THE GENETICS OF A SMALL AUTOSOMAL REGION OF *DROSOPHILA MELANOGASTER* CONTAINING THE STRUCTURAL GENE FOR ALCOHOL DEHYDROGENASE. VI. INDUCED REVERTANTS OF SCUTOID

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ABSTRACT

Twenty-six induced revertants of Scutoid (*Sco*), a dominant mutation of *Drosophila melanogaster*, have been characterized genetically. *Sco* is an unusual mutation, involving two small reciprocal transpositions within the region 35A4 to 35C5 of chromosome arm 2L. One of these transpositions juxtaposes the *noc* and *l(2)br28* loci. We suggested previously that the Sco phenotype results from the "fusion" of *noc* and *l(2)br28*. In support of this idea we now show that 23 of 26 revertants of *Sco* are *noc*$^-$, indeed the majority are either chromosome aberrations broken between *noc* and *l(2)br28* or deletions of these loci from the mutant chromosome. However, some revertants of *Sco* are rather more complex, and their properties suggest an interaction between the *pu-noc* and *l(2)br28-l(2)br37* regions of chromosome arm 2L and also demonstrate the genetic complexity of the *el-noc* region.

IN the previous paper of this series (ASHBURNER, TSUBOTA and WOODRUFF 1982), we described the formal genetics of Scutoid (*Sco*), an interesting dominant mutation mapping near to *Adh* on chromosome arm 2L of *D. melanogaster*. *Sco* is an unusual mutation since it maps proximal to *Adh* by recombination (O'DONNELL *et al.* 1977) but distal to *Adh* by deletion mapping (ASHBURNER, TSUBOTA and WOODRUFF 1982). Although the expressivity of *Sco* is enhanced by deletions that include the four loci *el*, *l(2)br22*, *l(2)br29* and *noc*, it cannot be mapped to any single interval, defined by deletion endpoints, within this region. Rather, the degree to which deletions enhance *Sco* depends upon just how much of this region they include. A rare recombinant between *el* and *Sco* (MARONI 1980) was found to be duplicated for *Adh* and *noc* and deleted for *rd* and two lethal loci adjacent to *rd*. *Adh* and *rd* are normally separated by at least nine genes.

The genetic properties of MARONI's recombinant can most easily be explained by the hypothesis that *Sco* itself is associated with two reciprocal transpositions, *noc*, *osp* and *Adh* exchanging places with *rd*, and two lethal loci, *l(2)br34* and

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l(2)br35. Since the *el-Sco* recombinant is phenotypically Scutoid, this phenotype must result from the transposition of the *noc-Adh* region to the chromosomal position normally occupied by *rd*, *l(2)br34* and *l(2)br35*. Mutations of *noc* enhance, and duplications of noc^+ suppress, the phenotype of *Sco*. These data indicate that the phenotype of *Sco* may result from a mutation, or position effect, of *noc*. More specifically, we have suggested (ASHBURNER, TSUBOTA and WOODRUFF 1982) that, in the *Sco* chromosome, *noc* has fused with *l(2)br28* and now codes for an altered NOC product that competes, in development, with the product of noc^+.

Evidence that supports our interpretation of *Sco* comes from the nature of induced revertants of *Sco*. Our experiments to revert *Sco* were based on the fact that, whereas *Sco*/+ flies have a very characteristic bristle phenotype, Sco^-/+ (where Sco^- indicates a deletion that includes *Sco*) are essentially wild type. Hence, deletion of the mutant *Sco* allele from the *Sco*-bearing chromosome should result in the reversion of the Sco phenotype. Since *Sco* maps close to *Adh* this appeared to be an easy way to obtain Adh^- deletions. However, most *Sco* revertants are considerably more complex than simply being deleted for *Sco*, and their structure throws light on the nature of the *Sco* mutation itself and on the normal genetic organization of the small chromosome region near to *Adh* on 2L. In particular we find that the majority of Sco revertants are chromosome aberrations and/or deletions that interrupt (or remove) the *noc/l(2)br28* junction of the *Sco* chromosome.

MATERIALS AND METHODS

Stocks: The genotypes (and extents) of deletions and other chromosomes are listed in Table 1.

Mutagenesis: Flies were treated with X-rays at 150 r/min (160 Kv, 14 mA with 1 mm Al + 0.5-mm Cu filtration) in air. For ethyl methane sulfonate (EMS) mutagenesis we fed adult males with a 0.025 M "solution" of EMS in 1% sucrose overnight.

Scoring of Sco and Sco^{R+} *phenotypes*: The bristles used to score Sco phenotypes were listed in ASHBURNER, TSUBOTA and WOODRUFF (1982). They were the pairs of major macrochaetae of the dorsal head and thorax (see also Table 3). Unless indicated otherwise we give the mean (± its standard error) of counts of ten flies of each sex. The viability of various genotypes is indicated as the number (or proportion) of Cy^+ progeny over total progeny number in crosses between mutations balanced over *Cy* balancers (usually *CyO*, sometimes *Cy Bl* or *Cy Roi*).

Recovery of Sco *revertants*: Table 2 lists the three experiments in which revertants of *Sco* were recovered. The first two experiments used X-rays (3.5 kr), and in each the frequency of phenotypically Sco^+ progeny was approximately 1/1000 flies. Of the 61 revertants recovered only 23 were successfully established in stock, and one of these (Sco^{R+5}) was lost well before the experiments were completed. It is important to note that revertants *R + 1* to *R + 14* (on *Sco*) were recovered in experiment 1 and revertants *R + 16* to *R + 27* (on *b Sco pr*) in experiment 2.

In a small experiment to see whether or not *Sco* could be reverted with EMS, two revertants, in 4193 progeny, were found. One (Sco^{R+15}) was established into a stock; the other one was apparently a mosaic, for it failed to breed true.

These experiments also lead to the reversion of the *Cy* mutation of the balancer chromosome, but at a far lesser frequency (about 1/8000 progeny) than that of *Sco*. Two of the eight *Cy* revertants were cytologically abnormal; Cy^{R+C1} was *In(2L)23B;24B* on $In(2L)Cy^Lt^R$ *In(2R)Cy,Cy Roi* and Cy^{R+C3} was *T(2;3)23B3.8;72F3.4* on the *In(2L)Cy* of a *Cy* L^4 chromosome. The other *Cy* revertants were cytologically similar to their parental *Cy* chromosomes.

Three other revertants of *Sco* have been analyzed here. One is the X-ray-induced Sco^{rev7} of E. H. GRELL (unpublished data), shown by O'DONNELL *et al.* (1977) to be a long deficiency of the region

around *Adh*. The second *Df(2L)el80f1* was recovered as an elbow mutation after treatment of *Sco/CyO* males with 4000 r of γ-rays (^{60}Co) in unrelated experiments. The third, *Df(2L)PA4*, was also γ-ray induced and was recovered as a fly that survived a pentynol selection screen for *Adh*$^-$ mutations.

For reasons of space much of the original data is not given in this paper. Supplementary tables of data are on file with *Genetics* or may be obtained from the senior author.

Nomenclature: The formal genetic description of *Sco* is *Tp(2; 2)Sco-1 Tp(2; 2)Sco-2, Sco*. The *b el Sco* crossover of MARONI (1980), recovered by exchange between *b el*^{1} *rd*^{s} and *Sco*, is *b el*^{1} *Df(2L)Sco-1 Dp(2; 2)Sco-2*. *el*^{2} was called *el*^{GM2} in previous papers.

RESULTS

Description of the revertants

Detailed cytological descriptions of all 26 revertants will be published by M. ASHBURNER. For the summary of these data that we include here we have assumed that the structure of the parental *Sco* chromosome is *21—35A4-B1|(35B10-C1—35C4-5)|35B4—35B10-C1|35B3—35A4-B1|35D1-2—60.*

Sco^{R+1}, genetically unusual in its complementation behaviour (see later in paper), is a pericentric inversion with the new order, *21—35A4-B1|(35B10-C1—35C4-5)|35B4—35B10-C1|35B3—35A4-B1|44C1-2—35D1-2|44C3-5—60.* *Sco*$^{R+1}$ is homozygous lethal (0/2146); the homozygotes die during the pupal stage. Genetically, a lethal on *Sco*$^{R+1}$ defines the locus *l(2)br29* (see later).

Sco^{R+2} is a small paracentric inversion with the new order *21—35A4-B1|(35B10-C1—35C4-5)|35B4—35B10-C1|35B3—35A4-B1|36D1-2—35D1-2|36D3—60.* *Sco*$^{R+2}$ is homozygous lethal (0/2944).

Sco^{R+4} is a complex paracentric inversion with the probable order *21—28F1-2|33A1-2—35B1-2|28F3-5—32F4|37A1-2—38F6|35D1-2—36F11|39A1-2—60.* Genetically, *Sco*$^{R+4}$ is a long contiguous deletion from *noc* to *l(2)br33*.

Sco^{R+5} was a small paracentric inversion with the order *21—35A4-B1|(35B10-C1—35C4-5)|35B4—35B10-C1|35B3—35A4-B1|38A3-8—35D1-2|38A3-8—60* associated with a *T(Y:2)* broken in the heterochromatin of chromosome 2 and a long pericentric inversion *In(2LR)23A;46E*. It was lost before completion of this study.

Sco^{R+7} is a reciprocal *T(2;3)* with the new orders *21—35A4-B1|(35B10-C1—35C4-5)|35B4—35B10-C1|35B3—35A4-B1|93F9-10—61; 60—35D1-2|94A1-4—100.* Genetically, it is a noncontiguous deletion, lacking *noc, osp* and *Adh* and at least three more proximal lethals, *l(2)br28* to *l(2)br37*.

Sco^{R+8} is a paracentric inversion with the order *21—34B6-7|35A4-B1—35B3|35B10-C1—35B4|(35C4-5—35B10-C1)|35A4-B1—34C1-2|35D1-2—60.* It is homozygous lethal (0/2530), and the homozygotes die as pharate adults.

