SEGMENTAL ANEUPLOIDY AND THE GENETIC GROSS STRUCTURE OF THE DROSOPHILA GENOME[1]

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ABSTRACT

By combining elements of two *Y*-autosome translocations with displaced autosomal breakpoints, it is possible to produce zygotes heterozygous for a deficiency for the region between the breakpoints, and also, as a complementary product, zygotes carrying a duplication for precisely the same region. A set of *Y*-autosome translocations with appropriately positioned breakpoints, therefore, can in principle be used to generate a non-overlapping set of deficiencies and duplications for the entire autosomal complement.——Using this method, we have succeeded in examining segmental aneuploids for 85% of chromosomes *2* and *3* in order to assess the effects of aneuploidy and to determine the number and location of dosage-sensitive loci in the Drosophila genome (Figure 5). Combining our data with previously reported results on the synthesis of Drosophila aneuploids (see LINDSLEY and GRELL 1968), the following generalities emerge.——1. The *X* chromosome contains no triplo-lethal loci, few or no haplo-lethal loci, at least seven Minute loci, one hyperploid-sensitive locus, and one locus that is both triplo-abnormal and haplo-abnormal. 2. Chromosome *2* contains no triplo-lethal loci, few or no haplo-lethal loci, at least 17 Minute loci, and at least four other haplo-abnormal loci. 3. Chromosome *3* contains one triplo-lethal locus that is also haplo-lethal, few or no other haplo-lethal loci, at least 16 Minute loci, and at least six other haplo-abnormal loci. 4. Chromosome *4* contains no triplo-lethal loci, no haplo-lethal loci, one Minute locus, and no other haplo-abnormal loci.——Thus, the Drosophila genome contains 57 loci, aneuploidy for which leads to a recognizable effect on

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the organism: one of these is triplo-lethal and haplo-lethal, one is triplo-abnormal and haplo-abnormal, one is hyperploid-sensitive, ten are haplo-abnormal, 41 are Minutes, and three are either haplo-lethals or Minutes. Because of the paucity of aneuploid-lethal loci, it may be concluded that the deleterious effects of aneuploidy are mostly the consequence of the additive effects of genes that are slightly sensitive to abnormal dosage. Moreover, except for the single triplo-lethal locus, the effects of hyperploidy are much less pronounced than those of the corresponding hypoploidy.

THE following is an account of a procedure designed to examine the effects, in heterozygous condition, of a set of non-overlapping autosomal deletions that cover the entire autosomal complement of Drosophila as well as the effects of the duplication corresponding to each deletion. The procedure is, in principle, quite simple: two *Y*-autosome translocations with different autosomal breakpoints that are determined cytologically are crossed to one another such that zygotes result carrying the *Y*-capped autosome of one translocation and the autosome-capped *Y* of the other. Of the two such combinations, one will be deficient for the region between the autosomal breakpoints while the other will carry the exactly corresponding duplication (see Figure 1). Three *Y*-autosome translocations can be used to construct two adjacent deletions (or duplications) by crossing the translocation with the middle autosomal breakpoint with each of the other two, while crosses between the two translocations with the most distant breakpoints result in individuals carrying a deletion (or a duplication) exactly equal in length to the sum of the two adjacent ones. Thus, a set of *Y*-autosome translocations with appropriately positioned autosomal breakpoints can be combined to synthesize any set of autosomal deletions and duplications.

T(2;4)'s and *T(3;4)*'s have been used in this way by DUBOVSKY and KELSTEIN (e.g. KELSTEIN 1938) and by PATTERSON, BROWN and STONE (1940) to generate autosomal deletions and duplications. The advantages of this method for the general survey envisioned here are: (1) duplication-bearing and deficiency-bearing zygotes are generated irrespective of the adult survival of these aneuploids, thus permitting identification of dominant lethal, as well as viable, aneuploids. (2) Corresponding duplications and deficiencies are of precisely the same length independently of the accuracy of the cytological determination of the breakpoints of the translocations involved; (3) also independent of cytological precision is the property that adjacent deficiencies or adjacent duplications are exactly adjacent and a large deficiency (or duplication) composed of two smaller adjacent ones is exactly equal in length to the sum of the smaller deficiencies; finally (4) the *T(Y;A)* method has the added virtue that while progeny will be simultaneously aneuploid for both autosomal and *Y*-chromosome segments, *Y*-chromosome aneuploidy will not affect survival owing to the inertness of the heterochromatic *Y* chromosome.

Interest in the construction of these aneuploids is both technical and theoretical. From the technical standpoint there are several uses to which the aneuploids can be put. First, an appropriate set of deletions can be used to find the cytological location of any recessive mutation. Second, in studies on gene action it is useful to be able to vary gene dosage, for which both duplications and deletions

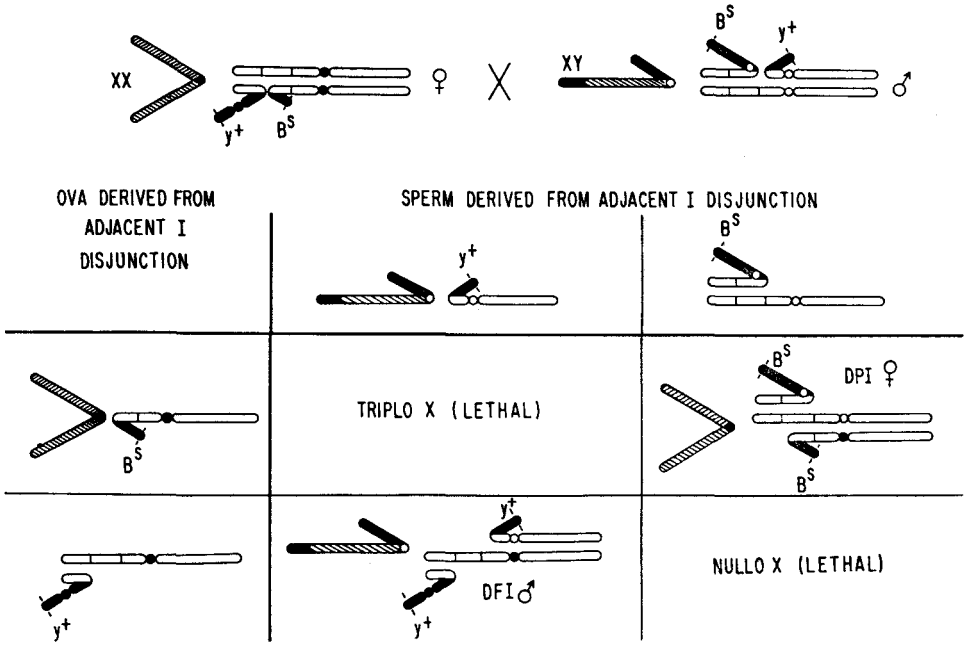


FIGURE 1.—The production of interstitial aneuploids by combining products of adjacent I disjunction from parents heterozygous for different *Y*-autosome translocations with displaced autosomal breakpoints and in which the translocated *Y* chromosome is marked with B^S at the end of Y^L and $y+$ at the end of Y^S. In the case diagrammed the autosomal breakpoint of the paternal *T(Y;A)* is proximal to that of the maternal *T(Y;A)*. *Y*-chromosome material is represented by solid bars, *X*-chromosome by shaded bars, and autosomal material by open bars. The centromeres of maternal elements are solid; those of paternal elements are open. Vertical lines on the autosomal elements designate the positions homologous to translocation breakpoints; in the intact autosomes the positions of both *T(Y;A)*'s are indicated, and in each translocated autosome the position of the breakpoint of the other *T(Y;A)* is indicated. DFI = Interstitial Deficiency; DPI = Interstitial Duplication.

can be utilized. For example, to the extent that enzyme levels are proportional to gene dosage, it should be possible to localize their structural genes without the need for a genetic variant. Third, in experiments requiring the collection of a large number of autosomal recessive mutations (e.g., behavioral mutants, meiotic mutants, etc.), the use of deletions can be advantageous in three distinct ways: (1) the laborious task of making autosomes homozygous can be obviated by examining the locus of interest in the haploid condition; (2) locus-specific mutants can be selected; and (3) autosomes treated with higher-than-usual doses of mutagen can be screened for new mutations because the treated autosome is tested in heterozygous condition except for the region of the deletion.

From the theoretical standpoint, constructions of this type provide insight into several questions relating to the effects of aneuploidy. First, duplications and deficiencies covering the entire autosomal complement would reveal all loci which when present in one or three doses result in lethality or a mutant phenotype. We may distinguish two general classes of such loci—those which result in

lethality when present in one dose (haplo-lethal) or three doses (triplo-lethal) and those which give a mutant phenotype when present in one dose (haplo-abnormal) or three doses (triplo-abnormal). It should be noted that our conclusions concerning aneuploid-lethal and aneuploid-abnormal loci are based only on adult survival and gross morphological changes associated with aneuploidy for specific regions, and it may well be the case that examination at more refined levels would reveal decreased survivals and altered phenotypes associated with aneuploidy at many if not all loci. Second, it is known generally that whereas small deficiencies survive fairly frequently in heterozygous condition, large deficiencies are almost invariably lethal. In an examination of the type envisaged here, it should be possible to decide how often a heterozygous deletion is lethal because of general genic inbalance and how often because the deletion contains one or more haplo-lethal loci. Third, the examination of the effects of one, two or three doses of many different loci might provide insights into the general question of dominance.

It was with these points in mind that we undertook to construct deletions and duplications for as large a fraction of the autosomal complement as possible. The principal generalizations that emerged are given below. (These generalizations and all others made in this report follow from the data presented here and the tabulation of Drosophila mutants by LINDSLEY and GRELL, 1968; we have not attempted an independent systematic search of the literature.)

Drosophila is quite insensitive to segmental trisomy; there appear to be but one triplo-lethal and only two triplo-abnormal loci in the genome. We have regularly observed individuals trisomic for the distal halves of each chromosome arm and occasional survivors for even greater degrees of terminal hyperploidy. Extensive hyperploidy is, however, generally accompanied by reduced survival and a variety of morphological anomalies.

Drosophila is much more sensitive to segmental monosomy than trisomy. Although haploidy for approximately one percent of the genome is frequently viable, three percent of the genome appears to be the upper limit that the fly can tolerate, while severely reduced viability and morphological abnormalities often accompany even smaller haploid regions. Nevertheless, there are very few, and conceivably but one, haplo-lethal loci. There are two classes of haplo-abnormal loci. The first of these is composed of the "Minutes." Heterozygous deficiencies for Minute loci produce flies with short, thin bristles, reduced developmental rate, low viability and fertility, and often other morphological abnormalities; there are at least 41 and probably not many more Minute loci in the Drosophila genome—7 on the *X* chromosome, 17 on chromosome *2*, 16 on chromosome *3*, and 1 on chromosome *4*. Haplo-abnormal loci of the second category produce a variety of other phenotypes; there are at least 13 of these in the Drosophila genome.[8]

PRODUCTION AND ANALYSIS OF *T(Y;A)*'S

The procedure for isolating *Y*-autosome translocations was the following: *y cv v f/B*^{s}*Yy*+

[8] The technical details of the stock constructions, the crosses, and the identification of aneuploid progeny are complex. The reader who has examined the introductory paragraphs and studies Figure 5 can leave the text here and rejoin it in the RESULTS section with little loss in comprehension.

males were irradiated with approximately 4000 r of X rays and crossed to *$Y^SX{\cdot}Y^L$, In(1)EN, y/In(1)dl-49, y Hw m^2 g^4; bw/bw; st/st* females. *B^SYy+* is a *Y* chromosome carrying the dominant marker, Bar-Stone, on the tip of its long arm and the normal allele of yellow at the terminus of its short arm; *$Y^SX{\cdot}Y^L$, In(1)EN, y* is a completely inverted *X* chromosome with *Y^S* attached distally and *Y^L* proximally; it is fertile in males in the absence of other *Y* chromosomes but sterile in homozygous females (sterility is not an inevitable consequence of homozygosity for *$Y^SX{\cdot}Y^L$*, and the reason for it in the stock used here was not investigated). The irradiated males were discarded within four days of treatment so that only post-meiotically irradiated cells were sampled. *F_1, $Y^SX{\cdot}Y^L$, (1)EN, y/B^SYy+; bw/+; st/+* males were individually crossed to females of the same constitution as their mothers, and the progeny examined. Because of the difficulty in distinguishing *bw* and *st* in the presence of *g* and *B^S*, the *Hw m^2 g^4 B^S* male progeny were ignored. An F_1 male was classified as normal if there was no evidence of linkage between any two chromosomes, and not more than one individual among his progeny in which *B^S* had segregated from *y+*. Linkage between *bw+* and *st+* indicates a translocation between chromosomes *2* and *3*; linkage between the *Y*-chromosome markers—*y+*, *B^S*—and *bw+* indicates a *Y-2* translocation; linkage of *y+ B^S* and *st+*, indicates a *Y-3* translocation; linkage of *y+ B^S*, *bw+*, and *st+* implies a *T(Y;2;3)*; independence of *y+* and *B^S* with respect both to each other and to the autosomal markers indicates a *T(Y;4)*. In all cases, the pseudolinkage is complete if all aneuploid progeny die, and partial if aneuploids survive. Aneuploid survival accompanied by segregation of *Y*-chromosome markers is indicative of a reciprocal translocation, whereas aneuploid survival unaccompanied by segregation of *Y*-chromosome markers indicates the insertion of an autosomal segment into the *Y* or a break in the *Y* chromosome distal to its markers. Even though reciprocal aneuploids survive, reciprocal translocations can be identified by the linkage of one *Y* marker to the autosome; insertional translocations in which both aneuploids survive cannot be so identified except in special cases where the surviving deficient aneuploid has a characteristic phenotype (e.g. *M*). The results of the examination of the progeny of backcrosses of 11,764 F_1 males are given in Table 1.

