The Molecular Analysis of the \textit{el-noc} Complex of \textit{Drosophila melanogaster}

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ABSTRACT

The \textit{el-noc} complex spans a distance of about 200 kb on chromosome 2L. It consists of three discrete genetic regions \textit{el}, \(l(2)35Ba\) and \textit{noc}, each of which has a distinct phenotype when mutant. The \textit{noc} locus itself is complex, including three separate regions. The \textit{el} locus has been characterized by mapping 30 aberration breakpoints to the DNA. It extends over a distance of about 80 kb. It can be divided into two parts by the aberrations \textit{In}(2LR)\textit{DTD}128 and \textit{T}(Y;2)\textit{ABO}. These break between two sets of \textit{el} alleles yet are both phenotypically wild type for elbow. The simplest explanation is that \textit{el} consists of two transcription units \textit{elA} and \textit{elB}. The locus \textit{pu}, which appears to be unrelated to the \textit{el-noc} complex, is found to map between the two \textit{el} loci very close to \textit{elB} (the distal \textit{el} locus). The loci \(l(2)35Ba\) and \textit{noc} have been separated by only two \(l(2)35Ba\)\textit{noc} deletions and a \textit{noc} inversion. No \(l(2)35Ba\)\textit{noc} aberrations have been found. At the molecular level these loci are found to occupy almost the same region, and are probably identical.

The \textit{el-noc} region has been extensively described (Woodruff and Ashburner 1979a; Ashburner, Aaron and Tsubota 1982; Ashburner, Tsubota and Woodruff 1982) and includes at least three distinct genetic loci, \textit{elbow (el)}, \(l(2)35Ba\) and \textit{no-ocelli (noc)}, all separable by chromosomal aberration breakpoints. Mutations of \textit{el} show a wing and haltere phenotype, mutations of \(l(2)35Ba\) are larval lethals and mutations of \textit{noc} lack the ocelli. All three of these loci show partial failure of complementation. Flies heterozygous for \(l(2)35Ba\) and \textit{noc} or \textit{el} alleles frequently have weak phenotypes characteristic of either, or both, of these loci. Alleles of \textit{noc} heterozygous with some \textit{el} alleles have weak no-ocelli phenotypes. \(l(2)35Ba\) homozygotes are usually lethal, but some, e.g., \(l(2)35Ba\text{GR}^{10}\), are hypomorphic lethals and can survive at low frequency when heterozygous with a \(l(2)35Ba\) "deletion. The escapers usually have a weak no-ocelli phenotype, if the deletion is also \textit{el} then they also have a weak elbow phenotype. Flies which are heterozygous for different mutant \(l(2)35Ba\) alleles may survive at low frequency and have a weak elbow and a weak no-ocelli phenotype. These three loci also interact with the dominant antimorphic mutation \textit{Tp}(2;2)\textit{Scutoid (Sco)}, an X-ray-induced mutation (Krivshenko 1959) that is associated with a reciprocal transposition between \textit{noc}, \textit{osp} (outsread) and \textit{Adh} (Alcohol dehydrogenase) and six loci from 35D (Ashburner, Tsubota and Woodruff 1982; McGill et al. 1988). The Scutoid phenotype is a loss of bristles from the head and thorax of the adult fly. The wild-type number of these bristles is 40, whereas in \textit{Sco}/+ flies the number is reduced to between 25 and 29 (Ashburner, Tsubota and Woodruff 1982). \textit{Sco} is a recessive lethal, although very occasional \textit{Sco} homozygotes (11/13,475) survive to adulthood. These flies have a very extreme Scutoid phenotype with only eight to ten bristles per fly. Some alleles of \textit{el}, all \(l(2)35Ba\) alleles and some \textit{noc} alleles act as dominant enhancers of the Scutoid phenotype, when heterozygous with the \textit{Sco} chromosome they reduce the number of bristles by between 5 and 15 per fly. When \textit{Sco} is heterozygous over deletions that include \textit{el}, \(l(2)35Ba\) or \textit{noc}, a progressive enhancement of the Scutoid phenotype is observed which is proportional to the number of these loci deleted. The maximal enhancement is observed when all three are deleted; these flies have a similar phenotype to \textit{Sco} homozygotes. Duplications of the region that include \textit{el}, \(l(2)35Ba\) and \textit{noc}, e.g., \textit{Dp}(2;2)\textit{Adh3}, partially suppress the Scutoid phenotype, increasing the number of bristles to about 34 per fly (Ashburner, Tsubota and Woodruff 1982). These data suggest that \textit{el}, \(l(2)35Ba\) and \textit{noc} comprise a genetic complex.

The proximal part of this complex, including the \textit{noc} locus, was cloned by Chia et al. (1985a). In this study the cloning of the remaining part of the complex is described. The relationships of the loci comprising the complex are discussed both with respect to each other and to the neighboring loci. The genetic relationships of these loci, based upon chromosomal aberrations broken within this region, are shown in Figure 1.

MATERIALS AND METHODS

\textbf{Drosophila stocks:} The chromosomes used in this study are listed in Table 1. The \textit{A} series of deletions were X-ray-induced \textit{Adh} null mutations (Aaron 1979; Ashburner,
T. Davis, J. Trenear and M. Ashburner

Figure 1.—Genetic map of the region surrounding el on chromosome 2L of D. melanogaster. The genetic loci are l(2)34Fb, l(2)34Fc, l(2)34Fd, ms(2)34Fe, elbow B (elB), pupal (pu), elbow A (elA), l(2)35Aa and no-ocelli A (nocA). The genetic breakpoints of deletions, inversions and translocations are shown; all aberrations are deletions unless otherwise indicated. Those breakpoints located on the DNA cloned are indicated by double vertical lines.

Preparation of DNA from Drosophila: DNA was prepared from adult flies as described by Chia et al. (1985a). Preparations of DNA from phage: Late log phase cultures of Escherichia coli K802 grown in 10 g/liter Bacto-tryptone, 5 g/liter yeast extract, 5 g/liter NaCl, 10 mM-MgSO4 were infected with phage at a multiplicity of infection of either 1 (Charon 4 phage) or 0.1 (EMBL3 or EMBL4 phage). Growth was allowed to proceed for 1 to 4 hr at 37° with aeration until cell lysis was apparent. Cellular debris was removed by centrifugation at 5000 X g for 10 min. The phage in the supernatant were concentrated by the addition of polyethylene glycol 6000 and NaCl as described by Maniatis, Fritsch and Sambrook (1982) and were further purified on a CsCl step gradient with step densities of 1.3, 1.5 and 1.7 g/ml followed by centrifugation on CsCl equilibrium gradient. The purified phage bands were dialyzed against 10 mM Tris-HCl (pH 8.0), 1 mM-EDTA.

