

subtype of a given meteorite. There are only three meteorites for which the literature data assignments differ systematically from the TL assignment; the Mezö-Madaras subtype could be decreased by 0.1, the Parnallee value increased by 0.1 and the Semarkona value increased by 0.2. Clearly there would be no problem in assigning a petrological subtype in the absence of TL data, provided there were other good quality data available.

Received 23 June; accepted 19 September 1980.

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We thank the donors of meteorites listed in Table 1 and John Wasson for many discussions and encouragement; also Drs J. A. Wood, R. T. Dodd, and W. R. Van Schmus for advice, and Joanna Pai and Clare Marshall for technical assistance. This research is supported by NASA grant NGR 05-007-329 (at UCLA) and NSF grant EAR78-22440 (at Washington University).

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Mutations affecting segment number and polarity in *Drosophila*

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①

In systematic searches for embryonic lethal mutants of *Drosophila melanogaster* we have identified 15 loci which when mutated alter the segmental pattern of the larva. These loci probably represent the majority of such genes in *Drosophila*. The phenotypes of the mutant embryos indicate that the process of segmentation involves at least three levels of spatial organization: the entire egg as developmental unit, a repeat unit with the length of two segments, and the individual segment.

② THE construction of complex form from similar repeating units is a basic feature of spatial organisation in all higher animals. Very little is known for any organism about the genes involved in this process. In *Drosophila*, the metameric nature of the pattern is most obvious in the thoracic and abdominal segments of the larva. We are attempting to identify all loci required for the establishment of this pattern. The identification of these genes and the description of their phenotypes should lead to a better understanding of the general mechanisms responsible for the formation of metameric patterns.

③ In *Drosophila*, the anlagen for the individual segments arise as equally sized subdivisions of the blastoderm, each segment represented by a transverse strip of about three or four cell diameters¹. A cell lineage restriction between neighbouring segments is established at or soon after this stage². Two basic types of mutation have been described which change the segmental pattern of the *Drosophila* larva. Maternal effect mutations like *bicaudal* lead to a global alteration of the embryonic pattern³. Bicaudal embryos develop two posterior ends arranged in mirror-image symmetry, and lack head, thorax and anterior abdomen. The *bicaudal* phenotype suggests that the initial spatial organisation of the egg established during oogenesis involves a morphogen gradient that defines antero-posterior coordinates in early embryonic pattern formation^{3,4}. The subdivision of the embryo into segments is thought to occur by a differential response of the zygotic genome to the maternal gradient. Homeotic mutations (for example, *bithorax*^{5,6}) seem to be involved in a final step of this response process. These mutations change the identity of individual segments; for example, *Ultrabithorax* transforms the metathoracic and first

④ abdominal segments into mesothoracic segments. However, the homeotic loci do not affect the total number, size or polarity of the segments, nor do they point to any other step which might intervene between the maternal gradient and the final pattern of segments.

We have undertaken a systematic search for mutations that affect the segmental pattern depending on the zygotic genome. We describe here mutations at 15 loci which show one of three novel types of pattern alteration: pattern duplication in each segment (segment polarity mutants; six loci), pattern deletion in alternating segments (pair-rule mutants; six loci) and deletion of a group of adjacent segments (gap mutants; three loci) (Table 1, Fig. 1).

The segmental pattern of the normal *Drosophila* larva

⑤ Figure 2 shows the cuticular pattern of a normal *Drosophila* larva shortly after hatching. The larval body is comprised of three thoracic and eight abdominal segments. Although differences are observed in different body regions, all segments have certain morphological features in common. The anterior of each segment is marked with a band of denticles, most of which point posteriorly. The posterior part of each segment is naked. The segment borders run along the anterior margins of the denticle bands⁷, they have no special morphological features. The polarity of the pattern is indicated by the orientation of the denticles and, in the abdomen, by the shape of the bands (Fig. 3). In the thoracic segments the bands are narrow with fine denticles whereas those in the abdominal segments are broader and comprised of thick pigmented denticles (for a detailed description of the cuticular pattern see ref. 1).

Segment polarity mutants: deletions in each segment

Mutants in this class have the normal number of segments. However, in each segment a defined fraction of the normal pattern is deleted and the remainder is present as a mirror-image duplication. The duplicated part is posterior to the 'normal' part and has reversed polarity (Figs 1-3).

⑪ Six such loci have been identified. Three loci, *fused*⁸, *wingless* (ref. 9 and G. Struhl, personal communication) and *cubitus interruptus*^D, were previously known whereas *gooseberry*, *hedgehog* and *patch* are new (Table 1). **All the mutations in this class are zygotic lethals** and the phenotypes are only produced in homozygous embryos. One of the loci, *fused*, also shows a maternal effect in that a wild-type allele in the mother is sufficient to rescue the mutant phenotype of the homozygous embryos.

⑫ In all mutants except *patch* the region deleted includes the naked posterior part of the pattern and the duplication involves a substantial fraction of the anterior denticle band. In mutant larvae, the ventral side of each segment is almost entirely covered with denticles, the posterior fraction of which point anteriorly. The segment identity seems to be normal, the denticles of the abdominal segments being large and pigmented whereas those of the thorax are short and pale (Figs 1, 2). The anterior margin of the region duplicated in these mutants coincides with the segment boundary in only two cases, *fused* and *gooseberry* (Fig. 3). In *wingless* and *hedgehog* it lies posterior to

the boundary, such that these larvae apparently lack all segment boundaries.

