Ectopic expression of homeotic genes caused by the elimination of the Polycamb gene in Drosophila imaginal epidermis

ANA BUSTURIA and GINES MORATA
Centro de Biología Molecular, Universidad Autónoma de Madrid, Canto Blanco, Madrid 28049, Spain

Summary

The morphological patterns in the adult cuticle of Drosophila are determined principally by the homeotic genes of the bithorax and Antennapedia complexes. We find that many of these genes become indiscriminately active in the adult epidermis when the Pc gene is eliminated. By using the Pc2 mutation and various BX-C mutant combinations, we have generated clones of imaginal cells possessing different combinations of active homeotic genes.

We find that, in the absence of BX-C genes, Pc- clones develop prothoracic patterns; this is probably due to the activity of Sex combs reduced which overrules Antennapedia. Adding contributions of Ultrabithorax, abdominal-A and Abdominal-B results in thoracic or abdominal patterns. We have established a hierarchical order among these genes: Antp < Scr < Ubx < abd-A < Abd-B. In addition, we show that the engrailed gene is ectopically active in Pc- imaginal cells.

Key words: homeotic genes, adult patterns, Polycamb, Drosophila epidermis, bithorax complex, Antennapedia complex, BX-C.

Introduction

The body of the adult Drosophila consists of a number of cephalic, thoracic and abdominal segments. Each segment consists of two compartments, one anterior and one posterior, which are independent lineage blocks (García-Bellido et al. 1976; Lawrence & Morata, 1977) set aside in early development. The characteristic morphological pattern of each segment constitutes the 'identity' of the segment and is determined by the activity of the homeotic genes, notably those of the bithorax (BX-C) and Antennapedia (ANT-C) complexes (Lewis, 1978; Sánchez-Herrero et al. 1985; Lewis et al. 1980; Kaufman et al. 1980).

Because many lethal homeotic mutants are able to secrete larval cuticle, the role of homeotic genes in specifying segment identity is better understood in the larva than in adult (Struhl, 1983). Trans-acting genes like extra sex combs (esc) (Struhl, 1981, 1983) or Polycamb (Pc) (Sato & Denell, 1985) have proved useful in understanding the morphogenetic role of homeotic genes. Mutations in these genes give rise to an indiscriminate and full activity of BX-C and some ANT-C genes, therefore esc- or Pc- embryos develop into a chain of metameric units each showing similar or identical pattern. As elegantly shown by Struhl (1981, 1983), the identity of the metameres in esc- embryos depends upon the combination of active BX-C and ANT-C genes present. Similar observations have been made with Pc- embryos (Sato & Denell, 1985; Casanova et al. 1986).

It is not clear, however, whether the larval and adult epidermis follow the same morphogenetic rules. As in the larvae, identities of the adult cells are principally determined by the ANT-C and BX-C genes (Struhl, 1982; Sánchez-Herrero et al. 1985), but the specific contributions of the different homeotic genes to the adult patterns have not been studied. In the experiments reported here, we have investigated these problems by using a Polycamb mutation. We show that the loss of Pc results in ectopic activity of BX-C genes, engrailed and probably Sex combs reduced. We have generated various combinations of active homeotic genes and have determined their different contributions to the adult patterns.

Materials and methods

Mutations, genetic combinations and mitotic recombination experiments
The Pc2 mutation has been described by Duncan & Lewis
(1982) and is the most extreme allele available. The cis-combinations of Pc3 with Abd-BM1 and abd-AM1 Abd-BM8 have already been reported (Casanova et al. 1986). The combination Pc3 UbXM12 abd-AM1 Abd-BM8 has been synthesized especially for this study.

For mitotic recombination experiments, the cuticle marker mutants multiple wing hairs (mwh) and javelin (jv) were introduced into all the Pc3 chromosomes. For the same purpose the stock y; DpscD Dp(I)6 M(3)P5 Ff115 e11/TM1 has been used (Morata et al. 1983). A scheme of the mitotic recombination method used to generate the mutant clones is shown in Fig. 1. Larvae (48–120 h of age) of genotype y; DpscD Dp(I)6 M(3)P5 Ff115 e11/mwh jv Pc3 BX-C were irradiated (1000 R) to induce mitotic recombination. In this genotype, BX-C denotes any of the combinations lacking the three or two or one or no BX-C genes.