Sco^{R+9} is a pericentric inversion, *21—35A4-B1|(35B10-C1—35C4-5)|35B4—35B10-C1|35B3—35A4-B1|41—35D1-2|41—60.* A few *Sco*$^{R+9}$ homozygotes hatch (the majority die as pharate adults) and have a Scutoid bristle phenotype (with 27.80 ± 0.38 (n = 20) bristles per fly). The 2*L* breakpoint of *In(2LR)Sco*$^{R+9}$ was mapped by CRAYMER's (1981) new method for combining the ends of different pericentric inversions. By exchange between *In(2LR)Sco*$^{R+9}$ and a wild-type sequence homologue CRAYMER (1981) had synthesized the autosynaptic form of this aberration, *LS(2)Sco*$^{R+9}$*/DS(2)Sco*$^{R+9}$ (with the order *21—35A4-B1|(35B10-*

TABLE 1

Description of chromosomes

Chromosomes	Cytology
Deletions	
Df(2L)fn1, pr cn	*Df(2L)34F4-A1; 35D5-7*
Df(2L)fn2, pr cn	*Df(2L)35A3; 35B2-4*
Df(2L)fn3, pr cn	*Df(2L)35B1; 35B3-4*
Df(2L)fn7, pr cn	*Df(2L)34E1.2; 35B3-5*
Df(2L)fn26, pr cn	*Df(2L)34E3; 35D8-E1.2*
Df(2L)fn27, pr cn	*Df(2L)35B1; 35D1.2*
Df(2L)A48, b cn bw	*Df(2L)35B1.2; 35D5-7*
Df(2L)A63, b cn bw	Not evident
Df(2L)A72, b cn bw	*Df(2L)35B1.2; 35B7*
Df(2L)A178, b rd^s *pr cn*	*Df(2L)35B2.3*
Df(2L)A245, b cn bw	*Df(2L)35A4; 35B2*
Df(2L)A260, b cn bw	*Df(2L)35B1.2*
Df(2L)A267, b cn bw	*Df(2L)35B2; 35B10*
Df(2L)A376, b cn bw	*Df(2L)34E3; 35C4.5*
Df(2L)A379, b cn bw	*In(2LR)35B3-5; 57A8-10 + 35B3-5; 40-41*
Df(2L)A400, b cn bw	*Df(2L)35A1.4; 35B10*
Df(2L)A446, b cn bw	*Df(2L)35B1; 35E1.2*
Df(2L)75c	*Df(2L)35A1.2; 35D4.7 + In(2L)27D1.2; 35A1.2*
Df(2L)Adh^{n78l3}	*Df(2L)35B1; 35D5-7*
Df(2L)do-1, pr cn	*Df(2L)35B1; 35D2*
Df(2L)b80e3, Adh^{n5} *pr*	*Df(2L)34C3; 35A4*
Df(2L)b81a1, Adh^{uf3} *cn*	*Df(2L)34D3; 35B1*
Df(2L)osp18, pr cn	*Df(2L)35B1.2; 35C4.5*
Df(2L)osp29, osp^{29} Adh^{uf3} *pr cn*	*Df(2L)35B3; 35E6*
Df(2L)TE36-GC, pr pk cn	*Df(2L)35C1; 35D2*
Df(2L)TE36-GD, pr pk cn	*Df(2L)34B4; 35C3*
Df(2L)W	*Df(2L)35A2.3; 35B3-5*
Duplications	
Dp(2; 2)Adh3, rd^s	*Dp(2; 2)34B1.2; 35B3*
Dp(2; 2)C163.41^L*C158.1*^{R}	*Dp(2; 2)35B3; 35E1.2 + Dp(2; 2)26D1.2; 27D1.2 + In(2L)27D1.2; 35E1.2*^{L}*26D1.2; 35B3*^{R}
Inversions and translocations	
In(2L)C158.1	*In(2L)26D1.2; 35B3*
In(2L)C163.41	*In(2L)27D1.2; 35E1.2*
In(2LR)O, Cy dp^{lwl} *pr* cn^2	
In(2L)Cy + In(2R)Cy, al^2 *Cy pr cn Bl* cn^2 *vg c* sp^2	
In (2L)Cy^L*t*^{R} *+ In(2R)Cy, Cy* $b^{77.1X}$ *Roi* cn^2 *bw* sp^2 *or*	
In(2LR)Gla, Gla l(2)br16^{SF16}	
T(2; 3)ML474	*T(2; 3)35B3-5; 94D5-13*
T(2; 3)TE36-V3, b pr pk cn sp	*T(2; 3)35B5-10; 81*
T(2; 3)TE146-V4, al dp b pr pwn cn	*T(2; 3)35B1-3; 81*
T(2; 3)H16, dpp^{ho2} *l(2)br37*^{GE1}	*T(2; 3)35D5-7; 86F6-8 + In(3R)86A; 87F*

Table 1—*Continued*

Chromosomes	Cytology
Lethal mutations	
l(2)br22^{AR10} Adh^{n11} cn vg	
l(2)br22^{FT1} Adh^{n11} cn vg	
l(2)br22^{HG33} Adh^{n7} cn vg	
l(2)br22^{HG46} Adh^{F} pr	
Adh^{n7} l(2)br28^{HG31} cn vg	
Adh^{n10} l(2)br33^{HG38} cn vg	
Adh^{n10} l(2)br34^{HG39} cn vg	
Adh^{n7} l(2)br35^{HG35} cn vg	
Adh^{n7} l(2)br36^{HG34} cn vg	
l(2)br7$^{Su(H)}$ l(2)br36^{AM1} l(2)Su(H) whd^{1}	
elbow alleles	
b el^{1} rd^{s} pr cn	
b el^{2} Adh^{F}	
el^{3} Adh^{uf3} cn	
T(Y; 2)el^{4}, b el^{4} Adh^{nC2} cn bw	
noc alleles	
In(2L)noc^{2}, b l(2)br1^{HG10} noc^{2} Adh^{nC1} pr cn bw	*In(2L)35B1.2; 36D3*
noc^{3} Adh^{n5} pr Df(2R)ST1	
In(2LR)noc^{4}, b noc^{4} cn bw	*In(2LR)35B1.2; 41*
al dp b noc^{TE146} pr l(2)pwn cn	insertion at *35B1.2*
b noc^{18} l(2)br4^{AR1} pr	
b noc^{19} l(2)br4^{AR1} pr	

TABLE 2

Recovery of revertants of Sco

Series	Mutagen	Chromosome	*Sco* → *Sco^{R+}*	Cy → Cy^{R+}	No. of progeny
1	X-rays	*Sco*	25 (12)	1 (1)	24,425
2	X-rays	*b Sco pr*	34 (11)	7 (7)	29,935
3	EMS	*Sco*	2 (1)	0	4,193
Total			61 (23)	8 (8)	58,553

The number of revertants is given as the number detected in the F_1 and, in parentheses, the number established in stock.

C1—35C4-5)|35B4—35B10-C1|35B3—35A4-B1|41—21; 60—41|35D1-2—60. An independent inversion *In(2LR)noc^{4}, b cn bw*, recovered as a γ-ray-induced *noc* allele, has similar breakpoints to *In(2LR)Sco^{R+9}*, *i.e.*, *In(2LR)35B1.2;41*. *In(2LR)noc^{4}, b cn bw/b cn bw* females were crossed to *LS(2)Sco^{R+9}/DS(2)Sco^{R+9}* males. This cross is expected to be sterile unless exchange occurs within *In(2LR)noc^{4}*, generating *LS(2)noc^{4}, b* and *DS(2)noc^{4}, cn bw* elements that can be rescued by the complementary *Sco^{R+9}* autosynaptic chromosomes. From ten bottles, each with 50 pairs of parents, only three flies hatched. Their phenotype (black) indicated that they should be *LS(2)noc^{4}, b/DS(2)Sco^{R+9}*. From a stock established from these flies four cultures of *LS(2)noc^{4}, b/DS(2)Sco^{R+9}* females crossed to *CyO/Gla* males were set up. This cross is

essentially sterile unless exchange occurs within region 35–41 between the autosynaptic elements to generate the heterosynaptic *In(2LR)noc*^{4L}*Sco*$^{R+9R}$ and its complement, a wild-type sequence chromosome. Three *Cy* and one *Gla* progeny were recovered from the cross, and the *Gla* fly proved to carry the required recombinant. Since the 2L breakpoint of *In(2LR)noc*^{4} is distal to that of *In(2LR)Sco*$^{R+9}$ this chromosome is deficient for *35B1; 35B10-C1*. The limits of the deletion were mapped in the usual way.

Sco^{R+10} is cytologically similar to *Sco* but is a noncontiguous deletion lacking *noc, osp, Adh; l(2)br28, l(2)br36* and *l(2)br37*.

Sco^{R+11} is a paracentric inversion *21—24C3-9|35A4-B1—35B3|35B10-C1—35B4|(35C4-5—35B10-C1)|35A4-B1—24C3-9|35D1-2—60*. *Sco*$^{R+11}$ homozygotes are almost lethal (5/3270), and those that do hatch have an extreme Scutoid bristle phenotype (with a mean of 14.83 (n = 5) bristles per fly). The genetic position of the *In(2L)Sco*$^{R+11}$ break was mapped by constructing a deletion with *In(2L)C158.1 (= In(2L)26D1.2; 35B3)*. The recombinant *In(2L)C158.1*^{L}-*Sco*$^{R+11R}$ can be recovered in two ways. On the one hand, it will be *Dp(2; 2)24C3.9; 26D1.2* and will, therefore, suppress *M(2)z*^{B}. On the other hand, it will be *Df(2L)35B3; 35C5* and will expose *rd*. The reciprocal recombinant, *In(2L)Sco*$^{R+11L}$*C158.1*^{R}, is a dominant lethal (VELISSARIOU and ASHBURNER 1980).

Sco^{R+12} is a transposition of the Scutoid region into 34B, *i.e., 21—34A8-B1|35A4-B1—35B3|35B10-C1—35B4|(35C4-5—35B10-C1)|35B1-A4—34F4-5|34B1-2—34F1-2|35D1-2—60*. We have failed to recover the products of exchange between *Tp(2; 2)Sco*$^{R+12}$ and a wild-type sequence homologue in the isosequential 34B-34F interval. However, only 11,606 progeny of *C(1)FMA3, b Adh*^{n2} *pr cn/Tp(2; 2)Sco*$^{R+12}$ females were scored in this attempt.

Sco^{R+13} is a homozygous lethal (0/527) translocation associated with an *In(3LR)* with the order *21—35A4-B1|(35B10-C1—35C4-5)|35B4—35B10-C1|35B3—35A4-B1|71B1-2—81|71B1-2—61; 60—35D1-2|81—100*. The genetic position of the region 35 breakpoint of this translocation was mapped by constructing deletions from the 2-proximal element of *T(2; 3)Sco*$^{R+13}$ and the 2-distal elements of *T(2; 3)TE146-VM4 (= T(2; 3)35B1-3; 81)* and *T(2; 3)TE36-V3 (= T(2; 3)35B10-C1; 81)*.

Sco^{R+14} is cytologically similar to *Sco* and genetically similar to *Sco*$^{R+7}$, *Sco*$^{R+10}$ and *Sco*$^{R+18}$, *i.e.*, a deficiency for *noc, osp, Adh, l(2)br28, l(2)br36* and *l(2)br37*.

Sco^{R+15} and *Sco*$^{R+16}$ are both cytologically similar to *Sco*. *Sco*$^{R+15}$ is mutant for *l(2)br28*, and *Sco*$^{R+16}$ is deficient for *l(2)br28, l(2)br36* and *l(2)br37*. Both are homozygous lethal.

Sco^{R+17} is a homozygous lethal (0/2699) paracentric inversion, *21–25D3-7|35A4-B1—35B3|35B10-C1—35B4|(35C4-5—35B10-C1)|35A4-B1—25D3-7|35D1-2—60*. From a cross of *In(2L)Sco*$^{R+17}$, *b pr/In(2L)C158.1* females × *In(2L)Cy, Cy dp*^{2} *b pr/M(2)z*^{B} *Sk b* males both the *In(2L)C158.1*^{L}*Sco*$^{R+17R}$, *pr* and *In(2L)Sco*$^{R+17L}$*C158.1*^{R}, *b* crossovers were recovered. The former is deleted for the region between the proximal limits of these inversions (*i.e., 35B3; 35C5*), and the latter duplicated for the same region and is also *Df(2L)25D3.7; 26D1.2*. As expected *In(2L)Sco*$^{R+17L}$*C158.1*^{R} is *cl*$^{-}$ (*cl* is in *25E1.2*), and its proximal duplication acts as a dominant enhancer of Hairless since it includes *l(2)br7* (see ASHBURNER 1982).

Sco^{R+18} and Sco^{R+19} are cytologically similar to *Sco*. Both are deletions, the former for *noc*, *osp* and *Adh* and from *l(2)br28* to *l(2)br37* and the latter for only *noc* and *l(2)br28*.

Sco^{R+21} is a small paracentric inversion with the order *21—35A4-B1|(35B10-C1—35C4-5)|35B4—35B10-C1|35B3—35A4-B1|36D7—35D1-2|36E1-2—60*. Sco^{R+21} is the only revertant that is reasonably viable when homozygous (452/2205). These homozygotes have a weak Scutoid phenotype (with a mean of 31.45 ± 0.51 (n = 20) bristles per fly) and are sterile.