Four hundred sixty-seven *Y-2* or *Y-3* translocations were recovered; for each, five operations were performed. First, the progeny of the cross *$Y^SX{\cdot}Y^L$, In(1)EN, y; bw* (or *st*)*/T(Y;A)* ♂ ♂ × *$Y^SX{\cdot}Y^L$, In (1)EN, y/In(1)dl-49, y Hw m^2 g^4; bw/bw; st/st* ♀ ♀ were scored. Second, each translocation, which was recovered and maintained in attached-*XY*-bearing males, was tested for fertility in the absence of the extra *Y* chromosome—that is, as *In(1)dl-49, y Hw m^2 g^4/T(Y;A)* ♂ ♂. Third, each *Y*-autosome translocation was balanced in an *attached-X/T(Y;A)/Balancer* ♀ × *attached-XY/T(Y;A)/Balancer* ♂ stock. The stocks were *C(1)RM, y/$Y^SX{\cdot}Y^L$, In(1)EN, y/T(Y;2)/In(2L+2R)Cy, Cy cn^2* (or *In(2LR)SM1, al^2 Cy cn^2 sp^2*) for the *T(Y;2)*'s and *C(1)M3, y^2 bb/$Y^SX{\cdot}Y^L$, In(1)EN, y/T(Y;3)/In(3LR)TM6, ss⁻ bx^{34e} Ubx^{67b} e* for the

TABLE 1

Translocations recovered from B^SYy+*-bearing mature sperm treated with approximately 4000 r of X rays*

Determinations are made on the basis of the segregation of the markers *y+*, *B^S*, *bw+* and *st+* among the progeny of F_1 *$Y^SX{\cdot}Y^L$, In(1)EN, y/B^SYy+; bw/+; st/+* ♂ ♂ by *$Y^SX{\cdot}Y^L$, In(1)EN, y/In(1)dl-49, y Hw m^2 g^4; bw/bw; st/st* ♀ ♀.

Type of progeny	Number	Relative frequency
Normal	8,525	0.725
Sterile	1,908	0.162
T(2;3)	754	0.064
T(Y;4)	46	0.004
T(Y;2;3)	64	0.005
T(Y;2)	233	0.020
T(Y;3)	234	0.020
Total	11,764	

T(Y;3)'s. Fourth, the *Y*-arm breakpoint for each translocation was determined in one of a number of ways. When the autosomal breakpoint is sufficiently distal to allow the survival of hyperploid progeny, the *Y*-arm break can be immediately inferred as being in the short arm if the hyperploids are marked by B^S and in the long arm if they are marked by $y+$. For those translocations that do not produce viable hyperploids, the determinations are made either cytologically or from the results of crosses between two translocations, with different *Y*-arm breakpoints or one of which has a known *Y*-arm break (for details, see Tables 5 and 7, and accompanying discussion below). Fifth, the autosomal breakpoint was cytologically localized on the standard salivary chromosome map. For cytological analysis, salivary preparations were made from male larvae taken from stock before balancing or from female larvae selected from a cross of $Y^SX{\cdot}Y^L$/ *T(Y;A)/Balancer* ♂ by *C(1)RM* ♀ after balancing; ideally, salivaries which had the full translocation complement were used to determine the breakpoints.

Salivary analysis allows detection of wholly euchromatic rearrangements and of euchromatic–heterochromatic rearrangements. In addition, the small *X*-chromosome segments associated with the termini of the *Y* chromosome have distinctive morphologies in polytene preparations; consequently, it was occasionally possible to make a tentative determination of the *Y*-chromosome breakpoint from the morphology of the tip of the proximal autosomal element. Complex *T(Y;A)*'s were frequently encountered, but when the autosome is divided into two contiguous segments it behaves as a reciprocal *T(Y;A)* and can be used in the scheme diagrammed in Figure 1. An example of a translocation that cannot be so used is an autosomal inversion with the *T(Y;A)* breakpoint included within it.

Because of the aggregation of all heterochromatic regions in the chromocenter, salivary analysis will not reveal a wholly heterochromatic aberration nor the presence of more than one heterochromatic break in a euchromatic–heterochromatic rearrangement. Consequently, translocations between the heterochromatic *Y* chromosome and the proximal heterochromatin of an autosome yields completely normal salivary-gland-chromosome configurations. A translocation was therefore classified as heterochromatic if no euchromatic breaks could be detected. Furthermore, the cytological undetectability of heterochromatic–heterochromatic rearrangements led to some *T(Y;A)*'s being diagnosed as reciprocal translocations with euchromatic autosomal breakpoints when they were in fact more complex. For example, an insertion of part of an autosome into the *Y* chromosome, where one of the autosomal breakpoints is euchromatic and the other heterochromatic, will appear cytologically as a reciprocal *T(Y;A)* with euchromatic breakpoint. The only indication of the presence of such a rearrangement prior to crossing different translocations *inter se* is the absence of terminal hyperploids even though the position of the euchromatic breakpoint identified cytologically suggests that they should survive. The breakpoints of each translocation were determined cytologically by two observers independently and concordance achieved before the breakpoint was considered established.

The results of these analyses are summarized in Figure 2 and Table 2[9]. The distribution of translocation breakpoints with respect to salivary-gland-chromosome length (Figure 2) reveals a striking nonrandomness. The recovered reciprocal *Y*-autosome translocations preferentially have the autosomal breakpoint either in the centromeric heterochromatin or in the distal euchromatin while the proximal euchromatin is much less frequently involved. Thus, of the 164 *T(Y;2)*'s for which the breakpoint was determined, 26 percent had a heterochromatic break, 48 percent were broken in the distal half of either *2L* or *2R*, while only 26 percent were broken in the proximal halves of the arms. In the case of chromosome 3, 177 *T(Y;3)*'s were examined; among these, 17 percent were broken in the heterochromatin, 55 percent in the distal halves, and 28 percent in the proximal halves of *3L* or *3R*.

The nonrandom distribution of breakpoints could reflect regional differences either in radiosensitivity or in the ability to form recoverable *Y*-autosome translocations. It is not possible to distinguish between these two possibilities, but the latter interpretation seems more reasonable.

[9] In addition, the results of these analyses, presented for each individual translocation separately along with stock designations and other particulars, can be found both in the appendix to this paper and in the "Report of the Seattle-La Jolla Drosophila Laboratories: The use of *Y*-autosome translocations in the construction of autosomal duplications and deficiencies", supplement to *Drosophila Information Service* 47 (1971).

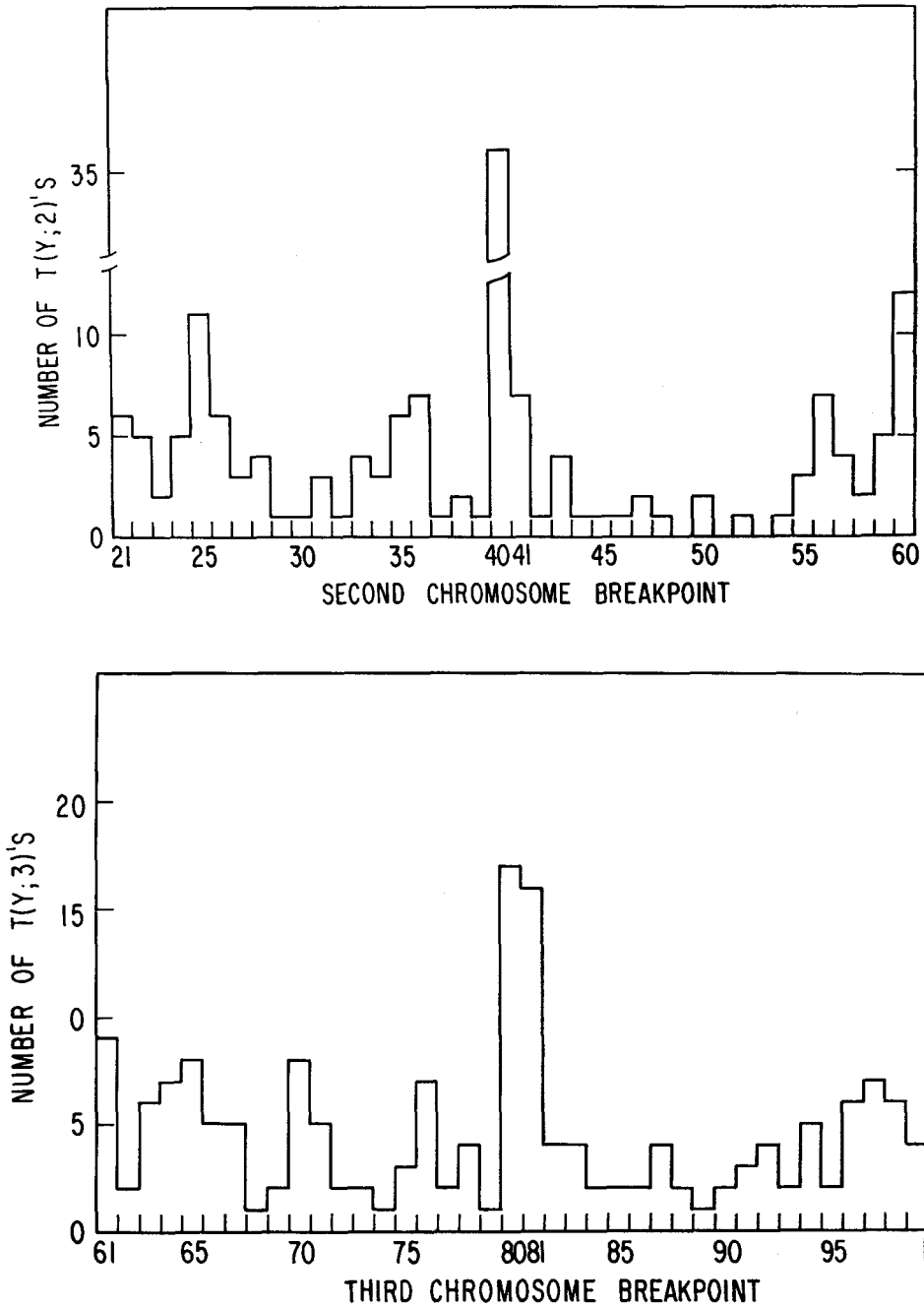


FIGURE 2.—The frequency distribution of autosomal breakpoints for 164 *T(Y;2)*'s and 177 *T(Y;3)*'s. For chromosome *3*, the heterochromatic breakpoints have not been located with respect to the centromere, and are consequently equally distributed in regions 80 and 81. In the case of chromosome *2*, the arm is known for all but eight cases; in the figure these eight are distributed by arm in the same proportions as the known cases.

TABLE 2

The cytogenetic characteristics of the 467 T(Y;A)*'s recovered*

"Insertions" are insertional translocations of an autosomal segment into the *Y* chromosome. The "Complex" class are multiple-break *T(Y;A)*'s either not usable in the construction of interstitial aneuploids or in which we have not been able to ascertain the new order. N.T. = not tested. $Y^D = Y$ chromosome breakpoint distal to *Y* markers.

	Y-arm breakpoint												
	Y^S			Y^L			Y^D			N.T.			
	Fertility of *X/T(Y;A)* ♂♂												
Autosomal breakpoint	Fert.	Ster.	N.T.	Fert.	Ster.	N.T.	Fert.	Ster.	N.T.	Fert.	Ster.	N.T.	Σ
2L	30	17	1	8	12	0	0	0	0	0	3	1	72
2 Het	11	8	0	7	9	0	0	0	0	3	5	0	43
2R	16	10	1	7	13	2	0	0	0	1	1	0	51
Subtotal		94			58			0			14		
Insertions	—	—	—	—	—	—	—	—	—	5	4	0	9
Complex	1	0	0	1	0	0	0	0	0	0	2	0	4
N.T.	4	6	1	1	1	1	0	0	0	11	24	5	54
Total													233
3L	20	14	0	19	18	1	1	0	0	2	6	0	81
3 Het	0	0	0	0	1	0	0	0	0	11	19	0	31
3R	15	13	0	17	15	0	2	0	0	2	2	0	66
Subtotal		62			71			3			42		
Insertions	—	—	—	—	—	—	—	—	—	1	11	3	15
Complex	0	0	0	0	1	0	0	0	0	4	4	1	10
N.T.	0	2	2	0	3	2	0	0	0	8	9	5	31
Total													234

In the first place, the data of BAUER, DEMEREC, and KAUFMANN (1938) indicate that the distribution of chromosome breaks induced by X rays in mature sperm and scored in the salivary-gland chromosomes of F_1 larvae is uniform with respect to the euchromatic subdivisions. Secondly, we have observed that all *Y*-autosome translocations with breakpoints in certain autosomal regions were almost impossible to keep in stock owing to extreme inviability or sterility (for example, five *T(Y;3)*'s in region 67D–69F). The matter must, however, remain equivocal because of the impossibility of a direct test and also because BAUER, DEMEREC, and KAUFMANN did note a consistent, but statistically non-significant, excess of very distal breakpoints.

Aside from the distribution of autosomal breakpoints, no other nonrandomness is evident from the data (Table 2).