The phenotype of embryos homozygous for *patch* contrasts with that produced by the five loci described above in that the duplicated region includes some naked cuticle anterior to each denticle band. The duplicated unit thus involves structures of two adjacent segments. *Patch* larvae, despite the normal number of denticle bands, have twice the normal number of segment boundaries (Figs 1, 3).

Despite these differences, the common feature of all mutants in this class is that a defined fraction of the pattern in each segment is **deleted. This deletion is associated with a mirror image duplication of the remaining part of the pattern**. We suggest that these loci are involved in the specification of the basic pattern of the segmental units.

Pair-rule mutants: deletions in alternating segments

In mutants of this class **homologous parts of the pattern are deleted in every other segment**. Each of the six loci is characterized by its own specific pattern of deletions (Table 1, Figs 1, 4). For example, in *even-skipped* larvae, the denticle bands and adjacent naked cuticle of the pro- and metathoracic, and the 2nd, 4th, 6th and 8th abdominal segments are lacking. **This results in larvae with half the normal number of denticle bands separated by enlarged regions of naked cuticle** (Figs 1, 4). In *paired* larvae the apparent reduction in segment number results

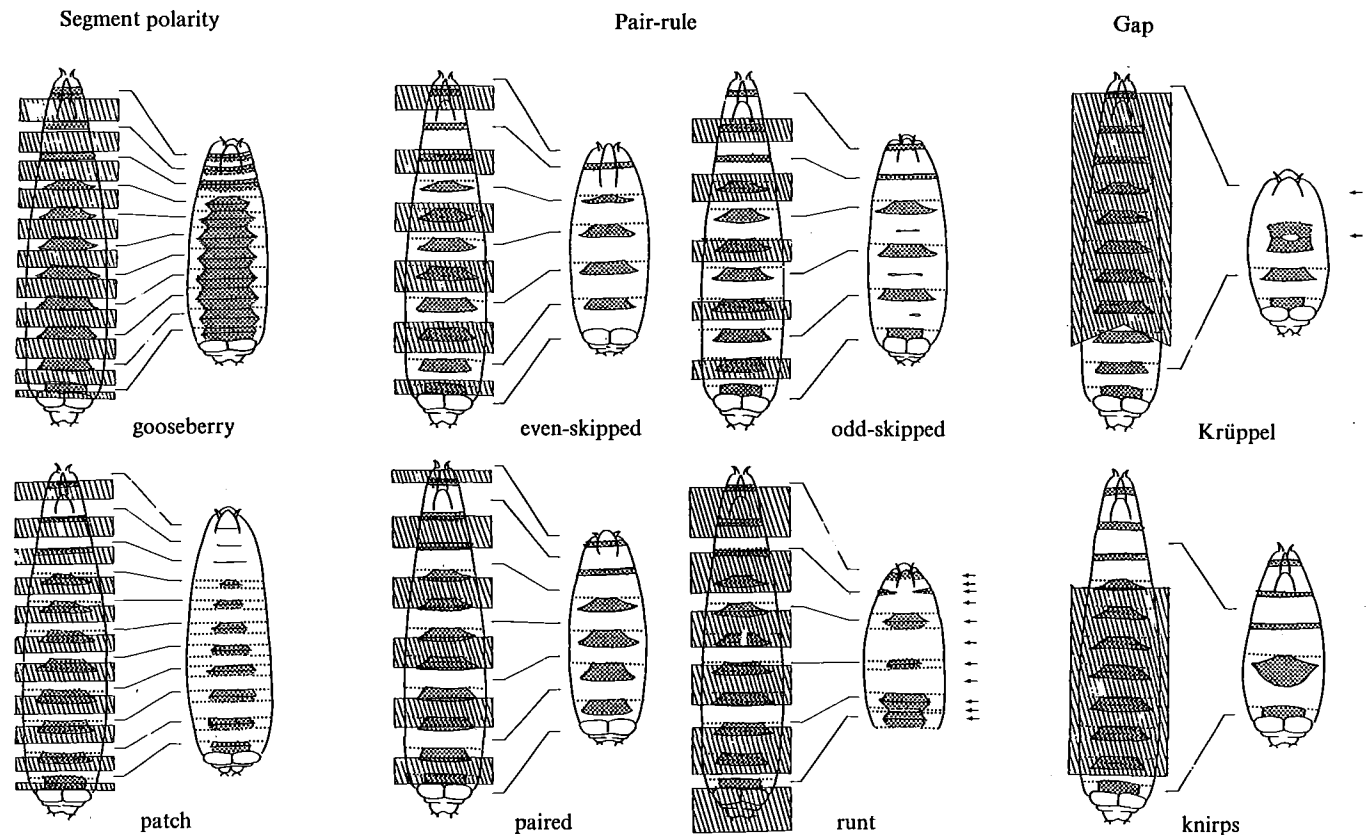


Fig. 1 Semi-schematic drawings indicating the regions deleted from the normal pattern in mutant larvae. Dotted regions indicate denticle bands, dotted lines the segmental boundaries. The regions missing in mutant larvae are indicated by the hatched bars. The transverse lines connect corresponding regions in mutant and normal larvae. For indication of the polarity of the patterns, see Fig. 3. The patterns of *fused* and *ci*^D (not shown) look similar to the *gooseberry* pattern, whereas in *hedgehog* and *wingless* the deleted regions are somewhat larger, cutting into the denticle bands at either