Genetic and Cytogenetic Analysis of the 43A-E Region Containing the Segment Polarity Gene costa and the Cellular Polarity Genes prickle and spiny-legs in Drosophila melanogaster

Pascal Heitzler,* Darin Coulson,† Maria-Theresa Saenz-Robles,* Michael Ashburner,† John Roote,‡ Pat Simpson* and David Gubb†

*Laboratoire de Génétique Moléculaire des Eucaryotes du CNRS, Unité 184 de Biologie Moléculaire et de Génie Génétique de l’INSERM, Faculté de Médecine, 67085 Strasbourg Cedex, France, and †Department of Genetics, University of Cambridge, Cambridge CB2 3EH, England

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ABSTRACT

A cytogenetic analysis of the 43A-E region of chromosome 2 in Drosophila melanogaster is presented. Within this interval 27 complementation groups have been identified by extensive F2 screens and ordered by deletion mapping. The region includes the cellular polarity genes prickle and spiny-legs, the segmentation genes costa and torso, the morphogenetic locus sine oculis and is bounded on its distal side by the eye-color gene cinnabar. In addition 19 novel lethal complementation groups and two semi-lethal complementation groups with morphogenetic escaper phenotypes are described.

The chromosomal region 43A-E lies within the proximal region of the right arm of chromosome two in Drosophila melanogaster. Seven genetic complementation groups have previously been described within this region: prickle (pk), spiny-legs (sple), l(2)pawn (l(2)pwn), costa (cos), sine oculis (so), torso (tor) and cinnabar (cn). Mutations at all of these loci give morphogenetic defects with the exception of cn, which gives an eye-color phenotype. The objective of this study was to provide a fine-scale genetic analysis of the region surrounding pk, sple and cos as a basis for the molecular analysis of these genes. A number of new complementation groups and chromosomal rearrangements are described and used to construct a genetic map and cytological map of the 43A-E region (Figure 1).

Mutant alleles of pk and sple affect the polarity of bristles and hairs (Gubb and García-Bellido 1982), whereas those of l(2)pwn affect bristle and hair morphology (García-Bellido and Dapena 1974). Embryos mutant for loss of function alleles of cos that are derived from a mutant female germ line display defects in the larval segments. A posterior part of the segmental denticle belt is missing and is replaced by a mirror-image duplication of the anterior part including the segment boundary (Grau and Simpson 1987). Embryos derived from females mutant for tor lack the terminal acron and telson (Schüpbach and Wieschaus 1986; Sprenger, Stevens and Nüsslein-Volhard 1989; Klingler et al. 1988; Strecker et al. 1989). In addition, a raf-related sequence (Draf2) has been mapped to 43A2-5 (Mark et al. 1987). Prior to this study only a small number of rearrangements had been described in this region (Grau and Simpson 1987).

MATERIALS AND METHODS

Stocks: The rearrangements used in this study are listed in Table 1. New mutations and aberrations recovered during this work will be described below and listed in Tables 1 and 2. Lethal loci are named according to their cytological position.

Crossovers: Crosses were set up in vials cultured at 25°. Complementation crosses were between mutant chromosomes balanced over In(2LR)/O, Cy dp16 pr cn2 (CyO). Mutations were classified as lethal if less than 1% of homozygotes survive to the adult stage and noncomplementary if transheterozygous flies expressed a mutant phenotype or no nonbalancer carrying progeny emerged among 150 or more progeny.

Mutagenesis: New mutations were induced in males with ethyl methanesulfonate (EMS) according to the protocol of Lewis and Bacher (1968) or by X rays at a dose of 4,000 rads (100 kV, 10 mA for 5 min, 1.5-mm aluminum filter, Philips MG102 constant potential X-ray system, beryllium window).

Cytology: Chromosome aberrations were analyzed cytologically with temporary propionic-orcein-carmine squash preparations of larval salivary gland chromosomes. The aberrations were interpreted with the aid of the revised polytene chromosome maps of Bridges (see Lefèvre 1976).

RESULTS

To characterize the pk-cos region, the first step was to recover a number of chromosome aberrations, in particular deletions, that could be used for the genetic mapping of lethal and visible mutations. The most productive of these screens was the reversion of Droplet, a gain-of-function mutation with a very clear dominant visible phenotype.
Reversion of Droplet: The dominant mutation Droplet (Drl) was induced by EMS and mapped to chromosome 2 to the left of Tufted (2-53.6 in region 37A3-6) by Craymer (1984). Drl causes an extreme reduction in the size of the eye when heterozygous with a wild-type chromosome. The first three X-ray induced revertants of Drl were recovered in a screen of 6,678 chromosomes in an experiment that was unrelated to this study. Of these revertants, two were found to be translocations each with one breakpoint in region 43B (T(2;3)DrZw1 and T(2;3)DrZw2, Table 1). This suggested that the original mapping of Drl might be incorrect and that the mutation could be related to so (2-57.1), which had been mapped to the 42E-43C interval (Yannopoulos et al. 1981). It was this experiment that suggested the use of Droplet reversion for the recovery of deletions in region 43.

