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Viability and fertility of sex-linked autosomal duplications in *Lucilia cuprina* (Wiedemann)

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Summary. Viable and fertile Y-linked duplications have been recovered in *Lucilia cuprina* for autosomal segments ranging in size from 2–12% of the autosomal polytene chromosome complement. No viable deficiency in this size range was recovered. Survival to adulthood of the duplications decreased with increasing duplication size. Genetic background also influenced recovery of some duplications. Recovery of duplications from fertile duplication-male parents was frequently much higher than from translocation-male parents, possibly due to low adjacent-1 segregation in some translocations or to meiotic-drive-type events. Chromosome 4R may contain a triplo-lethal locus. The use of sex-linked duplications in female-killing systems for genetic control programs may have considerable advantages over reciprocal sex-linked translocations, both in terms of fertility and strain stability.

Key words: Genetic control – Aneuploidy – Y chromosome – Sexing systems – *Lucilia cuprina*

Introduction

Studies aimed at developing genetic methods of controlling the Australian sheep blowfly, *Lucilia cuprina* (Wiedemann) have concentrated partly on the potential uses of sex-linked translocations. Such translocations can be used both as transport mechanisms to swamp populations with deleterious genes such as eye colour mutations (Whitten 1979), and in female-killing systems in conjunction with insecticide resistance mutations (Whitten and Foster 1975). However, the ability of translocations to revert to structurally normal arrangements with subsequent selection of these more fertile karyotypes, has presented difficulties in actual mass-rearing operations (Foster et al. 1980a).

One alternative to reciprocal Y-autosome translocations in female-killing systems, would be to construct a Y-linked duplication of an appropriate autosomal segment (i.e. to attach a segment containing a resistance locus to the portion of the Y chromosome which contains the male-determining locus or region). To evaluate this option it is necessary to determine whether even a small amount of autosomal trisomy may affect the performance of males carrying this type of rearrangement. In this report we present data from laboratory studies of viability and fertility of Y-linked autosomal duplications in *L. cuprina*, and discuss the implications of these results for programmes of autocidal pest control.

Materials and methods

Mutations and strains

The linkage groups, symbols and names of mutations cited in this report are listed below: chromosome 2 – *bp* (black puparium), *frag* (fragmented veins), *gla* (glazed eyes), *pb* (purple body); chromosome 3 – *yw* (yellowish eyes); chromosome 4 – *gl* (golden body); chromosome 5 – *mv* (Ml-veinless); chromosome 6 – *y* (yellow eyes), *st* (stumpy bristles). Details of the genetic and cytogenetic mapping of these mutations are contained in Foster et al. (1980b, 1981).

The Y-autosome translocations used in the present study were isolated in the offspring of γ-irradiated males mated to females marked genetically on each autosome, by utilising pseudolinkage of the appropriate marker (Fig. 3 of Foster and Whitten 1974) to sex. The Y chromosome determines maleness in this species (Ullerich 1963). Thus Y-autosome translocations isolated by this procedure give predominantly wild-type male and mutant female offspring.

Translocations were assessed for their ability to generate viable duplications by the procedure outlined in Fig. 1. Males heterozygous in repulsion for the translocation and genetic markers located on both arms of the appropriate structurally

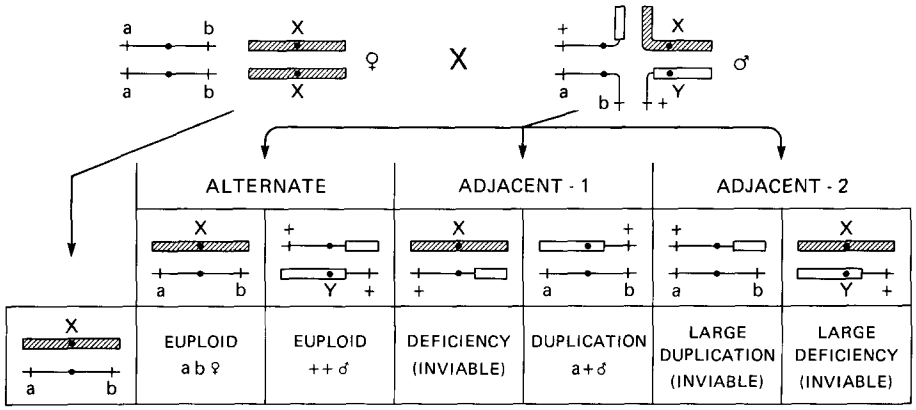
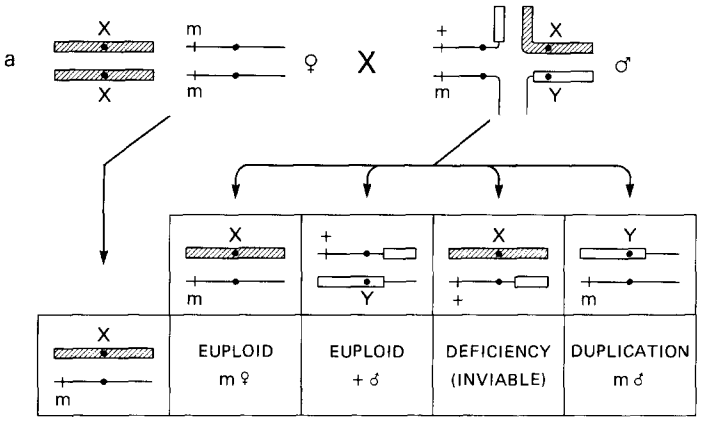