side. Four pair-rule mutants are shown in the centre. The interpretation of their phenotypes is based on the study of weak as well as strong alleles, combinations with *Ubx* (see text) and, in the case of *runt*, on gynandromorphs (unpublished). They probably represent the extreme mutant condition at the respective loci. The phenotypes of all known *barrel* and *engrailed* alleles (not shown) are somewhat variable and further studies are needed to deduce the typical phenotype. At the right, the two gap loci *Krüppel* and *knirps* are shown. Both patterns represent the amorphic phenotype as observed in embryos homozygous for deficiencies of the respective loci. The only known *hunchback* allele (not shown) deletes the meso- and metathorax.

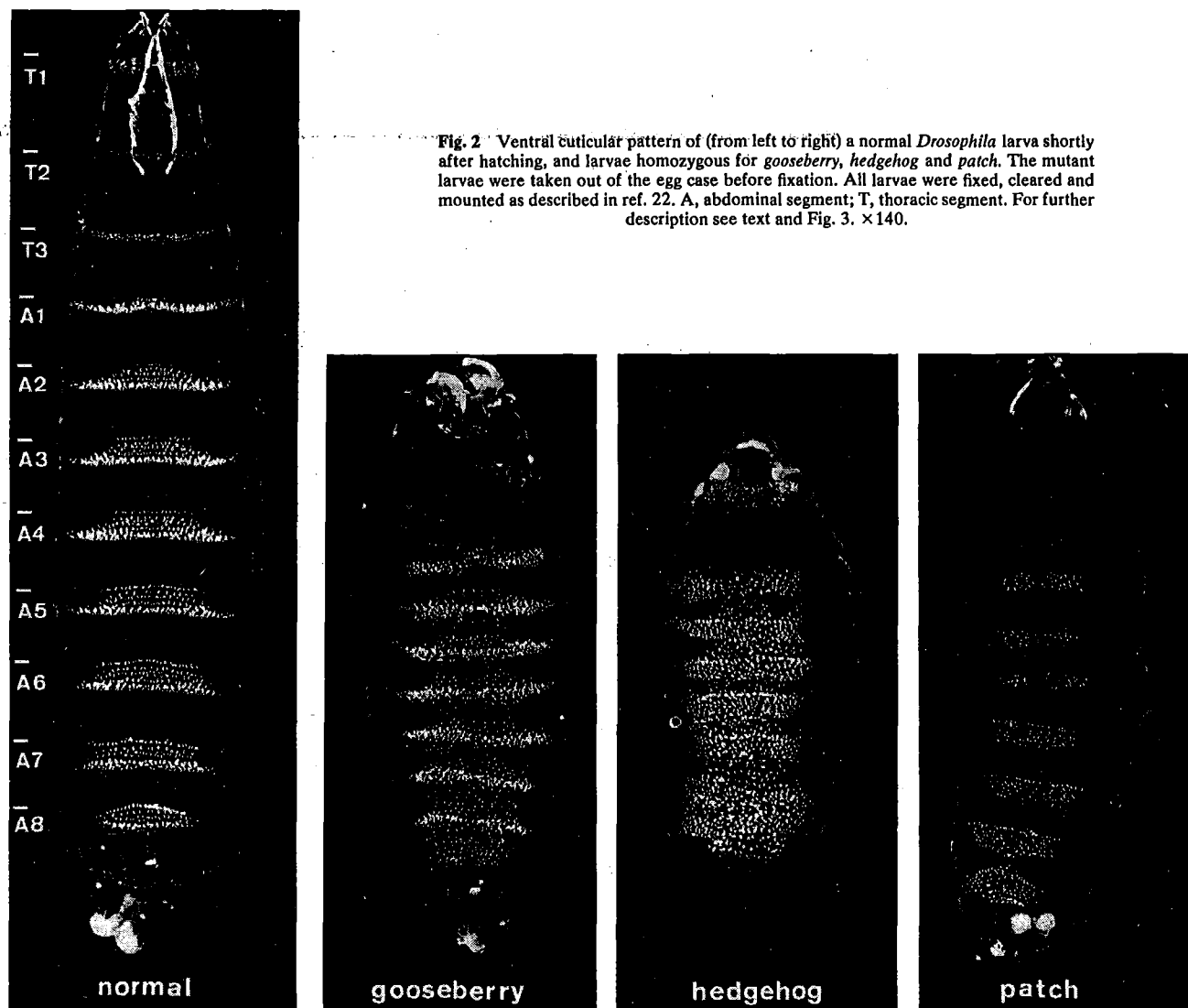


Fig. 2 Ventral cuticular pattern of (from left to right) a normal *Drosophila* larva shortly after hatching, and larvae homozygous for *gooseberry*, *hedgehog* and *patch*. The mutant larvae were taken out of the egg case before fixation. All larvae were fixed, cleared and mounted as described in ref. 22. A, abdominal segment; T, thoracic segment. For further description see text and Fig. 3. $\times 140$.

essentially from the deletion of the naked posterior part of the odd-numbered and the anterior denticle bands of the even-numbered segments (Figs 1, 4). The double segments thus formed are composites of anterior mesothorax and posterior metathorax, anterior first abdominal and posterior second abdominal segment, etc. The identification of the regions present or deleted in mutant larvae is based on the phenotypes produced by alleles with lower expressivity. In such embryos the deletions are in general smaller and variable in size. In a 'leaky' allele of *even-skipped*, the denticle bands of the even-numbered segments are frequently incomplete, whereas in *paired*² (*prd*²) the pattern deletions often involve only part of the naked cuticle of the odd-numbered segments, leading to a pairwise fusion of denticle bands (Fig. 4).