When Sco^{R+23}/CyO females were crossed to *b* el^1 rd^s *pr cn* males, a few (29) extreme elbow (and pr) flies were found among 5319 progeny. Backcrosses of the regular *b pr* F_1 sons and daughters of this cross to *b* el^1 rd^s *pr cn* immediately established that these exceptional flies resulted from the segregation of an el^- rd^- deficiency from an X-linked duplication of el^+ rd^+. The duplication bearing X is homozygous female and male viable and fertile, and Sco^{R+23} stock (*Dp(2; 1)*Sco^{R+23}; *Df(2L)*Sco^{R+23}/CyO) is homozygous for the duplication. The limits of the deletion were mapped by crossing Sco^{R+23} stock males to tester females and scoring the X/Y; *Df(2L)*Sco^{R+23}/tester and *Dp(2;1)*Sco^{R+23}/X; *Df(2L)*Sco^{R+23}/tester progeny. Crosses with *Dp(2; 1)*Sco^{R+23} refer to a stock bearing the duplication and recessively marked, but otherwise normal, second chromosomes. Cytologically Sco^{R+23} is *Df(2L)34F1-2; 35C5* and *Dp(2; 1)34F1-2—35A4-B1|(35B10-C1—35C4-5)|35B4—35B10-C1|35B3—35A4-B1*.

The limits of both *Dp(2; 1)*Sco^{R+23} and the corresponding deletion are shown in Figure 1. The duplication does not cover two lethal complementation groups that are exposed by the deficiency. Almost all Cy^+ progeny, both male and female, from crosses of *Dp(2; 1)*Sco^{R+23}/Y; *Df(2L)*Sco^{R+23}/CyO to *l(2)*$br28^{HG31}/CyO$ or *l(2)*$br36^{HG34}/CyO$ die. A few Cy^+ daughters (8/323) from the cross to *l(2)*$br28^{HG31}$ eclose, and they lack their halteres; a few Cy^+ sons (16/729) from the cross to *l(2)*$br36^{hG34}$ live, and they have abnormal bristles, disturbed acrostichial hairs and "netted" wing veins.

Sco^{R+24} is a small paracentric inversion with the new order *21—34A7-11|35A4-B1—35B3|35B10-C1—35B4|(35C4-5—35B10-C1)|35A4-B1—34B1-2|35D1-2—60*. This revertant is homozygous lethal (0/1916).

Sco^{R+25} is cytologically similar to *Sco* but is deleted for *l(2)br28* and *l(2)br36*.

Sco^{R+26} is a small inversion broken just distal to *35D1-2* and in the heterochromatin of chromosome 2. The inability to rescue gametes from *LS(2)*Sco^{R+9}/*DS(2)*Sco^{R+9} males crossed to *In(2)*Sco^{R+26}, *b* pr/+ females suggests that this revertant is broken in 2L (40 bottles of this cross were set up).

Sco^{R+27} is cytologically similar to *Sco*.

Finally both Sco^{rev7} and *Df(2L)el80f1* are long deletions of the entire Scutoid region, *Df(2L)34D5; 35D5-7* and *Df(2L)34E3; 35D7*, respectively. *Df(2L)PA4* is genetically similar to Sco^{R+7} but is cytologically a longer deletion, i.e., *Df(2L)35A4-B1; 36A1.2*.

In summary, 26 revertants of *Sco* have been analyzed cytologically. With respect to those changes in gene order that would appear to be relevant to their revertant genotype, 11 are inversions, three are translocations, one is a transposition and three are deletions. Of the remaining eight that are not obviously aberrant cytologically, or at least no more so than *Sco* itself, genetic data

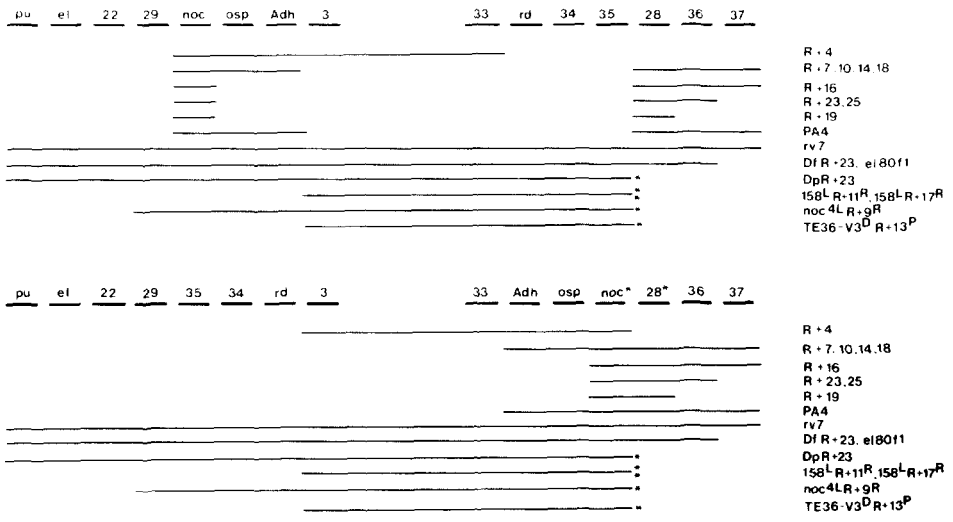

FIGURE 1.—A summary of the genetic extents of Sco^{R+} chromosomes and of the genetic positions of mapped Sco^{R+} breakpoints. Two maps are shown, above on a wild-type sequence and below on that proposed for *Sco*. Note that, when projected on a wild-type sequence, many of the deletions are noncontiguous but that they are contiguous on the *Sco* order. The extents of deletions synthesized from revertant chromosomes by recombination are also indicated and the positions of the breakpoints of the revertants indicated by asterisks. The common involvement of the *noc/br28* junction in these revertants is clear.

indicate that seven, at least, are deletions. The reversion of *Sco* is a very productive method for the detection of chromosome aberrations in the region of *Adh*.

The most remarkable feature of those revertants that are aberrations is that their *Sco* region breakpoint is always at or near the *35A4-B1|35D1-2* junction, *i.e.*, at the proximal margin of the right hand *Sco* transposition (M. ASHBURNER, unpublished results). In so far as it has been possible to determine the genetic location of these breakpoints all have mapped to the same genetic region, between *noc* and *1(2)br28* on the *Sco* map (Figure 1). The estimated cytological locations of *noc* and *1(2)br28*, from deletions independent of this study, are 35A4-B1 and 35D1.2, respectively.

Phenotypes of the revertants

In Table 3 we indicate the bristle phenotypes of all the revertants (except Sco^{R+5}) and compare these phenotypes with those of *Sco*/+ and *Sco*/− flies. *Sco*/+ heterozygotes usually lack between 13 and 15 of the normal 40 dorsal head and thoracic macrochaetae (Table 4). With two exceptions none of the revertants lack, on average, more than two of these bristles. Two bristle sites, the postverticals and anterior notopleurals, are seen to be most sensitive to loss. Sco^{R+9} is only very slightly more extreme in phenotype than the majority class (a mean of 37.60) but Sco^{R+11} is most definitely intermediate in phenotype between *Sco* and Sco^{+}, with a mean of about 34 bristles (*i.e.*, a loss of six) per fly.

TABLE 3

The pattern of bristle loss in Sco^{R+}/+ (Canton-S) *heterozygotes*

R+	MO	PVt	UH	AN	PN	AD	AP	ASc	PSc	Mean ± S.E.
R + 1	1.00	**0.84**	**0.95**	**0.60**	1.00	1.00	1.00	1.00	1.00	38.68 ± 0.17
R + 27	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	40.00 ± 0.00
R + 8	**0.93**	**0.53**	1.00	1.00	1.00	**0.95**	1.00	1.00	1.00	38.60 ± 0.17
R + 9	1.00	**0.80**	**0.94**	**0.45**	1.00	1.00	1.00	1.00	**0.63**	37.60 ± 0.24
R + 11	1.00	1.00	**0.49**	**0.00**	**0.94**	1.00	**0.14**	**0.86**	**0.66**	34.28 ± 0.17
R + 12	1.00	**0.76**	1.00	**0.66**	1.00	1.00	1.00	1.00	**0.94**	38.68 ± 0.17
R + 17	1.00	1.00	1.00	**0.04**	1.00	1.00	1.00	1.00	1.00	38.08 ± 0.08
R + 15	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	39.98 ± 0.03
R + 16	1.00	**0.95**	1.00	1.00	1.00	1.00	1.00	1.00	1.00	39.90 ± 0.05
R + 19	1.00	1.00	1.00	1.00	1.00	1.00	**0.95**	1.00	1.00	39.90 ± 0.05
R + 23	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	39.95 ± 0.30
R + 25	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	39.93 ± 0.04
R + 26	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	40.00 ± 0.00
R + 7	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	39.38 ± 0.14
R + 10	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	39.93 ± 0.43
R + 14	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	39.93 ± 0.04
R + 18	1.00	**0.95**	1.00	1.00	1.00	1.00	1.00	1.00	1.00	39.90 ± 0.05
R + 2	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	39.93 ± 0.04
R + 13	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	38.93 ± 0.04
R + 21	1.00	**0.33**	1.00	1.00	1.00	1.00	1.00	1.00	1.00	38.65 ± 0.30
R + 24	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	39.93 ± 0.06
R + 4	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	39.95 ± 0.03
Sco^{rev7}	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	40.00 ± 0.00
el80f1	1.00	0.99	1.00	1.00	1.00	1.00	1.00	1.00	1.00	39.98 ± 0.02
PA4	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	40.00 ± 0.00

Only those sites with a fractional occupancy of less than 0.95 in at least one genotype are shown. Occupancies of 0.95 or less are in **bold face**.

Some of the revertant chromosomes have other dominant phenotypes, for example Sco^{R+8} is associated with a rough eye effect and Sco^{R+1} with warped wing and pale scutellum phenotypes. The wing phenotype of Sco^{R+1} is especially strong when Sco^{R+1} is heterozygous with mutations that lower its viability [such as some *noc* alleles (see ASHBURNER, TSUBOTA and WOODRUFF 1982 and later in this paper)]; then, the flies have wings that have expanded longitudinally but not laterally.

The original *Sco* chromosome carries an active Adh^F allele. All of the revertants have been assayed for ADH activity as heterozygotes with an Adh^n allele, and five (Sco^{R+4}, Sco^{R+7}, Sco^{R+10}, Sco^{R+14} and Sco^{R+18}) are ADH null: these five are the only revertants that are also deficiencies for two loci very close to *Adh*, *i.e.*, *osp* and *noc*. The *Adh* allele of the *Dp(2; 1)*Sco^{R+23} is active.