TRANSIENT EFFECTS OF IRRADIATION

Two observations made during the course of the experiments deserve consideration before turning to the examination of the effects of aneuploidy. The first of these is that in the initial crosses of translocation-bearing males to normal females, the recoverable classes (from alternate disjunction) are translocation-bearing males and normal females. These two classes should appear equally frequently, but we often observed decided excesses of translocation-bearing progeny suggesting the possibility of meiotic drive in the translocation heterozygotes. Upon retesting a sample of the most extreme cases, however, the ratios were normal. Thus, either the original abnormal ratios were simply statistical fluctuations or else were cases of "transient" meiotic drive.

TABLE 3

The results of crosses of $Y^{S}X{\cdot}Y^{L}$, In(1)EN, y/B^{S}Yy+; bw/+; st/+ ♂ ♂ *by* $Y^{S}X{\cdot}Y^{L}$, In(1)EN, y/In(1)dl-49, y Hw m^2 g^4; bw/bw; st/st ♀ ♀

In the "experimental" set, the parental males resulted from a cross of *y cv v f*/$B^{S}Yy+$ males irradiated with approximately 4000 r of X-rays by $Y^{S}X{\cdot}Y^{L}$, *In(1)dl-49, y Hw* m^2 g^4; *bw/bw*; *st/st* females. The data presented exclude cultures scored as translocations or containing three or more exceptional progeny. The "control" set includes all the data of crosses from original males that had not received irradiation. The "selected" column gives the results of cultures derived from irradiated males and selected because they produce more than three exceptions; in these counts, *bw* and *st* were ignored. The distribution of exceptions by individual cultures for the control and selected sets are presented graphically in Figure 3.

	Control		Experimental		Selected	
Phenotype of progeny	Number	Frequency	Number	Frequency	Number	Frequency
y + + ♀ ♀	549	.221	1118	.227		
y bw + ♀ ♀	456	.183	1054	.214		
					4601	.614
y + *st* ♀ ♀	359	.144	682	.138		
y bw st ♀ ♀	320	.129	602	.122		
B^{S} + + ♂ ♂	255	.103	538	.109		
B^{S} *bw* + ♂ ♂	198	.080	355	.072		
					2154	.288
B^{S} + *st* ♂ ♂	171	.069	283	.057		
B^{S} *bw st* ♂ ♂	164	.066	226	.046		
B^{S} + + ♀ ♀	0	.000	5	.001		
B^{S} *bw* + ♀ ♀	0	.000	4	.001		
					144	.019
B^{S} + *st* ♀ ♀	1	.001	3	.001		
B^{S} *bw st* ♀ ♀	0	.000	0	.000		
y + + ♂ ♂	3	.001	22	.001		
y bw + ♂ ♂	4	.002	14	.003		
					591	.079
y + *st* ♂ ♂	2	.001	6	.001		
y bw st ♂ ♂	4	.002	14	.003		
♂ ♂/Total		.322		.296		.366
Percent nondisjunction		.006		.014		.098
♀ ♀/♂ ♂ in exceptions		.077		.214		.244

This latter possibility may not be dismissed out of hand because there definitely is a very striking, but transient, effect of irradiation on sex chromosome nondisjunction. In the crosses of F_1 sons ($Y^{S}X{\cdot}Y^{L}$, *In(1)EN*, *y*/$B^{S}Yy^{+}$; *bw*/+; *st*/+) of irradiated fathers by the tester females described above, some individual males exhibited very high frequencies of sex-chromosome nondisjunction suggesting the possibility that meiotic mutants had been induced by the irradiation. These results are presented in Table 3. The "control" column gives the results of a set of crosses in which the fathers of the parental males had not been irradiated. The "experimental" column gives the results of a set of crosses from irradiated fathers that were counted (the vast majority of the 11,764 crosses were

examined but not counted); these cultures had been discarded as non-translocated by the criteria used in Table 1 and in addition contained fewer than three sex-chromosome exceptions. The counts from all cultures from irradiated grandfathers exhibiting three or more exceptions at the time the cultures were first examined are given in the "selected" column.

It is evident that sex-chromosome nondisjunction is considerably higher in the sons of irradiated fathers than in the sons of untreated fathers. Moreover, the phenomenon under consideration is really nondisjunction and not merely chromosome loss because the fraction of exceptions receiving both homologs is at least as high in the experimental sets as in the control. The distribution of nondisjunction frequencies for individual males is shown in Figure 3. It can be seen that some sons of irradiated fathers produce nearly 50% exceptions.

A number of regular sons of males exhibiting high nondisjunction were retested in crosses identical to those of the preceding generation. The frequency and distribution of nondisjunction was the same as for the unirradiated control (Figure 3). That is, the high rate of nondisjunction induced by the irradiation persisted for only one generation.

There were two lines that were exceptions to this rule; in these the high rate of nondisjunction did not disappear after one generation. In one of the two lines,

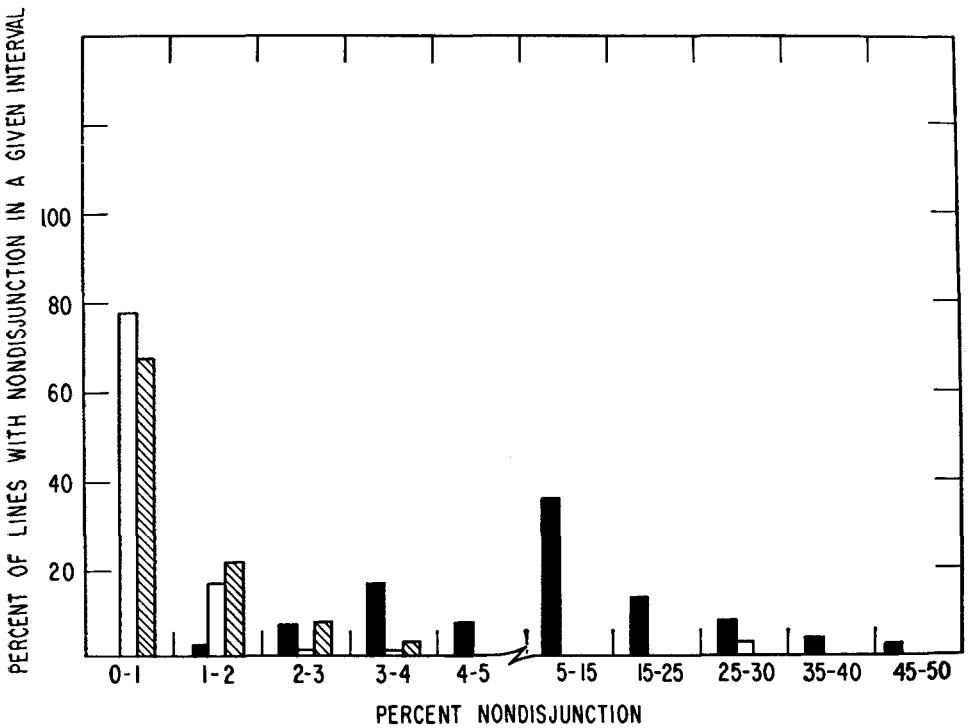


FIGURE 3.—The distribution of exceptions by individual cultures for the unirradiated control (shaded bars), the lines selected as showing high nondisjunction (solid bars) and retests of sons of males that showed high nondisjunction (open bars). Mean frequencies are shown in Table 3.

however, the effect was unstable—some males giving 30–40% exceptions and others none. This continued for several generations before the effect disappeared. The other line regularly produces a high frequency of nondisjunction and thus represents either some special chromosome aberration or else is a meiotic mutant.

It seems clear that transient high nondisjunction can be induced by X-irradiation and also perhaps transient forms of meiotic drive can be induced. Unfortunately, because the phenomena do not persist, they cannot easily be studied. It seems most reasonable to assume that these phenomena are caused by events occurring in the gonia of the F_1 males because they completely disappear in exactly one generation. If they are not gonial, then it is necessary to imagine some chromosomal instability induced in mature sperm that is not manifest during any of the immediately succeeding mitotic divisions, becomes manifest in the meiotic cycle, and then disappears permanently.

SYNTHESIS OF SEGMENTAL ANEUPLOIDS

As indicated in Figure 1, by combining elements of two different *Y*-autosome translocations whose autosomal breakpoints are displaced from one another, it is possible to synthesize interstitial duplications and deficiencies for the autosomal segment between the two breakpoints. A judicious choice of markers allows the recognition of these interstitial aneuploids. In general, the genotypes of the progeny recovered from crosses between heterozygotes for different *Y*-autosome translocations differ, depending on (1) which arm of $B^S Y\gamma+$ is broken in both the maternal and paternal *T(Y;A)*'s; (2) whether the autosomal breakpoint of the maternal *T(Y;A)* is distal or proximal to that of the paternal *T(Y;A)*; and (3) which types of aneuploid progeny survive. Furthermore, genotypes depend on whether chromosome *2* or *3* is involved in the translocation, as the markers used to identify the autosomal homolog of the translocation necessarily differ for the two autosomes. Considering all possible combinations of these parameters, sixteen different types of progenies are encountered. In addition, there are exceptional cases where the *Y* breakpoint is distal to $\gamma+$ or B^S or where the autosomal breakpoints are on opposite sides of the centromere; these cases will be considered separately. Rather than describe, as examples, one or several of the sixteen types of progenies, we have chosen instead to give a general decription that applies to all. For this exposition we adopt a notation somewhat at variance with standard Drosophila conventions; when it is necessary to differentiate maternally-derived from paternally-derived chromosomes, we italicize the former but not the latter. The cross utilized to generate interstitial aneuploids may, then, be designated as follows:

$$XX/T(Y;A)_i/Bal \; ♀ \times \mathrm{XY/T(Y;A)_j/Dom} \; ♂$$

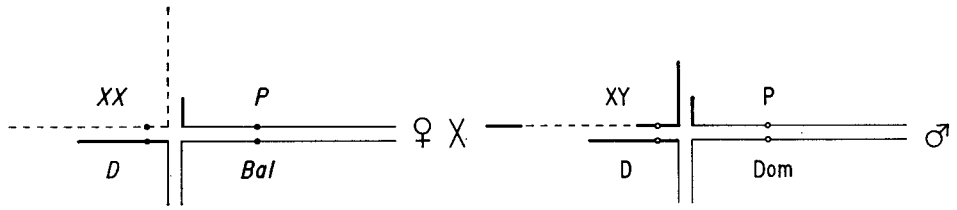
where *XX* signifies *C(1)RM, y; Bal* signifies a balancer chromosome that is either *In(2L+ 2R)Cy, Cy cn²* or *In(2LR)SM1, al² Cy cn² sp²* in crosses involving *T(Y;2)*'s and *In(3LR)TM6, ss⁻ bx³⁴ᵉ Ubx⁶⁷ᵇ e* in crosses involving *T(Y;3)*'s; *XY* signifies $Y^S X \cdot Y^L$, *In(1)EN, y;* and *Dom* signifies a dominant-bearing chromosome that is *Sco* in crosses involving *T(Y;2)*'s and *Sb* in crosses involving *T(Y;3)*'s.

Use of the compound-*X* chromosome permits the regular presence of the *T(Y;A)* in females and the $Y^S X \cdot Y^L$ chromosome insures fertility of males carrying translocations with impaired *Y*-fertility. Both *C(1)RM* and $Y^S X \cdot Y^L$ are marked with *y* to allow scoring of the $\gamma+$ marker on the translocated $B^S Y\gamma+$.

A reciprocal *Y*-autosome translocation divides the autosome into two elements: one may be symbolized $A^D Y^P$—it carries the autosomal terminus on the *Y* centromere and the marker of the *Y* arm not broken by the translocation; the other element may be symbolized $Y^D A^P$—it carries the *Y* terminus, with the marker of the *Y* arm broken by the translocation, on the centric fragment of the autosome. If we designate $A^D Y^P$ as *D* and $Y^D A^P$ as *P*, using *D* and *P* when they

TABLE 4

The disjunctional origin[1] of the euploid and possibly-surviving aneuploid zygotes from the following cross[2] where both maternal and paternal translocations are broken in the same autosomal arm



Zygotic genotypes are partitioned into translocated and non-translocated complements and the disjunctional origin of all potentially viable combinations indicated.