Preparation of probes: Probes were prepared as described in Chia et al. (1985a).

Chromosome walking, screening and restriction mapping: Chromosome walking was performed essentially as described by Bender, Spierer and Spierer (1983) and Bender et al. (1983). In this study three libraries have been used, the LAUER-MANIATIS library (Maniatis et al. 1978), a library made from Canton S DNA in EMBL4 phage (provided by M. Goldberg) and a library constructed by J. Tamkun from a stock made by J. Kennison carrying the markers y; en bu sp and isogenic for all four chromosomes (in EMBL3 phage) (J. Tamkun, personal communication). Phage were mapped with EcoRI, HindIII, Sal1 and XhoI by running all possible single and double digests on a single agarose gel.

Mapping breakpoints by Southern hybridization: Nick-translated clones were hybridized to enzyme-digested DNA prepared from flies bearing chromosomes that were suspected to have their breakpoints lying within the cloned region of DNA. All lethal chromosomes used were balanced over In(ZLR)O, Cy dppr AdhA pr cn2 (CyO nB) or In(ZLR)O, Cy dp + pr cn2 (CyO) unless otherwise stated. Parental chromosomes were homozygous except TE36(R) and TE146(Z), which carry lethal mutations. Since many of the aberrations that have been analyzed were induced on known progenitor chromosomes (Table 1), direct comparison of the aberrations with their precursors was usually possible. The Southern hybridizations were performed as described in Chia et al. (1985a). Restriction enzyme digests of genomic DNA were with EcoRI unless otherwise stated.
TABLE 1

Chromosomes used in this study, grouped according to their progenitor chromosomes

<table>
<thead>
<tr>
<th>Aberration</th>
<th>Cytology</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) On b Adh' cn bw</td>
<td>Df(2L)35A4;35B2</td>
</tr>
<tr>
<td>Df(2L)A245</td>
<td>Df(2L)35B1.2</td>
</tr>
<tr>
<td>Df(2L)A260</td>
<td>Df(2L)35B2.5</td>
</tr>
<tr>
<td>Df(2L)A266</td>
<td>Df(2L)35A1-4;35B10</td>
</tr>
<tr>
<td>In(2L)e16</td>
<td>In(2LR)35B1.3;57C3-9</td>
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<tr>
<td>(2) On Adh07 cn</td>
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<tr>
<td>Df(2L)e14</td>
<td>Not cytologically visible</td>
</tr>
<tr>
<td>Df(2L)e16</td>
<td>In(2L)34A2.3;35A3.4</td>
</tr>
<tr>
<td>T(2;3)p24</td>
<td>T(2;3)35B1.3;59C3-7</td>
</tr>
<tr>
<td>(3) On Adh07 pr cn</td>
<td>Df(2L)35B3;35D4</td>
</tr>
<tr>
<td>Df(2L)e28</td>
<td>T(Y;2)Y.35B1</td>
</tr>
<tr>
<td>(4) On b Adh' cn bw</td>
<td>Df(2L)35B1:35C4.5</td>
</tr>
<tr>
<td>T(Y;2)e24</td>
<td>Df(2L)35B1:35C1</td>
</tr>
<tr>
<td>(5) On al dp b TE146(Z) pr l(2)pwn cn</td>
<td>Df(2L)35A3.4;35B2</td>
</tr>
<tr>
<td>Df(2L)TE146(Z)GW1</td>
<td>Not cytologically visible</td>
</tr>
<tr>
<td>Df(2L)TE146(Z)GW6</td>
<td>In(2L)35B1.2;40 + not cytologically visible</td>
</tr>
<tr>
<td>Df(2L)TE146(Z)GW7</td>
<td>Df(2L)35B1.2;35B8.9-C1</td>
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<td>In(2L)TE146(Z)GW13 + Df(2L)TE146(Z)GW13</td>
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<tr>
<td>T(Y;2)TE146(Z)GR18.9R15*</td>
<td></td>
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<tr>
<td>(6) On b chTm3c00 pr pk cn sp</td>
<td>Df(2L)35B1.2;35E1.2</td>
</tr>
<tr>
<td>Df(2L)TE36(R)GW8</td>
<td></td>
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<tr>
<td>(7) On Adh' cn</td>
<td>Df(2L)35A3;35B2</td>
</tr>
<tr>
<td>Df(2L)n2</td>
<td>Df(2L)35B1;35B3.4</td>
</tr>
<tr>
<td>Df(2L)n3</td>
<td></td>
</tr>
<tr>
<td>(8) On Adh' pr pk cn</td>
<td>Df(2L)34C3;35A4</td>
</tr>
<tr>
<td>Df(2L)84a2</td>
<td>from the stock z w17X</td>
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<tr>
<td>(9) On a known wild-type chromosome 2</td>
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</tr>
<tr>
<td>T(Y;2)GT2</td>
<td>T(2;3)28BC;35B1.2;41;42F;98D</td>
</tr>
<tr>
<td>T(2;3)GT3</td>
<td>from the stock z w17X</td>
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<td>(10) On a known wild-type chromosome 2</td>
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</tr>
<tr>
<td>T(2;4)GT8</td>
<td>T(2;3)35A1-4;462F-6; + T(2;3)50B;81</td>
</tr>
<tr>
<td>T(2;3)GT10</td>
<td>T(2;3)35A1-4;76A-5</td>
</tr>
<tr>
<td>(11) No progenitor chromosomes available</td>
<td>Df(2L)35A4-B1;2;35B8.9-C1</td>
</tr>
<tr>
<td>Df(2L)35C7;8;35A-A + In(2L)35A-A + B;35D5-7</td>
<td>Df(2L)35A4-C1;35B8.9-C1</td>
</tr>
<tr>
<td>Df(2L)35D29a + In(2L)35D29a</td>
<td></td>
</tr>
<tr>
<td>Df(2L)35D29a + In(2L)83d29a</td>
<td></td>
</tr>
<tr>
<td>Df(2L)el'</td>
<td>Not cytologically visible</td>
</tr>
<tr>
<td>In(2L)83d22</td>
<td>In(2L)34D4.5;35C1.2</td>
</tr>
<tr>
<td>In(2L)DTD128</td>
<td>In(2LR)35B1.3;48C6-8</td>
</tr>
<tr>
<td>T(Y;2)A15</td>
<td>T(Y;2)B'VV'Y';35A4-B1.2</td>
</tr>
<tr>
<td>T(Y;2)A80</td>
<td>T(Y;2)YS;35A5.4</td>
</tr>
<tr>
<td>T(2;3)sho19</td>
<td>T(2;3)22F1.2;35A1-4;97A</td>
</tr>
</tbody>
</table>