Some Sco^{R+X}/Sco^{R+Y} heterozygotes show an interesting phenotype: the absence of one or both halteres and, often, a hemithorax. This phenotype is

TABLE 4

The pattern of bristle loss in Sco *genotypes*

Bristle site	*Sco/+*	*b el Sco/+*	*Sco/Df(2L)fn7*
AO	1.00	1.00	1.00
MO	**0.95**	0.99	**0.35**
PO	1.00	1.00	**0.20**
O	1.00	1.00	**0.15**
AV	**0.80**	1.00	**0.00**
PV	**0.65**	1.00	**0.00**
PVt	**0.80**	1.00	**0.20**
PSt	**0.90**	0.99	**0.00**
UH	**0.05**	**0.58**	**0.00**
LH	**0.55**	**0.95**	**0.00**
AN	**0.00**	**0.01**	**0.00**
PN	**0.00**	1.00	**0.00**
AS	**0.90**	1.00	0.98
PS	**0.90**	1.00	1.00
AD	**0.90**	0.99	1.00
PD	**0.85**	1.00	1.00
AP	**0.10**	**0.10**	**0.00**
PP	**0.51**	1.00	**0.00**
ASc	**0.00**	**0.09**	**0.00**
PSc	**0.00**	**0.54**	**0.00**
Mean	25.10	32.48	10.63
S.E.	0.40	0.19	0.22

The fractional occupancy of the 40 major head and thoracic bristle sites was scored in 20 flies of each sex. The "+" chromosome was from Canton-S. Scores of 0.95 or less are in **bold face.**

also seen in $Sco^{R+23}/l(2)br28^{HG31}$ and $Sco^{R+26}/l(2)br28^{HG31}$ heterozygotes and when several other revertants are hemizygous for mutant alleles or deficiencies of *l(2)br28*. Our only allele of *l(2)br28* fails to complement mutant alleles of snail (P. SIMPSON and M. ASHBURNER, unpublished observation). Moreover, $l(2)br28^{HG31}$ hemizygotes show the characteristic embryonic phenotype of snail homozygotes. A similar phenotype is seen in heterozygotes between different $l(2)br28^{-}$ *Sco* revertants (P. SIMPSON, personal communication). These data suggest that *l(2)br28* and snail are synonymous and, moreover, that *l(2)br28* is indeed the immediate proximal neighbor of *noc* in the *Sco* chromosome.

Interactions of the revertants and Sco

All of the revertants have been crossed to both *Sco* and *b el Sco*, and the results are shown in Table 5. The *b el Sco* chromosome differs from the *Sco* chromosome by virtue of the fact that it carries only the *noc, osp, Adh* transposition, and not the *rd, l(2)br34, l(2)br35* transposition, of *Sco*, in being duplicated for *noc, Adh*, and *osp* and in being deficient for *l(2)br34, l(2)br35* and *rd* (ASHBURNER, TSUBOTA and WOODRUFF 1982). With respect to their interaction with *Sco* the revertants fall into two clearly distinguishable classes: seven are lethal, or very nearly lethal (less than 1.8% viability), with *Sco*; these are Sco^{R+1}, Sco^{R+8}, Sco^{R+9}, Sco^{R+11}, Sco^{R+12}, Sco^{R+17} and Sco^{R+24}. The other revertants are only semilethal with *Sco*, and the Sco^{R+}/Sco heterozygotes survive with

TABLE 5

Viabilities and bristle phenotypes of Sco^{R+}/Sco *and* Sco^{R+}/b el Sco *heterozygotes*

	Sco^{R+}/Sco				Sco^{R+}/b el Sco			
R+	N	%	n	S.E.	N	%	n	S.E.
R + 1	0/1099	0			188/2225	8.4	24.85	0.36
R + 27	148/1545	9.6	26.26	0.52	107/8333	12.8	32.00	0.38
R + 8	0/3437	0			9/210	4.3	27.44	0.44
R + 9	0/2454	0.3	13.88	0.69	17/234	7.3	26.33	0.58
R + 11	1/1976	0.1	10		11/259	4.2	24.26	0.54
R + 12	0/1956	0			3/391	0.8	25.67	
R + 17	0/1126	0			25/670	3.7	24.85	0.54
R + 15	226/3029	7.5	11.10	0.43	98/533	18.4	26.15	0.53
R + 16	101/1031	9.8	11.60	0.45	108/524	20.6	24.55	0.33
R + 19	173/1563	11.1	10.90	0.28	74/405	18.3	24.50	0.46
R + 23	87/1324	6.6	10.67	0.27	74/633	11.7	28.75	0.45
R + 25	223/1260	17.7	12.20	0.36	76/359	21.1	27.90	0.40
R + 26	255/1261	20.2	11.60	0.30	111/557	19.9	24.15	0.42
R + 7	96/1151	8.3	11.27	0.20	84/477	17.6	25.32	0.41
R + 10	152/1681	9.0	13.45	0.38	67/627	10.7	26.40	0.42
R + 14	167/2417	6.9	12.30	0.28	27/264	10.2	26.20	0.38
R + 18	240/2411	10.0	13.15	0.42	69/494	14.0	27.00	0.46
R + 2	98/1183	8.3	11.38	0.38	40/346	11.6	26.65	0.35
R + 5	18/105	17.1						
R + 13	132/1180	11.2	14.33	0.48	43/360	11.9	28.70	0.34
R + 21	151/1204	12.5	11.85	0.33	44/360	17.1	26.75	0.24
R + 24	11/1709	0.6	13.18	0.64	72/721	10.0	27.40	0.32
R + 4	86/897	9.6	10.57	0.23	65/484	13.4	26.85	0.50
Sco	12/7700	0.2	7.91	0.31	21/1064	2.0	19.00	0.55

Twenty flies were counted (unless fewer found, in which case total). n = mean fractional occupancy of 40 major head and thoracic bristle sites (± standard error); N = the number of Sco^{R+}/ *Sco* (or Sco^{R+}/*b el Sco*) flies over the total progeny of crosses between *Cy* balanced stocks.

between 22 (for Sco^{R+15}) and 61% (for Sco^{R+26}) of the frequency expected were they fully viable genotypes.

In all but one case surviving *Sco*/Sco^{R+} genotypes are phenotypically extreme *Sco*, lacking between 23 and 30 bristles per fly: indeed, these phenotypes are very similar to those of *Sco* homozygotes and to those of *Sco*/Sco^{-} heterozygotes (ASHBURNER, TSUBOTA and WOODRUFF 1982). The only exceptional revertant in this respect is Sco^{R+27}; as shown by the data of Table 5 *Sco*/Sco^{R+27} heterozygotes are just like *Sco*/+ in phenotype: three independent scores of this genotype gave means of 25.85 ± 0.35, 25.30 ± 0.40 and 25.35 ± 0.28 bristles/ fly.

Sco^{R+}/*b el Sco* heterozygotes are similar to Sco^{R+}/*Sco* heterozygotes except that they are more viable and have a weaker Scutoid phenotype. Those revertants that are quite lethal with *Sco* are viable with *b el Sco*, although their

relative viabilities are, in general, less than that of those revertants that are viable with *Sco*. Sco^{R+}/*b el Sco* flies have 25–27 bristles/fly, that is to say, a similar phenotype to *Sco*/+ or *b el Sco*/Sco^{-}. As before, only Sco^{R+27} is an exception: Sco^{R+27}/*b el Sco* flies have the same bristle number as do *b el Sco*/+ flies.

In summary, the revertants fall into three classes with respect to their interactions with *Sco* and *b el Sco*. The majority behave just like a Sco^{-} deletion, being semilethal with *Sco* and enhancing the *Sco* phenotype by the loss of an extra 15 bristles or so and the *b el Sco* phenotype by the loss of some eight bristles. Seven are almost completely lethal with *Sco*; in fact, they are less viable with *Sco* or *b el Sco* than any known region 34D-35D deficiency. Yet, the very few escapers in this class also show a phenotype similar to that of *Sco*/Sco^{-} or *b el Sco*/Sco^{-} flies. The third class is represented uniquely by Sco^{R+27} which, although semilethal with both *Sco* and *b el Sco*, does not enhance the bristle phenotype of these mutations.

Complementation pattern between revertants

All of the revertants have been crossed *inter se* and the viabilities and phenotypes of the various heterozygotes scored. The interpretation of the lethal complementation data is not straightforward, for it is confounded by the existence on the *Sco* or *b Sco pr* chromosomes of preexisting lethals unrelated to *Sco*. To a large extent complications arising from such sources can be identified and cleared up by studying the relative viabilities of revertants with region 34D-35D deficiencies.

Unless they involve Sco^{R+27} all viable Sco^{R+X}/Sco^{R+Y} heterozygotes have a phenotype similar to *Sco*/+ and, often, the irregular eye phenotype characteristic of *Sco*/*Sco*. For example, the mean bristle number of Sco^{R+2}/Sco^{R+17} was 27.30 ± 0.36, but that of Sco^{R+27}/Sco^{R+17} was 39.00 ± 0.40.

The genetic behavior of the revertants, both when crossed *inter se* and when crossed to deletions or lethal mutations in region 34D-35D, allows them to be conveniently grouped into several distinct classes.

Group 1: Two revertants, Sco^{R+1} and Sco^{R+27}, are included in this group. This may seem paradoxical since Sco^{R+1} is completely lethal with all revertants except Sco^{R+27}. Sco^{R+1}/Sco^{R+27} are only semilethal (239/3234; 7.4%). Sco^{R+27}, on the other hand, shows no obviously consistent pattern of lethality with the other revertants (but see later) (See Tables 6–8). The inclusion of these two revertants in group 1 is, however, based on the deletion mapping of their lethal or semilethal phenotypes.

The consequences of crossing both Sco^{R+1} and Sco^{R+27} to deletions of region 34-35 are illustrated in Figure 2. If we consider first those deletions that do not extend proximally beyond *Adh*, then it is clear that Sco^{R+1} is lethal, and Sco^{R+27} semilethal, with all deletions that include both *l(2)br22* and *noc* (e.g., *Df(2L)fn2*, *Df(2L)A245*). Sco^{R+1}, but not Sco^{R+27}, is semilethal with the $l(2)br22^{+}$ noc^{-} deletion *Df(2L)A178*, and neither revertant is lethal with *Df(2L)A379* or *Df(2L)A267* ($br22^{+}noc^{-}$) or the more proximally broken *Df(2L)A72* ($br22^{+}noc^{+}$).

These data define a lethal locus between *noc* and *l(2)br22*: this has been called *l(2)br29* (ASHBURNER, TSUBOTA and WOODRUFF 1982), and these two revertants

TABLE 6

Complementation for viability between group 2 revertants inter se and between representatives of other revertant groups

	R + 8	R + 9	R + 11	R + 12	R + 17
R + 8	0/2530	0/250	1/248	0/190	4/329
R + 9		37/2746	0/198	0/504	0/127
R + 11			5/3272	0/257	0/689
R + 12				0/2072	0/217
R + 17					0/2699
R + 1	0	0	0	0	0
R + 27	0.20	0.17	0.15	0.15	0.01
R + 2	0.14	0.21	0.19	0.25	0.15
R + 13	0.20	0.22	0.14	0.22	0.18
R + 21	0.45	0.25	0.24	0.27	0.01
R + 24	0.24	0.11	0.06	0.03	0.01

Above, showing the number of Cy^+/total progeny and, below, Cy^+ as a fraction of total progeny. (See also Tables 7 and 8).