		Zygotic genotype partitioned with respect to							
		Non-translocated complement							
Aneuploid types	Translocated complement	XY *Bal*	XY Dom	XY $\frac{Bal}{Dom}$	XY	*XX Bal*	*XX* Dom	*XX* $\frac{Bal}{Dom}$	*XX*
+	*DP*	*1:3*/1:3	*alt*/alt			*4:0*/4:0	*3:1*/3:1		
	DP	*3:1*/3:1	*4:0*/4:0			*alt*/alt	*1:3*/1:3		
	DDPP				*alt*/3:1				*3:1*/alt
	O			*3:1*/alt				*alt*/3:1	
Ani	*DP*	*ad1*/ad1	*1:3*/1:3			*3:1*/3:1	*ad2*/ad2		
	D*P*	*ad2*/ad2	*3:1*/3:1			*1:3*/1:3	*ad1*/ad1		
Tpt	*D*			*ad1*/alt				*3:1*/3:1	
	DDP	*ad1*/3:1	*1:3*/4:0			*3:1*/alt	*ad2*/1:3		
Tpt	D			*3:1*/3:1				*alt*/ad1	
	DDP	*1:3*/ad2	*alt*/3:1			*4:0*/1:3	*3:1*/ad1		
Tpt/Tpt	*DD*			*ad1*/3:1				*3:1*/ad1	
Dft	*P*	*ad2*/1:3	*3:1*/alt			*1:3*/4:0	*ad1*/3:1		
	DPP				*3:1*/3:1				*ad1*/alt
Dft	P	*3:1*/ad1	*4:0*/1:3			*alt*/3:1	*1:3*/ad2		
	DPP				*alt*/ad1				*3:1*/3:1
Dft/Dft	*PP*				*3:1*/ad1				*ad1*/3:1

[1] Disjunction notation is maternal type of disjunction followed, after a slash bar, by the paternal type of disjunction.
[2] Dashed line = *X* chromosome; heavy line = *Y* chromosome; fine line = autosome. See text for explanation of symbol conventions and detailed discussion of table.

are maternally derived and D and P when paternally derived, we can represent the basic aneuploid-generating cross as:

$$XX/D_i;\ Bal/P_i\ ♀ \times XY/D_j;\ Dom/P_j\ ♂$$

The next conventions to be established concern disjunction in such translocation heterozygotes; we consider all possible modes of segregation of the four elements in the above genotypes—two from two, three from one, and four from zero. In females, alternate disjunction (*alt*) produces

XX Bal and *D P* gametes; adjacent I disjunction (*ad1*) produces *XX P* and *D Bal* gametes; adjacent II disjunction (*ad2*) produces *XX D* and *Bal P* gametes; nondisjunction of sex-chromosome centromeres (*3:1*) produces *XX D Bal* and *P* or *XX D P* and *Bal* gametes; nondisjunction of autosomal centromeres (*1:3*) produces *XX Bal P* and *D* or *XX* and *D Bal P* gametes; passage of all four elements to the same pole (*4:0*) produces *XX D Bal P* and *O* gametes. There are comparable segregation possibilities in the male. Thus for each parent there are sixteen possible gametic types and hence 256 possible zygotic genotypes. Of these, all euploid zygotes survive. Aneuploid zygotes fall into two classes: (1) those considered invariably lethal because they carry either two or no *X*-chromosome centromeres (i.e. triplo-*X* or nullo-*X*) or one or three autosomal centromeres; and (2) those with one *X*-chromosome and two autosomal centromeres, the survival of which depends upon the autosomal breakpoints of the translocations. Table 4 summarizes all possibly-surviving progeny types among the 256 possible zygotes in the basic cross.

The possibly-surviving aneuploid types are as follows: (1) Interstitial aneuploids (*Ani*) are trisomic or monosomic for the segment between the autosomal breakpoints of the translocations used in the cross. The interstitial aneuploid carrying A^DY^P from the parent with the more proximal, and Y^DA^P from the parent with the more distal, autosomal breakpoint is trisomic for the region between the two translocation breakpoints (*Tpi*). Those carrying A^DY^P from the parent with the more distal, and Y^DA^P from the parent with the more proximal, autosomal breakpoint carry the corresponding interstitial deficiency (*Dfi*). (2) Terminal triplications carry, in addition to a diploid complement, the A^DY^P element of either the maternal (*Tpt*) or paternal (Tpt) translocation. (3) Terminal quadruplications (*Tpt*/Tpt) carry, in addition to a normal diploid complement, the A^DY^P element of both the maternal and the paternal translocation; they are consequently tetrasomic for the terminal region up to the point of the more distal autosomal translocation breakpoint and trisomic from that point to the more proximal autosomal breakpoint. (4) Terminal hypoploids carry one complete autosome and the Y^DA^P (but neither A^DY^P element) from either the mother (*Dft*) or the father (Dft). (5) Homozygous terminal deficiencies (*Dft*/Dft) are nullosomic for the region from the tip to the more distal autosomal breakpoint and monosomic from that point to the more proximal autosomal breakpoint. It is conceivable that this class would survive when the autosomal breakpoints are very nearly terminal.

Thus, starting from the top of Table 4, the number of rows containing surviving classes is a function of the autosomal breakpoints of the translocations used in the cross, with distal breakpoints producing more surviving classes than proximal ones. The recovery of the various zygotic classes is influenced not only by viability but also by the relative frequencies of the various types of segregation. Our impression is that roughly alt > adl > 3:1 ⩾ ad2 > 1:3 > 4:0, so that the vast majority of progeny are produced by either alternate or adjacent I disjunction in one or both parents.

The phenotypes of the surviving classes in each column of Table 4 are unique with respect to sex and the autosomal dominant markers. What phenotypic differentiation is possible among the rows is based on the B^S and $y+$ markers on the translocated B^SYy+ chromosome. How these markers are distributed among the rows depends upon which *Y* arm is broken in each translocation, since the *D* elements carry the marker from the unbroken *Y* arm and the *P* elements carry the marker from the broken arm. Because there are only two different *Y* arm markers, there is some among-row ambiguity. Fortunately, however, it is possible to distinguish one from two doses of each marker; B^S on the basis of the smaller eye of B^SB^S than B^S and $y+$ because $y+y+$ produces a more extreme *Hw* effect than $y+$. Some translocations, however, modify these phenotypes in troublesome ways.

With respect to $y+$ and B^S, we recognize nine phenotypes which are listed in the first column of Table 5. The genetic constitutions of flies of each phenotype vary with respect to the translocated complement and thus with respect to autosomal aneuploidy depending on which arm of the *Y* chromosome is broken in both the maternal and the paternal translocation. The relations between *Y*-marker phenotype and genotype for the different *Y*-chromosome-breakpoint combinations are indicated in the table. Thus, in scoring progeny, the 8 × 16 table (Table 4) is condensed into an 8 × 9 table by combining the rows as indicated in Table 5.

TABLE 5

The translocated complements carried by, and the corresponding genotype of, the nine different Y-marker phenotypes resulting from different combinations of maternal and paternal Y-chromosome breakpoints, when the autosomal breakpoints are in the same arm

A zero in the "Translocated Complement" column means that the zygote has no translocated element. A plus sign in the column marked "Genotype" signifies a euploid constitution which may be *Bal/Dom, Bal/T(Y;A), T(Y;A)/Dom,* or *T(Y;A)/T(Y;A).*

	Y-arm breakpoint of maternal (italics) and paternal T(Y;A)							
	LL		*LS*		*SL*		*SS*	
Y-marker phenotype	Translocated complement	Genotype	Translocated complement	Genotype	Translocated complement	Genotype	Translocated complement	Genotype
O	O	+	O	+	O	+	O	+
y+	*D*	*Tpt*	*D*	*Tpt*	D	Tpt	*P*	*Dft*
	D	Tpt	P	Dft	*P*	*Dft*	P	Dft
B^S	*P*	*Dft*	D	Tpt	*D*	*Tpt*	*D*	*Tpt*
	P	Dft	*P*	*Dft*	P	Dft	D	Tpt
y+y+	*D*D	*Tpt*/Tpt	*D*P	Ani	D*P*	Ani	*P*P	*Dft*/Dft
y+B^S	*DP*	+	*D*D	*Tpt*/Tpt	*D*D	*Tpt*/Tpt	*DP*	+
	*D*P	Ani	*DP*	+	*DP*	+	*D*P	Ani
	D*P*	Ani	DP	+	DP	+	D*P*	Ani
	DP	+	*P*P	*Dft*/Dft	*P*P	*Dft*/Dft	DP	+
B^SB^S	*P*P	*Dft*/Dft	D*P*	Ani	*D*P	Ani	*D*D	*Tpt*/Tpt
y+y+B^S	*DD*P	Tpt	*D*DP	*Tpt*	*DD*P	Tpt	*DP*P	Dft
	*D*DP	*Tpt*	*DP*P	Dft	D*P*P	*Dft*	D*P*P	*Dft*
y+B^SB^S	*DP*P	Dft	*DD*P	Tpt	*D*DP	*Tpt*	*DD*P	Tpt
	D*P*P	*Dft*	D*P*P	*Dft*	*DP*P	Dft	*D*DP	*Tpt*
y+y+B^SB^S	*DDPP*	+	*DDPP*	+	*DDPP*	+	*DDPP*	+

As the major thrust of this article is the recovery of interstitial aneuploids, let us now examine how combining rows in Table 4 affects our ability to detect such aneuploids. In the first place it can be seen in Table 5 that when the *Y* chromosomes of the maternal and paternal *T(Y;A)*'s are broken in different arms, the reciprocal interstitially-aneuploid genotypes are unambiguously marked with $y+\ y+$ and B^SB^S, and from Table 4 it is evident that interstitial aneuploids of four different disjunctional origins may be differentiated on the basis of their non-translocated complements. (Because it is possible to determine which interstitial aneuploid carries the deficiency on the basis of the concordance of its sex with that of the parent with the more proximal autosomal breakpoint, the *Y*-marker phenotype of the interstitial aneuploids identifies the *Y* arm involved in each of the component translocations; e.g. if the deficiency is marked by $y+y+$, then the *P* element of the translocation with the more proximal autosomal breakpoint is marked with $y+$ and consequently the *Y* chromosome is broken in the short arm.) The situation is less clear when both maternal and paternal translocations are broken in the same *Y*-chromosome arm. In this case, reciprocal interstitial aneuploids are $y+B^S$ as are sibs carrying either the maternal or the paternal *T(Y;A)* intact and the non-translocated complement does not completely differentiate among these genotypes. For example, the $y+B^S$ classes indicated in Table 5 can be seen to correspond to rows 1, 2, 5, and 6 in Table 4. Combining these rows, the first column shows that $y+\ B^S$ *Bal* males can be any of four different genotypes. Only one class, an interstitial aneuploid, however, is the consequence of frequent disjunctional events, (*ad1*/ad1), and thus,

if $y+$ B^S *Bal* males are a common class, one can be reasonably sure that they are preponderantly the interstitial aneuploid. If, on the other hand, $y+$ B^S *Bal* males are infrequent, they may be either rare survivors of a poorly viable aneuploid genotype or rare segregants with normal viability. Without further crossing it is not possible to distinguish between these alternatives unless the $y+$ B^S *Bal* males have an aneuploid phenotype such as Minute. Thus, it is difficult to determine whether some interstitial deficiencies survived. Identical arguments apply to identification of $y+$ B^S *Dom* females, $y+$ B^S *Dom* males, and $y+$ B^S *Bal* females in crosses in which the two translocations are broken in the same *Y* arm.

Tables 6 and 7 are the counterparts of Tables 4 and 5, but where the autosomal breakpoints of the translocations are on opposite sides of the centromere. Since in these crosses such breakpoints are also in the vicinity of the centromeres, none of the terminal aneuploids survive, considerably shortening Table 6 compared with Table 4. It will be noticed, comparing Table 4 and Table 6, that although the origin of euploid genotypes is identical when autosomal breakpoints are in the same or opposite arms, the interstitial aneuploids are of different origin and of different constitution with respect to the translocated complement. When the breakpoints are in different autosomal arms, the interstital aneuploids are uniquely marked when both translocations are broken in the same *Y*-chromosome arm unlike the situation in Tables 4 and 5. Thus, interstitial aneuploids may be unambiguously recognized on the basis of *Y*-chromosome markers when the arms broken in two *T(Y;A)*'s are the same for the *Y* but not the autosome or the same for the autosome but not the *Y* chromosome. When these conditions are satisfied, information about the nontranslocated complement is dispensible and interstitial aneuploids can be recognized in crosses between stock males and females where both sexes carry *Bal*.

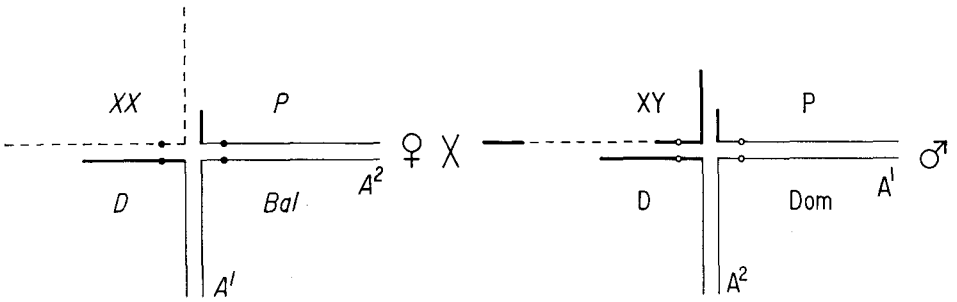
In practice, translocations whose breakpoints are in the vicinity of the centromere present a problem because it is not possible to determine cytologically the positions of the autosomal breakpoints either with respect to the centromere or with respect to each other. However, by examining the progenies of crosses of these translocations with those having the proximalmost euchromatic breakpoints and assuming that adjacent II, but not adjacent I, disjunction is extremely rare in males but not in females—an assumption that appears to be born out for the vast majority of *T(Y;2)*'s with heterochromatic second-chromosome breakpoints (GETHMANN, unpublished)—it has been possible to order heterochromatic autosomal breakpoints on chromosome 2 with respect to the centromere. This permits the construction of aneuploids for the centromere region.

A second exceptional case presents itself if a translocation is broken in the *Y* chromosome distal to one of the *Y*-chromosome markers rather than between them. In this case, instead of *D* and *P* each having one of the *Y*-chromosome markers, *D* is marked with $y+$ and B^S while *P* is unmarked. Complete description of the results with such translocations would require the expansion of Tables 5 and 7 from four to nine columns.