Among approximately 100,000 non-Cy progeny of X-irradiated Drl/CyO males, 67 revertants of Drl were recovered and 29 stocks (Drl<sup>no</sup> to Drl<sup>ns3</sup>) were established. Sixteen of the 29 mutant second chromosomes were found to carry cytologically visible aberrations or were identified as chromosomal deletions by their failure to complement several loci adjacent to so (Table 1 and Figure 1). In view of our major objective, the recovery of deletions in region 43, we have not excluded the possibility that some of the chromosomally nonaberrant reversions result from dominant second-site suppressors of Droplet. In addition, four deletions including the cn locus were recovered among the CyO progeny in this screen (Df(2R)cnS3, Df(2R)cnS5, Df(2R)cnS6 and Df(2R)cnS8, Table 1).

**F<sub>2</sub> lethal screens:** Four F<sub>2</sub> screens were performed to recover lethal mutations in the 43A to 43E region. In the first two screens, 3,400 EMS-treated bu<sup>o</sup> chromosomes and 1,200 X-irradiated cn bw sp chromosomes were tested for visible and lethal mutations when heterozygous with the large deletion Df(2R)ST1. The first screen gave 39 EMS-induced lethal mutations and a single female sterile mutation. The second screen gave eight X-ray-induced lethals. To select mutations in region 43A-43E stocks of these mutations were crossed to Df(2R)pk-78k and Df(2R)CA58 (Figure 1) and mutations that were viable with both these deletions, but lethal with Df(2R)ST1, were discarded. These crosses allowed us to discard any lethal chromosomes that might carry mutations elsewhere on chromosome 2. These screens gave 31 EMS and seven X-ray-induced lethal mutations within the 43A to 43E interval (Table 2).

A further two screens were designed to recover lethal mutations mapping proximal to pk. In these
screens, chromosomes were tested for lethality with $Df(2R)pk$-78k and counter-screened with $Df(2R)ST1$, so as to eliminate any lethals allelic to extraneous mutations on the $Df(2R)pk$-78k chromosome. A total of 5,470 EMS and 2,359 X-ray-mutagenized $b$ cn chromosomes were tested giving a further 42 EMS-induced and eight X-ray-induced lethal mutations (Table 2). These new mutations were mapped with the available region 43A-43E deletions. Surprisingly, since the proximal cytological limits of both $Df(2R)pk$-78k and $Df(2R)ST1$ are well proximal to 43B, all these mutations map distal to 43B, in the interval between $pk$ and so, and define no additional complementation groups.

In addition to lethal point mutations, 17 visible

<table>
<thead>
<tr>
<th>Chromosomal rearrangements used in this study</th>
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<tr>
<td><strong>Rearrangement</strong></td>
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<tr>
<td>$Df(2R)CA38$</td>
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<tr>
<td>$Df(2R)cn-S3 + In(2LR)O$, $Cy dp^w$ pr cn</td>
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<td>$Df(2R)cn-S5 + In(2LR)O$, $Cy dp^w$ pr cn</td>
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<tr>
<td>$Df(2R)cos3$, $bw$ sp</td>
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<tr>
<td>$Df(2R)cos13-rv1$, $bw$ sp</td>
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<td>$Df(2R)HZ3$</td>
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<tr>
<td>$Df(2R)pm-5$, $bw^o$</td>
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<tr>
<td>$Df(2R)nap-2$, $dp^+$</td>
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</tbody>
</table>

All chromosomes in Table 1 were obtained in this study unless otherwise indicated:

- $Df(2R)CA38$ was found in a laboratory stock of $Dp(2;3)P32$ and separated from the duplication. The origin of the deletion is unclear, but is probably $Df(2R)P32$.
- $Df(2R)Drl-rv1$ and $Df(2R)pk-N6$ were lost before cytological analysis.
- $Df(2R)cos13-rv1$ failed to complement $pk$ and $cos$; $Df(2R)pk-N6$ failed to complement all mutant chromosomes tested in the nec-cn interval.

mutations and one female sterile mutation were recovered in these F2 screens, as well as five chromosomal deletions, two inversions and one transposition ($Df(2R)pm-5$, $Df(2R)ST1$, $Df(2R)GW60$, $Df(2R)DX14$, $Df(2R)DX1$, $Df(2R)DX14$, $In(2R)DX4$, $In(2R)DX8$ and $T(2;3)DX11$).

**F. screens for visible mutations:** Two screens were made for mutations that failed to complement the wing phenotype of a hypomorphic $cos$ allele (see below); 8,812 EMS-treated and 27,310 X-irradiated $bw^o$ chromosomes were scored heterozygous with the $cos^{11}$ cn $bw$ sp chromosome. One new $cos$ allele, $cos^{12}$, was recovered from the EMS screen. All putative new $cos$ alleles from the X-ray screen were sterile. The $cn$ mutation carried on the $cos^{11}$ cn $bw$ sp chromosome
also allowed the recovery of five X-ray-induced deletions that include the cn locus, Df(2R)NCX8, Df(2R)NCX9, Df(2R)NCX10, Df(2R)NCX11, and Df(2R)NCX13, and one new cn allele, cnNCX12.