As illustrated in Fig. 1, a recombination event proximal to Pc3 will result in clones marked with y mwh jv M* that are homozygous for Pc3 and lose the Dp(1)6, which carries a complete functional copy of BX-C. Notice that given the position of Pc3 and the Dp(1)6, there is a number of equally marked y mwh jv M* clones that retain the Pc* gene. Several of these experiments were repeated to generate flies of the same genotype in the third chromosome as above, but that also carried in the second chromosome a y* P-element construct in which the bacterial lacZ gene is linked to 5–7 kb of engrailed promoter (Hama & Kornberg, personal communication). The particular line we have used (ry-Xh025) has the transposon inserted less than 3 kb upstream of the start of transcription of the resident engrailed gene, thus in this case the expression of β-gal is presumably directed by the normal engrailed promoter (Tom Kornberg, personal communication).

**Immunocytochemistry**

Embryos aged 0–14 h from the appropriate stocks (approximately 25% of them were homozygous for the selected mutant combinations) were collected, dechorionated in sodium hypochlorite for 3 min and washed with water. The fixation, devitellinization and staining was carried out as described previously (Mitchison & Sedat, 1983; Carroll & Scott, 1985; DiNardo et al. 1985). The primary antibodies were used at 1:50 for en ascites and 1:750 for fts-polycional serum. Commercial biotinylated secondary antibodies were used (Amersham) in conjunction with streptavidin–horseradish peroxidase. The cytological stain was developed with (Sigma) diaminobenzidine (0.5 mg ml⁻¹) and hydrogen peroxide (0.03%) in PBS. Embryos were embedded in acetone: vestopal (1:1) overnight at 4°C; acetone was evaporated and the polymerization reaction was carried out at 60°C.

**β-gal staining**

Wings and abdomens were cut from freshly eclosed flies, submerged in EM grade 50%, glutaraldehyde (Fluka) and immediately washed once with PBS. They were transferred into X-gal solution (0.2 % 5-bromo-4-chloro-3-indolyl-β-d-galacto-pyranoside in 10 mm-sodium pyrophosphate (pH 7.0), 0.15 M-NaCl, 1 mm-MgCl2, 5 mm-potassium ferricyanide, 5 mm-potassium ferrocyanide) and staining was at 37°C overnight. They were dehydrated and mounted in Canada Balsam.

**Preparation of adult and larval cuticle**

For the larval epidermis, late embryos were dechorionated with dilute hypochlorite and the vitelline membrane removed using a glass needle. They were mounted in Hoyer’s medium according to van der Meer (1977). After incubation at 60°C, slides were studied under bright-field phase or dark-field optics.

The adult cuticle was prepared by cutting the appropriate pieces under the dissecting microscope. The internal organs were digested with 10% KOH, and the cuticle washed in alcohol and mounted in Euparal for examination.

**Results**

**Interactions of Polycomb with BX-C and some ANT-C genes during the embryonic period. Design of the experiments**

There is well-documented evidence (Lewis, 1978;
Duncan & Lewis, 1982; Sato & Denell, 1985; Casanova et al. 1986) that the elimination of Pc\(^+\) gives rise in the embryo to an indiscriminate expression of all the BX-C and some ANT-C genes like Antp and Scr. Other ANT-C genes like fushi tarazu (ftz) or zerknäult (zen) do not seem to be affected in Pc embryos since there is no phenotypic evidence for their over-expression. In the case of ftz, for example, one would expect an anti-ftz phenotype (Struhl, 1985) and this is not observed. Furthermore, we have stained Pc\(^-\) Abd-B\(^{M1}\) embryos with ftz antibody and found that they show the normal seven stripes of ftz expression (Hafen et al. 1984a; Carroll & Scott, 1985). No label was found in the epidermal cells after germ band elongation.

The embryonic phenotypes of Pc\(^-\) Scr\(^-\) Antp\(^-\) Ubx\(^-\) Abd-A\(^-\) Abd-B\(^-\) and Pc\(^+\) Scr\(^+\) Antp\(^+\) Ubx\(^+\) Abd-A\(^+\) Abd-B\(^+\) combinations are very similar (Sato & Denell, 1985), although not identical. Therefore, we consider that these five genes are the only homeotics affected by the elimination of P; if other genes are involved, their effects must be minor.

The objective of our experiments is to test whether the elimination of Pc in the adult cells results in ectopic expression of homeotic genes and, if so, to generate clones of epidermal cells containing different combinations of active homeotics. We thus can investigate the contribution of each one of the BX-C genes to the adult pattern and the pattern formed in their absence.