Further support for the composite nature of the segments in *prd* larvae was obtained in combinations with *Ultrabithorax* (*Ubx*), a homeotic mutation which when homozygous causes the transformation of the first abdominal segment into mesothorax⁵. In such *prd; Ubx* larvae, only the denticle band of the first double segment in the abdomen is transformed. The posterior margin of this band and the naked cuticle which follows remain abdominal in character and lack, for example, the typical mesothoracic sense organs. The composite nature of the segments in *paired* shows that the establishment of segmental identity does not require the establishment of individual segments as such.

The segmentation pattern in *prd; Ubx* larvae is typical of that of *paired* alone and is not affected by the *Ubx* homeotic trans-

formation. Similarly, in *prd; Polycomb* larvae the naked cuticle of alternating segments is deleted just as it is in *paired* alone, even though in *Polycomb* embryos all the thoracic and abdominal segments have an 8th abdominal character⁵. These combinations, and similar ones with *even-skipped*, indicate that the observed grouping of segments in pairs depends on the position of segments within the segmental array rather than the segmental identity. These combinations thus provide evidence that mutations such as *paired* and *even-skipped* affect different processes from those altered in homeotic mutants.

Other mutants in this class show different deletion patterns. The phenotype of *odd-skipped* is similar to that of *even-skipped*. However, in this case it is the odd-numbered denticle bands that are affected. In *odd-skipped*, the deleted region is smaller and is restricted to a more posterior part of the segment than in *even-skipped* (Figs 1, 4). *Barrel* has a phenotype similar to *paired* although the pattern is often less regular (Fig. 4). *Runt*, on the X chromosome, is the only pair-rule mutant showing mirror-image duplications. *Runt* embryos have half the normal number of denticle bands, each a mirror-image duplication of the anterior part of a normal band (similar to the duplications found in *patch*). The bands in *runt* embryos, as well as the region of naked cuticle separating them, are of unequal sizes (Figs 1, 4).

To the list of pair-rule loci we have added the *engrailed*¹⁰ locus. Lethal *engrailed* alleles¹¹ lead to a substantial deletion of the posterior region of even-numbered segments. In addition, the anterior margin and adjacent cuticle of each segment are

affected. Thus, the defect pattern in *engrailed* shows repeats which are spaced at both one- and two-segment intervals.

Each of the six different pair-rule loci affects a different region within a double segmental repeat. In no case does the margin of the deleted region coincide with a segment boundary. When the deleted region corresponds in size up to one entire segment (paired, even-skipped) it includes parts of two adjacent segments.

The phenotypes of the pair-rule mutants suggest that at some stage during normal development the embryo is organized in repeating units, the length of which corresponds to two segmental anlagen.

Gap mutants: one continuous stretch of segments deleted

One of the striking features of the mutations of the first two classes is that the alteration in the pattern is repeated at specific intervals along the antero-posterior axis of the embryo. No such repeated pattern is found in mutants of the third class and instead a single group of up to eight adjacent segments is deleted from the final pattern. Three loci have been identified which cause such gaps in the pattern (Table 1, Figs 1, 5). *Krüppel* (*Kr*) was originally described by Gloor¹². Embryos homozygous for *Kr* lack thorax and anterior abdomen. The posterior-terminal region with the abdominal segments 8, 7 and 6 is normal, although probably somewhat enlarged. Anterior to the 6th abdominal segment is a plane of mirror-image symmetry followed by one further segment band with the character of a 6th segment oriented in reversed polarity. The exact position of the plane of symmetry varies and does not usually coincide with a segmental boundary. A large part of the *Krüppel* pattern is reminiscent of the pattern observed in embryos produced by the maternal-effect mutant *bicaudal*, although no maternal