A similar screen to revert the adult phenotype of cos13/cos14 gave a single deletion, Df(2R)cos13-rol, among 2,090 X-irradiated chromosomes. (The adult phenotype of cos13/cos14 flies resembles that of fused and is described below.)

Ten chromosomal aberrations in the region were recovered in screens for new prickle and sple alleles (D. Coulson, N. Chapman and J. Regan, unpublished data). Six of these aberrations were recovered from 48,374 X-irradiated chromosomes, Df(2R)sple-D1, Df(2R)sple-N3, Df(2R)sple-N5, Df(2R)sple-N6, Tp(2;2)pk-sple22 and Tp(2;3)pk-sple24. The remaining four, Df(2R)sple-D2, Df(2R)sple-D3, Df(2R)sple-j1 and Df(2R)sple-J2 were recovered from 32,380 chromosomes, by failure to complement the sple1 mutation following the imprecise excision of a P element in the pk locus. The P element was mobilized by crossing the pk15 chromosome (D. Coulson, unpublished data) to a b pr sple1; P[ry*(Δ2-3)](99B) stock. The P[ry*(Δ2-3)](99B) chromosome carries a P-element insertion that provides constitutive high levels of expression of the P element transposase (Robertson et al. 1988). Progeny of this cross were scored for the sple phenotype.

In addition, a viable allele of so (so5) was recovered heterozygous with Df(2R)NCX9 among 3,000 EMS mutagenized chromosomes. Individual b cn/ Df(2R)NCX9 males from this cross were mated to Df(2R)ST1 virgin females in order to recover lethal mutations proximal to the 43B region (J. Roote, G. Johnson and T. Morley, unpublished data).

From these screens and existing mutant strains, 150 visible and lethal mutations were available for study.

### Maternal effect lethals

Mutations affecting female fertility were not screened for systematically. However, one of the novel lethal chromosomes recovered in this study, humilis, displayed a maternal effect (see below for description of humilis, hum). An F2 screen of 2,700 EMS-treated chromosomes for mutations affecting female fertility was undertaken to isolate additional alleles of hum. This screen also produced six lethal mutations, which are included in the data in Table 2.

Alleles of the maternal-effect gene torso (tor) were obtained from C. Nüsslein-Volhard and localized precisely within the 43A-E region. The maternal effect lethal, scraps (screps), described by Schüpbach and Wieschaus (1989), also maps within this region and four novel zygotic lethal alleles were recovered in this study.

### Deletion mapping of the 43A-E region

Mutations were first crossed to the Df(2R)pk-78k and Df(2R)CA38 deletions to assign them to one of three regions (see Figure 1). The mutations in each group

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**TABLE 2**

Mutant alleles recovered in this study

<table>
<thead>
<tr>
<th>Complementation group</th>
<th>Allele</th>
<th>Progenitor chromosome</th>
<th>Mutagen</th>
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<tr>
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<td>EMS</td>
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were then crossed inter se and to the other deletions recovered in this study. Twenty-seven complementation groups were identified and ordered relative to each other by deletion mapping and recombination (Table 2).

This allows 16 of the complementation groups to be assigned a precise linear order with respect to adjacent loci (Figure 1). The remaining 11 loci occur in four pairs and a single group of three that are not ordered with respect to each other. The proximal extent of this region overlaps the distal boundary of the Df(2R)nup-2 deletion recovered by B. GANETZKY (Table 1).

Exchange mapping: Several mutations surrounding cos were ordered by recombination (Table 3), giving results consistent with the deletion mapping. The region within which the largest number of mutations was recovered, between the proximal breakpoint of Df(2R)CA58 and the distal breakpoint of Df(2R)pk-78k (Figure 1), corresponds to a genetic distance of about 0.24 map units, estimated as the distance between the sple and so loci (Table 3). The overall genetic extent of the region studied, between the proximal and distal breakpoints of Df(2R)ST1, corresponds to about 2.2 map units, estimated as the distance between the pk (2-55.3) and cn (2-57.5) loci.

### Description of mutant phenotypes

**necrotic**: Amorphic mutations of necrotic are either pharate adult lethal, or die within 24 hr of eclosion. The adult flies look relatively normal at eclosion but, after a few hours, develop brown spots principally at the bases of the legs and around the ocelli. These patches become more extensive and darker as the flies age. Thirteen alleles of this gene have been studied (Table 2). With two exceptions, the EMS-induced nec and the X-ray induced nec, the nec mutations appear to be amorphic, giving the same phenotype whether homozygous or when heterozygous with each other or a deletion.

Complete deletions of necrotic, indeed of necrotic, prickle and spiny-legs, may survive as adults (Table 4). These have a strong necrotic phenotype, and soon die. They also have a prickle phenotype (e.g., if the proximal deletion is Df(2R)nup-2) or a prickle-spiny-leg phenotype (if Df(2R)pk-N3). Heterozygotes between Df(2R)nup-2 and Df(2R)pk-78k are viable (3.8%) despite being homozygously deleted for the 7-8 bands between 42E3 and 45A1.2—a remarkably long region. This accounts for our failure to recover any lethal mutations in this interval (see above, also J. ROOTE, M. ASHBURNER and D. SEGAL, unpublished data).