Ideally we planned to choose translocations with autosomal breakpoints at intervals of three lettered subdivisions on the salivary gland chromosome in order to divide each autosome into 80 approximately equal segments. We then planned to cross all adjacent pairs of translocations in order to generate an uninterrupted series of adjacent non-overlapping interstitial aneuploids, each one-eightieth of a chromosome in length. We also planned to cross all adjacent-but-one pairs of translocations to produce a series of combinations of adjacent pairs of interstitial deficiencies one-fortieth of a chromosome in length and overlapping in the manner, a + b, b + c, c + d, etc. This ideal could not be entirely realized owing to our failure to completely saturate the autosomal complement with *T(Y;A)* breakpoints. However, we adhered to these criteria insofar as possible in our crossing design. Fifteen pair matings of each cross were made in vials and the progenies of each vial scored separately. All crosses were made reciprocally. This procedure provides insurance against the possible failure to recover a deficiency, not because the deficiency is inviable *per se*, but owing to hemizygosity for a recessive lethal in the chromosome in combination with which the deficiency is recovered; this ambiguity is avoided because deficiencies are recovered over *Bal* and *Dom* in reciprocal crosses. In fact, our observations confirmed the known locations of *al*, *Sco*, *sp*, *Sb*, *Ubx*, and *e*, as well as providing a cytological location for *Cy* (between 22D and 23C) and *cn* (between 43A and 43F). Moreover, we identified the

TABLE 6

The disjunctional origin[1] *of the euploid and possibly-surviving aneuploid zygotes from the following cross*[2] *where the maternal and paternal translocations are broken in different autosomal arms, designed* A^1 *and* A^2



Zygotic genotypes are partitioned into translocated and non-translocated complements and the disjunctional origin of all potentially viable combinations indicated.

		Zygotic genotype partitioned with respect to							
		Non-translocated complement							
Aneuploid types	Translocated complement	XY *Bal*	XY Dom	XY *Bal*/Dom	XY	*XX Bal*	*XX* Dom	*XX Bal*/Dom	*XX*
+	*DP*	*1:3*/1:3	*alt*/alt			*4:0*/4:0	*3:1*/3:1		
	DP	*3:1*/3:1	*4:0*/4:0			*alt*/alt	*1:3*/1:3		
	DDPP				*alt*/3:1				*3:1*/alt
	O			*3:1*/alt				*alt*/3:1	
Dpi	*PP*	*ad2*/ad1	*3:1*/1:3			*1:3*/3:1	*ad1*/ad2		
Dfi	*DD*	*ad1*/ad2	*1:3*/3:1			*3:1*/1:3	*ad2*/ad1		

[1] Disjunctional notation is maternal type of disjunction followed, after a slash bar, by the paternal type of disjunction.

[2] Dashed line = *X* chromosome; heavy line = *Y* chromosome; fine line = autosome. See text for explanation of symbol conventions and detailed discussion of table.

TABLE 7

The translocated complement carried by, and the corresponding genotype of, the nine different Y-marker phenotypes resulting from different combinations of maternal and paternal Y-chromosome breakpoints, when the autosomal breakpoints are in different arms

	Y-arm breakpoint of maternal (italics) and paternal T(Y;A)							
	LL		*LS*		*SL*		*SS*	
Y-marker phenotype	Translocated complement	Genotype	Translocated complement	Genotype	Translocated complement	Genotype	Translocated complement	Genotype
O	O	+	O	+	O	+	O	+
y^+y^+	*DD*	Dfi					*PP*	Tpi
			DD	Dfi	*DD*	Dfi		
y^+B^S	*DP*	+	*DP*	+	*DP*	+	*DP*	+
	DP	+	DP	+	DP	+	DP	+
			PP	Tpi	*PP*	Tpi		
B^SB^S	*PP*	Tpi					*DD*	Dfi
$y^+y^+B^SB^S$	*DDPP*	+	*DDPP*	+	*DDPP*	+	*DDPP*	+

positions of two recessive lethals on the *Sb* chromosome (73D–74A and 92D–94A). Both *Sb* and *TM6* carry recessive lethals in region 88C–89C; consequently it was necessary to replace the *Sb*-bearing chromosome with a homozygous-viable third chromosome marked with *Ki* in order to show that heterozygotes for that deficiency are not recovered.

As a control, for every translocation utilized in the crossing design, males and females carrying the same translocation were also crossed in 15 pairs. This cross, of course, produces no interstitial aneuploids. In general, then, each translocation was crossed to two adjacent translocations to its left, to itself, and to two adjacent translocations to its right.

One final complication that should be mentioned arises from the inadvertant use of translocations that have, in addition to the euchromatic break scored cytologically, an undetected heterochromatic break in the autosome, and in which the effective translocation breakpoint is in the proximal heterochromatin and not in the euchromatin as supposed. The consequence of this is that instead of generating duplications and deficiencies for small regions on either side of the supposed euchromatic breakpoint as intended, the interstitial aneuploids are deficient or duplicated for a segment extending from the euchromatic region in question to the centromere. This often results in lethality which could be erroneously interpreted as inviability of small duplications or deficiencies. We have eliminated from the results all crosses involving such translocations.

For most uses, duplicated and deficient chromosomes would be easier to handle were they simple rather than translocated chromosomes. Irradiation of oocytes of *T(X;A)/Bal* interstitially aneuploid females should occasionally result in a translocation between the proximal *Y* heterochromatin of the *D* element and the distal *Y* heterochromatin of the *P* element. Such a translocation would produce an autosome with a small heterochromatic segment at the point where *D* and *P* are rejoined (i.e. at the point of deficiency or between tandemly-duplicated segments) and a doubly marked *Y*-chromosome derivative. These two elements will then segregate from one another at the ensuing first meiotic division and the desired derivative may be recognized as an offspring carrying neither *Y*-arm marker nor the Balancer chromosome. Putative cases of spontaneously occurring reattachments were recorded among the progenies of the aneuploid-generating crosses, but none were confirmed.

RESULTS

Over 140,000 progeny of 6,885 pair matings from crosses of 555 combinations of *Y*-autosome translocations of the type diagrammed in Figure 1 were scored in this experiment. The results with respect to hyperploidy can be dealt with directly. All segmental hyperploids survived with the exception of the region from 83D to 83E, which was triplo-lethal. Since triplo-4 individuals regularly survive and triplo-*X* metafemales sometimes survive, it follows that 83D–E contains the only triplo-lethal locus in the Drosophila genome. (It should perhaps be noted that triploid Drosophila are viable.) Furthermore, there are but two other hyperploid-sensitive loci, both of which are on the *X* chromosome: band 3C7 which produces the Confluens phenotype when present in two doses in males or three doses in females and region 17A–C which produces a Beadex phenotype when present in two doses in males and four doses in females but not when present in three doses in females.

Extensive hyperploidy, on the other hand, is lethal while intermediate levels cause reduced survival, small size and a variety of morphological anomalies such as rough eyes, abnormal wings and bristle patterns, and a misshapen abdomen. These morphological effects do not seem to be characteristic of any particular region of the chromosome, but are more appropriately thought of as a hyperploid syndrome. One measure of the reduction in viability as a function of the extent

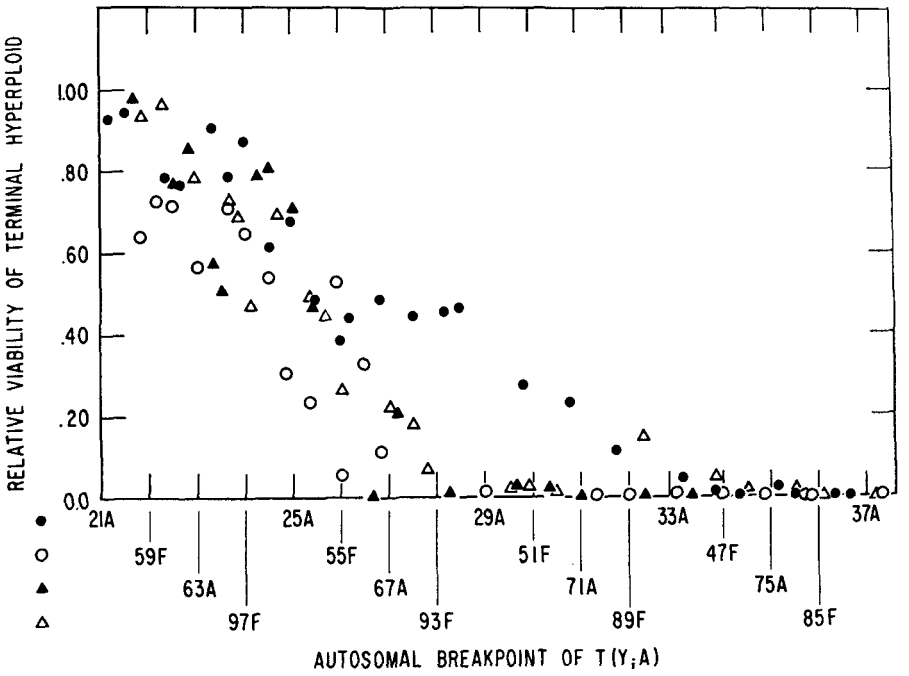


FIGURE 4.—Survival of terminal hyperploids for the four major autosomal arms as a function of the length of the trisomic region (in numbered salivary subdivisions). Circles represent second-chromosome and triangles third-chromosome arms; solid symbols represent left and open symbols right arms.

of the hyperploid region is illustrated in Figure 4. There we plot the survival of individuals hyperploid for $A^{D}Y^{P}$ (i.e. terminal trisomics) for each translocation used in the aneuploid-generating crosses against the autosomal breakpoint of the translocation. Hyperploid survival is taken as the recovery of hyperploids from adjacent I disjunction in one parent and alternate disjunction in the other from all crosses involving the translocation in question, relative to euploids of the same sex as the hyperploids in the same crosses. It can be seen that the viability of terminal hyperploids declines regularly with the amount of triplicated material, becoming generally lethal when more than one-half an autosomal arm is present in three doses. However, we have observed sporadic survivors for two-thirds and more of an autosomal arm. The apparent resistance of *2L* to terminal hyperploidy when compared with other arms disappears when viability is plotted against the number of bands in the trisomic region rather than salivary subdivision; viability reaches zero when 500 bands are included in the terminally hyperploid region irrespective of arm. Results very similar to these have been reported for terminal *X*-chromosome hyperploidy in Drosophila females by PATTERSON, STONE and BEDICHEK (1935).

The results with respect to segmental monosomy are shown in Figure 5. We consider a region adequately tested when (a) the translocation breakpoints defining the region are four or fewer lettered subdivisions apart (there are six lettered subdivisions per numbered division and twenty numbered divisions per

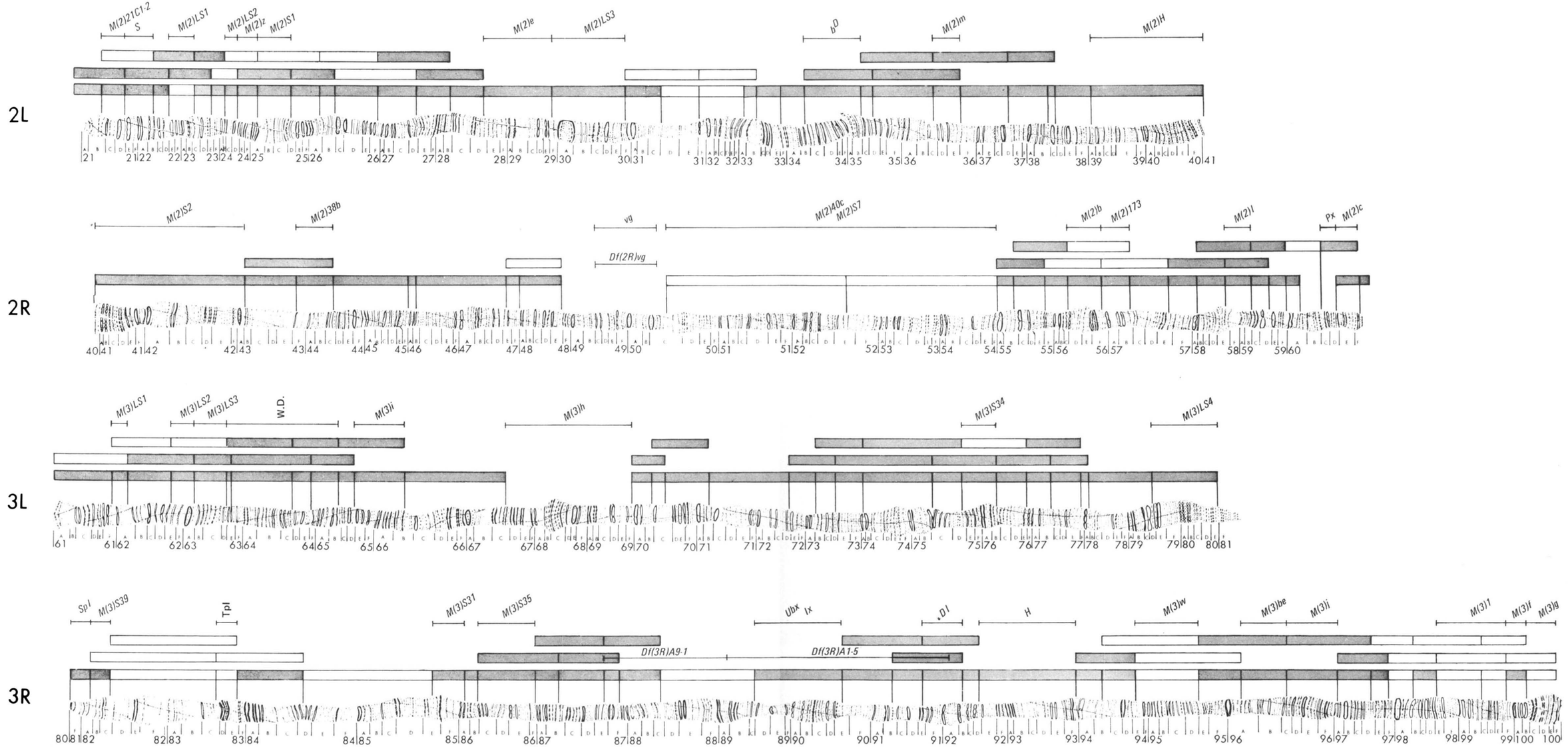


FIGURE 5.—The results of the constructions of interstitial deficiencies. Shaded segments of the horizontal bars above each autosomal arm indicate regions for which heterozygous deficiencies survived; open segments, regions for which they failed to survive. The vertical lines defining the limits of the segments indicate the positions of the autosomal breakpoints of the *Y*-autosome translocations used in the synthesis of the deficiencies. Horizontal lines define three previously reported deficiencies that survive in heterozygotes and which cover regions not adequately tested in the present experiments. The positions of the haplo-sensitive loci, as defined by the present experiments, are indicated in the top line above each arm. (The chromosomes are taken from drawings by R. C. KING 1970, after BRIDGES 1935).

chromosome arm), and (b) the progeny of a reasonable number of pair matings have been examined, or (c) when it survived in the monosomic condition. By these criteria we have adequately tested 85% (400 of the 480 lettered subdivisions) of chromosomes *2* and *3*. In the 14% of the autosomal genome that we were unable to properly examine, PATTERSON, BROWN and STONE (1940) report two adjacent deficiencies covering 6 of the remaining lettered subdivisions [*Df(3R)A9–1* and *Df(3R)A1–5*], and several vestigial deficiencies taken together cover another seven divisions; thus, approximately 86% of the autosomal complement has been examined. Of this 86%, only 19 lettered subdivisions, or 4.6%, failed to produce viable hypoploids. Thus, the most striking inference from the data is that there are very few loci in the entire Drosophila genome that must be present in two doses for survival of the organism.

