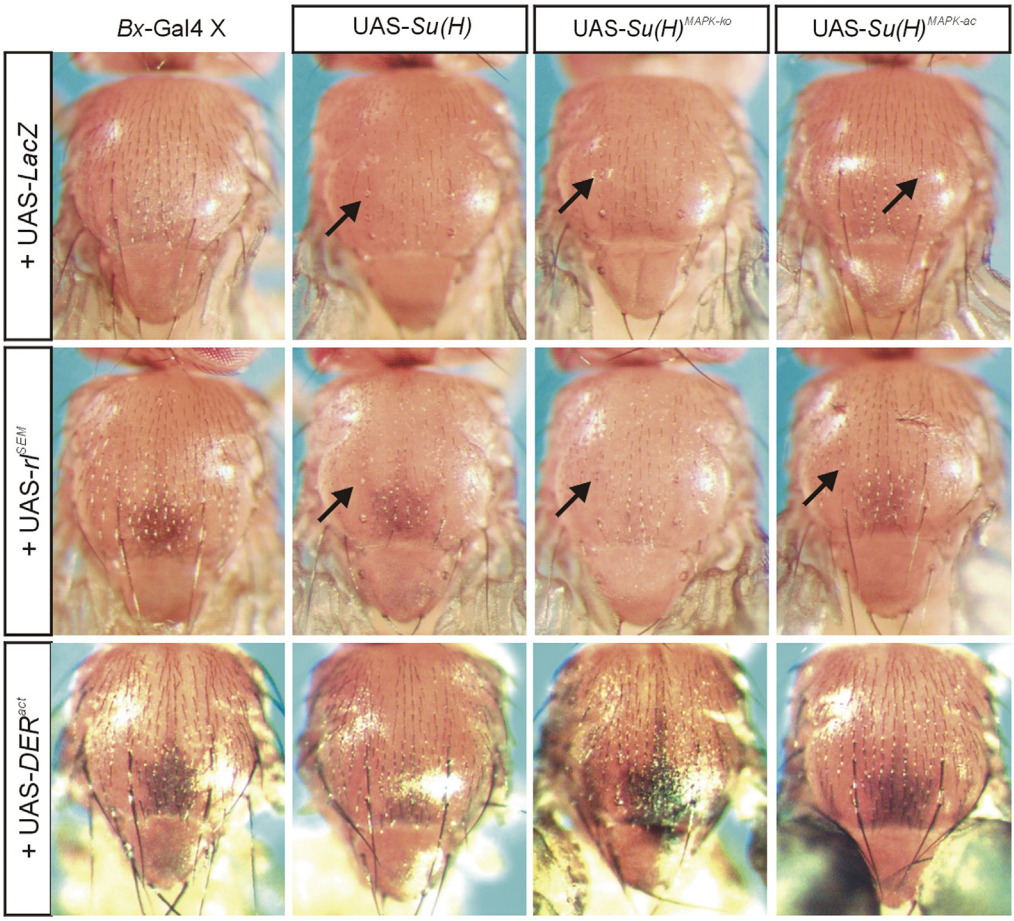


**Fig. 6.** Overexpression consequences of *Su*(*H*), $DER^{act}$ and $rl^{SEM}$ during thorax development. Co-overexpression of UAS-*Su*(*H*) variants together with UAS-*lacZ* (control), UAS-$rl^{SEM}$ or UAS-$DER^{act}$ was driven in the developing thorax using *Bx*-Gal4 at 18 °C. Arrows point to examples of shaft to socket transformations that affected the majority of macrochaetae when UAS-*Su*(*H*) or UAS-$Su(H)^{MAPK\text{-}ko}$ were overexpressed, but were rarely observed upon UAS-$Su(H)^{MAPK\text{-}ac}$ ectopic expression. Simultaneous overexpression of UAS-$rl^{SEM}$ had little influence on each of these specific phenotypes. In contrast UAS-$DER^{act}$ phenotypes were epistatic to the overexpression of any the respective Su(H) constructs, i.e. all the resultant flies resembled those of the single $DER^{act}$ overexpression. Typical representatives are shown in each case.

dried over night. Pictures of wings or adult flies were taken on a Zeiss Axiophot or a Wild 5M stereomicroscope, respectively, using an ES120 camera (Optronics, Goleta CA, USA) and Pixera Viewfinder software, version 2.0.