Mapping breakpoints by hybridization in situ: Stocks in which the aberration of interest was balanced over a suitable balancer [usually CyO or In(2LR)Gla] were used for the mapping of the clones to polytene chromosomes. Salivary gland polytene chromosomes were prepared and hybridized with labeled probes as described by Gubb et al. (1984). Probes were labeled by nick-translation using DNA polymerase I according to the procedure of Gubb et al. (1984) using Biotin-16-UTP (Boehringer) at a final concentration of 0.1 μmol per ml in the presence of 30 μM each of dATP, dGTP and dCTP for 90 min at 16 °C. After hybridization to polytene chromosomes the hybridized DNA was visualized by staining with horseradish peroxidase. Chromosomes were washed twice in phosphate buffered saline (PBS) at room temperature for 5 min, followed by PBS containing 0.1% Triton X-100 for 2 min and rinsing in PBS. Chromosomes were then incubated in the presence of a 1:250 dilution of streptavidin-biotinylated peroxidase complex (Uniscience, Ltd.) for 30 min at 37 °C. After incubation the above wash steps were repeated. Chromosomes were then incubated at room temperature for 1–10 min in the presence of 0.5 mg per ml 3,3'-diaminobenzidine (Sigma) in PBS containing 0.01% H2O2. The staining reaction was stopped by immersion in water followed by immersion in PBS. Chromosomes were then stained with 5% Giemsa (Gubb et al. 1984).

RESULTS

The map: This study is an extension of the work started by Chia et al. (1985a) and McGill et al. (1988)
involving the molecular analysis of the Adh region of the genome of Drosophila melanogaster. CHIA et al. (1985a) cloned the genetic loci Adh, osp and noc in overlapping lambda phage by chromosomal walking. This walk finished 124 kb distal to the Adh locus with the phage λob10.02. In this study this walk has now been extended to 360 kb distal to Adh (Figure 2). Two strategies were employed: (1) the walk was extended from λob10.02 by chromosomal walking in the distal direction; (2) a bidirectional walk was initiated from the phage λds635 (provided by D. ANDREWS) which was thought to map to one of the loci immediately distal to the el-noc complex. The phage λds635 maps at -286.5 to -298.0 (Figure 2). The map coordinates are negative in the direction of the telomere.

All phage between λob10.02 and λb30.012 inclusive have been checked by hybridization to polytene chromosomes of a wild-type stock (Canton S) and cover the region 35A1.2 to 35B1.2. The phage λob18.42 has a second minor site at the polytene chromosome region 95E1.2 of 3R and the phage λds635 has a second site at 57E1-4 of 2R.

The positions of all breakpoints which are not deletions of genomic DNA have been confirmed by in situ hybridization to polytene chromosomes with a suitable probe.

The -110.0- to -150.0-kb region: This region is covered by the phage λob10.02 to λb14.04 (Figure 2) and includes the distal part of nocA, l(2)35Ba and the proximal part of the el locus. The proximal limit for the locus l(2)35Ba is defined by the l(2)35Ba+ noc- deletion Df(2L)A446 at coordinates -113.5 to -117.5 (CHIA et al. 1985a) and the most proximal of the l(2)35Ba+ noc- deletions Df(2L)Adh741 at -119.5 to -121.8 (McGill 1985). The distal limit of l(2)35Ba is defined by the el+ l(2)35Ba+ deletion Df(2L)b84a2. When DNA from Df(2L)b84a2/CyO digested with EcoRI is probed with the 4.6-kb EcoRI fragment of λob11.02 (Figure 4) two fragments are seen, the 4.6-kb parental fragment and a fusion fragment of 3.3 kb (Figure 3, lane 4). This is not seen in the parental DNA (Figure 3, lane 5). When Df(2L)b84a2 is heterozygous over Df(2L)fn3 (an el+ l(2)35Ba- deletion) the 4.6-kb fragment is absent but the 3.3-kb fragment is present. In addition a second fragment of 1.5 kb is found (Figure 3, lane 3). This is also present in the Df(2L)fn3/CyO lane (Figure 3, lane 2) and is thus the fusion fragment of Df(2L)fn3. Therefore both Df(2L)b84a2 and Df(2L)fn3 are broken in the 4.6-kb EcoRI fragment at -121.8 to -125.1 and -124.9 to -126.4, respectively.

Four other el- l(2)35Ba- aberrations are broken within this region, Df(2L)A245, Df(2L)A260, Df(2L)-A266 and the synthetic deletion T(Y;2)TE146(Z)GR18 R15P. Df(2Lvn3/CyO nB EcoRI-digested DNA probed with the 7.2-kb EcoRI fragment of λob12.07 (Figure 2) gives the 7.2-kb parental and a 3.2-kb fusion fragments (Figure 3, lane 7). This deletion is broken between -134.6 and -137.8. DNA from Df(2L)A260 and Df(2L)A266 [both heterozygous with Df(2L)b84a2] probed with the 1.9-kb and 2.4-kb EcoRI fragments of λob14.21 (Figure 5) show only the 2.4-kb fragment (Figure 3, lanes 9 and 10). There are no fusion fragments visible, therefore these deletions are broken close to the EcoRI site at -144.4. These deletions include the genetic loci l(2)35Ba, noc, osp and Adh but do not include l(2)35Bb the next most proximal locus. As the distal endpoints of these deletions are indistinguishable, it is possible that they are re-isolates of the same event.

DNA from flies heterozygous for T(Y;2)TE146(Z)GR18 R15 and Df(2L)b84a2 lacks the 2.8-kb, 1.5-kb and 7.2-kb fragments when probed with λob12.07 but retains the distal 3.9-kb fragment (Fig-
The EcoRI fragment at -141.7 is present on the EcoRI-digested genomic DNA probed with purified DNA fragments or λ phage covering the region of the walk between -110.0 and -150.0. The sizes of wild-type restriction enzyme fragments are indicated; f indicates a fusion fragment. The EcoRI site at -19.5. The significance of this translocation is discussed later.