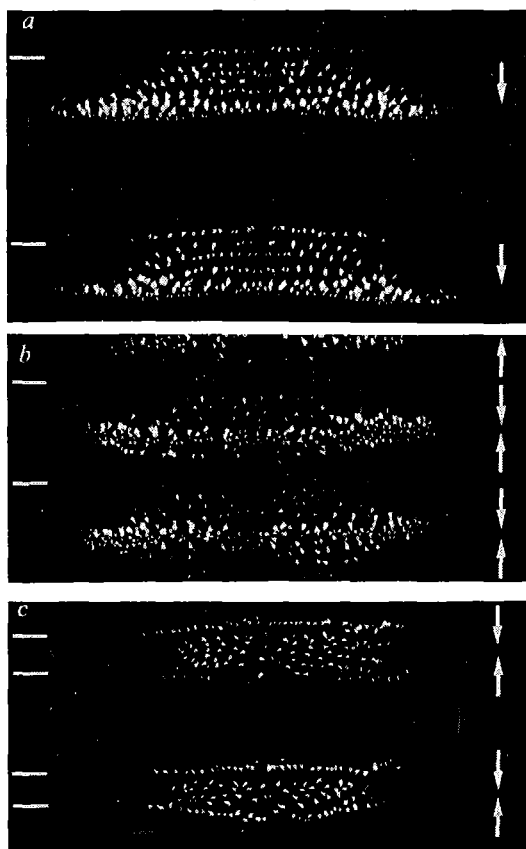


Fig. 3 Details from the ventral abdomen of a normal (a), a *gooseberry* (b), and a *patch* (c) larva. The positions of the segment boundaries are indicated at the left by the transverse lines. The arrows at the right indicate the polarity of the pattern as judged by the orientation of the denticles as well as the shape of the denticle bands.

Table 1 Loci affecting segmentation in *Drosophila*

Class	Locus	Map position*	No. of allelest	Ref.
Segment-polarity	<i>cubitus interruptus</i> ^D (<i>ci</i> ^D)	4-0	(2)	20
	<i>wingless</i> (<i>wg</i>)	2-30	6	9
	<i>gooseberry</i> (<i>gsb</i>)	2-104	1	This work
	<i>hedgehog</i> (<i>hh</i>)	3-90	2	This work
	<i>fused</i> (<i>fu</i>)†	1-59.5	(9)	8, 20
	<i>patch</i> (<i>pat</i>)	2-55	8	This work
Pair-rule	<i>paired</i> (<i>prd</i>)	2-45	3	This work
	<i>even-skipped</i> (<i>eve</i>)	2-55	2	This work
	<i>odd-skipped</i> (<i>odd</i>)	2-8	2	This work
	<i>barrel</i> (<i>brr</i>)	3-27	2	This work
	<i>runt</i> (<i>run</i>)	1-65	1	This work
	<i>engrailed</i> (<i>en</i>)	2-62	6	11, 20
Gap	<i>Krüppel</i> (<i>Kr</i>)	2-107.6	6	12, 20
	<i>knirps</i> (<i>kni</i>)	3-47	5	This work
	<i>hunchback</i> (<i>hb</i>)	3-48	1	This work

* For the new loci (see last column) the map positions are based on recombination between the markers *S*, *Sp*, *Bl*, *cn*, *bw* for the second chromosome, and *ru*, *h*, *th*, *sl*, *cu*, *sr*, *e*⁵, *ca* for the third chromosome. For description of markers see ref. 20. The loci *runt*, *Krüppel* and *knirps* were further mapped using the breakpoints of deficiencies and duplications for the respective regions. All mutants were mapped by scoring the embryonic progeny of single recombinant males backcrossed to heterozygous females from the original mutant stocks.

† The numbers in parentheses refer to the alleles listed in Lindsley and Grell²⁰. All other alleles, except the *runt* allele, three *Kr* alleles and one *knirps* allele, were isolated in a screen for embryonic lethal mutants on the second chromosome. 5,800 balanced stocks were established from individual males heterozygous for an ethylmethane sulphonate-treated *cn bw sp* chromosome using the DTS-procedure suggested by Wright²¹. 4,500 of the stocks had one or more new lethal mutations. Unhatched embryos from 2,600 putative embryonic lethal stocks were inspected for cuticular abnormalities²². Third chromosomal mutants discovered in the second chromosomal balanced lines were recovered after selection through individual females by balancing individual third chromosomes over TM3. Complementation tests were carried out between mutants with similar phenotypes whereby the occurrence of mutant embryos among the progeny of the crosses served as the criterion for allelism. Three new *Kr* alleles were isolated in a screen for lethals over the original *Kr* of Gloor¹², and one *knirps* allele of presumably spontaneous origin was discovered on a TM1 chromosome. The *runt* allele was isolated in a screen for X-linked lethals.

‡ *fused* is a male-rescueable maternal-effect locus⁸. Thus, the segment polarity reversal is observed in *fu/fu* embryos from *fu/fu* mothers. The progeny of *fu/+* females show a normal embryonic pattern regardless of embryonic genotype.

component is involved in the production of the *Krüppel* phenotype (in preparation).

In the other two loci of this class, the gap in the pattern occurs in specific morphologically defined subregions of the larval pattern, in the thorax and in the abdomen, respectively. In *hunchback*, the meso- and metathoracic segments are deleted. In embryos homozygous for *knirps*, only two rather than eight denticle bands are formed in the abdomen. The posterior-terminal region including the 8th abdominal segment seems normal whereas the anterior abdominal denticle band is considerably enlarged. The anterior margin of the denticle band is morphologically similar to the first abdominal segment but combinations with *Ubx* show that the band is a composite with more than one segmental identity.

All three loci are required for a normal segmental subdivision of one continuous body region. The lack of a repeated pattern of defects suggests that the loci are involved in processes in which position along the antero-posterior axis of the embryo is defined by unique values.

When are genes affecting segment number active?