Generation of UAS-*Su*(*H*), UAS-$Su(H)^{MAPK\text{-}ko}$ (T426A) and UAS-$Su(H)^{MAPK\text{-}ac}$ (T426E) was described earlier [3,20]. UAS-$rl^{SEM}$ was provided by Martín-Blanco [15] and UAS-$DER^{act}$ by Freeman [14]. LacZ-reporter gene constructs $vg^{BE}$-*lacZ* and *E*(*spl*)*m8-lacZ* were kindly provided by Bray and Schweisguth [11,13]. Tissue-specific expression of respective transgenes was induced with the Gal4:: UAS-system [6] using *omb*-Gal4 [7], *Bx*-Gal4 [17], *sca*-Gal4 [21] and *gmr*-Gal4 [19]. Overexpression clones were induced by the flip-out technique [16] with the following fly lines: *y w* $flp^{1.22}$; UAS-*Su*(*H*) or UAS-*Su*(*H*) mutants, *y w* $flp^{1.22}$; UAS-$rl^{SEM}$ and *y w* $flp^{1.22}$; UAS-$rl^{SEM}$ UAS-*Su*(*H*); UAS-$DER^{act}$ and *y w* $flp^{1.22}$; UAS-$DER^{act}$ UAS-*Su*(*H*) or UAS-*Su*(*H*) mutants and *y w* Act > CD2 > Gal4, UAS-*GFP*-nls (kindly provided by K. Basler).