Using marker and Minute mutants (see Fig. 1 and Materials and Methods for details) in the same chromosome as Polycomb and a duplication for the entire BX-C, we can generate marked clones that lose simultaneously Pc and a duplication for the BX-C. In these clones, there should be an indiscriminate expression of the BX-C and ANT-C genes still present in the cells. If, for example, we use the genotype y; mwh jv Pc\(^3\) abd-A\(^{M1}\) Abd-B\(^{M8}\)/Dpsc\(^{M1}\) Dp146 M(3)\(^{15}\) Df115, the y, mwh, jv, M\(^+\) clones resulting from mitotic recombination proximal to Pc\(^3\) will possess Ubx\(^+\), Scr\(^+\) and Antp\(^+\) activity.

These experiments can be done using Pc mutants because Pc, unlike esc (Struhl & Brower, 1982), is required during the larval period (Struhl, 1981). The allele used (Pc\(^3\)) is the most extreme available (Duncan & Lewis, 1982). There is evidence (Denell, 1982; Lawrence et al. 1983) for some maternal function of Pc. This might rescue, in part, the embryonic phenotype, but it should not affect our Pc\(^-\) clones, which are generated during the larval period (48–96 h after egg laying) well past any influence of maternal product.

We have restricted our analysis to the main trunk (thorax and abdomen) of the body because, in this region, the ANT-C and BX-C genes are the principal
Simultaneous elimination of Pc and the entire BX-C
In embryos of genotype $Pc^{-} Ubx^{-} abd-A^{-} Abd-B^{-}$, thoracic and abdominal segments present an identity intermediate between prothorax and mesothorax, very similar to $Pc^{-}DfP9$ described by Sato & Denell (1985).

Cell clones deficient for $Pc$ and the BX-C genes were screened in all thoracic and abdominal segments. In 992 wings, we found a total of 124 $y$, $mwh$, $jv$, $M^{+}$ clones. There is a group that retains $Pc^{+}$ (45) and develops normally (BX-C function is not required in the wing) and a second group that differentiates atypically. The pattern shown by these clones does not correspond to any cuticular pattern in the body. The majority contain only trichomes and sensilla (Fig. 4A). The trichomes are of smaller size and more densely packed than normal wing trichomes. They tend to run along veins and are elongated in the proximodistal axis. Sometimes they produce thin bristles that are more or less evenly spaced. These clones are often invaginated and form vesicles that segregate from the normal wing territory (Fig. 4D). This phenomenon probably reflects differential cell affinities of the clone cells. The pattern observed in the clones is the same in the anterior (parasegment 4) and the posterior compartment (parasegment 5). One significant (and unexpected) feature of these clones is that they transgress the anteroposterior compartment boundary (Fig. 4C) although they respect the dorsoventral one. This observation suggested an effect on the engrailed (or engrailed-like) gene (Morata & Lawrence, 1975); either it is eliminated in the posterior compartment or it is adventitiously expressed in the anterior one. Very few mutant clones were found in the thoracic part (notum) of the wing segment.

Unlike those in the wing, $Pc^{-} Ubx^{-} abd-A^{-} Abd-B^{-}$ clones in the legs show an identifiable pattern, although sometimes it is not entirely normal. This pattern corresponds to the posterior compartment of the prothorax and appears in clones located in anterior or in posterior compartments of any of the three pairs of legs. This can easily be identified in the femur region where the posterior prothoracic pattern is very distinctive (Fig. 2A). A total of 40 $Pc^{-}$ clones have been found in the legs of 400 flies examined (X-rays 72–120h of development). The pattern shown by the clones strongly indicates that both Scr and en, which promote, respectively, prothoracic and posterior patterns, are derepressed in the absence of $Pc$ and BX-C function. This is consistent with our observation that mutant clones cross the A/P boundary in the wing. However, they do not transgress the

Fig. 3. $Pc^{-}$ clones in abdominal segments. (A) $Pc^{-} Ubx^{+} abd-A^{-} Abd-B^{-}$ clone in the third tergite. The thin small bristles are typical of the first tergite. Notice the presence of a cuticular fold (arrow). This abnormal structure appears very frequently associated with the clones. (B) $Pc^{-} Ubx^{+} abd-A^{-} Abd-B^{-}$ clone in the first tergite. The clone bristles (arrows) are longer than those of the first tergite and similar to those of more posterior segments. (C) $Pc^{-} Ubx^{+} abd-A^{+} Abd-B^{+}$ in the fifth tergite. The bristles are long and thin resembling those of analia. Also the cuticle is without trichomes, like that of analia or some parts of genitalia.
A/P boundary in the legs, except in the prothoracic one where we found two cases of transgression.