The phenotypes described above are observed only in homozygous embryos, indicating that the loci identified by these mutations are active after fertilization and are crucial for the normal segmental organisation of the embryo. We have described the mutations in terms of their effect on the differentiated pattern. However, in many instances their effect can be observed much earlier in development. In normal embryos segmentation is first visible 1 h after the onset of gastrulation as a repeated pattern of

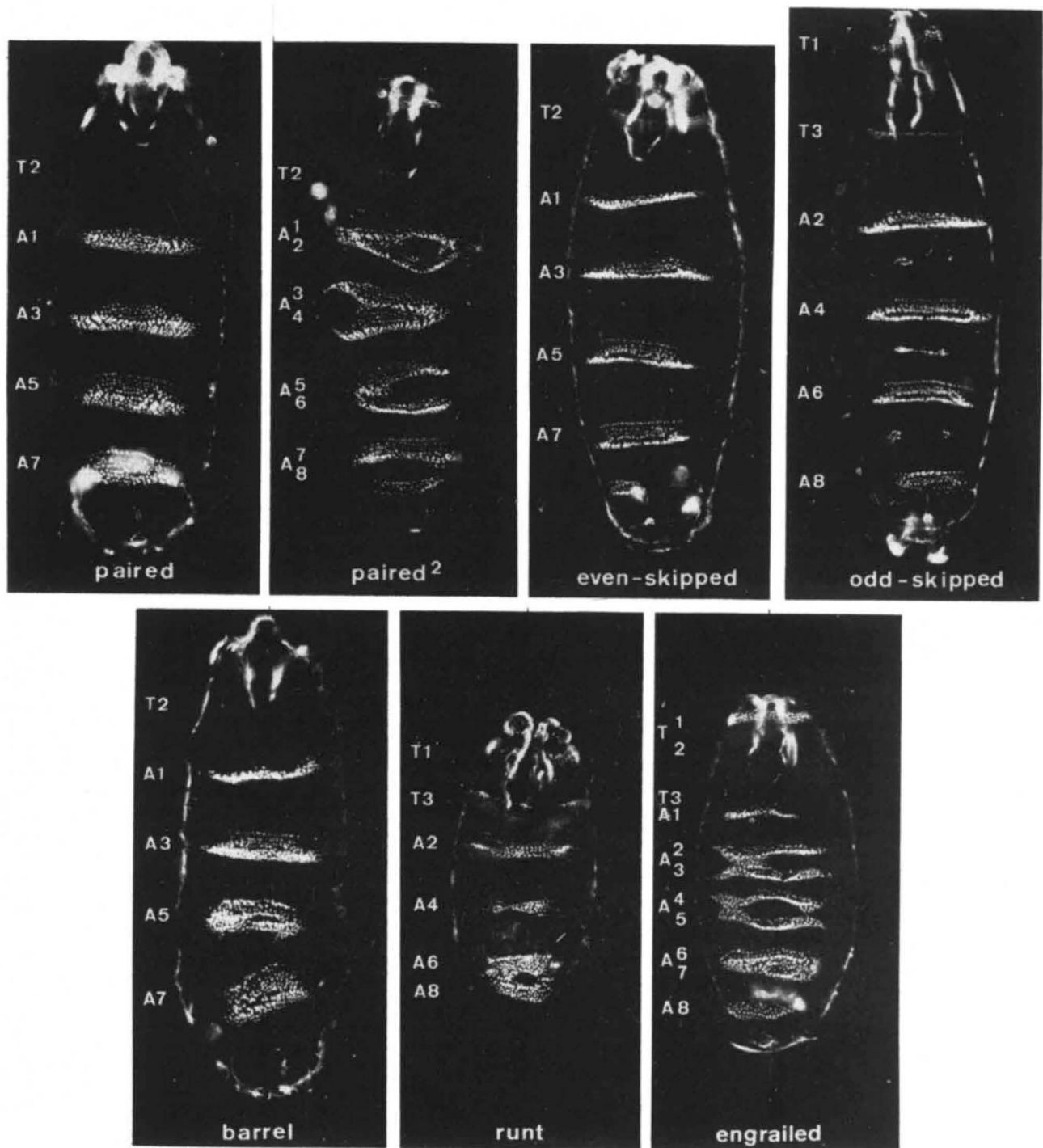


Fig. 4 Larvae homozygous for mutations at the six pair-rule loci. The segmental identity of the denticle bands is indicated at the left of each picture. A, abdominal band; T, thoracic band. For comparison with the normal pattern see Figs 1 and 2.

bulges in the ventral ectoderm (unpublished observations). In *paired* and *even-skipped* embryos the number of bulges is reduced and corresponds to the number of segments observed in the differentiated mutant embryos. *Krüppel*, *runt* and *knirps* embryos can be identified 15 min after the onset of gastrulation. All three mutations cause shorter germ bands, a phenomenon clearly related to the strong reduction in segment number observed in the differentiated larvae. Further evidence for an early activity at the *paired* locus was obtained using a temperature-sensitive allele. The extreme phenotype is only obtained when the embryo is kept at the restrictive temperature during the blastoderm stage. **All these results indicate that the wild-**

type genes defined by the mutations are active before the end of gastrulation during normal development.