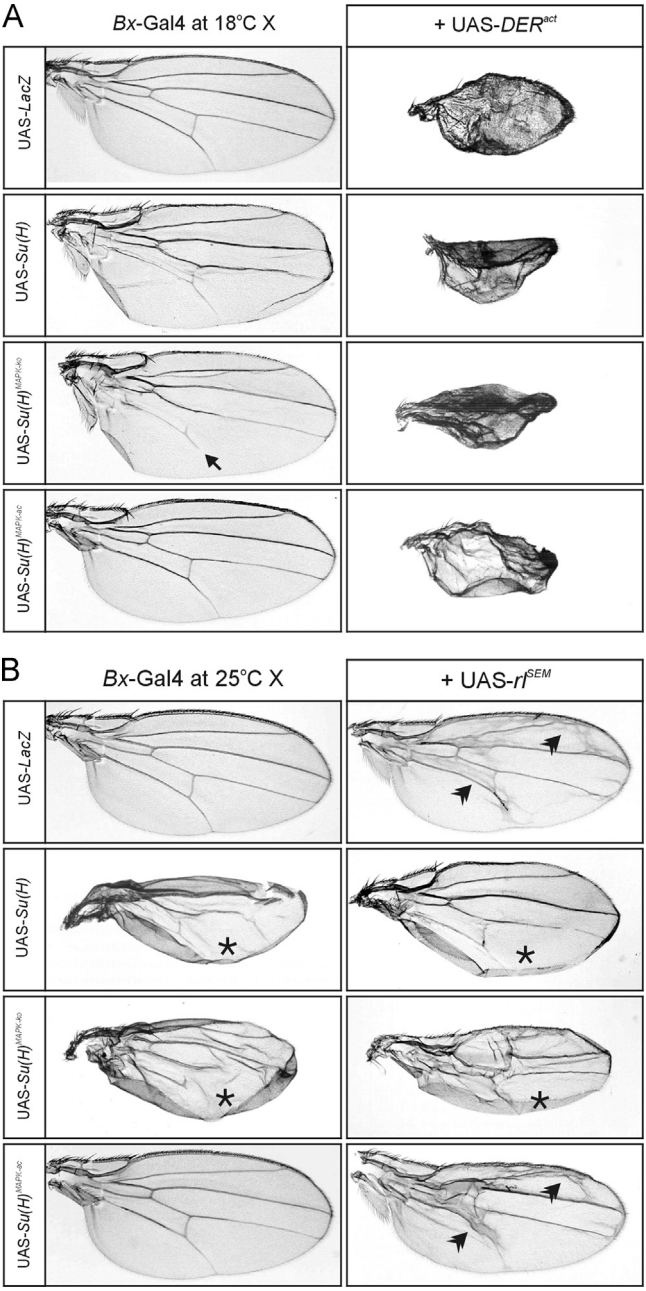


**Fig. 7.** Overexpression consequences of *Su*(*H*), *DER*$^{act}$ and *rl*$^{SEM}$ during wing development. Co-overexpression of UAS-*Su*(*H*) variants together with UAS-*lacZ* (control), UAS-*DER*$^{act}$ (at 18 °C) (A) or UAS-*rl*$^{SEM}$ (at 25 °C) (B) was driven in the developing wing using *Bx*-Gal4. (A) At 18 °C, *Su*(*H*)$^{MAPK\text{-}ko}$ repressed vein formation (arrow) which was not observed for either *Su*(*H*) or *Su*(*H*)$^{MAPK\text{-}ac}$. Induction of UAS-*DER*$^{act}$ resulted in very small wings mainly consisting of vein material, which was independent of *Su*(*H*) overexpression. As a consequence, the wings resulting from the combined overexpression were indistinguishable from those of the single *DER*$^{act}$ overexpression. (B) At 25 °C, overexpression of either *Su*(*H*) or *Su*(*H*)$^{MAPK\text{-}ko}$ but not *Su*(*H*)$^{MAPK\text{-}ac}$ induced tissue overgrowth typified by wing blisters (asterisks). Induction of UAS-*rl*$^{SEM}$ caused a network of veins (double arrowheads) which was repressed by the presence of ectopic *Su*(*H*) or *Su*(*H*)$^{MAPK\text{-}ko}$ but not by *Su*(*H*)$^{MAPK\text{-}ac}$. At the same time *Su*(*H*) and *Su*(*H*)$^{MAPK\text{-}ko}$ gain of function phenotypes prevailed. Typical representatives are shown in each case.

