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We have examined the pattern of abundant protein synthesis in various homeotic imaginal discs. The mutants used were: bithorax (bx^3), which replaces anterior haltere structures with anterior wing; postbithorax (pbx), transforming posterior haltere to posterior wing;

and engrailed (en^1), in which posterior wing structures take on characteristics of anterior wing. Mutant stocks used were bx^3/bx^3 , red $pbx/TM9$, and $cn\ en^1/CyO, 1(2)513^{DTS}$. All mutations are listed in Lindsley & Grell (1968) with the exception of $TM9$, a balancer third chromosome containing a dominant temperature-sensitive lethal. Homozygous $cn\ en^1$ larvae were obtained by crossing the balanced stock inter se and pulsing the embryos and early larvae for two days at $30^\circ C$. Because the engrailed phenotype is not expressed strongly at $30^\circ C$ (Lawrence & Morata 1976), larvae were raised at $25^\circ C$ following the $30^\circ C$ pulse. Although non-engrailed larvae survived with this protocol, they developed more slowly than $cn\ en^1$ wing disc morphology differed subtly, but nonetheless perceptibly, from wild-type. Discs were dissected, labelled with ^{35}S -methionine, and their proteins run on 2D gels as described in Greenberg & Adler (1982).

Neither bx^3 , nor pbx haltere discs differed in their patterns of abundant protein synthesis from bx^3 or pbx wing discs. Since we also find no reproducible differences between wild type wing and haltere discs, it stands to reason that these two mutations do not alter the wild type patterns of abundant protein synthesis.

Wild type wing and haltere discs synthesize a protein (RG38) with a nonuniform, homologous spatial distribution (Greenberg & Adler 1982). Thus, RG38 is synthesized preferentially in presumptive anterior, dorsal, and proximal regions of the wing disc and in anterior, dorsal haltere disc. Because bx^3 and pbx change the size of particular disc regions (i.e., a bx^3 anterior haltere disc is larger than wild type), these mutations might also be expected to result in a concomitant alteration in the level of RG38 synthesis. We have, however, observed no such result.

Since en^1 directly affects the pattern of wing disc derivatives, it might also be expected to alter the pattern of RG38 synthesis. Wing discs from $cn\ en^1$ larvae were labeled as usual and cut into anterior and posterior fragments. Examination of 2D PAGE protein patterns revealed no difference from wild type. Thus, it appears that, although en^1 causes major changes in fly morphology, the mutation does not affect pattern as assayed by RG38 level of synthesis. A trivial explanation for this result may reside in the fact that only more distal derivatives of the wing exhibit the engrailed phenotype. Since only low levels of RG38 synthesis can be found in the presumptive distal region of wild type wing discs, an anterior/posterior transformation there would be swamped by the much larger amounts of RG38 in other wing disc regions; the change would be undetectable.

We thank Margaret MacQueen for expert technical assistance. Dr. T.R.F. Wright & Dr. Michael Russell generously provided *Drosophila* stocks. RMG was supported by NIH training grant #HD07192. PNA was supported by a research career development award (NIH KHD00361), a grant from NIH (HD11763), and a grant from NSF (PCM 8203205).

References: Greenberg, R.M. & P.N. Adler 1982, *Devel. Biol.* 89:273-286; Lawrence, P.A. & G. Morata 1976, *Devel. Biol.* 50:321-337; Lindsley, D.L. & E.H. Grell 1968, *Genetic Variations of Drosophila melanogaster*, Carnegie Institute of Washington Publ. #627; Marsh, J.L. 1978, *DIS* 53:155-156.