Discussion

Segmentation in *Drosophila* proceeds by the transition from a single field into a repeated pattern of homologous smaller subfields. Mutant alleles at the 15 loci we have described interfere with this process at various points. Although each locus has its own distinct phenotype, we were able to distribute the mutations in three classes. In one class only a single large subregion of the embryo is affected, whereas in mutants of the other two classes a reiteration of defects is produced with a

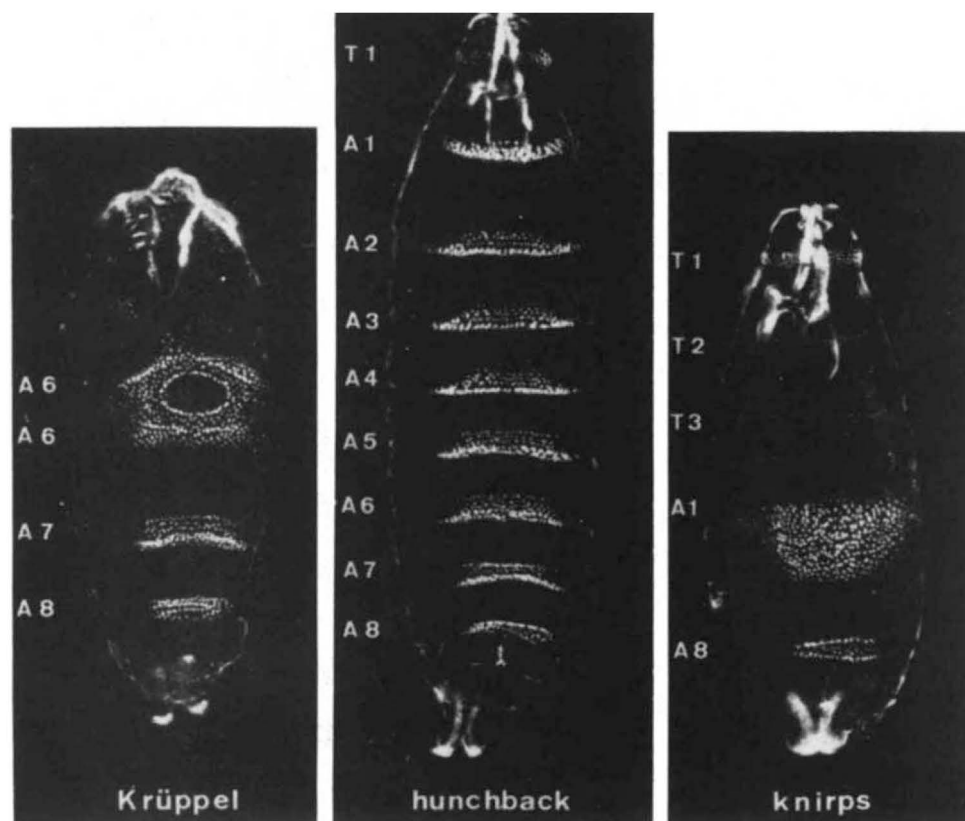


Fig. 5 Larvae homozygous for mutations at the three gap loci. A, abdominal segment; T, thoracic segment.

repeat length of one or two segments, respectively. This suggests that the process of segmentation involves three different units of spatial organization.

The organization of the egg is thought to be controlled by a monotonic gradient set up during oogenesis under the control of the maternal genome^{3,4}. All the mutants we have described depend on the embryonic rather than the maternal genome. None of them alters the overall polarity of the embryo, the head always being at the anterior and the telson at the posterior end of the egg. The most dramatic alterations of the pattern are produced in the gap mutants and involve one large subregion of the egg. *Hunchback* and *knirps* affect the development of thorax and abdomen, respectively, and might be involved in the establishment of these large morphologically unique subregions of the embryo. The large mirror-image duplications found in the posterior pattern of *Krüppel* embryos are similar to those of *bicaudal*. The *bicaudal* phenotype has been interpreted as resulting from an instability in the maternal gradient^{3,13}, and thus *Krüppel* might be involved in the maintenance or elaboration of this gradient in the posterior egg region after fertilization.

The smallest repeat unit, the individual segment, is affected in mutants at the six segment-polarity loci. The pattern alteration consists of a mirror-image duplication of part of the normal pattern with the remainder deleted. In these mutants the deleted region corresponds in size to more than half a segment. Mutations causing smaller deletions in the segmental pattern do not lead to polarity reversals (unpublished observations). The mirror-image duplications produced by the pair-rule mutation *runt* are also associated with a deletion of more than half a repeat unit, that is, more than one segment. The tendency of partial fields containing less than half the positional values to duplicate is well described for imaginal disks in *Drosophila*¹⁴, as well as for the larval epidermis in other insects^{15,16}. On the other hand, the same types of pattern duplication are also produced in conditions which do not involve cell death and regeneration, but rather a reorganization of the positional information in the entire field^{3,17}. More detailed studies of early development in mutant embryos may reveal which mechanism is responsible for the different mutant phenotypes.

Given the evidence for a homology between segments, the existence of a class of mutants affecting corresponding regions in each individual segment is perhaps not surprising. The discovery of a mutant class affecting corresponding regions in every other segment was not expected and suggests the existence at some time during development of homologous units with the size of two segmental anlagen. It is possible that the double segmental unit corresponds to transitory double segmental fields which are established early during embryogenesis and are later subdivided into individual segments. At the blastoderm stage the epidermal primordium giving rise to thorax and abdomen is only about 40 cells long¹. An initial subdivision into double segments would avoid problems of accuracy encountered in a simultaneous establishment of segment boundaries every three to four cells. A stepwise establishment of segments implies that the borders defining double segments be made before the intervening ones. The mutant phenotypes do not definitely show which, if any, of the segment borders define a primary double segmental division in normal development. The mutant phenotypes which come closest to a pattern one would expect if the transition from the double segment to the individual segment stage were blocked are the patterns of *paired* and *even-skipped*. Both suggest the frame meso- and methathorax, 1st and 2nd, 3rd and 4th abdominal segment, etc.