The hypomorphic alleles, nec and nec, were both recovered on chromosomes carrying an inversion in the region and an associated pk mutation (Table 1). In(2R)nec pk gives a slight nec phenotype when homozygous, whereas In(2R)nec pk is nec when homozygous. When heterozygous with a deletion, both the In(2R)nec pk and In(2R)nec pk chromosomes give fertile adults that survive for more than a week. These flies have swollen abdomens, reddish spots that darken with age and a variable pk phenotype ranging from wild-type to weak pk. The viabilities of these two alleles on eclosion, heterozygous with a deletion, are somewhat higher than those of the amorphic alleles (Table 5).

Both nec and nec almost complement the amorphic nec alleles, giving a phenotype showing dark spots near the ocelli at a low penetrance. This is surprising and suggests that the 11 amorphic nec mutations might retain enough residual nec activity to complement the hypomorphic alleles.

**prickle and spiny-legs**: pk and sple mutations affect the polarity of bristles and other cuticular processes.
single-mutant alleles of pk give a polarity phenotype similar to pk'/Df(2R)pk-78k or Df(2R)nap-2/Df(2R)pk-78k.

Flies homozygous for a double-mutant allele have a less extreme prickle phenotype than pk'/pk' and a very weak polarity pattern in the leg. The abdominal polarity and the tarsal duplication phenotypes, however, remain similar to that of sple single-mutant flies (D. Coulson and D. Gubb, unpublished data). This surprising result does not reflect the double-mutant alleles being hypomorphic. When heterozygous with deletions including both pk and sple, the double-mutant alleles give a phenotype that is indistinguishable from that given by homozygous double-mutant alleles or that given by the overlapping deletion in Df(2R)pk-N5/Df(2R)sple-D1 flies.

SHARMA, CHITNIS and SHYNGLE (1985) reported an unusual sple mutant, sple\textsuperscript{bor}, isolated from an EMS screen. When homozygous this has a stunted leg phenotype, as well as one affecting cell polarity on the legs. The sple mutation on this chromosome can, however, be separated from a second site mutation responsible for the stunted legs. This second mutation is allelic to dachshund (dac = l(2)36Ae of Ashburner et al. 1990) mapping to 35F on the left arm of chromosome 2. Once separated from dac, the sple\textsuperscript{bor} chromosome retains an amorphic sple mutation (renamed sple\textsuperscript{6b} by Lindsley and Zimm 1992), giving a phenotype similar to other sple alleles. A further single mutant allele of pk, pk\textsuperscript{22}, was identified within Dp(2;3)P32. This duplication is nec\textsuperscript{+} pk\textsuperscript{22} sple\textsuperscript{*} pwn\textsuperscript{+}, the pk mutation presumably mapping to the 42F breakpoint within the transposed segment.

\textit{l(2)pawn}. The original allele of \textit{l(2)pawn} was recovered in a screen for cell-viable marker mutations (GARCÍA-BELLIDO and DAPENA 1974). Homozygous clones within heterozygous flies give truncated bristles with transparent tips and thin, transparent hairs. The mutation is lethal in homozygous flies during the zygotic stage (GARCÍA-BELLIDO and DAPENA 1973). In

---

**TABLE 4**

<table>
<thead>
<tr>
<th>Df(2R)sple-11</th>
<th>Df(2R)sple-f2</th>
<th>Df(2R)sple-D1</th>
<th>Df(2R)pk-78k</th>
</tr>
</thead>
<tbody>
<tr>
<td>15/211 7.1%</td>
<td>19/124 15.0%</td>
<td>65/402 16.0%</td>
<td>20/217 9.2%</td>
</tr>
<tr>
<td>17/593 4.3%</td>
<td>64/971 6.6%</td>
<td>22/586 3.8%</td>
<td>4/167 2.4%</td>
</tr>
</tbody>
</table>

\* Survivors necrotic and prickle. 
\* Survivors necrotic, prickle and spiny legs.

Numbers of Cy\textsuperscript{*} progeny, over total numbers of progeny, from crosses between CyO balanced chromosomes.

---

**TABLE 5**

| nec\textsuperscript{1} | 180/1382 13% |
| nec\textsuperscript{2} | 127/1024 12% |
| nec\textsuperscript{3} | 121/357 34%  |
| nec\textsuperscript{4} | 110/565 26%  |
| nec\textsuperscript{5} | 75/252 30%   |
| nec\textsuperscript{6} | 44/445 10%   |
| nec\textsuperscript{7} | 47/379 11%   |
| nec\textsuperscript{8} | 35/187 19%   |
| nec\textsuperscript{9} | 38/418 12%   |

Viabilities of adult flies heterozygous for nec alleles and nec\textsuperscript{*} deletions at eclosion; all mutant chromosomes were balanced over CyO and the numbers of non-Cy flies, over the totals, are shown. All survivors were phenotypically necrotic.