Two el(l(2)35Ba" deletions, Df(2L)TE146(Z)GW1 and Df(2L)TE146(Z)GW6 are broken in the 4.0-kb EcoRI fragment of λob14.21 (Figure 5) and have fusion fragments of 4.5 kb and 5.2 kb, respectively (Figure 3, lanes 14 and 15). The fusion fragment of Df(2L)TE146(Z)GW6 gives a slightly weaker band of hybridization than that of Df(2L)TE146(Z)GW1, suggesting that the distal breakpoint of Df(2L)TE146(Z)GW6 is more distal than that of Df(2L)TE146(Z)GW1. The distal limits of these deletions are between -146.8 and -150.8.

The l(2)35Ba" noc- translocation T(2;3)GT8 probed with the 8.5-kb EcoRI fragment of λob10.02 (Figure 4) has the parental fragment of 13.0 kb and fusion fragments of 1.5 kb and 20.0 kb (Figure 3, lane 17). This aberration is therefore broken within 1.5 kb of the EcoRI site at -119.5. The significance of this translocation is discussed later.

Two el" alleles also have their breakpoints in this region. The spontaneous allele el" is homozygous viable and has a strong elbow phenotype. The DNA from el" completely lacks homology with λob14.21 (data not shown). Thus the el" chromosome carries a deletion in this region. The proximal end of the el" deletion is found in the 6.4-kb EcoRI fragment of λob13.01 (Figure 5). When el" DNA is probed with this fragment, a 3.9-kb EcoRI fragment is present (Figure 6, lane 1). The 3.9-kb fragment is due to a polymorphic site at -141.7. DNA also lacks the 5.6-kb HindIII fragment but retains the adjacent 5.8-kb fragment (Figure 6, lane 2). With XhoI only a fusion fragment of 19.0 kb is seen (Figure 6, lane 3). Therefore el" is broken in the 11.5-kb XhoI fragment close to the EcoRI site at -141.7. DNA from T(2;3)el24/CyO probed with the 5.4-kb HindIII fragment of λob11.02 (Figure 4) has fusion fragments in a XhoI digest of 8.4 kb and 13.0 kb (Figure 3, lane 19). The parental XhoI fragment is 6.5 kb. This translocation is broken between -126.0 and -131.4.

The -150.0- to -200.0-kb region: This region is covered by the phage λob15.85 to λob17.41 (Figure 2) and includes most of the el locus and the proximal part of the pu locus. Four el" l(2)35Ba" deletions that are wild type for pu have been analyzed, Df(2L)el28,
Figure 4.—Expanded map of the region covering the phage \( \text{ob10.02} \) and \( \text{ob11.02} \). The coordinates are shown above the EcoRI (E) sites. Only the relevant HindIII (H) and XhoI (X) sites are shown.

Figure 5.—Expanded map of the region covering the phage \( \text{ob13.01}, \text{ob14.21} \) and \( \text{ob15.85} \). The coordinates are shown above the EcoRI sites. Sites for HindIII (H), SalI (S) and XhoI (X) are also shown.

\( \text{Df(2L)TE36(R)GW8}, \text{Df(2L)el14} \) and \( \text{Df(2L)el16} \). DNA from \( \text{Df(2L)el28(Df(2L)h84a2} \) heterozygotes when probed with \( \text{ob14.21} \) (Figure 5) lacks the 6.4-kb, 2.4-kb and 4.0-kb fragments but retains the 3.6-kb and 18.0-kb fragments (Figure 7, lane 2). The 0.5-kb fragment is not seen and there is no fusion fragment, therefore the breakpoint of \( \text{Df(2L)el28} \) is between \(-150.8 \) and \(-151.3 \).

DNA from \( \text{Df(2L)TE36(R)GW8(CyO)} \) has been digested with SalI and XhoI and probed with the 8.7-kb XhoI fragment of \( \text{ob15.85} \) (Figure 5). The parental strain has 26.0-kb, 13.5-kb and 12.5-kb SalI fragments (Figure 7, lane 3). The 13.5-kb and 12.5-kb fragments are due to a polymorphic site on the CyO nB chromosome within the 26.0-kb fragment (Figure 5). The \( \text{Df(2L)TE36(R)GW8} \) chromosome lacks the 26.0 kb and has a 9.0-kb fusion fragment (Figure 7, lane 4). This chromosome also has a 7.5-kb XhoI fusion fragment (Figure 7, lane 6). The deletion is broken between \(-166.1 \) and \(-170.1 \).

\( \text{Df(2L)el16(CyO)} \) gives a 7.8-kb fusion fragment when probed with the 5.4-kb EcoRI fragment of \( \text{ob17.41} \) (Figure 8a) and is therefore broken between \(-187.9 \) and \(-193.3 \) (Figure 7, lane 8). HindIII-digested DNA from \( \text{Df(2L)el14(CyO)} \) gives a fusion fragment of 5.8-kb (Figure 7, lane 12) when probed with the 4.2-kb HindIII fragment of \( \text{ob18.42} \) (Figure 8a). This deletion is broken between \(-196.0 \) and \(-200.2 \).

The neutron-induced deletion \( \text{Df(2L)h83d29a} \) includes \( \text{pu} \) and at least 18 more distal loci. The deletion is associated with a small inversion between 35A-B (proximal to \( \text{pu} \)) and 35D5-7. As there is no parental chromosome available for this deletion, the inversion \( \text{In(2L)b83b22} \), which was induced on the same progenitor chromosome, has been used as a control. When DNA from \( \text{Df(2L)b83d29a(In(2LR)SM5)} \), \( \text{at}^2 \text{Cy} \) \( \text{el}^2 \) \( \text{cn}^2 \) \( \text{sp}^2 \) is probed with the 9.6-kb EcoRI fragment of \( \text{ob17.36} \) (Figure 8b) a fusion fragment of 7.4 kb is seen (Figure 7, lane 13). The 9.6-kb fragment is due to a polymorphic EcoRI site in the 16.0-kb EcoRI fragment at \(-178.3 \) (Figure 8b). With DNA cut with XhoI a fusion fragment of 16.0 kb is seen (Figure 7, lane 14). These are not found with \( \text{Zn(2LR)el6} \) (Figure 7, lanes 15 and 16) therefore this aberration breaks between \(-178.3 \) and \(-185.7 \). The fusion fragments are from the 35A-B:35D5-7 inversion.