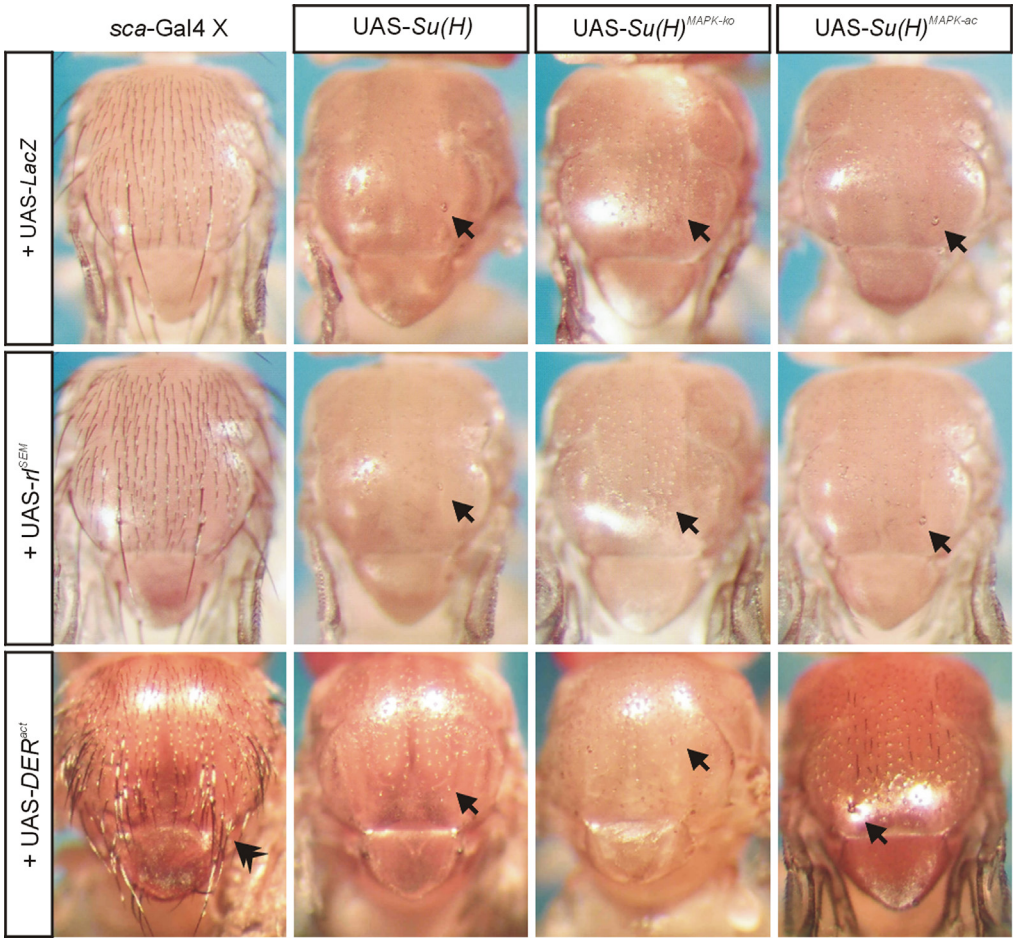


**Fig. 8.** Overexpression consequences of *Su*(*H*), $DER^{act}$ and $rl^{SEM}$ in the developing bristle organs. Co-overexpression of UAS-*Su*(*H*) variants together with UAS-*lacZ* (control), UAS-$rl^{SEM}$ or UAS-$DER^{act}$ was driven in the developing bristle organs using *sca*-Gal4 at 25 °C. Overexpression of any of the *Su*(*H*) variants within the developing bristle organ caused a near complete transformation of bristle shafts to sockets of micro- or macrochaetae. Examples of the resultant double sockets are highlighted by arrows. The phenotypes were nearly indistinguishable between the three Su(H) variants. Whereas flies overexpressing of *sca*::$rl^{SEM}$ matched the control phenotype, *sca*:: $DER^{act}$ developed tufts of macrochaetae on the posterior thorax (double arrowhead). Each of these phenotypes disappeared completely in a combination with any *Su*(*H*) variant. Typical representatives are shown in each case.

### 2.2. Immunohistochemistry

Imaginal discs were stained according to standard protocols using mouse monoclonal antibodies directed against Cut, Wingless or beta-Galactosidase (developed by G.M. Rubin, S.M. Cohen, and J.R. Sanes respectively, and obtained from DSHB or using a polyclonal antiserum directed against Su(H)) [22]. Secondary antibodies coupled to FITC, Cy3 or Cy5 (1:200) were obtained from Jackson Immuno-Research Laboratories (Dianova, Hamburg, Germany). Samples were mounted in Vectashield (Vector Lab) and examined on a Zeiss Axioskop coupled to a BioRad MRC1024 confocal microscope using LaserSharp 2000TM software (Carl Zeiss, Jena, Germany).