---

(GUBB and GARCÍA-BELLIDO 1982). These alterations in polarity differ from the mirror-image pattern duplications caused by segment polarity mutations in that the topological polarity of cuticular processes is affected without other elements of the pattern being altered. In the tarsi of sple mutations, however, extra tarsal joints are formed as mirror-image duplications (Held, Duarte and Derakhshian 1986). These tarsal duplications are analogous to the intersegmental duplications seen in \textit{cos}.

All known alleles of pk and sple are homozygous viable. Of nine new alleles of sple and two recovered as pk, eight were mutant for both pk and sple (pk-sple\textsuperscript{6}, pk-sple\textsuperscript{7}, pk-sple\textsuperscript{8}, pk-sple\textsuperscript{9}, pk-sple\textsuperscript{10}, pk-sple\textsuperscript{14}, pk-sple\textsuperscript{15} and pk-sple\textsuperscript{25}). Seven of these have a double-mutant phenotype indistinguishable from each other and that of sple\textsuperscript{78k}, described in Gubb and GARCÍA-BELLIDO (1982). Double-mutant flies have rough eyes and weak pk and sple phenotypes. The double-mutant allele, pk-sple\textsuperscript{23}, has the rough-eyes typical of other double-mutant alleles of pk-sple, but a much weaker pk phenotype, visible only in mounted wings and affecting the polarity of hairs in the region of the posterior cross-vein. Three novel amorphic alleles of sple that were not also mutant for pk, sple\textsuperscript{6}, sple\textsuperscript{7} and sple\textsuperscript{27}, were recovered in this study (Table 2).

This tendency to recover double-mutant alleles from an EMS screen suggests that pk and sple may be related genetic functions as EMS causes preferentially single base substitutions (Williams and Shaw 1987). In classical genetic terms, however, pk and sple are not allelic, the phenotype of pk/sple flies being wild-type (GUBB and GARCÍA-BELLIDO 1982). This is true for the original allele of sple, and also for the sple\textsuperscript{6}, sple\textsuperscript{7} and sple\textsuperscript{27} alleles, when heterozygous with pk\textsuperscript{1} or other single-mutant alleles of pk. When heterozygous with deletions that include both pk and sple, such as Df(2R)pk-N5, the new single-mutant alleles of sple give the amorphic sple phenotype described for sple\textsuperscript{6}/Df(2R)pk-78k (Gubb and GARCÍA-BELLIDO 1982) and...
uncrowded cultures, occasional escapers survive, these show thin bristles and hairs and have dark brown eyes with melanic patches in the corneal cuticle (GARCÍA-BELLIDO and DAPENA 1974). Five new alleles were recovered in the present study (Table 2); all are phenotypically similar to the original allele.

costa: cos is a member of the segment polarity class of genes (GRAU and SIMPSON 1987; SIMPSON and GRAU 1987). The gene exhibits a strong maternal effect. Homozygous embryos from heterozygous mothers are lethal but develop until the end of embryogenesis and look normal except for occasional perturbations of the polarity of the ventral denticle belts. When derived from homozygous germ-line clones, however, homozygous cos embryos show extensive morphogenetic abnormalities. The thoracic denticle belts are absent and the abdominal denticle belts are reduced in width (GRAU and SIMPSON 1987).

A series of wild-type iso-alleles (the V alleles) were described that fail to complement lethal alleles only when heterozygous with dominant mutations of the Costal (Cos) (2-68) locus. One of the new alleles, cos13, is unusual in that it fails to complement the wild-type iso-alleles in a Cos+ background. Such cos13/cosY flies lack ocelli, ocellar and post-vertical bristles and wing veins L3 and L4 are partially fused and lie closer together. In addition, the scutellum is reduced in size, scutellar bristles are missing and the wings are held-out. These phenotypes are characteristic of fused (BUSSON et al. 1988).

cos13 was recovered in a screen for lethal mutations in the Adh region (M. ASHBURNER, unpublished data). It maps genetically and by reversion between pk and en and is lethal when heterozygous with lethal cos mutations or deletions that include the cos locus. The revertant Df(2R)cos13-ru1 failed to complement pk and cos. It was suspected to be a very large deletion but was lost soon after recovery. It had a Minute phenotype that was suppressed by the presence of Dp(2;3)P32. Heterozygous cos13/+ flies are essentially wild-type but have occasional held-out wings and lack some post-vertical and scutellar bristles. This dominant phenotype, together with the failure to complement iso-alleles (in cosY/cos13 flies), suggests that cos13 may represent a gain of function mutation.

Seven new lethal alleles, cos8, cos9, cos10, cos12, cos14, cos15 and cos16, in addition to a hypomorphic allele, cos11, were recovered in the present screens (Table 2). cos11 is homozygous viable and wild-type, but when heterozygous with a deletion gives flies bearing pattern duplications. These flies are fertile, unlike flies homozygous for a second hypomorphic allele, cos7 (GRAU and SIMPSON 1987).