TABLE 7

Complementation for viability between group 3 revertants inter se and between group 3 and representative other revertants

	R + 7	R + 10	R + 14	R + 18	R + 15	R + 16	R + 19	R + 23	R + 25	R + 26
R + 7		0/531	0/424	0/224	0/139	0/250	0/114	0/228	0/306	0/341
R + 10			0/852	0/328	0/449	0/447	0/298	0/324	0/179	0/434
R + 14				0/264	0/330	1/393	0/230	0/336	0/186	0/215
R + 18					0/267	0/242	0/189	0/293	0/201	0/223
R + 15						0/224	0/114	1/380	0/287	0/234
R + 16							0/289	0/354	0/537	0/430
R + 19								0/168	0/327	0/133
R + 23									0/318	1/444
R + 25										0/183
R + 26										
R + 1	0	0	0	0	0	0	0	0	0	0
R + 27	0.07	0.04	0.06	0	0.23	0.05	0.11	0.03	0.04	0.19
R + 12	0.17	0.19	0.16	0.54	0.22	0.56	0.22	0.22	0.28	0.34
R + 2	0.15	0.15	0.18	0.26	0.20	0.48	0.43	0.40	0.50	0.37
R + 13	0.10	0.12	0.11	0.34	0.16	0.34	0.31	0.26	0.34	0.28
R + 21	0.37	0.29	0.52	0.13	0.49	0.13	0.43	0.04	0.22	0.17
R + 24	0.22	0.22	0.14	0.01	0.09	0	0	0	0.02	0

See legend to Table 6.

can be regarded as partially complementing alleles of this locus, with *Df(2L)A178* having its distal limit "between" the two "complementation groups." Despite the fact that alleles of *l(2)br22* show a partial failure of complementation, with respect to viability, with both Sco^{R+1} and Sco^{R+27} (see Table 12) *l(2)br22* and *l(2)br29* are different loci. In Table 9 the results of crossing a series of deletions, all with their distal breakpoints mapping between elbow and outspread, to *l(2)br22* alleles and to these revertants are summarized. It is obvious that the

TABLE 8

Complementation for viability between group 4 revertant. inter se (above) and between group 4 and group 1 revertants (below)

	R + 2	R + 13	R + 21	R + 24
R + 2	0/2944 (0)	191/597 (0.32)	108/287 (0.38)	4/760 (0.01)
R + 13		0/527 (0)	100/365 (0.27)	38/109 (0.35)
R + 21			452/2205 (0.20)	0/389 (0)
R + 24				0/2517 (0)
R + 1	0	0	0	0
R + 27	0.06	0.18	0.03	0.08

See legend to Figure 6.

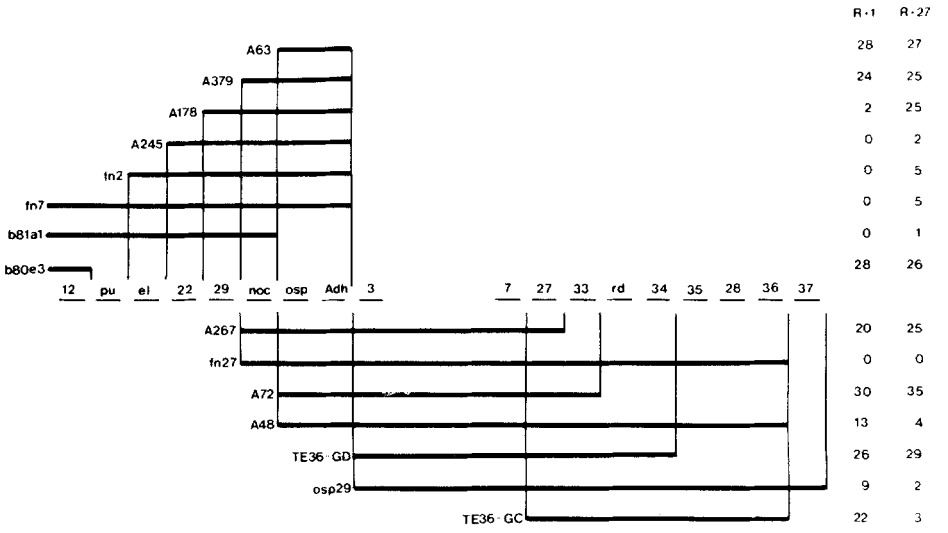

FIGURE 2.—The relative viabilities of Sco^{R+1} and Sco^{R+27} heterozygous with deletions of the *Adh* region. A genetic map from *l(2)br12* (12) to *l(2)br37* (37) is drawn, truncated between *l(2)br3* and *l(2)br7*. The limits of each deletion are indicated and the viability of each with the revertants shown in the right-hand columns. These data demonstrate a lethal on each revertant between *l(2)br22* and *noc*. This is indicated as *l(2)br29*. In the proximal part of the region note that Sco^{R+27} is a lethal with all deletions that include *l(2)br36* but that Sco^{R+1} is viable with *Df(2L)TE36-GC* (see text). This indicates different proximal lethals on these revertant chromosomes.

distal limits of *Df(2L)fn3* and *Df(2L)A446* map *l(2)br22* and that the distal limits of *Df(2L)A446* and *Df(2L)A379* define *l(2)br29*. The existence of a vital locus between the *A379* (or *A178*) and *A446* limits is also very clear from crosses of these deletions to any one of 14 different deletions broken proximally to *Adh* and extending distally. Three examples are shown in Table 9. *Df(2L)A446* (or any more distal deletion) is quite lethal with, for example, *Df(2L)fn7, Df(2L)W* or *Df(2L)A260*. *Df(2L)A178* (or any more proximal deletion) is viable with these. The viability of these genotypes may be depressed; in part this is due to their extreme osp phenotype which causes the flies to get trapped in the food.

The unusual nature of the br29 lethality associated with Sco^{R+1} (at least) is shown by the fact that we have never recovered an induced mutation that maps

TABLE 9

Deletion mapping of l(2)br22 *and* l(2)br29

	Df(2L)fn2	*Df(2L)fn3*	*Df(2L)A446*	*Df(2L)A178*	*Df(2L)A379*	*Df(2L)A72*
el	*el*	+	+	+	+	+
l(2)br22	31/1894[a] 0.02	65/1678[b] 0.04	335/1167 0.29	682/2001 0.34	271/905 0.30	216/823 0.26
Sco^{R+1}	0/528 0	0/1008 0	9/1863 0.01	42/2758 0.01	89/368 0.24	355/1152 0.31
Sco^{R+27}	36/796 0.05	65/662 0.10	2/399 0.01	112/451 0.25	82/322 0.25	265/765 0.35
noc	*noc*	*noc*	*noc*	*noc*	*noc*	+
A260	0/278 0	0/316 0	0/309 0	22/362[c] 0.06	17/384[c] 0.04	1270/8517[d] 0.15
fn7	0/970 0	0/1046 0	0/263 0	76/326[c] 0.23	41/279[c] 0.15	160/3654[d] 0.05
W	0/519 0	0/661 0	0/516 0	60/396[c] 0.15	51/209[c] 0.24	162/501[d] 0.32

The relative viabilities of deletions with distal endpoints in the elbow to outspread interval when heterozygous with mutant alleles of *l(2)br22* and *l(2)br29* and with deletions whose proximal breakpoints are between *Adh* and *l(2)br3*, showing the "independence" of *l(2)br22* and *l(2)br29* and the mapping of group 1 lethality to between the *Df(2L)A446* and *Df(2L)A379* limits.

[a] elbow; [b] elbow$^+$; [c] *noc, osp, ADH*$^-$; [d] *osp, ADH*$^-$.

to the *br29* site despite screening more than 26,000 chromosomes (after EMS treatment of males) across $br29^-$ deletions. Moreover, in an EMS screen designed to recover lethal alleles of Sco^{R+1} none that mapped to *br29* were found in 6854 chromosomes tested, although this screen did yield a new allele of *l(2)br22*.

Sco^{R+1} shows negative complementation when heterozygous with *Df(2L)A178* and *noc* alleles. *Df(2L)A178* is deleted for *noc*, *osp* and *Adh* and is semiviable when homozygous (62/487; 12.7%). Yet, heterozygotes between *A178* and Sco^{R+1} are semilethal (32/1240; 2.6%). Similarly, Sco^{R+1} is lethal, or semilethal, when heterozygous with some alleles of *noc* (see later).

In addition to being mutant for *l(2)br29*, both Sco^{R+1} and Sco^{R+27} must also carry second lethals, mapping to the right of *Adh*, since both are semilethal when heterozygous with, for example, the deficiency *Df(2L)osp29* (which is $br22^+$ to Adh^+). The fact that Sco^{R+27}/*Df(2L)TE36-GC* is semilethal but Sco^{R+1}/*Df(2L)TE36-GC* is viable (see also data with *Df(2L)A48*) suggests that their proximal lethals differ. Neither revertant is lethal with any of the identified lethal loci mapping to the proximal 34-35 region.

The inviability of Sco^{R+27} with all $l(2)br28^-$ and $l(2)br28^-l(2)br36^-$ deletions, and the inviability of Sco^{R+27} with the $l(2)br28^-l(2)br36^-$ group 3 revertants (but not with $l(2)br28^-l(2)br36^+$ group 3 revertants) suggests that there is a vital locus between *br28* and *br36* that is mutant on Sco^{R+27}.

The location of the proximal lethal on Sco^{R+1} is more difficult to estimate. Since Sco^{R+1}/*Df(2L)TE36-GC* are viable, but Sco^{R+27}/*Df(2L)TE36-GC* lethal, and since *Df(2L)TE36-GC* is $l(2)br28^-l(2)br36^-$, then a vital locus proximal to *br36*, but included within the $l(2)br36^-$ deletions *Df(2L)fn27* and *Df(2L)osp29*, is indicated. However, although Sco^{R+1}/*Df(2L)fn27* is a lethal genotype (0/451), Sco^{R+1}/*Df(2L)osp29* is not quite (89/992). These two deletions differ in that *fn27*, but not *osp29*, is also noc^-. This may indicate an interaction between the *noc* and *br36–br37* regions (see DISCUSSION).

Group 2: Five revertants, Sco^{R+8}, Sco^{R+9}, Sco^{R+11}, Sco^{R+12} and Sco^{R+17}, are included in this group. They are all lethal with *Sco* and, moreover, are lethal *inter se*. They do not show any consistent pattern of lethality with any other revertants (except Sco^{R+1}) (Tables 6 to 8).

The lethality of this group of revertants with *Sco* cannot be trivial, *i.e.*, it cannot be due to lethals unrelated to *Sco* on the revertant chromosomes, as the following argument shows: were the lethality of group 2 revertants and *Sco* due to an unrelated lethal, then such a lethal must have been polymorphic in the *Sco* and *b Sco pr* stocks, because, were it fixed, all of the revertants would have carried it. Yet, if only some *Sco* chromosomes in the *Sco* stocks carried this lethal, then these revertants would not be completely lethal with *Sco*, because they would survive with those *Sco* chromosomes that happened to lack the unrelated lethal. All of group 2 revertants are also lethal with various recombinant *Sco* chromosomes from which extraneous lethals have been removed by exchange.

This leads us to suppose that the lethality of the group 2 revertant with *Sco* is a specific property of these revertants. They cannot simply be Sco^- deletions,

because, if so, they would be far more viable with *Sco* than they are (see ASHBURNER, TSUBOTA and WOODRUFF 1982). Moreover, were they deletions, they would be expected to be deleted for loci other than *Sco*: with the exception of *noc* (see later), they are not. Moreover, they show no consistent pattern of lethality with deletions that span the 34D-35D region (Figure 3); group 2 revertants are viable with deletions which, in sum, cover the entire 34C3 to 35E6 interval [*i.e., Df(2L)b80e3, Df(2L)fn7, Df(2L)A72* and *Df(2L)osp29*]. These revertants are, however, all semilethal with deletions that include both the *pu* to *el* and the *br35* to *br36* intervals [contrast *Df(2L)fnl* and *Df(2L)A376*]. Although not as clear as would be ideal, these data emphasize the "synthetic" nature of the lethality of group 2 revertants with *Sco*, a lethality, we note, that is to some extent covered by the $noc^{+}osp^{+}Adh^{+}$ duplication of the *b el Sco* crossover.