We have found very few clones in the abdomen and they are small and associated with vesicles and invaginations. In a few cases, they differentiate bristles that do not look of abdominal type. Our interpretation is that these clones are of thoracic type but cannot develop in an abdominal environment (Morata & García-Bellido, 1976). The comparison of the frequency of clones in the abdomen in this experiment (0.07 per abdomen) with control animals where the clones do not produce a homeotic transformation (0.57 per abdomen) indicates that most of the mutant clones fail to develop.

**Simultaneous elimination of Pc, abd-A and Abd-B**

The larval phenotype of 
Pc~ abd-A~ Abd-B~ (Casanova et al. 1986) is a reiteration of parasegment 6, formed by T3p-Ala compartments. In this genotype, Scr+, Antp+ and Ubx+ become inappropriately active.

Pc~ abd-A~ Abd-B~ clones in the wing (n = 76) show a phenotype very similar to the one found in the previous case in which Ubx+ was also lacking. It was unexpected and indicates that either Ubx+ is not derepressed in the wing or its activity has no major effect in the wing pattern. As in the previous case, the clones show the same pattern in anterior and posterior compartments and are also able to transgress the anteroposterior compartment boundary. We find no clones in the thoracic part of the wing segment.

In all three legs, the mutant clones (n = 32) develop a pattern which can be interpreted as posterior compartment of the third leg. The bristles are thin, some have bracts and, in the femur region, the clones often differentiate cuticle without trichomes as in the T3p leg compartment (Fig. 2B).

The comparison of these patterns with those described in the previous experiment (Pc~ Ubx~ abd-A~ Abd-B~) indicates that the presence of Ubx+ is having a morphological effect and overrides Scr+. The pattern shown by the mutant clones is the same in the anterior and the posterior compartment of the legs. In the abdomen, Pc~ abd-A~ Abd-B~ clones differentiate bristle patterns characteristic of A1, as expected for Ubx+ overexpression. Nevertheless the clones are not entirely normal; they are frequently depigmented and form cuticle invaginations and local pattern aberrations (Fig. 3A). As we show below this is probably due to engrailed overexpression.

**Simultaneous elimination of Pc and Abd-B**

This should result in the ectopic activation of Scr, Antp, Ubx and abd-A. Embryos of genotype Pc~ Abd-B~ secrete a reiterated chain of parasegment 9 (A3p–A4a).

Mutant clones in the wing (n = 125) exhibit a phenotype very similar to that reported in previous experiments including the transgression of the A/P border, while in the thorax we fail to find any Pc~ Abd-B~ clone.

23 mutant clones were found in the legs and they show a phenotype different from those of previous experiments. They often form vesicles or local outgrowths that disrupt the normal leg pattern. Some of the bristles are bracted indicating that they have leg properties but the pattern cannot be assimilated to any normal leg pattern. The fact that the mutant clones tend to sort out from the surrounding leg territory suggests that their identity is unlike any of the legs. In this genotype, the presence of abd-A+ probably confers to the cells an unusual identity (a nonsense codeword, Struhl, 1982) which is unlike any segment identity.

In the abdomen, Pc~ Abd-B~ clones (n = 47) differentiate abdominal patterns of A2–A5 type. This is expected if abd-A+ is overexpressed. The transformation is particularly clear in the A1 segment (Fig. 3B) where the size of the clone bristles contrasts with the surrounding ones. Our interpretation is that these clones develop an A4 pattern in correspondence with that observed in embryos of the same genotype. Many of these clones are associated with loss of pigment and some cases of anomalous bristle differentiation which may be caused by ectopic engrailed activity. In this case, however, the effect of engrailed overexpression appears to be minor in comparison with previous experiments.

**Elimination of Pc**

This should result in the derepression of the five genes concerned. In Pc~ embryos, thoracic and abdominal metameres develop as PS13, A7p–A8a. The behaviour of Pc~ cell clones in the adult cuticle has been reported by Struhl, 1981. He found some clones in the leg developing into structures resembling analia. Little was known about the pattern differentiated by Pc~ clones in wings and in abdominal segments.