The fused-like phenotype of cos13 may be significant as fused is also a segment polarity gene. Furthermore, there is a dominant interaction between fu and cos (B.

**TABLE 6**

Viability of *humilis* heterozygotes; numbers of Cy+ flies from crosses between alleles balanced over CyO

<table>
<thead>
<tr>
<th>Allele</th>
<th>cy0</th>
<th>Cy0</th>
<th>cy0</th>
<th>cy0</th>
<th>cy0</th>
</tr>
</thead>
<tbody>
<tr>
<td>hum1</td>
<td>2/566</td>
<td>1/595</td>
<td>1/502</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>hum2</td>
<td>2/650</td>
<td>3/508</td>
<td>0/306</td>
<td>99/218</td>
<td>11595</td>
</tr>
<tr>
<td>hum3</td>
<td>3/798</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>hum4</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>hum5</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
</tr>
</tbody>
</table>

Escapers are very small flies, with short legs and wings. When heterozygous with Df(2R)ST1 all alleles are lethal (n > 2,000 CyO progeny).

**LIMBOURG-BOUCHON** and T. PRÉAT, personal data): fu1/+: cos+/+ females have reduced or absent ocelli and ocellar bristles. This ocellar phenotype is also shown by fu1/+: cos2/+, Df(2)fu2/+; cos2/+, fuA/Y; cos2/+ females and fuA/Y; cos2/+ males. In fuA/Y; cos2/+ males, however, there is an extreme enhancement of the recessive cos phenotype to give a pupal lethal phenotype, with pharate adults expressing the pattern duplications typical of cosY flies. In addition, all fu alleles are lethal when hemizygous males carry the cos13 allele.

**humilis**: Mutant alleles of *humilis* are either recessive lethal or recessive female-sterile. Eight alleles of this locus have been studied. Five, hum1, hum2, hum3, hum4 and hum6, are completely lethal when heterozygous with Df(2R)ST1, but give escapers when heterozygous with each other (Table 6). Escaper flies are tiny with disproportionately short legs and wings. The hum1 chromosome is female sterile when homozygous or when heterozygous with deletions that include hum. It also carries a maternal effect segmentation mutation that causes embryonic shortening and the loss of thoracic segments in embryos from homozygous hum1 females. This defective segmentation phenotype is not shown in embryos from the cross of hum1/Df(2R)NCX11 females to homozygous hum1 males or in crosses with any of the other hum alleles and so is unlikely to map to the hum locus.

Three viable but female sterile alleles of *humilis*, hum12, hum26 and hum66 were recovered in a screen for chromosomes that were sterile in females heterozygous for hum1. These sterile alleles are viable, but female sterile, when heterozygous with deletions of the hum locus and when heterozygous either with each other or with lethal hum alleles; the flies are otherwise wild-type. These chromosomes are all homozygous lethal, presumably due to the presence of additional lethal mutations on the second chromosome.

In a three-factor cross the hum1 mutation was mapped by recombination between 1(2)43Bd and so (Table 3). Since Df(2R)NCX9 and Df(2R)NCX11 complement lethal hum alleles for viability, but are so,
The viabilities of so alleles interact with so deletions; numbers of non-Cy progeny from crosses between mutations balanced over CyO, except for those involving so1 and so5, which were homozygous

<table>
<thead>
<tr>
<th>so1</th>
<th>so2</th>
<th>so3</th>
<th>so4</th>
<th>so5</th>
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<tbody>
<tr>
<td>197/477</td>
<td>133/308</td>
<td>0/1324</td>
<td>0/1159</td>
<td>136/437</td>
</tr>
<tr>
<td>0/205</td>
<td></td>
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</table>

TABLE 8

Viabilities of Drl with mutations of so; number of non-Cy progeny between crosses of Drl to so/CyO or so homozygotes

<table>
<thead>
<tr>
<th>so1</th>
<th>Drl</th>
</tr>
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<tbody>
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</tbody>
</table>

<table>
<thead>
<tr>
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<th>Drl</th>
</tr>
</thead>
<tbody>
<tr>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>so3</th>
<th>Drl</th>
</tr>
</thead>
<tbody>
<tr>
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<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>so4</th>
<th>Drl</th>
</tr>
</thead>
<tbody>
<tr>
<td>61/161</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>so5</th>
<th>Drl</th>
</tr>
</thead>
<tbody>
<tr>
<td>27/356</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>so6</th>
<th>Drl</th>
</tr>
</thead>
<tbody>
<tr>
<td>0/108</td>
<td></td>
</tr>
</tbody>
</table>

* Eyes absent or very small, some eye outgrowths, no ocelli.  
* Eyes absent or very small, ocelli reduced in size, but present.  
* Eyes very small, ocelli reduced in size, but present.

their breakpoints lie between hum and so and the hum lethality maps to the left of so. These deletions, however, fail to complement the sterility of hum1 and hum65 strains. Thus, the female sterility maps distal to the lethal function. All so alleles are fertile in hum1/so females, so that the distal, female-sterile, function of hum is distinct from the so locus. However, Drl and Drl-revertant chromosomes fail to complement the female sterility of the hum1 and hum65 chromosomes (see below). The Drl chromosome may, therefore, carry a lesion affecting the distal, female-sterile, function of the hum locus, in addition to its so mutation.