Four other breakpoints are located in this region, two of which \( \text{[el' and In(2LR)el6]} \) are associated with \( \text{el}^2 \) alleles. The distal breakpoint of \( \text{el}^2 \) is broken in the 8.7-kb XhoI fragment of \( \text{ob15.85} \) (Figure 5). When DNA from \( \text{el}^2 \) is probed with this fragment, the only fragments seen are the 8.7-kb EcoRI (due to a polymorphic EcoRI site at \(-164.4 \), see Figure 5) and 20.7-kb HindIII parental fragments and a 19.0-kb XhoI fusion fragment (Figure 6, lanes 4, 5 and 6). The 19.0-kb XhoI fragment is the fusion fragment seen with the 6.4-kb EcoRI fragment of \( \text{ob13.01} \) (Figure 6, lane 3). Therefore \( \text{el}^2 \) is broken close to the HindIII
In(2LR)DTD128/CyO has been digested with four different restriction enzymes. The sizes in kb of wild-type restriction enzyme fragments are indicated; f indicates a fusion fragment. The el" chromosome has an EcoRI site at -141.7. (a) Probed with the 6.4-kb EcoRI fragment of λob13.01; EcoRI-digested DNA (track 1); HindIII-digested DNA (track 2); XhoI-digested DNA (track 3); (b) probed with the 8.7-kb XhoI fragment of λob15.85 (tracks 4, 5 and 6 as for tracks 1, 2 and 3).

FIGURE 6.—Southern transfers of genomic DNA from Df(2L)el'. The sizes in kb of wild-type restriction enzyme fragments are indicated; f indicates a fusion fragment. The el" chromosome has an EcoRI site at -141.7. (a) Probed with the 6.4-kb EcoRI fragment of λob13.01; EcoRI-digested DNA (track 1); HindIII-digested DNA (track 2); XhoI-digested DNA (track 3); (b) probed with the 8.7-kb XhoI fragment of λob15.85 (tracks 4, 5 and 6 as for tracks 1, 2 and 3).

The sizes in kb of wild-type restriction enzyme fragments are indicated; f indicates a fusion fragment. The el" chromosome has an EcoRI site at -141.7. (a) Probed with the 6.4-kb EcoRI fragment of λob13.01; EcoRI-digested DNA (track 1); HindIII-digested DNA (track 2); XhoI-digested DNA (track 3); (b) probed with the 8.7-kb XhoI fragment of λob15.85 (tracks 4, 5 and 6 as for tracks 1, 2 and 3).

The other el" defining breakpoint, T(Y;2)A80, induced by LINDSLEY et al. (1972), was also genetically mapped between pu and el. DNA from T(Y;2)A80/ Df(2L)b84a2 probed with λob16.19 (Figure 8b) lacks the 8.5-kb fragment but retains the 5.4- and 9.5-kb fragments (due to a polymorphic site at -178.3 in the 16.0-kb fragment, Figure 8b). The 16.0-kb fragment is also present in this DNA therefore some of the chromosomes lack this polymorphic site. Fusion fragments of 19.0 and 20.0 kb are present (Figure 9, lane 5). With HindIII-digested DNA the parental 20.7-kb fragment is missing but fusion fragments of 11.5 and 25.0 kb are found (Figure 9, lane 6). Thus the translocation is broken between -167.1 and -172.9. This has been confirmed by in situ hybridization to T(Y;2)A80 polytene chromosomes using λob16.19 as a probe (Figure 11c). The deficiency Df(2L)ARR1 is an X-ray-induced reconstitution of chromosome 2 from the Y2o element of the translocation T(Y;2)A80 and the 2n0 element of the translocation T(Y;2)RI5 (WOODRUFF and ASHBURNER 1979a; GUBB et al. 1984). Thus it has only the distal element of the translocation T(Y;2)A80. When DNA from Df(2L)ARR1/CyO is probed with λob16.19 the EcoRI fusion fragment of 20.0 kb and the HindIII fusion fragment of 25.0 kb are seen (Figure 9, lanes 7 and 8). This confirms that T(Y;2)A80 breaks between -167.1 and -172.9.

The -200.0- to -260.0-kb region: This region is covered by the phage λob18.42 to λob22.36 (Figure 2) and includes the distal part of the el locus, the pu locus and the proximal part of the locus l(2)35Aa. Four aberrations T(Y;2)el4, T(Y;2)A15, T(2;3)shv19 and In(2L)el9 are broken in this region. DNA from flies heterozygous for T(Y;2)el4 with Df(2L)b84a2 when probed with λob18.42 (Figure 8a) lacks the 2.7-kb and 0.5-kb fragments but retains the 1.9-kb, 5.2-kb, 2.5-kb, 1.7-kb and the 18.0-kb fragments (Figure 10, lane 2). When probed with only the 2.7-kb fragment it lacks this fragment and no fusion fragments are present (data not shown). When λob18.42 is hybridized to polytene chromosomes of T(Y;2)el4 the T(Y;2) break is seen (Figure 11a). Thus the distal translocation breakpoint of T(Y;2)el4 is between -207.3 and -207.8, and the proximal translocation breakpoint is close to the EcoRI site at -204.6. This event is associated with a small deletion of about 2.7 kb. DNA from T(Y;2)A15/Df(2L)b84a2 was digested with EcoRI and SalI and probed with the 8.5-kb XhoI fragment of λob18.42 (Figure 8a). With EcoRI the 2.7-kb fragment is missing but the 2.5-kb, 1.7-kb and 18.0-kb fragments are retained (Figure 10, lane 3). In addition a single fusion fragment of 27.0 kb is found. With SalI the 8.1-kb fragment is missing and a fusion fragment of 27.0 kb is found (Figure 10, lane 4). As only one fusion fragment is found with each enzyme, it is
Figure 7.—Southern transfers of restriction enzyme-digested genomic DNA probed with purified DNA fragments or phage covering the region between −150.0 and −200.0. The sizes in kb of wild-type restriction enzyme fragments are indicated; f indicates a fusion fragment. The SalI site at −163.4 is present on the In(2LR)0, CyO dp14 pr cn chromosome. (a) EcoRI-digested DNA probed with λob14.04: AdhEnpr cn (track 1); Df(2L)el14, pr cn/ Df(2L)b83d2, AdhEnpr pk cn sp (track 2); (b) DNA probed with the 6.7-kb EcoRI fragment of λob13.85: SalI-digested b cy18.46 pr cn/sp/CyO nB DNA (track 3); SalI-digested Df(2L)TE16(R)GW8, b pr pk cn/sp/CyO nB DNA (track 4); Xhol-digested Df(2L)TE16(R)GW8, b pr pk cn/sp/CyO nB DNA (track 5); Xhol-digested Df(2L)TE16(R)GW8, b pr pk cn/sp/CyO nB DNA (track 6); (c) EcoRI-digested DNA probed with the 5.4-kb EcoRI fragment of λob18.41: AdhEnpr cn (track 7); Df(2L)el16, cn/CyO (track 8); (d) EcoRI-digested DNA probed with the 5.4-kb EcoRI fragment of λob18.41: b AdhEn cn bw (track 9); In(2LR)el6, b AdhEn cn bw/CyO (track 10); (e) HindIII-digested DNA probed with the 4.2-kb HindIII fragment of λob18.42: AdhEnpr cn (track 11); Df(2L)el14, AdhEnpr cn/CyO (track 12); (f) DNA probed with the 9.6-kb EcoRI fragment of λob17.36: Xhol-digested Df(2L)b83d29a, In(2L)b83d89a/SM5 DNA (track 13); EcoRI-digested Df(2L)b83d29a, In(2L)b83d29a/SM5 DNA (track 14); Xhol-digested In(2L)b83b22/SM5 DNA (track 15); EcoRI-digested In(2L)b83b22/SM5 DNA (track 16).