The complexity of the lethality associated with the group 2 revertants is seen in the results of crosses with crossovers that separate the left and right-hand ends of $In(2L)Sco^{R+17}$ and $In(2L)Sco^{R+11}$. Neither $In(2L)C158.1^{L}Sco^{R+17R}$ nor $In(2L)Sco^{R+17L}C158.1^{R}$ are fully lethal with *Sco* (8/892 and 7/172, respectively). $In(2L)C158.1^{L}Sco^{R+17R}$ has a wild-type *el-noc* region from *In(2L)C158.1*, and $Sco/In(2L)C158.1^{L}Sco^{R+17R}$ have a mean of 22.5 bristles/fly. $Sco/In(2L)Sco^{R+17L}C158.1^{R}$ (with a revertant derived *el-noc* region) have only 11.71 bristles/fly. These phenotypes are expected from the model of *Sco* proposed by ASHBURNER, TSUBOTA and WOODRUFF (1982).

Unlike $In(2L)Sco^{R+17}$ itself, neither recombinant derivative is fully lethal with *Sco* nor with all other group 2 revertants. For example, $In(2L)Sco^{R+17L}C158.1^{R}/Sco^{R+11}$ are only semilethal (15/266; 23.1 bristles/fly) as are $In(2L)C158.1^{L}Sco^{R+17R}/Sco^{R+11}$ (10/596; 32.4 bristles/fly). Data from crosses with $In(2L)C158.1^{L}Sco^{R+11R}$ (its reciprocal cannot be recovered with any ease) are similar; thus, $In(2L)C158.1^{L}Sco^{R+11R}/Sco$ are only semilethal (14/624; 18.4 bristles/fly), as are $In(2L)C158.1^{L}Sco^{R+11R}/Sco^{R+11}$ (5/575; 25.8 bristles/fly).

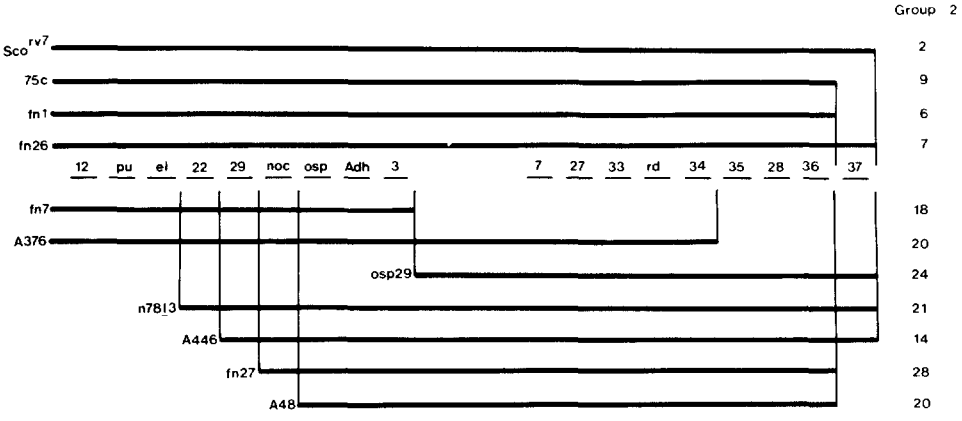

FIGURE 3.—The relative viabilities of group 2 revertants heterozygous with deletions of the *Adh* region. See Figure 2 legend. Data from all five group 2 revertants have been pooled. Note that the revertants are far less viable with deletions that include both the *pu-el* and the *br35-br36* regions than with deletions of either region alone.

In(2L)Sco^{R+17L}*C158.1*^{R} is lethal with *Sco*$^{R+1}$ (0/243), as is *In(2L)Sco*$^{R+17}$ itself. Its reciprocal recombinant, *In(2L)C158.1*^{L}*Sco*$^{R+17R}$ is only semilethal with *Sco*$^{R+1}$ (29/1000).

Group 3: The ten revertants included in group 3 have in common the feature that they fall into a single lethal complementation group (Table 8), and that their common lethal is that defined as *l(2)br28*. An EMS-induced allele of *l(2)br28* (*HG31*) was selected as a lethal included with *Df(2L)fn1*. Subsequently, other alleles were identified as alleles of the embryonic lethal snail (C. NUSSLEIN and P. SIMPSON, personal communication). *Sco*$^{R+26}$/*l(2)br28*^{HG31} are only semilethal (Table 10); the escapers lack one, often both, halteres and are often hemithorax in phenotype. A similar phenotype was seen in *Dp(2; 1)Sco*$^{R+23}$/*l(2)br28*^{HG31} escapers.

At least eight of the group 3 revertants can be shown to be deletions by the criteria that they are lethal with alleles of *l(2)br36* and *l(2)br37*, which map (using unrelated deletions) proximal to *br28*. In addition, all of these revertants, except *Sco*$^{R+15}$ and *Sco*$^{R+26}$, are *noc*$^{-}$ and four (*Sco*$^{R+7}$, *Sco*$^{R+10}$, *Sco*$^{R+14}$ and *Sco*$^{R+18}$) (*i.e.*, group 3a) are also *osp*$^{-}$ and *Adh*$^{-}$.

The simultaneous mutation of *noc* and *l(2)br28* (at least) in ten *Sco* revertants would be unusual were the *Sco* chromosome normal in sequence, since then these loci would be separated by 14 identified complementation groups. Such revertants are recovered with a frequency of once in 6000 chromosomes after X-ray treatment of *Sco* males, far too high a frequency for the mutation of both of these loci to be independent events.

Four of the group 3 revertants (group 3a) are deleted for the three contiguous loci *noc, osp* and *Adh*. These differ from the *osp*$^{+}$ *Adh*$^{+}$ revertants (group 3b) in having a reduced viability with deletions that include the *br22* region (*e.g., Df(2L)fn2, Df(2L)A245, Df(2L)fn7*) (see Figure 4).

Group 4: Four revertants defy simple categorization since each has unique genetic properties. Unlike the previous revertants these do not form a "natural" group, since they are all more or less viable *inter se* (Tables 6–8) (exceptions involve revertant *b Sco pr* chromosomes and are probably due to unrelated lethals), and none are lethal with any identified complementation group in the region (Figure 5).

Sco^{R+2} is viable with all revertants except *Sco*$^{R+24}$ and *Sco*$^{R+27}$. It is semilethal with deletions that lack *l(2)br28* or more proximal loci. *Sco*$^{R+13}$ is not dissimilar to *Sco*$^{R+2}$ in its pattern of lethality.

Unique among the revertants studied, *Sco*$^{R+21}$ is neither lethal nor semilethal when heterozygous with a chromosome deleted for any part or all of the 34D-35D region. Furthermore, *Sco*$^{R+21}$ is the only revertant that is reasonably viable as a homozygote.

Finally, *Sco*$^{R+24}$ resembles the group 2 revertants, in its almost complete lethality with *Sco* (indeed, *Sco*$^{R+24}$ is almost lethal with three group 2 revertants), and the group 1 revertants in having semilethal mutations that map to both the *el-osp* region and to the *br28-br37* region. However, the distal semilethal of *Sco*$^{R+24}$ is not due to mutation of *br29*, nor indeed of *br22*, but maps between the distal breakpoints of *Df(2L)fn2* and *Df(2L)fn3*, *i.e.*, to the neighborhood of

TABLE 10

The relative viabilities of Sco *revertants and lethal alleles of complementation groups in the region of reduced. All revertants are* rd^+

	br33	*br34*	*br35*	*br28*	*br36*	*br37*
R + 1	0.33	0.28	0.38	0.29	0.34	0.37
R + 27	0.35	0.32	0.28	0.26	0.24	0.37
R + 8	0.34	0.38	0.28	0.36	0.36	0.34
R + 9	0.33	0.30	0.32	0.29	0.46	0.31
R + 11	0.31	0.29	0.38	0.30	0.32	0.28
R + 12	0.29	0.37	0.35	0.27	0.29	0.30
R + 17	0.34	0.31	0.38	0.29	0.33	0.35
R + 15	0.36	0.33	0.41	**0.00**	0.35	0.35
R + 19	0.47	0.39	0.45	**0.00**	0.30	0.21
R + 26	0.32	0.31	0.35	**0.10**[a]	0.36	0.33
R + 23	0.37	0.32	0.45	**0.02**[a]	**0.02**[a]	0.31
R + 25	0.33	0.35	0.35	**0.00**	**0.00**	0.37
R + 16	0.31	0.36	0.34	**0.00**	**0.00**	**0.00**
R + 7	0.32	0.41	0.39	**0.00**	**0.00**	**0.00**
R + 10	0.27	0.32	0.32	**0.00**	**0.00**	**0.00**
R + 14	0.30	0.29	0.33	**0.00**	**0.00**	**0.00**
R + 18	0.32	0.31	0.31	**0.00**	**0.00**	**0.00**
R + 2	0.30	0.38	0.35	0.37	0.33	0.34
R + 13	0.33	0.32	0.36	0.27	0.29	0.30
R + 21	0.31	0.32	0.26	0.30	0.34	0.41
R + 24	0.33	0.35	0.29	0.35	0.30	0.28
R + 4	**0.06**[a]	0.44	0.31	0.28	0.37	0.29

[a] See text.

elbow. Sco^{R+24} is not, however, mutant for elbow, nor is it semilethal with any known elbow allele.

Deletion mapping of the recessive bristle phenotype of the revertants

The bristle phenotypes of the revertants are often enhanced by heterozygous deletions; indeed, some $Sco^{R+}/-$ genotypes are *Sco* in phenotype. It can easily be shown that the region responsible for the enhancement of bristle loss of the revertants is the region between *l(2)br22* and *noc* (data not shown).

Duplications and the revertants

The importance of the *br22-noc* region for the phenotype of the revertants can also be deduced from a study of the interaction between the revertants and two duplications of this general region of *2L*. *Dp(2; 2)Adh3*, a tandem duplication from *34B1.2* to *35B3*, suppresses the bristle phenotype of Sco^{R+1} and Sco^{R+11}. This duplication covers both *el* and *Adh* and, presumably, the entire interval between. On the other hand, $Dp(2; 2)C163.41^{L}C158.1^{R}$ does not cover the *el* to

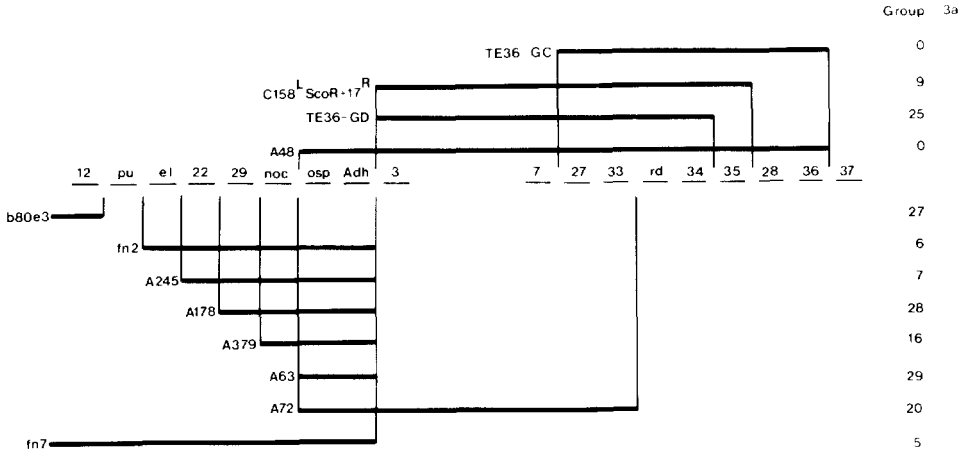

FIGURE 4.—The relative viabilities of group 3a revertants heterozygous with deletions of the *Adh* region. See Figure 2 legend. Data from all four group 3a revertants pooled (*ScoR + 7, 10, 14, 18*). Note the absolute inviability of these revertants with *br28*$^{-}$ deletions and the reduced viability with deletions that include *el* and *l(2)br22*. The viability of heterozygotes with *Df(2L)C158.1*^{L}*ScoR + 17*^{R} is low since this deficiency was synthesized from *Sco*$^{R+17}$.