Drop elite is a gain of function allele of sine oculis:
The most obvious phenotypes of flies mutant for sine oculis are the loss of their ocelli and partial or complete loss of their eyes. Both viable and recessive lethal alleles of so have been recovered in this study (Table 7). Overlapping deletions that include the so locus are lethal, e.g., Df(2R)NCX9 or Df(2R)NCX11 heterozygous with Df(2R)sple-D2, Df(2R)sple-J1 or Df(2R)sple-J2 (totals summed over all the sple deletions with Df(2R)NCX9 and Df(2R)NCX11 respectively being 0/618 and 0/401). All alleles of so that are lethal with so7 deletions are also lethal inter se.

Three alleles of so (soo1, soo2 and soo5) have been recovered in F2 screens of EMS-treated chromosomes over Df(2R)ST1 (soo1, soo4) or Df(2R)pk-78k (soo6). In addition a weak, viable allele (soo5) was recovered in an F1 EMS screen against Df(2R)NCX9.

soo1 is a spontaneous mutation. About 80% of homozygous soo1 adults completely lack eyes and ocelli. In the remaining flies, one or both eyes are reduced to isolated ommatidia in the posterior region of the normal eye. About 5% of the eyes have outgrowths of omatidial-like cells, these tend to be at the posterior region of the eye. In place of corneal tissue, a disorganized patch of bristle-bearing cuticle is formed. It seems likely, therefore, that the eyeless phenotype may be associated with an alteration in cell fate.

The sooo1 mutation is not completely recessive as about 10% of eyes in sooo1 flies show ectopic bristles or limited corneal outgrowths. sooo1 or sooo7 flies have eyes of intermediate size, slightly reduced ocelli and 90% of the flies show corneal outgrowths. Similar phenotypes are seen in soo1/Df(2R)sooo7 flies but not in +/Df(2R)sooo7 flies. Since partial dominance and corneal outgrowths of soo1 are not seen in Df(2R)sooo7/+ flies, sooo1 is not a simple hypomorphic mutation. Either sooo1 has acquired a new function (it is a neomorph) or there is an unlinked dominant enhancer of the mutant phenotype in the soo1 stock. The sooo1 chromosome has reduced viability at high temperatures (RANSOM 1980), but is not lethal (LINDSLEY and ZIMM 1992).

A hypomorphic allele, sooo2, is a spontaneous partial reversion of sooo1 (LINDSLEY and ZIMM 1992). Homozygous sooo2 flies have normal, or slightly reduced, eyes but no ocelli. Df(2R)sooo2/sooo2 flies have small rough eyes and no ocelli. In sooo7/sooo7 flies the eyes are of intermediate size and the dominant sooo7 phenotype of corneal disruption and outgrowths is enhanced, so that 50% of eyes are affected to a variable extent. A third viable allele, sooo5, gives small rough eyes and reduced ocelli in Df(2R)sooo5/sooo5, sooo7/sooo7 and sooo1/sooo7 flies. The sooo7 mutation, however, complements the sooo1 and sooo2 mutations completely, i.e., sooo1/sooo2 and sooo7/sooo7 flies are indistinguishable from sooo1/+ and wild-type flies, respectively.

The dominant EMS-induced Drl mutation causes an extreme reduction in the size of the eye when heterozygous, similar to that of homozygous sooo1 flies. However, the ocelli are not severely reduced in Drl/+ flies. Drl is lethal when homozygous, when heterozygous with sooo7 deletions or with lethal alleles of so (Table 8). In Drl/sooo1 flies both the eyes and ocelli are very reduced. Drl flies with three copies of sooo1 (e.g., Drl/Dp(2;2)Scooo1TE35A-14R46, Dp(2;2)P32+/-) have a weak eye phenotype that is approaching wild-type. Drl flies with two copies of sooo1 (e.g., Drl/Dp(2;2)Scooo1TE35A-4R or Drl/Dp(2;2)Scooo1TE35A-4R46) have a moderate eye phenotype, less severe than that of Drl/+ . The suppression of the phenotype of
Drl by extra copies of so+ suggests that Drl may be an antimorphic allele of so.

A number of phenotypic revertants of Drl were recovered in X-ray screens (see above). Twenty-six of these are phenotypically wild-type in Drl+/+ flies. Three of the revertant chromosomes, however, are only partial and retain mutations giving a reduction in eye size (T(2;3)Dr1^w10, Dr1^w8 and Dr1^w27, Table 1). All except four of the revertants that are associated with chromosome aberrations have a breakpoint close to, or delete, 43B1-3.

