Figure S1.—Genetic and molecular approaches used to map the hlfi mutation. (Top) Genetic map of hlfi relative to the dominant alleles Lobe (L), Black cells (Bc) and Sternopleural (Sp or wg^Sp1). (Middle) Complementation analysis of hlfi with a series of chromosomal deficiencies covering the polytenic region between the 48 division and the centromere of the second chromosome. Grey bars represent the complementing deficiencies and the black bar indicates the non-complementing Df(2R)ST1 deficiency. (Bottom) Molecular map of the putative interval between the distal breakpoint of Df(2R)nap1 and the proximal breakpoint of the Df(2R)Drlrv17. The black triangles on molecular map represent the positions of three of the five P-element insertions used to generate local deficiencies by P-mediated imprecise excision experiments. To obtain excision events, disgenic P/CyO; P(Δ2-3), Sb/+ flies (in y^566 or a^2118 genetic background, depending on the P dominant marker) were crossed with a w^1118; CyO/Sco or y^1118; CyO/Sco; y^566 mapping strain and, among the non-Sb progeny, single Cy non Sco flies, lacking the P dominant marker (excision event), were recovered and crossed again with the mapping strain to obtain a stock. About 40 excision events per P-element were recovered and each tested for their ability to complement hlfi mutation. Only one non-complementing excision event (obtained with the P(EP)Vha16^EP2372) was recovered and named hlfi^17. Molecular mapping of hlfi^17 breakpoints (see material and methods) allowed to determine its extension (represented at the bottom of the figure). The lines below the molecular map represent the genomic fragments used for transgene-mediated rescue of hlfi mutants (see material and methods). Black lines represent the non-rescuing fragments; the grey line indicates the rescuing fragment containing the bap170 gene.