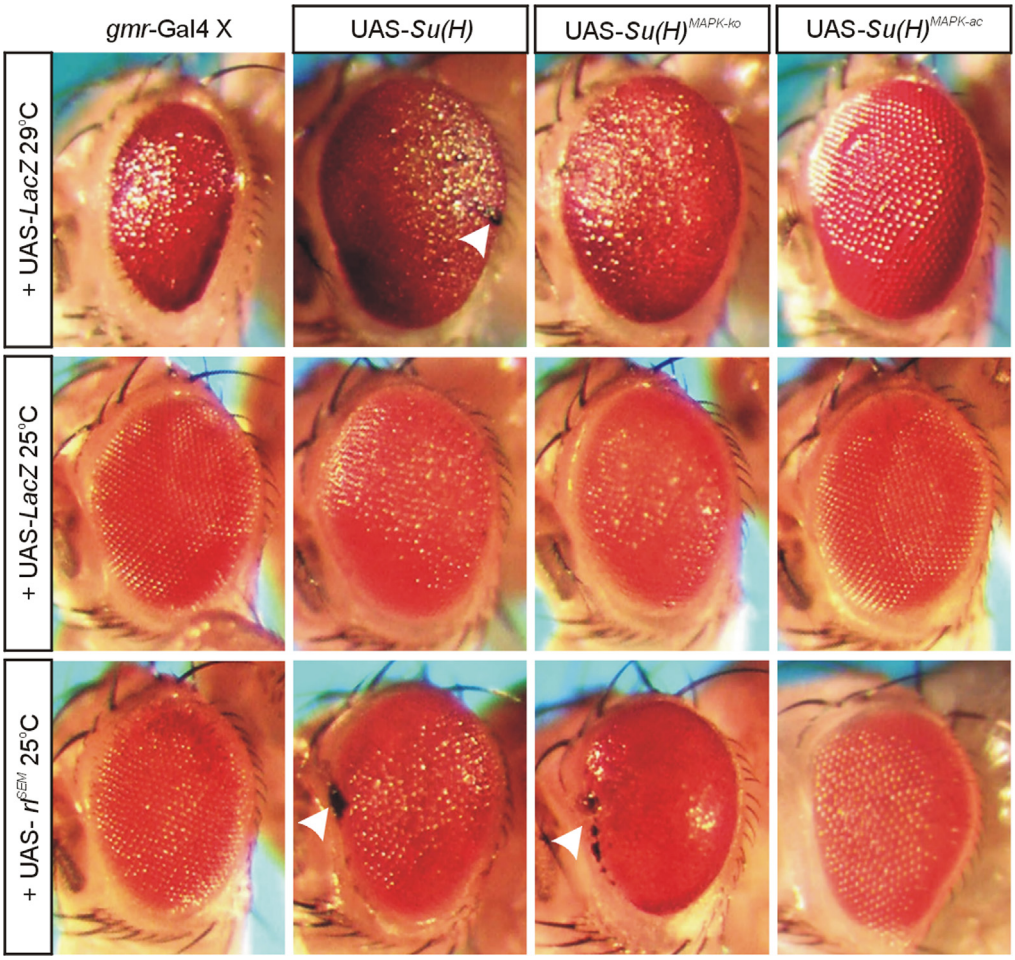


**Fig. 9.** Overexpression consequences of *Su*(*H*), *DER*$^{act}$ and *rl*$^{SEM}$ in the developing eye. Co-overexpression of UAS-*Su*(*H*) variants together with UAS-lacZ (control) or UAS-*rl*$^{SEM}$ was driven in the developing eye using *gmr*-Gal4. At 29 °C, *gmr*::lacZ flies have smaller eyes with irregular facets giving the eye a rough appearance. In contrast, overexpression of *Su*(*H*) variants at this temperature causes enlarged eyes that appear slightly bulgy. Both *Su*(*H*) and *Su*(*H*)$^{MAPK\text{-}ko}$ induced irregularities in the arrangement of the facets and necrosis (arrowhead), in contrast to *Su*(*H*)$^{MAPK\text{-}ac}$. At 25 °C the phenotypes are much milder, and eyes appear like wild type (*Su*(*H*)$^{MAPK\text{-}ac}$) or slightly rough (*Su*(*H*) and *Su*(*H*)$^{MAPK\text{-}ko}$). A similar rough eye phenotype was observed upon induction of *rl*$^{SEM}$ at 25 °C. The combined overexpression of *Su*(*H*) and *rl*$^{SEM}$ gave a mixed phenotype, i.e. eyes were smaller, rough and necrotic (arrowhead). Similar necrotic patches (arrowhead) and size decrease were also observed in the eyes of *gmr*:: *Su*(*H*)$^{MAPK\text{-}ko}$+*rl*$^{SEM}$ animals, which in addition had a glossy appearance. In contrast, the eyes of the *gmr*:: *Su*(*H*)$^{MAPK\text{-}ac}$+*rl*$^{SEM}$ animals looked similar to *gmr*:: *rl*$^{SEM}$. Typical representatives are shown in each case.

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## Appendix A. Supplementary material

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.dib.2015.11.004.