Pc~ clones in the wings show a phenotype very similar to that described in previous combinations (Fig. 4C). In the abdomen, clones frequently lack trichomes and pigment (Fig. 3C), sometimes analia-like structures can be seen.

Engrailed is ectopically expressed in Pc~ imaginal cells

Some of the results, particularly the identity of Pc~BX-C~ clones in the legs and their transgression of A/P border in the wing suggested that the engrailed gene is derepressed in imaginal Pc~ cells. On the other hand, phenotypic analyses of Pc~
embryos with different mutant combinations for BX-C genes do not give any indication of ectopic expression of engrailed (Casanova et al. 1986). We have tested the expression of en in Pc− and Pc−/abd-A− Abd-B− embryos using an anti-engrailed antibody (provided by Drs T. Kornberg & D. Brower) and found no detectable change in the normal pattern of engrailed activity.

We also tested the expression of en in Pc− BX-C− and Pc−/abd-A− Abd-B− cell clones in the wing and abdominal tissue. This has been carried out by using an en-lacZ fusion gene (generously provided by Dr T. Kornberg) in which the engrailed promoter is linked to the E. coli lacZ gene coding for β-galactosidase. In this series of experiments, the mutant clones were generated in larvae carrying the en-lacZ gene in the II chromosome. The rationale was that, if Pc− cells showed ectopic engrailed activity in the anterior compartment, they should contain β-gal activity. This activity can be detected in adult cells due to the stability of the β-gal protein. As shown in Fig. 4E,F, anterior Pc− clones in the wing show β-gal activity and the limit of the galactosidase stain coincides with the marked clone. In abdominal segments, we found a large number of Pc−/abd-A− Abd-B− clones and none of them was as convincingly stained with β-gal as those in the wing. It is possible that the amount of overexpression of engrailed in the Pc− abdominal cells is less than in the thoracic cells.

Discussion

The elimination of Pc function derepresses engrailed in imaginal but not in embryonic cells

The finding that Pc− BX-C− clones in the legs, whether of anterior or posterior provenance, develop a posterior pattern indicates that engrailed or an engrailed-like gene is being derepressed in anterior compartments. The fact that Pc− clones in the wing transgress the A/P border points to the same conclusion. That the gene in question is engrailed is demonstrated using en-lacZ flies in which anterior Pc clones show ectopic β-gal activity (Fig. 4E,F). However, there is no indication of en activity in Pc− embryos. The size and aspect of engrailed stripes in these embryos is normal as visualized with anti-engrailed antibody.

Since Antp and BX-C genes are overexpressed in Pc− embryos, the late effect on engrailed is probably not due to any maternal rescue of Pc phenotype. Why en is only derepressed in Pc− imaginal cells whereas the other homeotics are in larval and imaginal cells is not at all understood. Mutations of the trithorax (trx) gene (Ingham, 1985) also have a pleiotropic effect, although of opposite type, on some ANT-C and BX-C genes, and probably also engrailed. These mutations also present different homeotic transformations in embryonic and in adult segments, indicating a differential expression of trx in imaginal cells. The ectopic expression of engrailed in Pc− clones of anterior thoracic compartments is the first report of gain of function of engrailed in imaginal cells. The observation that these clones develop posterior pattern emphasizes the role of engrailed as a major determinant of the posterior component of the segment identity. It also indicates that the engrailed gene is not simply required in posterior compartments but that it must also be inactive in anterior ones.

The elimination of Pc derepresses Scr (most probably) and all the BX-C genes in adult cells

Our experiments strongly suggest, but do not conclusively prove, that Pc− Ubx−/abd-A− Abd-B− clones contain ectopic Scr activity. The observation is that these clones develop prothoracic patterns in the three legs. Since Scr promotes prothoracic pattern in adult (Struhl, 1982) and larval cells (Wakimoto & Kaufman, 1981) and is derepressed in Pc− and esc− embryos (Sato & Denell, 1985; Struhl, 1983), it is very likely that it is responsible for the prothoracic pattern seen in the clones.

Since this pattern is produced in segments like T2 where there is normal Antp activity, this suggests that either Scr suppresses Antp or, if both are present, Scr exerts the dominant role. This conclusion is supported by previous experiments (Morata & Kerridge, 1981) showing that T2 and T3 legs that contain Antp activity (Struhl, 1982; Wirz et al. 1986) can develop prothoracic patterns if Scr is ectopically active in those legs (Struhl, 1982; Casanova et al. 1985).