The majority of revertants, including all those that carry cytologically visible deletions in 43B-C (Table 1), give a typical so1 phenotype, with neither eyes nor ocelli, when heterozygous with the so1 chromosome. That is to say, these revertants behave as so− chromosomes. Nine revertants, however, give either normal eyes and reduced ocelli (Dr1^w9, Dr1^w10, Dr1^w11 and Dr1^w16) or reduced eyes and reduced (Dr1^w8, Dr1^w31) or absent (T(2;3)Dr1^w1, Tp(2;2)Dr1^w23) ocelli in Dr1+/so1 flies. Thus, the eye and ocellar phenotypes of so are genetically separable.

All the revertant chromosomes, including the four (ru4, ru10, ru11 and ru14) on cytologically normal chromosomes that give normal eyes when heterozygous with so1, remain lethal when heterozygous with an so− deletion. Thus, although some of the revertants almost completely complement the visible phenotype of so1, the recessive lethality of the Drl mutation is retained.

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**A set of 175 mutations, including 46 chromosomal deletions define 27 complementation groups within the 43A-E interval. In the proximal part of this region, it is likely that most of the complementation groups have been identified, as multiple alleles of most have been recovered (Table 2). In the distal region, however, several loci are defined by only single alleles and additional complementation groups may remain to be discovered.**

**Mutant alleles of necrotic result in an early adult death, within 24 hr of eclosion the flies develop melanicotic spots. The complementation seen between weak and strong nec alleles is peculiar, since it indicates that the strong alleles retain some nec function. Nevertheless, the strong alleles of nec have, when heterozygous with nec− a similar phenotype to that seen in nec− homozygotes. A pattern of complementation between amorphic and hypomorphic alleles similar to that seen at nec has been described at the engrailed (en) locus (Gubb 1985). The en1 mutation gives a strong phenotype when heterozygous with a deletion, but when heterozygous with amorphic, lethal alleles the en1/i(2)en flies are almost wild-type. This pattern of complementation does not reflect residual en+ activity in the lethal alleles (Condé and Brower 1989).**

If some of the amorphic nec alleles do correspond to complete lack of nec+ activity, then the partial complementation between amorphic and hypomorphic alleles requires an alternative explanation. The formal possibilities are either that the deletion chromosomes remove a related gene that acts as a suppressor of nec, or that the level of nec+ activity is modulated by feedback repression that is sensitive to the number of copies of the locus.

Given that the nec and pk alleles on the In(2R) nec^pk1 and In(2R) nec^pk2 chromosomes are hypomorphic, the double mutations cannot result from small deletions that include both loci being associated with the inversion breakpoints. A likely explanation is that the double mutant phenotypes result from position effect variegation, which can spread linearly along a chromosome from a heterochromatic breakpoint (see Henikoff 1990 for a recent review); both the In(2R) nec^pk1 and In(2R) nec^pk2 have breakpoints that juxtapose the 43A region to the centric 2R heterochromatin (Table 1).

There is a complex pattern of complementation between pk and sple alleles. In classical genetic terms these correspond to two adjacent genes that can be separated either by recombination (Gubb and García-Bellido 1982) or by aberration breakpoints. Moreover, there is complete complementation between single alleles at these two loci. The two mutant phenotypes are similar, however, although they affect reciprocal regions of the body. The striking observation is that double-mutant alleles, which fail to complement both single pk and single sple alleles, can be recovered at a high frequency.

These three classes of mutation, two single and one double, might be affecting a single, complex, transcription unit. An alternative possibility is that a cluster of transcription units controlling related genetic functions is involved. Distinguishing these possibilities must await the molecular analysis of these genes.

The sine oculis gene, hitherto known for its visible phenotypes (the loss of the eyes and ocelli) is shown to be a vital locus. Not only are deletions that only, as far as is known, overlap the so locus lethal, but also lethal EMS-induced so alleles are not uncommon.

During pupal development, the so1 phenotype is associated with extensive cell death in the optic lobes (Fischbach and Technau 1984). This cellular degener-
eration results from an increase in death of the precursor neurons compared with normal development (Fischbach 1983) and leads to a reduced number of axons in the anterior optic tract (Fischbach and Lyly-Hünerberg 1983). A discrete group of lamina precursor cells fail to enter their final S phase in the brains of so larvae (Selleck et al. 1992). A similar failure in the final divisions posterior and anterior to the morphogenetic furrow during imaginal disc development might result in incorrect specification of the presumptive corneal cells.

The Drl mutation is a dominant antimorphic allele of so. This is shown by the interactions between Drl and so mutations and by the properties of Drl revertants. Drl fails to complement the sterility of female sterile mutations of the adjacent hum locus and may be associated with a small deletion or other chromosome aberration or be genetically complex. The complex pattern of complementation between the Drl, Df(2R)NCX9 and Df(2R)NCX11 chromosomes and the different classes of hum and so alleles suggests that there may be several functionally related transcripts in the hum-so interval. In particular, there appear to be lethal and viable morphogenetic functions at the so locus that are partially complementary.

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LITERATURE CITED


Sharma, R. P., P. R. Chittnis and J. Shyngle, 1985 Spiny ingransite orientation reversal (spil°)—a new temperature sensitive


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