## References

[1] S.J. Bray, Notch signalling: a simple pathway becomes complex, Nat. Rev. Mol. Cell Biol. 7 (2006) 678–689.
[2] R. Kopan, M.X. Ilagan, The canonical Notch signaling pathway: unfolding the activation mechanism, Cell 137 (2009) 216–233.
[3] J.S. Auer, A.C. Nagel, A. Schulz, V. Wahl, A. Preiss, MAPK-dependent phosphorylation modulates the activity of suppressor of hairless in *Drosophila*, Cell. Signal. 27 (2015) 115–124.
[4] M.V. Sundaram, The love–hate relationships between Ras and Notch, Genes Dev. 19 (2005) 1825–1839.
[5] D.B. Doroquez, I. Rebay, Signal integration during development: mechanisms of EGFR and Notch pathway function and cross-talk, Crit. Rev. Biochem. Mol. Biol. 41 (2006) 339–385.
[6] A.H. Brand, N. Perrimon, Targeted gene expression as a means of altering cell fates and generating dominant phenotypes, Development 118 (1993) 401–415.
[7] T. Lecuit, W.J. Brook, M. Ng, M. Calleja, H. Sun, S.M. Cohen, Two distinct mechanisms for long-range patterning by *decapentaplegic* in the *Drosophila* wing, Nature 381 (1996) 387–393.
[8] F.J. Diaz-Benjumea, S.M. Cohen, Serrate signals through Notch to establish a Wingless-dependent organizer at the dorsal/ventral compartment boundary of the *Drosophila* wing, Development 121 (1995) 4215–4225.
[9] C.A. Micchelli, E.J. Rulifson, S.S. Blair, The function and regulation of *cut* expression of the wing margin of *Drosophila*: Notch, Wingless and a dominant negative role for Delta and Serrate, Development 124 (1997) 1485–1495.
[10] J.F. de Celis, J. de Celis, P. Ligoxygakis, A. Preiss, C. Delidakis, S. Bray, Functional relationships between *Notch*, *Su*(*H*) and the bHLH genes of the *E*(*spl*) complex: the *E*(*spl*) genes mediate only a subset of Notch activities during imaginal development, Development 122 (1996) 2719–2728.
[11] B. Kramatschek, J.A. Campos-Ortega, Neuroectodermal transcription of the *Drosophila* neurogenic genes *E*(*spl*) and *HLH-m5* is regulated by proneural genes, Development 120 (1994) 815–826.
[12] J.P. Couso, E. Knust, A. Martinez-Arias, *Serrate* and *wingless* cooperate to induce *vestigial* gene expression and wing formation in *Drosophila*, Curr. Biol. 5 (1995) 1437–1448.
[13] J. Kim, A. Sebring, J.J. Esch, M.E. Kraus, K. Vorwerk, J. Magee, S.B. Carroll, Integration of positional signals and regulation of wing formation and identity by *Drosophila vestigial* gene, Nature 382 (1996) 133–138.
[14] M. Freeman, Reiterative use of the EGF receptor triggers differentiation of all cell types in the *Drosophila* eye, Cell 87 (1996) 651–660.
[15] E. Martín-Blanco, Regulatory control of signal transduction during morphogenesis in *Drosophila*, Int. J. Dev. Biol. 42 (1998) 363–368.
[16] G. Struhl, K. Basler, Organizing activity of wingless protein in *Drosophila*, Cell 72 (1993) 527–540.
[17] J. Capdevila, I. Guerrero, Targeted expression of the signaling molecule decapentaplegic induces pattern duplications and growth alterations in *Drosophila* wings, EMBO J. 13 (1994) 4459–4468.
[18] A.C. Nagel, D. Maier, A. Preiss, Su(H)-independent activity of Hairless during mechano-sensory organ formation in *Drosophila*, Mech. Dev. 94 (2000) 3–12.
[19] B.A. Hay, R. Maile, G.M. Rubin, P-element mediated insertion-dependent gene activation in the *Drosophila* eye, Proc. Natl. Acad. Sci. USA 94 (1997) 5195–5200.
[20] A.C. Nagel, A. Krejci, G. Tenin, A. Bravo-Patiño, S. Bray, D. Maier, A. Preiss, Hairless-mediated repression of Notch target genes requires the combined activity of Groucho and CtBP corepressors, Mol. Cell. Biol. 25 (2005) 10433–10441.
[21] U. Hinz, B. Giebel, J.A. Campos-Ortega, The basic-helix-loop-helix domain of *Drosophila* lethal of scute protein is sufficient for proneural function and activates neurogenic genes, Cell 76 (1994) 77–87.
[22] D. Maier, H. Praxenthaler, A. Schulz, A. Preiss, Gain of function Notch phenotypes associated with the ectopic expression of the Su(H) C-terminal domain illustrate separability of Notch and Hairless-mediated activities, PLoS One 8 (11) (2013) e81578.