Ninety-five regions of four or fewer lettered subdivisions were tested. Eight of these regions (31D-E; 60A-C; 67C-D; 83D-E; 97F-98B; 98F-99C; 99D-F; 100C-F) did not survive as monosomics and therefore might contain haplo-lethal loci. The 95 tested regions comprise 242 subdivisions, or approximately 40% of the haploid complement. If this sample can be taken as representative, then there might be as many as 20 (8÷0.4) haplo-lethal loci in the entire Drosophila genome. The *X* chromosome has not been systematically examined; although it might be special because of the property of dosage compensation, our general impression is that with respect to aneuploidy it behaves very much like the autosomes.

In fact, there are most probably fewer than this number because certainly some, and conceivably most, of the haplo-lethal regions pictured in Figure 5 are extreme Minutes that reduce viability such that the hypoploids did not appear in our sample. The evidence for this is that in our initial experiments there were two additional haplo-lethal regions—one at 56C-56F and one at 29F-30F. The first of these was resolved as being *M(2)b* as the result of an intensive examination of regions 55 to 57 using twenty translocation combinations (Figure 6). In that experiment, several thousand progeny were examined, among which the *M(2)b*-bearing hypoploid appeared only twice. The second region was shown to carry a new very extreme Minute, *M(2)LS3*, when the deficiency-generating crosses were made in large numbers in another connection.

In summary, then, there are fewer than twenty, probably one (83D-E), and conceivably no other, haplo-lethal loci in the Drosophila genome.

The most striking class of haplo-abnormal loci is composed of the Minutes—wild-type loci that when hemizygous produce a small bristle phenotype, and also result in a decreased developmental rate, variable (generally quite low) viability, and often other morphological abnormalities. The Minutes on chromosomes *2* and *3* are localized in Figure 5. Our procedure for these assignments was as follows. First, Minutes with known cytological locations that were confirmed by our results or are in a region that we found to be haplo-lethal or that we could not adequately test are entered in Figure 5 with limits set by our breakpoints (even when the location is known more precisely). Second, Minutes with known genetic, but not cytological, positions were assigned to the most probable regions in which we had found Minutes (or were haplo-lethal or that we were unable to

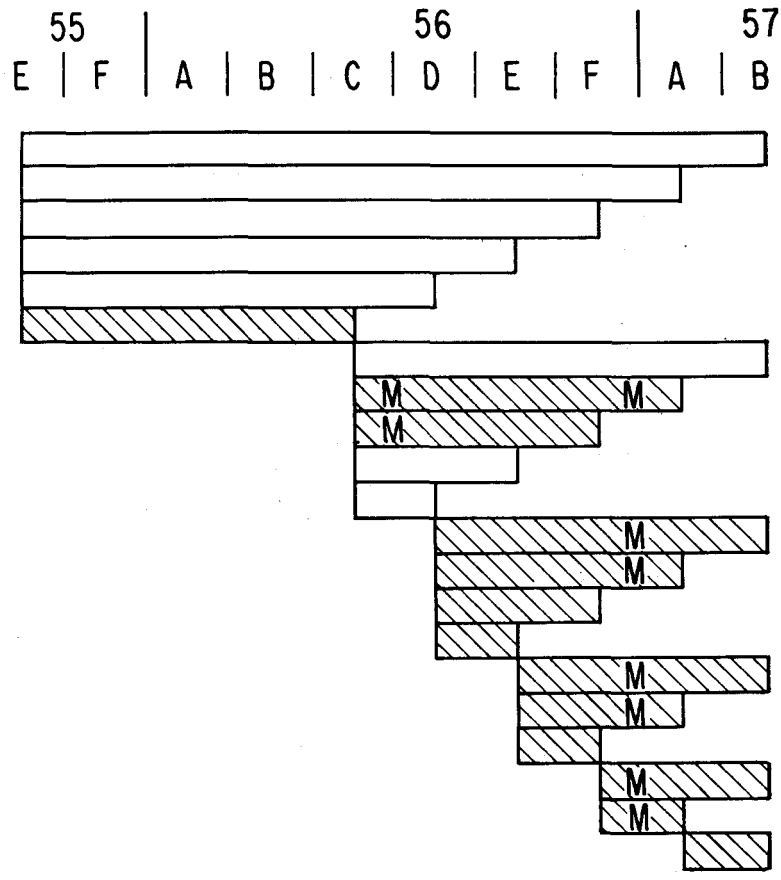


FIGURE 6.—A detailed examination of region 55E to 57B. The survival (shaded bars) or lack of survival (open bars) for 21 synthesized deficiencies is shown. *M* indicates the positions of the two Minutes in the region, *M(2)b* (to the left) and *M(2)173* (to the right).

test). This procedure worked well in the sense that there were no M^+ regions by our test that should have contained a known *M*; also, the procedure accounted for all the genetically and cytologically located Minutes and assigned a function to two of the five apparent haplo-lethals. The remaining Minutes that we found are designated *LS* followed by a number. *M(3)LS4*, in the centromere region of chromosome *3*, was inferred from the recovery of but a single *M* individual. It is included in the figure because it likely corresponds to the *M* recovered by BALDWIN and SUZUKI (1971) in this same region.

There are, therefore, at least 41 Minutes in the Drosophila genome—seven on the *X* chromosome, 17 on chromosome *2*, 16 on chromosome *3* and one on chromosome *4*. The actual number of Minutes must be greater than this for three reasons: (1) Some regions that result in a Minute phenotype will contain more than one Minute locus. (2) One or more of the three regions that we scored as haplo-lethal, and to which we were unable to assign a Minute locus, may actu-

ally contain an extreme Minute, as discussed above. (3) There is one recorded case of a synthetic Minute; that is, two elements, *M(2)d* and *M(3)d*, both of which must be present to produce the phenotype. While the first two sources of error most probably result in a rather small underestimate of the actual number of Minute loci, it is impossible to assess the possible number of synthetic Minutes. Synthetic Minutes are, therefore, not pictured in Figure 5, nor are they considered further in this report.

Finally, the haplo-abnormal loci are indicated on Figure 5. On chromosome *2* there are four, *S*, *b*, *vg*, and *Px*, all of which were previously known to be dosage-sensitive loci. The effect of a heterozygous deficiency for *b* was described as $Df(2L)b^{D}$ and of *vg* as $Df(2R)vg^{B}$, $Df(2R)vg^{C}$, $Df(2R)vg^{D}$, $Df(2R)vg^{I}$, and $Df(2R)vg^{S}$. Of the four haplo-abnormals, only *vg* was in a region that we were unable to test adequately. There are six haplo-abnormal loci on chromosome *3*. Three of these, *Ubx*, *Ix*, and *Dl*, were previously described. *Ix*: Intersex is a new symbol for the intersexual phenotype previously described for both *Df(3R)89 E-F*/+ males and females. *H*, although a known dominant mutant, is shown here for the first time to be a haplo-abnormal phenotype. "W.D." refers to a region that appears to be involved in wing development. Heterozygotes for different deficiencies in this region exhibit various combinations of wing abnormalities; however, there are no mutants affecting wing morphology located in the corresponding region of the genetic map. *Spl*: Splayed, refers to the phenotype associated with heterozygosity for a deficiency for 81F-82A; *Spl* flies haxe extended legs which they seem to bend with difficulty and which sometimes have accumulations of dried black material at the joints. This haplo-abnormal phenotype is previously undescribed. The dominant mutation, Lyra, is associated with a deficiency for bands 70A3, 4, and 5. Although these bands are at a boundary between a tested and an untested region according to our cytological determination, they fall within a surviving deficiency whose phenotype was normal (both with respect to wing shape and bristle size). This, taken together with the observation that *Ly* interacts lethally with $M(3)w^{ssJ}$ which itself does not produce a *Ly* phenotype in combination with +, leads us to conclude that *Ly* is not a hypoploid phenotype.

In summary, then, the *X* chromosome contains no triplo-lethals, few or no haplo-lethals, at least seven Minutes, one hyperploid sensitive (*Bx*), and one locus (band 3C7) that is both triplo-abnormal (*Co*) and haplo-abnormal (*N*); chromosome *2* contains no triplo-lethals, two apparent haplo-lethals (31D-E and 60A-C), at least 17 Minutes, and at least four other haplo-abnormals; chromosome *3* contains one triplo-lethal that is also haplo-lethal (83D-E), three other apparent haplo-lethals (67C-D, 97F-98B and 98F-99F), at least 16 Minutes, and at least six other haplo-abnormals; and chromosome *4* contains no triplo-lethals, one Minute, no haplo-lethals, and no other haplo-abnormals.

DISCUSSION

The results of the crosses reported in this article clearly demonstrate the potential utility of a large array of *Y*-autosome translocations for producing seg-

mentally monosomic or trisomic individuals. The viability in heterozygous condition of the majority of deficiencies (and all save one of the duplications) for approximately one-eightieth of an autosome and of something over half the deficiencies (and all but one of the duplications) for one-fortieth of an autosome means that the dosage of virtually all autosomal genes is amenable to genetic manipulation.

Although most small deficiencies survive in heterozygotes, many of these genotypes are sterile in one or both sexes. This sterility is probably of dual origin, at least in males. In addition to the effects of autosomal aneuploidy, *Y*-chromosome hyperploidy must be considered a possible source of male sterility because the synthetic translocations are at the same time aneuploid for *Y*-chromosome material and carried in combination with a $Y^{S}X{\cdot}Y^{L}$ chromosome. In some cases amelioration of the male sterility should be achieved by replacement of $Y^{S}X{\cdot}Y^{L}$ by a normal *X*. The sterility of flies carrying duplicated and deficient translocations prevents their being carried in balanced stocks; consequently, segmental aneuploids in many cases must be created anew for each use to which they are to be put. Also, the translocated nature of the duplicated and deficient chromosomes makes them cumbersome to use in many crossing schemes. In principle this shortcoming can be overcome by reattaching the autosomal portions of the translocation in the manner suggested at the end of the section entitled "SYNTHESIS OF SEGMENTAL ANEUPLOIDS." Besides circumventing the problem posed by the translocated nature of the duplications and deficiencies, this procedure has the added virtue of eliminating some of the *Y*-chromosome hyperploidy that characterizes the aneuploid complements.

The primary point of theoretical interest to emerge from these studies is that the deleterious effects of aneuploidy are, in the main, caused by the additive effects of genes that slightly reduce viability and not by the individual effects of a few aneuploid-lethal genes among a large array of dosage insensitive loci. This conclusion is most clearly demonstrated in Figure 4 for the case of hyperploidy; gradually increasing amounts of terminal trisomy cause a gradual reduction in viability with no evidence of sudden decreases in recovery as might be expected if there were extremely dosage-sensitive loci. The same point can be made for the case of hypoploidy. There are numerous deficiencies of approximately one-fortieth of a chromosome in length that are lethal when heterozygous, but that have been subdivided into two smaller deficiencies (approximately one-eightieth of a chromosome long), both of which are heterozygous viable. Thus, the inviability of the larger deficiency cannot imply the existence of a haplo-lethal locus. In general it seems that the vast majority of deficiencies one-eightieth of an autosome long, and something over half those two-eightieths long, survive in the heterozygous state. The largest recorded deficiency (*Df(2L)H*) that survives as a heterozygote is six-eightieths long.