possible that the breakpoint is close to the EcoRI site at −207.6 and the fusion fragments with SalI are both the same size. This breakpoint is confirmed by in situ hybridization to polytene chromosomes, using λob18.42 as a probe (Figure 11d). The translocation T(2;3)shv19 is broken in the 8.2-kb SalI fragment of λob18.42 (Figure 8a). When DNA from T(2;3)shv19/CyO digested with EcoRI is probed with this fragment a single fusion fragment of 7.9 kb is found (Figure 10, lane 5). With SalI-digested DNA, fusion fragments of 3.7 kb and 14.0 kb are seen (Figure 10, lane 6). Therefore this translocation is broken close to one of the EcoRI sites between the limits −199.9 and −208.1. This is also confirmed by in situ hybridization (Figure 11f). In(2L)el9 is also broken in the 8.2-kb SalI fragment giving fusion fragments in a SalI digest of 6.1 kb and 11.0 kb (Figure 10, lane 10). No fusion fragments are seen in an EcoRI digest (Figure 10, lane 8). This inversion is broken between −199.9 and −208.1.

Also broken in this region are a λ(2)35Aa pu el-deletion, Df(2L)A400, and a λ(2)35Aa pu el− deletion, Df(2L)TE146(Z)GW7. DNA from Df(2L)A400/ CyO nB digested with Xhol and probed with the 5.0-kb EcoRI fragment of λob21.11 (Figure 2) has the parental fragments of 8.0 kb and 5.6 kb and an additional 6.5-kb fusion fragment (Figure 10, lane 12). The deletion is not broken in the 8.0-kb fragment (data not shown) so is therefore broken in the adjacent 5.6-kb XhoI fragment between −238.6 and −243.5.

Df(2L)TE146(Z)GW7/CyO DNA has a fusion fragment of 0.8 kb when probed with the 2.8-kb EcoRI fragment of λob22.35 (Figure 2) and is thus broken between −247.0 and −247.8 (Figure 10, lane 14).

The breakpoints of two translocations map genetically between the λ(2)35Aa and pu loci, T(Y;2)GT2 and T(2;3)GT3. Both of these are γ-ray-induced translocations, and neither are mutant for pu or λ(2)35Aa (GuBB et al., unpublished data). DNA from T(Y;2)GT2/Df(2L)b83a2 digested with EcoRI and probed with the 9.0-kb EcoRI fragment of λob20.43 (Figure 2), lacks the parental fragment of 18.0 kb and has fusion fragments of 19.5 kb and 28.0 kb (Figure 10, lane 16). Only the 19.5-kb fusion fragment is seen with λob19.48 (data not shown) therefore this translocation breaks between the end of λob19.48 (−220.9) and the EcoRI site at −226.0 (see Figure 11c for in
situ results). When DNA from $T(2;3)GT3/CyO$ is probed with the 5.6-kb EcoRI/HindIII fragment of $\lambda ob21.11$ (Figure 2), EcoRI fusion fragments of 5.3 kb and 8.8 kb are found (Figure 10, lane 18). Thus the breakpoint of $T(2;3)GT3$ is between $-234.4$ and $-240.0$.

The $\gamma$-ray-induced translocation $T(2;3)GT10$ is broken in the 6.5-kb SalI fragment of $\lambda ob22.35$ (Figure 2) giving SalI fusion fragments of 6.2 kb and 11.5 kb (Figure 10, lane 20) and is therefore broken between $-253.5$ and $-260.0$. This translocation is broken distal to the locus $l(2)34Fd$ (Figure 12).

Distal to $\lambda ob22.35$ a further 100 kb of DNA have been cloned in overlapping phage (Figure 2). The only aberration that has been mapped to this DNA is the translocation $T(2;4)GT6$. This translocation falls between the loci $l(2)34Fb$ and $l(2)34Fc$ (Figure 1). By in situ hybridization to polytene chromosomes, the breakpoint of $T(2;4)GT6$ is broken within the DNA covered by the phage $\lambda ob22.35$ and $\lambda ob22.29$, i.e., between $-241.0$ and $-273.8$ (data not shown).

**FIGURE 8.**—Expanded maps of the regions covered by (a) the phage $\lambda ob17.41$, $\lambda ob18.41$ and $\lambda ob18.42$. (b) The phage $\lambda ob16.19$ and $\lambda ob17.36$. The coordinates are shown above the EcoRI (E) sites, only relevant HindIII (H), SalI (S), XbaI (Xb) and XhoI (X) sites are shown.

**DISCUSSION**

A summary of the molecular relationships of the aberrations mapped in this study to each other and to the genetic loci is given in Figure 12.