It is also possible that the double segmental units are never defined by distinct borders in normal development. The existence of a double segmental homology unit may merely reflect a continuous property such as a wave with a double segmental period responsible for correct spacing of segmental boundaries (see, for example, refs 18, 19). We have not found any mutations showing a repeat unit larger than two segments. This may indicate that the subdivision of the blastoderm proceeds directly by the double segmental repeat with no larger intervening homology units. However, the failure to identify such larger units may reflect the incompleteness of our data.

Drosophila has been estimated to have about 5,000 genes and only a very small fraction of these when mutated result in a change of the segmental pattern of the larva. Some of the loci

described here were known previously but only in the case of *Krüppel* has the embryonic lethal phenotype been recognized as affecting segmentation.¹² The majority of the mutants described here have been isolated in systematic searches for mutations affecting the segmentation pattern of the *Drosophila* larva. These experiments are still incomplete. Most of the alleles on the second chromosome were isolated in one experiment which yielded an average allele frequency of four or five alleles per locus (based on 42 embryonic lethal loci). From this yield and similar calculations for the third and first chromosomes, we estimate that we have identified almost all segmentation loci on the second chromosome and about 50% each of those on the third and first chromosome. Our sample of 15 loci should therefore represent the majority of the loci affecting segmentation in the *Drosophila* genome. Thus, in *Drosophila* it

would seem feasible to identify all genetic components involved in the complex process of embryonic pattern formation.

We thank Hildegard Kluding for excellent technical assistance, Adelheid Schneider, Maria Weber and Gary Struhl for help during various parts of the mutant screens, Gerd Jürgens for stimulating discussion and our colleagues from the *Drosophila* laboratories in Cambridge, Freiburg, Heidelberg and Zürich for critical comments on the manuscript, and Claus Christensen for the photographic prints. Thomas Kornberg and Gary Struhl provided us with lethal alleles of *engrailed* and *wingless* respectively which facilitated the identification of our alleles. All mutants are available on request.

Note added in proof: All known *barrel* alleles fail to complement *hairy*²⁰, suggesting that the *barrel* mutations are alleles at the *hairy* locus.

Received 26 June; accepted 29 August 1980

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Chemical synthesis of a polypeptide predicted from nucleotide sequence allows detection of a new retroviral gene product

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We previously determined the nucleotide sequence of the 3' end of Moloney leukaemia virus and discovered the potential coding region for an unknown protein, R. We now show that this region does encode a protein. A pentadecapeptide of R was chemically synthesized and antibodies raised against it. Antisera to the synthetic peptide recognize the R protein and the env precursor polyprotein in infected cells. The strategy presented here should provide a general method for accessing proteins predicted by nucleotide sequences.

THE accepted genetic structure of replication-competent murine leukaemia viruses, such as Moloney, Rauscher, Friend and AKV, includes three genes. These genes, *gag*, *pol* and *env*, are arranged, in respective order, along the single-stranded RNA genome. They are transcribed from the integrated double-stranded DNA provirus¹. The protein products of these three genes are understood in considerable detail. The *gag* gene encodes a polyprotein of about 65 kilodaltons which is proteolytically processed to proteins, amino to carboxy terminal, of 15, 12, 30 and 10 kilodaltons, respectively². These proteins are found in the core of the virion, and one, p30, has been associated with virus tropism³. The second gene, *pol*, is expressed as an apparent extension of the *gag* gene such that an approximately 180-kilodalton polyprotein containing both *gag* and *pol* components is observed. The polyprotein is processed to a 70-kilodalton protein which is the reverse transcriptase⁴. This enzyme is responsible for copying the single-stranded RNA genome of an infecting virus into the double-stranded DNA structure which can integrate into host DNA. The third gene, *env*, encodes a polyprotein which is glycosylated and processed into components gp70 and p15E (ref. 5). gp70 is the major envelope component and determines viral host range. It is sometimes found disulphide linked to p15E, a hydrophobic

protein which may anchor gp70 to viral and cellular membranes⁶. Messenger RNA molecules have been described which can account for the expression of these three genes⁷.

To understand fully the genetic structure of this class of viruses, we have been analysing their nucleotide sequences. We began our study at the 3' end of Moloney murine leukaemia virus (Mo-MuLV) because of our particular interest in the *env* gene which was thought to be the most 3'-proximal gene. Surprisingly, we found an open reading frame which extended beyond the coding region for known viral products⁸. Even though we then had no evidence for a gene product, we provisionally named this the R-region because it is the coding region that is the furthest to the right in the genome. Here we report the detection of the R product. We chemically synthesized part of the R protein, raised antibodies to the synthetic peptide and detected immunologically cross-reactive material in infected cells.

The DNA sequence

For orientation purposes, several features of the previously reported⁸ DNA sequence are presented here (Fig. 1). The DNA sequence was initially generated from an 1,108-base pair cDNA clone obtained by cloning the product of reverse transcription of