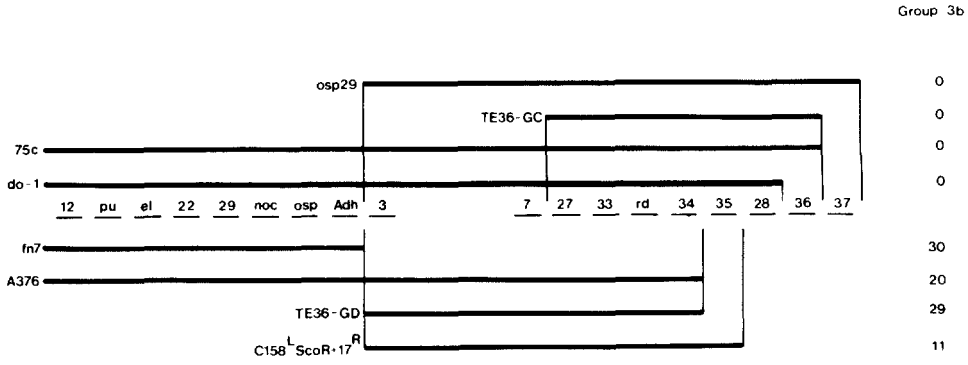

FIGURE 5.—The relative viabilities of group 3b revertants heterozygous with deletions of the *Adh* region. See Figure 2 legend. Data from the six group 3a revertants (*ScoR + 15, 16, 19, 23, 25, 26*) pooled. See Figure 4 legend for *Df(2L)C158*^{L}*ScoR + 17*^{R}. These data map the common recessive lethality of these revertants to *br28*.

Adh region but only the *br3-br36* interval; it has no effect on the revertant's phenotype (Table 11).

The interaction of the Sco *revertants and other mutant alleles in the* Adh *region*

Some 29 lethal and nine "visible" complementation groups have been identified by mutation in the 34D-35D region. Representative alleles of each have been crossed to all revertants. Other than those failures of complementation between revertants and lethal alleles which indicate the extent of the revertant's deficiency, the only interactions we have noted are between *Su(H)* and some group 3 revertants, between revertants and mutations in the *el* to *noc* interval, and those involving *l(2)br28* mentioned before.

Sco^{R+7} and six other group 3 revertants are lethal with the *Su(H)* chromosome. Since *Su(H)* is a lethal allele of *l(2)br7*, a locus that maps to the left of

TABLE 11

The interaction of Sco^{R+1} *and* Sco^{R+11} *with two duplications of region 35*

	Sco^{R+1}			Sco^{R+11}		
	n	x	S.E.	n	x	S.E.
Dp(2; 2)Adh3	81	1.914	0.03	20	37.70	0.14
Cy Roi	57	1.491	0.08	20	33.25	0.33
Difference		+0.423			+4.45	
Dp(2; 2)C163.41^{L}C158.1^{R}	60	1.483	0.08	20	34.80	0.53
Cy Bl	60	1.483	0.09	20	34.70	0.40
Difference		0			+0.10	

The data show the mean number of postvertical bristles in the Sco^{R+1} genotypes and the mean number of head and thoracic bristles in the Sco^{R+11} genotypes. n = number of flies scored; x = the mean bristle number and its standard error (S.E.).

l(2)br33 (with whose lethal alleles these revertants are all viable) this was a puzzling observation. However, it can be shown to be relatively trivial and due to an independent lethal allele of *l(2)br36* on the *Su(H)* chromosome. These revertants are viable with all other *l(2)br7* alleles.

In the preceding paper (ASHBURNER, TSUBOTA and WOODRUFF 1982), we showed that some mutant alleles of elbow, all four alleles of *l(2)br22* and some alleles of *noc* enhance the phenotype of *Sco*. We regarded these effects to be due to the fact that those elbow alleles that enhance *Sco* and all *l(2)br22* alleles are partial noncomplementors of *noc*. It was, therefore, of interest to see just how these mutations interacted with the revertants of *Sco*.

Four alleles of elbow are known; all except el^1 enhance the expressivity of *Sco* (ASHBURNER, TSUBOTA and WOODRUFF 1982). Similarly, they (el^2, el^3 and el^4) enhance the bristle phenotype of *Sco* revertants (Table 12). In addition, el^2, an elbow allele that is semilethal with el^- deletions and *l(2)br22* alleles, has a reduced viability with Sco^{R+4}, with both group 1 and with all but two group 3 revertants (Table 13). el^2/Sco^{R+15} and el^2/Sco^{R+26} do not have a lowered viability. It is interesting that these are the only two group 3 revertants that are both noc^+ and $l(2)br36^+$: more evidence for an interaction of the *el-noc* and *br36* regions.

All four known alleles of *l(2)br22* enhance *Sco* (ASHBURNER, TSUBOTA and WOODRUFF 1982) and are semilethal with group 1 but no other revertants (Table 13). However, *br22* alleles do enhance the bristle phenotype of other revertants, especially those of group 2 (Table 12). It is to be noted that reversion of *Sco* has, except for the two group 1 revertants, also reverted the semilethal interaction between *br22* alleles and *Sco*.

Several EMS and γ-ray-induced alleles of *noc* have been isolated in addition to an allele resulting from the insertion of the w^+rst^+ Transposing Element of ISING and BLOCK (1981). Some *noc* alleles (e.g., noc^4, noc^{TE146}, noc^{19}) but not all (e.g., noc^2, noc^3, noc^{18}) are semilethal with Sco^{R+1}. This is paradoxical since *noc* is not a vital locus, and all known alleles are homozygous or hemizygous viable. Most, but not all, alleles of *noc* enhance the bristle phenotype of the

TABLE 12

The enhancement of the bristle phenotypes of two revertants by mutant alleles of el, l(2)br22 *and* noc, *expressed as the difference in mean bristle number between* Sco^{R+}/*tester and their* Sco^{R+}/Cy *sibs*

	Sco^{R+1}	Sco^{R+11}
el^1	−0.45	+0.60
el^2	−4.60	−4.50
$l(2)br22^{FT1}$	−6.68	−8.03
noc^3	−0.27	−0.55
noc^4	−5.46	−8.45

TABLE 13

The interaction, with respect to viability, of group 1 revertants and alleles of el, 1(2)br22 *and* noc

	Sco^{R+1}		Sco^{R+27}	
	n	%	*n*	%
el				
el^1	234/444	52.7	152/399	38.1
el^2	157/1091	14.4	107/769	13.9
el^3	275/1444	19.0	108/479	22.5
el^4	338/1186	28.5	154/752	20.5
br22				
AR10	135/3061	4.4	75/889	8.4
FT1	323/3442	9.4	53/770	6.8
HG33	93/965	9.6	60/902	6.7
HG46	96/2181	4.4	81/656	12.3
noc				
noc^2	225/1073	21.0	130/446	29.1
noc^3	192/640	30.0	268/929	28.8
noc^4	21/611	3.4	227/884	25.7
noc^{18}	158/764	20.7	82/469	17.5
noc^{19}	98/987	9.9	237/1136	20.9
noc^{TE146}	13/9063	0.1	742/2847	26.1

The number (*n*) and % of nonbalancer progeny are shown over the total progeny number. All chromosomes were balanced over *Cy* balancers with two exceptions, el^1, which was homozygous, and $l(2)br22^{AR10}$, which was balanced over *In(2LR)Gla*.

revertants [seen most clearly with Sco^{R+11} (Table 12)], but there is no correlation between this effect and their viability with Sco^{R+1} (Table 13). Some *noc* alleles are aberrations, e.g., both noc^2 and noc^4 are inversions, but the interaction of *noc* alleles does not correlate with their cytological nature.

Sco^{R+}/noc^- genotypes usually show a typical noc phenotype suggesting that, unlike *Sco* itself, they are mutant for *noc*. The expressivity of the noc phenotype is, under the best of circumstances, rather variable, and a conclusion as to the

state of the *noc* allele of a revertant is based on the phenotypes of many different Sco^{R+1}/noc^{-} genotypes. Only Sco^{R+15}, Sco^{R+26} and Sco^{R+27} seem to be noc^{+}. Note that Sco^{R+15} and Sco^{R+26} differ from other revertants of their group in being both $l(2)br36^{+}$ and fully viable with el^{2}.

The statement that *Sco* is noc^{+} is apparently contradicted by one result: *Df(2L)A446/Sco* flies show a strong noc phenotype. The distal breakpoint of *Df(2L)A446* has been mapped between *l(2)br29* and *l(2)br22* on the basis of the lethality of $Df(2L)A446/Sco^{R+1}$ and the viability of this deficiency with all four *l(2)br22* alleles (Table 9). However, since *Sco* is noc^{+} when heterozygous with any one of many deletions that remove *noc, br29, br22* and *el*, we suspect that the noc^{-} phenotype of *Sco/Df(2L)A446* is a specific consequence of the *A446* breakpoint; that is to say, it is an example of negative complementation.

DISCUSSION

The analysis of the induced revertants of *Sco* is complicated by three factors. One of these is uninteresting, that many of the revertants chromosomes carry lethal mutations quite unrelated to *Sco*, but the other two are of greater importance: (1) *Sco* itself is an unusually complex mutation—a small reciprocal transposition (ASHBURNER, TSUBOTA and WOODRUFF 1982), (2) part of the region involved in the transpositions, that between *pu* and *osp*, is itself genetically complex. There can be no doubt that our interpretation of both *Sco* and its revertants will remain incomplete until we have a better understanding of the genetic structure of the *pu-osp* region. Be that as it may, we can, for the present purposes, proceed as before on the assumption that those alleles of *el* and *br22* that interact with *Sco* do so as a consequence of their "polar" effects on *noc* (see ASHBURNER, TSUBOTA and WOODRUFF 1982 for discussion).

In our previous paper on *Sco* (ASHBURNER, TSUBOTA and WOODRUFF 1982) we interpreted the structure of the mutant *Sco* chromosome in terms of two reciprocal transpositions. Not only does this structure explain the paradoxical observation that deletion and exchange mapping of *Sco* give different "positions" of *Sco*, but it also accounts for the facts that an *el-Sco* crossover chromosome (MARONI 1980) is deleted for three identified loci (*br34, br35* and *rd*) which normally map in the proximal part of the 34D-35D region and is duplicated for both *Adh* and *noc* (MARONI 1980; ASHBURNER, TSUBOTA and WOODRUFF 1982). As we discussed before, strong evidence that this model for the *Sco* chromosome is not too far from the mark comes from the genetic analysis of induced revertants of *Sco*. This evidence has been presented: most germane is the fact that about half of the revertants are deleted, or mutant, for both *noc* and *l(2)br28*, loci at least 14 complementation groups apart on a wild-type chromosome. Although the *Sco* chromosome itself is certainly $br28^{+}$, its status with respect to *noc* requires some further consideration. The few *Sco* homozygotes that we have seen are noc^{+} in phenotype; so are the great majority of heterozygotes between *Sco* and noc^{-} deletions. An exception is the combination of *Sco* and *Df(2L)A446*, a deletion that, uniquely, is broken between *l(2)br22* and *l(2)br29*. The most economical interpretation of this result is that *Df(2L)A446* is not, in fact, completely deleted for noc^{+} but that, as a consequence

of its distal breakpoint, it carries a mutant *noc* allele which shows negative complementation with the transposed *noc* allele of the *Sco* chromosome (see ASHBURNER, AARON and TSUBOTA 1982). We had earlier (ASHBURNER, TSUBOTA and WOODRUFF 1982) suggested that the Sco phenotype results from a position effect at the *noc/br28* boundary of the right-hand transposition, and that the *Sco* chromosome codes for an abnormal *noc* product that, although normally competent with respect to the ocellar phenotype, competes with the product of a normal noc^+ allele to produce the bristle phenotype.