**The relationship of noc to $l(2)35Ba$:** The molecular limits of the noc locus are between the noc$^+\ osp^-$ deletion $Df(2L)f{n}52$ at $-52.8$ to $-57.4$ (proximal limit) (CHIA et al. 1985a) and the $l(2)35Ba^-\ noc^-$ deletion $Df(2L)Adh^n$ at $-119.5$ to $-121.8$ (distal limit) (MCGILL 1985). However, the chromosome 2 breakpoint of the translocation $T(2;3)Mpe$ is between $-98.5$ and $-103.0$ (CHIA et al. 1985a) yet this translocation is completely wild type for no-ocelli. In other words a translocation which breaks within sequences required for noc function is not itself mutant for noc. This apparent contradiction was explained as being due to a dominant suppressor of noc on the chromosome associated with $T(2;3)Mpe$. However, a more detailed analysis of the noc locus (MCGILL 1985; MCGILL et al. 1988) indicates that noc does not form...
Alleles of nocA have a strong no-ocelli phenotype when homozygous or when heterozygous with deletions that include nocA.

The locus l(2)35Ba was originally defined on the basis that the EMS-induced mutation l(2)35Ba<sup>AR10</sup> maps within the deletion Df(2L)fn2, which is mutant for the loci el to Adh (WOODRUFF and ASHBURNER 1979b). More detailed analysis showed that l(2)35Ba<sup>AR10</sup> maps distal to the breakpoints of the l(2)35Ba<sup>noc</sup> deletions Df(2L)A446 and Df(2L)A178 (ASHBURNER, AARON and TSUBOTA 1982). At the molecular level l(2)35Ba is defined by the distal breakpoint of the deletion Df(2L)A446 (above) and the proximal breakpoint of the deletion Df(2L)B84a2 (121.8 to -125.1). Therefore the areas of uncertainty of the loci nocA and l(2)35Ba overlap to a considerable extent (Figure 13).

The translocation T(2;3)GT8 is lethal when heterozygous with alleles of l(2)35Ba, suggesting that this is an allele of l(2)35Ba, but it has a strong no-ocelli phenotype when heterozygous with l(2)35Ba<sup>noc</sup> deletions, e.g., Df(2L)A178 (our unpublished data). The breakpoint of T(2;3)GT8 is between -118.0 and -119.5, within the area of overlap of nocA and l(2)35Ba (Figure 13). One possible explanation of these data is that the phenotype of T(2;3)GT8 is due to a position effect of the translocation on one or both of the loci nocA and l(2)35Ba. An alternative explanation, however, is that nocA and l(2)35Ba are a single transcription unit. It would then follow that mutations that disrupt or delete the distal end of l(2)35Ba/nocA are lethal (e.g., Df(2L)Adh<sup>7813</sup>, T(2;3)GT8) but mutations that disrupt or delete the proximal end of this gene are viable and phenotypically no-ocelli (e.g., Df(2L)A178, In(2LR)noca). With respect to these two groups of mutations, Df(2L)A446 is intermediate, both in respect to the position of its breakpoint (Figure 13) and by the fact that it is semilethal with l(2)35Ba alleles or when heterozygous with deletions which include l(2)35Ba.

The model that l(2)35Ba and nocA might be identical is supported by the presence of a transcript starting close to the EcoRI site at -119.5 and extending in the 3' direction to about -112.0. The direction of transcription is distal to proximal (BUO MENG, unpublished data) (Figure 13). If this is the transcript of the l(2)35Ba/nocA locus, then T(2;3)GT8 would be broken within the 5' region of the transcript. The breakpoint of the deletion Df(2L)A446 is in, or near, the 3' end of the transcript, whereas Df(2L)A178 is proximal but close to the 3' end of the transcript (Figure 13). A thorough search for transcripts in this region has been made and only the one transcript has been found (B. MENG, W. CHIA and M. ASHBURNER, and colleagues, unpublished data). Further evidence for this model is that the locus l(2)35Ba has not been
separated from noeA by any inversion or translocation event, i.e., no l(2)35Ba- noeA* aberrations have been found. Further, all deletions that affect one but not both of these functions are l(2)35Ba+ noeA-, no l(2)35Ba- noeA* deletions are known.

According to this model the pleiotropy of "l(2)35Ba/noeA" mutations is due to the temporal or spatial specificity of this gene's expression. For example, mutations which result in the loss of gene function during larval development could lead to lethality, mutations which lead to the loss of gene function only later in development could lead to the ocellar-phenotype.

The el locus and its relationship to pu: The el locus was originally discovered by E. M. WALLACE in 1935 (BRIDGES and BREHME 1944) as a spontaneous mutation (el'). Phenotypically, elbow flies have small, bent wings due to the absence or reduced size of the posterior compartment. The alulae are reduced in size with a reduced number of marginal bristles, and are often fused with the wing blade. Nineteen further alleles of el have subsequently been isolated (11 EMS-induced, one X-ray-induced and eight γ-ray-induced). When heterozygous with a deletion of the entire el region, el alleles vary in their phenotypic severity from very strong alleles, such as el' and T(2;3)el24, to those which are almost wild type, such as T(Y;2)A15 and In(2LR)el9. These alleles also vary in their interactions with Sco, some [e.g., T(Y;2)A15], strongly enhance the Scutoid phenotype, but others (e.g., el'), have no effect on the Scutoid phenotype (ASHBURNER, TSUBOTA and WOODRUFF 1982; M. ASHURNER, J. ROOTE, G. JOHN- son, D. KIMBRELL and T. DAVIS, unpublished data). Genetically all el mutations behave as if they fall into a single complementation group (M. ASHBURNER, J. ROOTE, G. JOHN- son, D. KIMBRELL and T. DAVIS, unpublished data).