Our experiments also demonstrate that the BX-C...
genes are ectopically expressed in the absence of P{c}. The P{c}Ubx\textsuperscript{-}\textasciitilde{abd-A}\textasciitilde{Abd-B}\textasciitilde clones in the legs generate a pattern closely resembling T3p and clearly different from that produced by P{c}Ubx\textasciitilde{abd-A}\textasciitilde{Abd-B}\textasciitilde clones. Thus the presence of Ubx\textasciitilde establishes a difference in identity even in those territories like T2p and T1 leg compartments anterior to P55 where Ubx is not normally expressed and that contain Scr and Antp activities. The pattern observed in these clones indicates that the presence of Ubx function overrides Scr\textsuperscript{+}, probably suppressing its expression (Struhl, 1982).

These clones develop, in the abdomen, a defective but recognizable A1 identity. The defects associated with these clones are probably due to the ectopic expression of en which promotes posterior identity.

The pattern produced by P{c}Ubx\textasciitilde{abd-A}\textasciitilde{Abd-B}\textasciitilde clones is different in the legs and abdomen from that of P{c}Ubx\textasciitilde{abd-A}\textasciitilde{Abd-B}\textasciitilde clones indicating that abd-A\textasciitilde is also derepressed in imaginal cells when P{c} is absent. In the abdomen, the identity of the clones can be clearly seen to correspond to an intermediate abdominal segment, even if appearing in A1a (Fig. 4B), a compartment that normally does not contain abd-A activity. This abdominal pattern is characteristic of the abd-A domain (Sánchez-Herrero et al. 1985) and indicates that abd-A activity overrides Ubx.

Finally, P{c}Ubx\textasciitilde{abd-A}\textasciitilde{Abd-B}\textasciitilde clones show a identity very different from the previous ones in all legs and abdominal segments indicating that Abd-B is becoming inappropriately active and is the major contributor to the pattern.

Taken together, our results suggest that when homeotic genes are active together in imaginal cells, they contribute differently to the pattern. They can be ordered in a series Antp < Scr < Ubx < abd-A < Abd-B. For any combination of these genes, the identity will be principally determined by the highest member of the series present. This hierarchy explains why, in some instances, the presence of a given homeotic gene product may be largely irrelevant in certain segments. For example, Antp product is present in abdominal segments (Hafen et al. 1984b; Wirz et al. 1986) but the elimination of the product (Struhl, 1982) or its overexpression (Schnewly et al. 1987) does not affect the identity of those segments. Here the major determinants are the BX-C genes. Similarly, Ubx (White & Wilcox, 1984) is expressed in abdominal segments A2–A8 (domains of abd-A and Abd-B) yet the elimination of Ubx in the adult cells of these segments has no effect (Kerridge & Morata, 1982).

Our results suggest that the mesothoracic identity, determined by Antp, is the ground one for the trunk segments. This is modified by Scr in the anterior direction and by the BX-C genes in the posterior one.

It is not clear why the identity of P{c} clones appears to be different in thoracic and abdominal segments whereas in P{c}– embryos thoracic and abdominal metameres develop similarly (although not identically). This may indicate that P{c}– causes a different amount of ectopic expression along the body or perhaps that there are other region-specific patterning genes in adult cells whose presence yields different combinations of active genes in thorax and in abdomen. We believe the first possibility unlikely because there is, for example in P{c}– Abd-B\textsuperscript{M1} clones, a clear-cut difference of identity in T3p leg compartment and A1a tergite even though these two compartments belong to parasegment 6 and should have a similar level of P{c} requirement.

The hierarchy of function that can be seen in the legs and abdomen is not observed in the wing. The pattern shown by P{c}– clones in the wing is not a normal wing pattern, but it is the same whether BX-C genes are present or absent. Although it is possible that the absence of P{c} does not derepress the BX-C genes in the wing, we think this is unlikely since they are activated everywhere else, including the larval mesothorax. It seems more probable that other morphogenetic genes, derepressed in P{c}– clones, are suppressing or obliterating the BX-C genes. We do not understand this dorsoventral difference in identity of the clones. However, it has been observed (Morata & Kerridge, 1981) that the elimination of the Ubx function transforms the leg cells of T2p compartment but has no effect on the wing cells of the same compartment. This also points to a different genetic specification of wing and leg imaginal cells.

References


(Accepted 27 September 1988)