Finally, a dominant mutation can result from either a change in the dosage of a gene or a change that results in uncontrolled gene function or the production of an abnormal gene product. These two classes of dominants can be distinguished operationally as follows: dosage dominants (1) may be synthesized from aber-

rations that themselves have a normal phenotype, (2) are easily X-ray inducible but not usually X-ray revertable, and (3) are generally recessive in one dose in triploids. Dominants that result in uncontrolled or abnormal gene function, on the other hand, (1) cannot be synthesized from phenotypically normal components, (2) are not readily X-ray induced but are easily X-ray reverted (LIFSCHYTZ and FALK 1969), and (3) are generally dominant in one dose in triploids. Our data imply that very few of the loci of *Drosophila melanogaster* can produce dominant phenotypes owing to changes in dosage.

We dedicate this work to the memory of Jack Schultz whose pioneering research on the structure of the Drosophila genome forms so much of the background for these experiments. We fondly remember his excitement and enthusiasm at seeing these results that confirm and extend the foundation that he was so instrumental in building.

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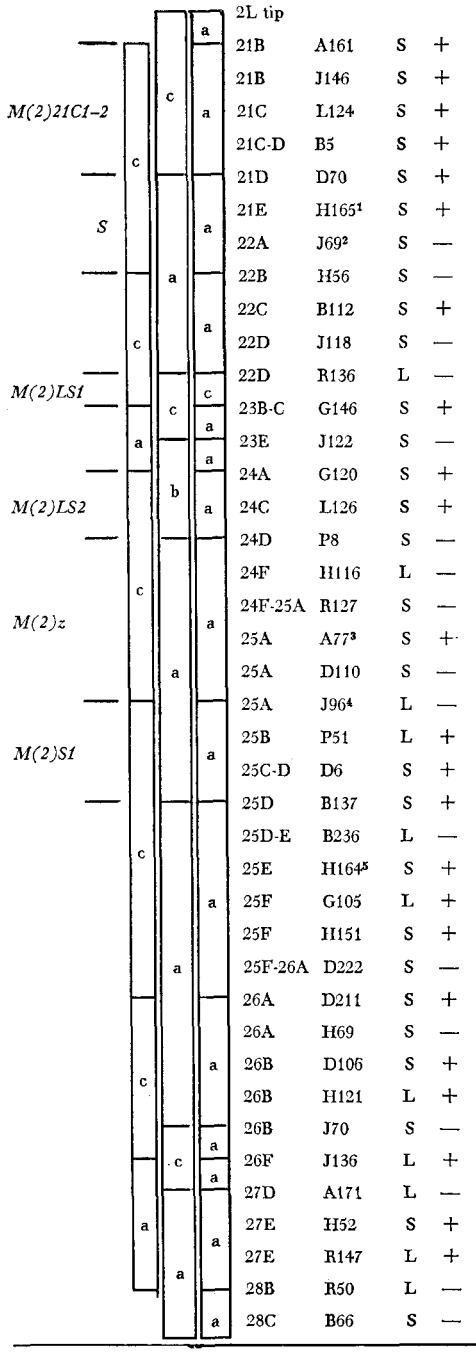
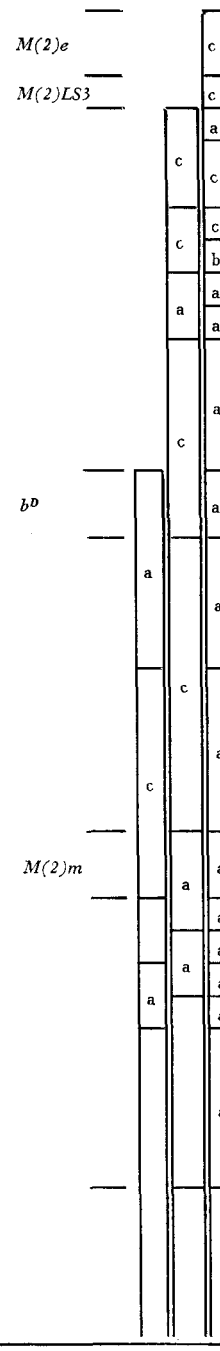
APPENDIX TABLES 1 AND 2

The breakpoints of reciprocal *T(Y;A)*'s that may be used in the construction of interstitial duplications and deficiencies. The three vertical bars correspond to the horizontal bars in text Figure 5 and indicate which combinations of translocations have been utilized in the production of interstitial aneuploids. The letters in the bar segments indicate the survival of segmental monosomics as a proportion of the surviving trisomics for the same chromosome segment; the two segmental aneuploids are produced as reciprocal products of the same types of segregations. A proportion of 1.0–0.1 is designated by the letter a, 0.1–0.01 by b, and < 0.01 by c. The *T(Y;A)* divides the autosome into two segments, one extending from region 21 for chromosome *2* or 61 for chromosome *3* to the point designated in column 6 and the other extending from this point through region 60 for chromosome *2* or 100 for chromosome *3*. Either of these segments may be further rearranged; such further rearrangements are specified in the footnotes. The rightmost column indicates whether the fertility factors on the translocated *Y* chromosome function normally. The data in these tables are slightly more up to date than those in text Figure 5.

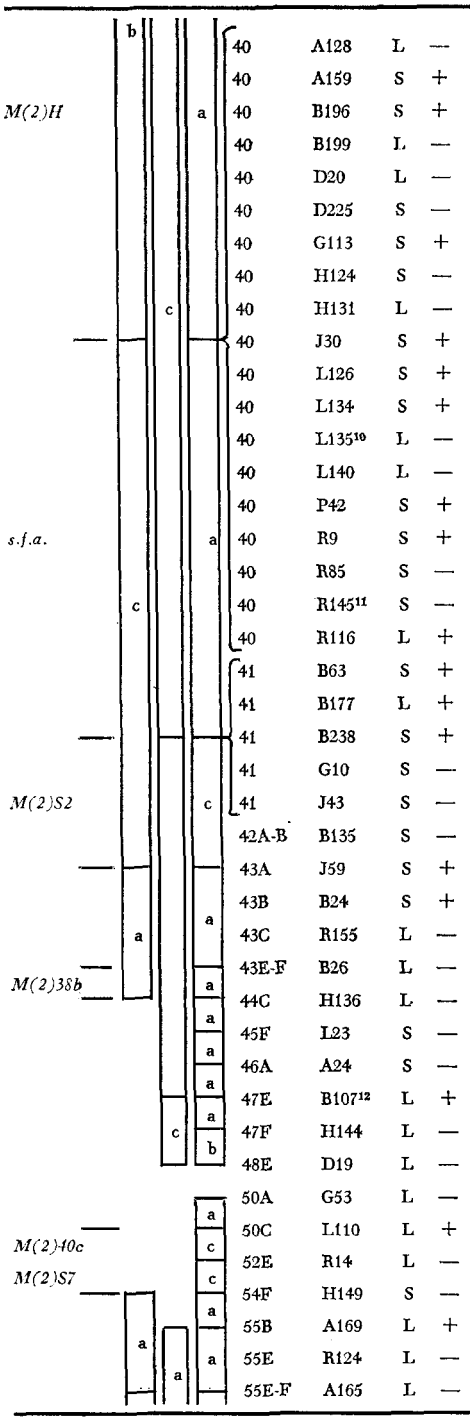
TABLE 1



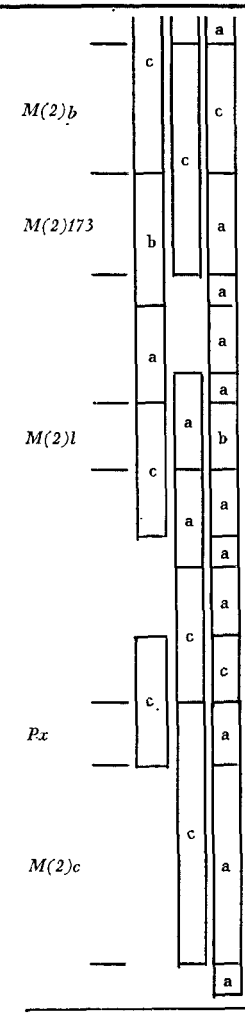


Haploid phenotype	Results	Autosomal breakpoint	Translocation designation	Y breakpoint	X/T (Y;A) ♂fertility
		2L tip			
		21B	A161	S	+
		21B	J146	S	+
M(2)21C1–2		21C	L124	S	+
		21C-D	B5	S	+
		21D	D70	S	+
		21E	H165[1]	S	+
S		22A	J69[2]	S	—
		22B	H56	S	—
		22C	B112	S	+
		22D	J118	S	—
		22D	R136	L	—
M(2)LS1		23B-C	G146	S	+
		23E	J122	S	—
		24A	G120	S	+
M(2)LS2		24C	L126	S	+
		24D	P8	S	—
		24F	H116	L	—
M(2)z		24F-25A	R127	S	—
		25A	A77[3]	S	+
		25A	D110	S	—
		25A	J96[4]	L	—
M(2)S1		25B	P51	L	+
		25C-D	D6	S	+
		25D	B137	S	+
		25D-E	B236	L	—
		25E	H164[5]	S	+
		25F	G105	L	+
		25F	H151	S	+
		25F-26A	D222	S	—
		26A	D211	S	+
		26A	H69	S	—
		26B	D106	S	+
		26B	H121	L	+
		26B	J70	S	—
		26F	J136	L	+
		27D	A171	L	—
		27E	H52	S	+
		27E	R147	L	+
		28B	R50	L	—
		28C	B66	S	—
		28D	A111	L	—
M(2)e		28D	B104	L	+
		29F	A145	S	+
M(2)LS3		30F	L52	L	+
		31C-D	G20[6]	S	+
		31D-E	J166	S	+
		31E-F	A162	S	—
		32F-33A	R158	S	+
		33B	B92	L	—
		33E-34A	J160	S	+
		33F	D212	L	—
		33F-34A	*B212		
		34A	*A97		—
		34A	B224	S	+
		34B	G74	L	—
b^D		35A-B	H161[7]	S	
		35B	A80	S	—
		35B-C	R15	S	+
		35C-D	J165	S	—
		35D	D217		—
		35D	P58	S	+
		36A-B	B210	S	+
		36A-B	B214	S	+
		36B	A62	L	+
		36C	B4	S	—
		36C	B242	L	—
M(2)m		36D-E	*A139		—
		36E-F	D219[8]	L	—
		37D	H174	S	—
		38B	P57	L	—
		38C	B110	L	—
		39C	L138	S	+
		40	A87	L	+
		40	A107	L	+
		40	B190	S	+
		40	B209	L	—
		40	B251	S	—
		40	H54	L	—
		40	H118	L	+
		40	L67[9]	S	—
		40	R146	L	+

TABLE 1—Continued



40	A128	L	—	
40	A159	S	+	
40	B196	S	+	
40	B199	L	—	
40	D20	L	—	
40	D225	S	—	
40	G113	S	+	
40	H124	S	—	
40	H131	L	—	
40	J30	S	+	
40	L126	S	+	
40	L134	S	+	
40	L135[10]	L	—	
40	L140	L	—	
40	P42	S	+	
40	R9	S	+	
40	R85	S	—	
40	R145[11]	S	—	
40	R116	L	+	
41	B63	S	+	
41	B177	L	+	
41	B238	S	+	
41	G10	S	—	
41	J43	S	—	
42A-B	B135	S	—	
43A	J59	S	+	
43B	B24	S	+	
43C	R155	L	—	
43E-F	B26	L	—	
44C	H136	L	—	
45F	L23	S	—	
46A	A24	S	—	
47E	B107[12]	L	+	
47F	H144	L	—	
48E	D19	L	—	
50A	G53	L	—	
50C	L110	L	+	
52E	R14	L	—	
54F	H149	S	—	
55B	A169	L	+	
55E	R124	L	—	
55E-F	A165	L	—	



56C	B184	S	—	
56D	G100	S	+	
56E	L139	S	+	
56E-F	L62	S	—	
56F	L141	S	+	
56F	*S14	L		
57A	R93	S	—	
57B	L107	L	—	
57F	A120	S	—	
57F	J163	S	—	
58A	R104	S	+	
58D	H158	S	+	
59A-B	B202	S	+	
59B	P59	S	+	
59D	*A96[13]	L	+	
59D	J131	L	+	
59F	H143	L	+	
60?	*J157	L	+	
60A	L137[14]	S	+	
60B	*B106	S	—	
60B-C	A160	S	+	
60C	L11	S	+	
60D	H137	S		
60E-F	*R103	S	+	
60F	A146	S	+	
60F	B80	S	+	
60F	B185[15]	S	—	
60F	B228	S	+	
60F	R123	L	—	
2R tip				