At the molecular level, the el alleles that are associated with aberration breakpoints are found to map to two clusters. The distal cluster includes the aberrations T(Y;2)el4, T(Y;2)A15, In(2LR)el6, T(2;3)el9 and In(2LR)el9, all of which are dominant enhancers of the Scutoid phenotype. The proximal cluster includes the aberration T(2;3)el24 and the spontaneous allele el', neither of which enhance the Scutoid phenotype. The aberrations T(Y;2)A80 and In(2LR)DTD128 have
FIGURE 11.—Hybridization in situ to the polytene chromosomes of aberrations broken in the el region. 
(a) T(Y;2)e4/CyO hybridized with λob18.42. There are three sites of hybridization to region 35, one to the CyO chromosome (Cy) and the other two to the Y2α element of the translocation and the Y2β element of the translocation. This translocation is broken approximately one-third of the way along this phage. (b) In(2LR)DTD128/CyO hybridized with λob16.19. There are three sites of homology to this phage; one to the CyO site (Cy) and the others to the DTD128α and the DTD128β sites. (c) T(Y;2)GT2/In(2LR)Gla hybridized with λob20.43. There are three sites of hybridization to region 35. The Y2α and the Y2β elements are labeled to equal extents showing that the aberration is broken in the middle of this phage. (d) T(Y;2)A15/cn bw hybridized with λob18.42. Three sites are seen; the site on the cn bw chromosome and the sites due to the Y2α and the Y2β sites. This translocation is broken approximately one-third of the way along this phage. (e) T(Y;2)A80/CyO hybridized with λob16.19. Three sites of hybridization are seen; the CyO site (Cy), the Y2α and the Y2β sites. (f) T(2;3)shv19/CyO hybridized with λob18.42. There are three sites of hybridization; the CyO site (Cy), the Y2α part of the translocation and the site due to the fusion of 35A to 22F (the new order of these chromosomes is 21–22F/35A–22F/97A–60F).

their breakpoints between these two clusters, both are phenotypically elbow+, and both fail to enhance the scutoid phenotype.

These data could be explained in several ways, of which the most parsimonious is that there are two elbow genes. Genetically this can be most obviously seen from the facts that: T(Y;2)A80 is el+, yet synthetic deletions extending both proximally (e.g., T(Y;2)-A80α/TE146(Z)GR155) and distally (e.g., T(Y;2)el4p A80p) from the T(Y;2)A80 breakpoint are both phenotypically elbow when heterozygous with el alleles. The only anomalous el mutant is T(2;3)el24, whose breakpoint maps to a region of the DNA (−126.0 and −131.4) not required for el function. Presumably this translocation is associated with a second lesion, close to that of el1.

The model that el comprises two genes, a proximal elA and a distal elB, is supported by genetic data from combinations of deletions. The heterozygote Df(2L)b83d29a/Df(2L)TE36(R)GWR8 is phenotypically elbow+, yet both of these deletions are phenotypically elbow when heterozygous with el alleles (ASHBURNER et al., unpublished data). The el alleles that are associated with aberrations can be allocated to either elA or elB on the basis of phenotypic strength. The strong alleles map to the elA region and the weak alleles map to the elB region.

The elA locus is defined by the deletions Df(2L)A260 and Df(2L)A266 at −144.4 and the translocation T(Y;2)A80 at −167.2 to −172.9. The elB locus is defined by the translocation T(Y;2)A80 (−167.2 to −172.9) and the translocation T(Y;2)GT2 (−220.9 to −226.0) (Figure 12).

Flies which are heterozygous for alleles of elA and elB are phenotypically elbow. This could be explained by a model based on structural interactions between the products of the two genes. For example the protein products of elA and elB might form a heterodimer.
A fly is also heterozygous for elB, the amount heterodimer would decrease to 25% of the wild-type result from the amount of wild-type heterodimer decreasing below the threshold required for normal function. This would predict that both Df(2L)elB−elA−/+ and Df(2L)elB−elA+/elB+Df(2L)elA− genotypes would be wild type, since both would make 50% of the normal heterodimer and no inactive mutant heterodimer (Figure 14, d and e). This is indeed the case, no elB-elA-deletion has any dominant phenotype and the heterozygote Df(2L)elB−elA−/Df(2L)elA− flies (Figure 14g). This model is similar to that proposed by REGAN and FULLER (1988) to explain the failure of complementation of mutations of the testis specific gene coding for β-tubulin (B2t) by point mutations of the gene haywire (hay), but not by hay"null" mutations. These workers have now shown that the products of the α-tubulin and β-tubulin genes in Drosophila testis do form a heterodimer and that mutations of one gene fail to complement mutations of the other in this way (HAYS et al. 1989).

The pu locus was discovered in 1925 (MORGAN, BRIDGES and STURTEVANT 1925). In pupal flies the wings do not expand after eclosion of the adult. The five known pu alleles all fall into a single simple complementation group. The pu locus maps genetically to be distal to elA, on the basis of its inclusion within the synthetic deletion T(Y;2)A80pTE146(Z) A80p but not within the synthetic deletion T(Y;2)A80pTE146(Z)GR18 (ASHBURNER, TSUBOTA and WOODRUFF 1982 and unpublished data). Both of

![Figure 12](image_url)

**Figure 12.** The relationship between the genetic and molecular maps of the el region of chromosome 2L. The limits of the deletions and the breakpoints of other aberrations are shown, with the limits of uncertainty (narrow lines). The minimum limits are indicated by the bold lines (see the text).

![Figure 13](image_url)

**Figure 13.** The relationship between l(2)35Ba and nocA. The limits of nocA are defined by the insertion point of TE146 (nocA−) at −107.5 and the l(2)35Ba−nocA− deletion Df(2L)A80p at −119.5 to −121.8. The limits of l(2)35Ba are defined by the l(2)35Ba−nocA− deletion Df(2L)A446 at −113.5 to −117.5 and the l(2)35Ba nocA+ deletion Df(2L)b83d29a at −125.1 to −121.8. The amount of overlap of the regions of uncertainty of these loci is 8.3 kb. The putative l(2)35Ba/nocA transcript is indicated by a bold line, the direction of transcription is shown by the arrow (Beco MENG, unpublished data). The narrow lines indicate the limits of uncertainty of the aberration breakpoints. Only EcoRI sites are shown.
The locus l(2)35Aa: This locus is not part of the elnec complex and is included here because this locus indicates the extreme distal end of the complex. The proximal limit of l(2)35Aa is defined by the distal breakpoint of the l(2)35Aa deletion Df(l)TE146(Z)GW7 at -247.0 to -247.8 and the distal breakpoint of the l(2)35Aa+ deletion Df(l)A400 at -238.6 to -243.5. The distal limit of this locus has not been defined but must be proximal to -260.0 on the basis that the translocation T(2;3)GT10 maps at -235.3 to -260.0 and breaks distal to the locus l(2)34Fd which is distal to l(2)35Aa (Figure 12). Also mapping between -247.0 and -260.0 is the male sterile locus ms(l)34Fe (Figure 12).

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