Reversion of the dominant bristle phenotype of *Sco* would appear to result from one, or more, of three types of genetic event. The most common of these is the interruption of the juxtaposition, in *Sco*, of *noc* and *l(2)br28*. It is striking that the great majority of revertants are noc^- and that all except one (Sco^{R+27}) have either been broken between *noc* and *br28* or are mutant for one or both of these loci. If the Sco phenotype does result, as suggested (ASHBURNER, TSUBOTA and WOODRUFF 1982), from "competition" between a normal noc^+ gene product and an altered *noc** product coded for by the transposed *noc* allele of *Sco*, then this result is readily understandable. It cannot, however, be quite that simple since the complete lethality of the group 2 revertants with *Sco*, a lethality that contrasts with the absence of any lethal mutation mapping to the region on these chromosomes, argues that these revertants, at least, are not amorphic mutations. The fact that the group 2 revertants are more viable when heterozygous with the *b el Sco* chromosome than with *Sco* suggests that the synthetic lethal interaction between them and *Sco* can, in part, be compensated for by wild-type *el-noc* function. This would appear to be supported by the observation that the viability of group 2 revertants when heterozygous with deletions is only strongly impaired if the deficiency lacks both the *pu-el* region and more proximal functions in the *br35-br36* region. The normal viability of the group 2 revertants with, for example, *Df(2L)TE36-GC* (which only includes the proximal loci) argues against any proximal lethal (in the *br35-br37* region) on these revertants, despite strong evidence that two of them (Sco^{R+11} and Sco^{R+17}) are inversions broken just distal to *br28*. It is interesting that the only revertant that does not behave as a recessive *Sco* mutation (with respect to the phenotype of Sco^{R+}/Sco heterozygotes and of Sco^{R+}/Df heterozygotes), *i.e.*, Sco^{R+27}, is also the only revertant that is both noc^+ and $l(2)br28^+$.

More rarely does reversion of the Sco phenotype appear to result from genetic events that map, in the main, to the *el-noc* region. Both Sco^{R+1} and Sco^{R+27}, despite evidence for proximal lethals, do carry lethal (or semilethal) mutations that map, by deletion analysis, to this region and, more strikingly, both interact with alleles of loci that map to this region. Moreover, Sco^{R+1} is an inversion broken in the general region of *noc* and *l(2)br28*. None of the revertants are simply deleted for the *el-noc* region; deletion of these loci from *Sco* would, therefore, appear to be insufficient to revert this mutation's phenotype. Indeed, there is considerable evidence that neither Sco^{R+1} nor Sco^{R+27} are simply amorphic alleles: were they so their interaction with alleles of *noc* could not simply be explained. Both group 1 revertants must have mutant *br29* (and *noc*?) functions that positively interfere with the product of the wild-type alleles of these loci.

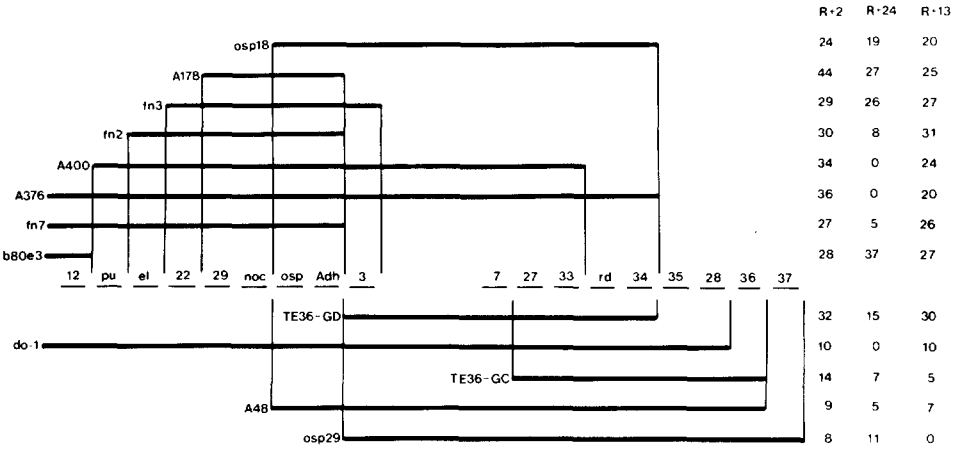

FIGURE 6.—The relative viabilities of three group 4 revertants heterozygous with deletions of the *Adh* region. See Figure 2 legend. All three revertants have a lethal to the right of *br35*; Sco^{R+24} also has a lethal in the region of *el*.

It might be argued that the data we have presented mitigate against the interpretation that *br22*, *br29*, *noc* and *el* are distinct gene loci. Despite the fact that deletions can be used to "separate" these functions, the four loci are not independent. This can be seen, not only by the negative complementation between *l(2)br29* and some alleles of *noc*, but also by the partial failure of complementation for viability between *l(2)br22* and *l(2)br29* (Table 13), between el^2 and *l(2)br22* and between el^2 and *l(2)br29*. Finally, some *el* alleles (e.g., el^2, el^3 and el^4) and all *l(2)br22* alleles show a weak noc phenotype when heterozygous with strong *noc* alleles or noc^- deficiencies.

Evidence for an interaction between these loci is also provided by *Sco* and its revertants. Not only do recessive alleles of all four loci interact with *Sco*, with respect to viability or bristle phenotype (or both), but reversion of *Sco* does, in the majority of cases, relieve this interaction. Thus, for example, *Sco/br22* have a reduced viability and more severe bristle phenotype than *Sco/+*: yet, most revertants are fully viable when heterozygous with *br22* alleles, and only those of group 2 (and Sco^{R+1}) have their bristle phenotype enhanced by mutant *br22* alleles. It is significant that, as discussed before, the evidence suggests that the group 2 revertants cannot be amorphic "alleles" of *Sco*.

Deletion of the entire *pu-noc* region enhances *Sco* to a far greater extent than deletion of only *br22* and *noc* or of *noc* alone (ASHBURNER, AARON and TSUBOTA 1982) (see Figure 7). Indeed, the phenotypes of heterozygotes between *Sco* and deletions in this region identify three critical regions or "incremental points" which, when deleted, progressively enhance the expressivity of *Sco*. These are (1) between *pu* and *el*, (2) between *br29* and *noc* and (3) between *noc* and *osp*. Duplication of the *pu-noc* region suppresses *Sco* (ASHBURNER, TSUBOTA and WOODRUFF 1982). Therefore, a mutation that results in overproduction of one (or more) of the gene products of this region would be expected to suppress, or revert, *Sco*. The negative complementation seen between both Sco^{R+1} and Sco^{R+27} and *noc* indicates that neither revertant is an amorphic allele. Were they to be *br29* hypermorphs and were the *br29* and *noc* products to retain a

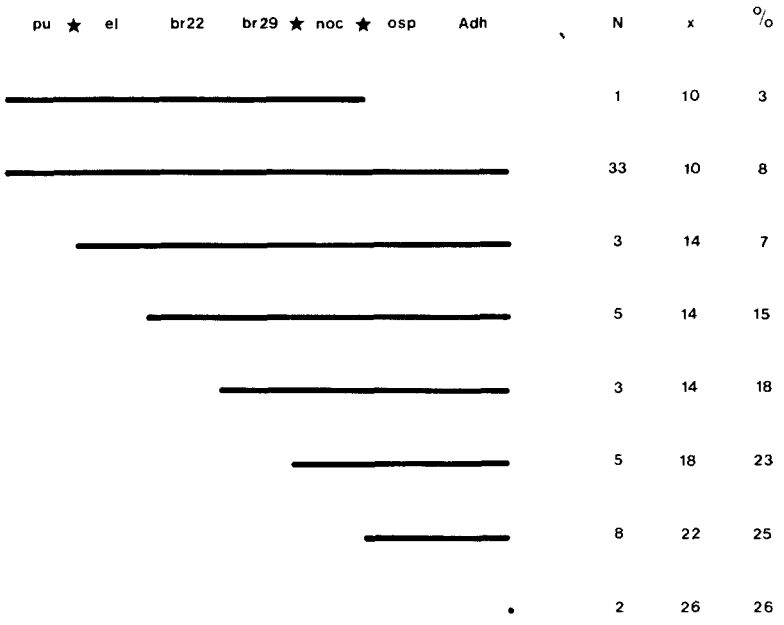

FIGURE 7.—A summary of the enhancement of the *Sco* phenotype by heterozygous deletions (original data of ASHBURNER, TSUBOTA and WOODRUFF 1982; ASHBURNER, AARON and TSUBOTA 1982; and unpublished results). The extent of each group of deficiencies is indicated on a genetic map of the *pu* to *Adh* interval. "N" is the number of different deficiencies in each group studied, "x" the mean bristle number of *Sco/Df* flies, and "%" the viabilities of these genotypes. The three "incremental points" (see text) are marked on the map with asterisks. The mean bristle number of *Sco/Sco* is 7.91 and of *Sco/+* within the range 24–28.

degree of functional homology, then their *Sco* revertant phenotype would be explicable. However, duplications of $l(2)br29^{+}$ are not recessive lethal (M. ASHBURNER, unpublished results). Since the *Sco* chromosome is broken "between" *l(2)br29* and *noc*, it may well not be $br29^{+}$, and overproduction of a mutant *br29* product may account for the recessive lethality of Sco^{R+1} and Sco^{R+27}.

One revertant, Sco^{R+24}, carries a recessive lethal that maps to the left of *el* (or at least, between the distal limits of *Df(2L)fn2* and *Df(2L)fn3*). This corresponds to the most distal of the sites, whose deletion enhances the expressivity of *Sco* (see Figure 7).

The three events that result in the reversion of *Sco* are, therefore, (1) inactivation of the *noc* allele of *Sco* (with or without adjacent deletion); (2) mutation of *br29*, perhaps to a hypermorphic allele; and (3) mutation of a site near elbow.

There are persistent, but poorly understood, indications for some sort of interaction between the *el-noc* and *br35-br37* regions. The viabilities of many revertants, when heterozygous with $br35^{-}$ to $br37^{-}$ deletions, are severely depressed by simultaneous deletion of the *el-noc* region. Moreover, there is the interaction of, for example, el^{2}, with $br35^{-}$ $br36^{-}$ (but not $br35^{-}$ $br36^{+}$), revertants and the so-far undefined, lethals between *br28* and *br37* on both Sco^{R+1} and Sco^{R+27}. Further study, in particular the identification of more loci in the

neighborhood of *br28*, is needed before we can assert, as the data indicate, that these two genetic regions interact in normal development.

Our original intention was to use *Sco* as a convenient dominant marker for studies of the genetic organization of the region surrounding *Adh* and its reversion as a simple and convenient way of generating deletions. In the event *Sco* has proven to be far from a "simple" mutation and its analysis has revealed not only this fact but also an unexpected complexity of the normal genome in the region between (and including) the four contiguous loci *el, l(2)br22, l(2)br29* and *noc*. This region is about 0.1% map units long and is clearly "complex", in the same sense as bithorax or Antennapedia are complex. It differs from these, however, in that there is no obvious similarity between the mutant phenotypes of different components of the complex.

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