* Lost

1. *In(2L)21E;28D*
2. *In(2L)*
3. *In(2L)23F;25A*
4. *T(Y;2;4)*

Y^{L}|25A—60; $Y^{S}.Y^{L}$|102; 21—25A|102—101

5. *In(2)40–41;44F*
6. *T(Y;2;3)*

$Y^{L}.Y^{S}$|81F—82F|30D—21; Y^{S}|30D—60

7. *T(Y;2;4)*

$Y^{L}.Y^{S}$|102; Y^{S}|35B—60; 21—35A|101

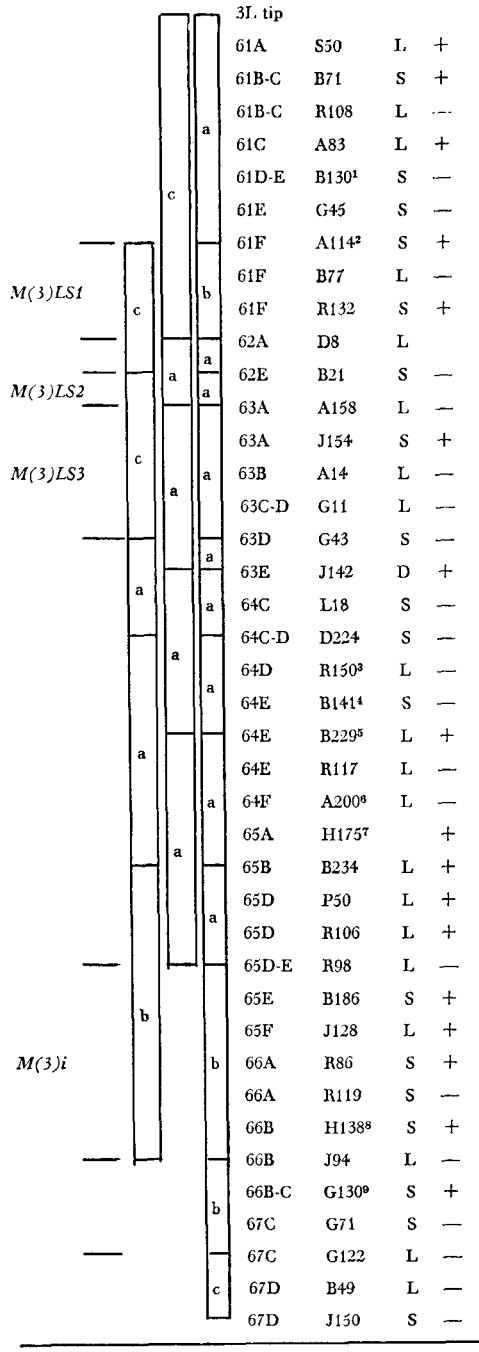
8. *In(2L)35F–36A;36E–37A*
9. *In(2L)25E;40*
10. *In(2L)31F;40*
11. *In(2L)23F;40*
12. *In(2LR)29C;47E*
13. *T(Y;2;4)*

Y^{L}|101; $Y^{S}.Y^{L}$|59D—60; 21—59D|102

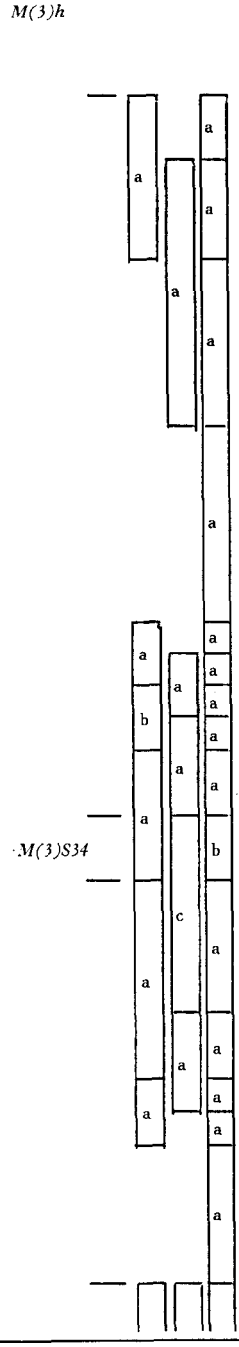
14. *In(2R)43E;60A*
15. *In(2LR)34A;51A;53C-D*

$Y^{L}.Y^{S}$|60F; Y^{S}|60F—53D|51A—53C|34A—51A|34A—21

TABLE 2

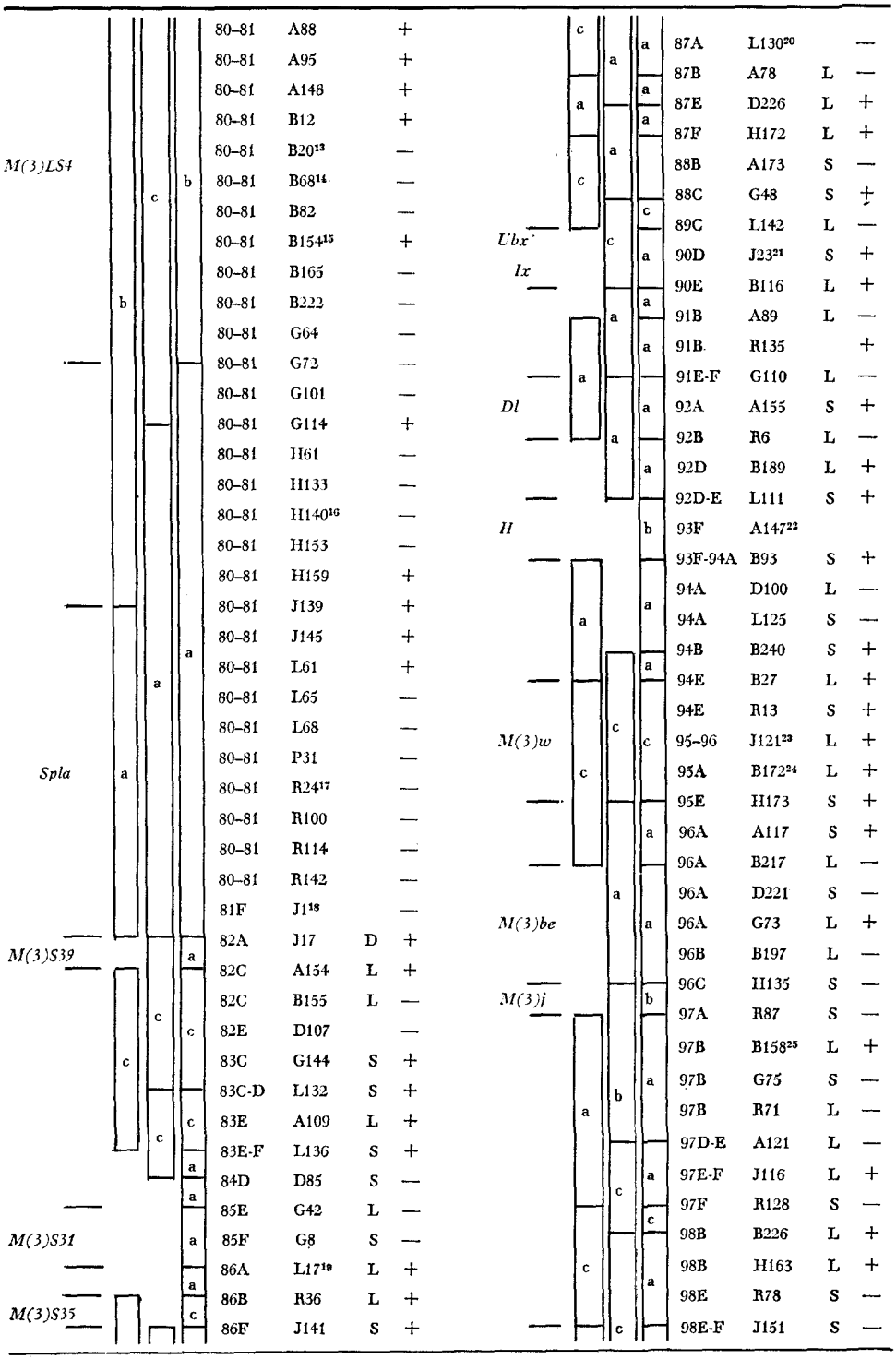


Autosomal breakpoint	Translocation designation	Y breakpoint	X/T (Y;A) ♂ fertility
3L tip			
61A	S50	L	+
61B-C	B71	S	+
61B-C	R108	L	—
61C	A83	L	+
61D-E	B130[1]	S	—
61E	G45	S	—
61F	A114[2]	S	+
61F	B77	L	—
61F	R132	S	+
62A	D8	L	
62E	B21	S	—
63A	A158	L	—
63A	J154	S	+
63B	A14	L	—
63C-D	G11	L	—
63D	G43	S	—
63E	J142	D	+
64C	L18	S	—
64C-D	D224	S	—
64D	R150[3]	L	—
64E	B141[4]	S	—
64E	B229[5]	L	+
64E	R117	L	—
64F	A200[6]	L	—
65A	H175[7]		+
65B	B234	L	+
65D	P50	L	+
65D	R106	L	+
65D-E	R98	L	—
65E	B186	S	+
65F	J128	L	+
66A	R86	S	+
66A	R119	S	—
66B	H138[8]	S	+
66B	J94	L	—
66B-C	G130[9]	S	+
67C	G71	S	—
67C	G122	L	—
67D	B49	L	—
67D	J150	S	—



Autosomal breakpoint	Translocation designation	Y breakpoint	X/T (Y;A) ♂ fertility
67D-E	*L60	S	—
68B	A13	L	+
69F	R7[10]	L	+
69F	R122	S	+
70A-C	A31	S	—
70B-C	A23		—
70B-C	R83	L	+
70C	H156	S	+
70C	J132	L	+
70C-D	R91	L	+
70D	B170		+
70D-E	A150	S	+
71B	A60	S	+
71B-C	J112	S	+
71B-C	L113	S	+
71F	B99	L	—
71F	D210		—
72A-B	B223	L	+
72D	B207	S	+
73A-B	B96	L	+
73D	B225	L	+
74A	D228	L	+
75C	G7	L	—
75C	J100		—
75D	L131	S	+
76A	B132	S	—
76B	L14	L	—
76C	A176	L	—
76C-D	R92	S	+
76D	B115	L	+
76E	A112	L	+
76F-77A	J147	S	+
77B	H147	L	—
77E-F	B108	S	+
78A	R153	L	—
78C	J95	S	—
78C-D	J44		—
78F-79A	R59		—
79D	J162[11]	S	+
80–81	A63[12]	L	—

TABLE 2—Continued



80–81	A88		+	87A	L130[20]		—
80–81	A95		+	87B	A78	L	—
80–81	A148		+	87E	D226	L	+
80–81	B12		+	87F	H172	L	+
80–81	B20[13]		—	88B	A173	S	—
80–81	B68[14]		—	88C	G48	S	±
80–81	B82		—	89C	L142	L	—
80–81	B154[15]		+	90D	J23[21]	S	+
80–81	B165		—	90E	B116	L	+
80–81	B222		—	91B	A89	L	—
80–81	G64		—	91B	R135		+
80–81	G72		—	91E-F	G110	L	—
80–81	G101		—	92A	A155	S	+
80–81	G114		+	92B	R6	L	—
80–81	H61		—	92D	B189	L	+
80–81	H133		—	92D-E	L111	S	+
80–81	H140[16]		—	93F	A147[22]		
80–81	H153		—	93F-94A	B93	S	+
80–81	H159		+	94A	D100	L	—
80–81	J139		+	94A	L125	S	—
80–81	J145		+	94B	B240	S	+
80–81	L61		+	94E	B27	L	+
80–81	L65		—	94E	R13	S	+
80–81	L68		—	95–96	J121[23]	L	+
80–81	P31		—	95A	B172[24]	L	+
80–81	R24[17]		—	95E	H173	S	+
80–81	R100		—	96A	A117	S	+
80–81	R114		—	96A	B217	L	—
80–81	R142		—	96A	D221	S	—
81F	J1[18]		—	96A	G73	L	+
82A	J17	D	+	96B	B197	L	—
82C	A154	L	+	96C	H135	S	—
82C	B155	L	—	97A	R87	S	—
82E	D107		—	97B	B158[25]	L	+
83C	G144	S	+	97B	G75	S	—
83C-D	L132	S	+	97B	R71	L	—
83E	A109	L	+	97D-E	A121	L	—
83E-F	L136	S	+	97E-F	J116	L	+
84D	D85	S	—	97F	R128	S	—
85E	G42	L	—	98B	B226	L	+
85F	G8	S	—	98B	H163	L	+
86A	L17[19]	L	+	98E	R78	S	—
86B	R36	L	+	98E-F	J151	S	—
86F	J141	S	+				

TABLE 2—Continued

Haploid phenotype	Results	Autosomal breakpoint	Translocation designation	Y breakpoint	X/T (Y;A) ♂ fertility	
M(3)1	c	98F	A82[26]	S	+	6. *In(3LR)64F;99E*
		98F	B152	L	—	7. *In(3LR)65A;81F*
		99C-D	B81[27]	L	+	8. *In(3L)66B;66F*
	c					9. *In(3L)61E;66B-C*
		99E	R133	L	+	10. *T(Y;3;4)*
	c					Y^{L}|101; $Y^{S}.Y^{L}$|69F—61; 100—69F|102
		99F	G116[28]	L	—	11. *In(3LR)79D;99E*
						12. *In(3)68C-D;80–81*
	c	99F	P60	L	—	13. *In(3)65F;80–81*
						14. *In(3)80–81;87A.*
		100A	A113	S	+	15. *In(3)64C-D;80–81*
M(3)f	a					16. *In(3)66F;80–81*
	c	100A	R130	S	—	17. *In(3R)91B-C;94C-E;98E*
						61—80|Y; Y|81—91B|94E—98E|94E—91C|98E—100
		100B-C	L129	S	—	18. *In(3LR)75C;81F*
						19. *In(3L)68B;73F*
M(3)g	c	3R tip				20. *In(3LR)70D-F;87A*
						21. *T(Y;3;4)*
						$Y^{L}.Y^{S}$|90D—100; Y^{S}|101; 61—90D|102
						22. *T(2;3)60B;61A*
						23. *In(3LR)61–65;95–96*
						24. *In(3R)93B-C;99A*
						25. *In(3LR)76A;93B*
						26. *In(3L)70D-F;79B-C*
						27. *In(3)80–81;87–88*
						28. Complex; 1 breakpoint in 64C

* Lost
1. *In(3LR)61E;* other break not determined
2. *In(3LR)80;* other break in middle of 3R
3. *In(3L)64D;71B?*
4. *In(3L)64E;68A*
5. *In(3R)94E-F;97C-D*