Fig. 1. Detection of autosomal duplications generated by Y-autosome translocations



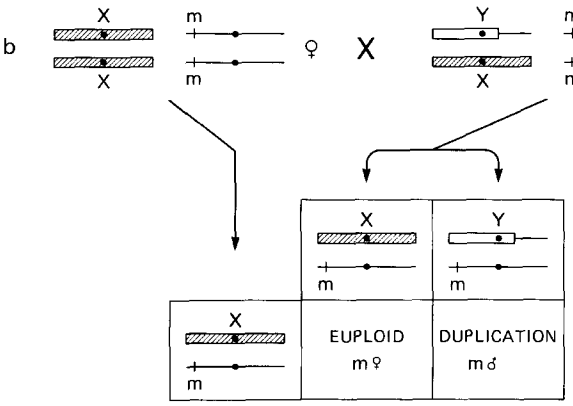


Fig. 2a, b. Crosses used to estimate viability of duplication-bearing males: **a** from translocation – ♂ parents, **b** from duplication – ♂ parents

normal autosome were mated to females homozygous for both markers. Crossing over is rare or absent in *L. cuprina* males (Foster et al. 1980a). The appearance of one (as shown in Fig. 1) or both markers in a portion of the male offspring therefore indicated the presence of a viable duplication.

Autosomal breakpoints of most of the translocations which yielded viable duplications were determined by cytological examination of pupal trichogen cell polytene chromosomes prepared as described by Foster et al. (1976). Mitotic preparations of these translocations were not examined. However, from existing knowledge of the positions of the autosome centromeres (Foster et al. 1980b) and the position of the sex-determining factor near the Y-chromosome centromere (Foster and Bedo unpublished), it was possible to infer which elements carried the Y-chromosome centromere.

Estimation of length of duplications and deficiencies

Estimates of the relative lengths of duplications and deficiencies were obtained for each using photographs of 4 whole trichogen-cell nuclei in which there was no evidence of stretching of the chromosomes. The position of the break point of each duplication was marked on each photograph and the length of each duplication was measured. This was then expressed as a

portion of the length of the entire polytene chromosome complement for each nucleus, and an average proportion was derived from the 4 measurements.

Rearing procedures

The general rearing procedures and conditions used in the present study have been described by Foster et al. (1980b, 1981). Unless otherwise noted all crosses were performed using single-pair cultures.

Viability of aneuploid genotypes

Data concerning viability (i.e. survival to adulthood) and fertility of aneuploid genotypes were obtained by counting the offspring of two types of crosses. In the first type of cross (Fig. 2a), males heterozygous in repulsion for a Y-autosome translocation and a mutation whose locus was not covered by the expected (adjacent – 1) duplication, were mated to homozygous mutant females. In this type of cross, translocation-bearing male offspring are wild type and duplication-bearing males are mutant in phenotype. In the second type of cross (Fig. 2b), fertile mutant duplication-bearing males were mated to mutant females. All male offspring of this type of cross carry the duplication.

Results

1 Size and recovery of duplications

The cytological limits and estimated proportions of the autosomal polytene chromosome complement included in each duplication for which quantitative viability data are available, are presented in Table 1. For convenience of analysis, duplications arising from Y-single-autosome (Y; SA) translocations are grouped separately from those arising from Y-multiple-autosome translocations. Except for those complex rearrangements whose structures are shown in Fig. 3, all of the translocations appeared to result from a single autosomal break and at least one Y-chromosome break. Most of the translocations studied generated only the terminal hyperploid (Lindsley et al. 1972) type of duplication. Of the four exceptions, *Dp(5L;5R;Y)39*, *Dp(4L;5R;Y)22* and *Dp(5R^P;2R;Y)44*, each contained both a terminal hyperploid and an interstitial segment, and *Dp(3L;Y)34* contained only an interstitial autosomal segment (Table 1, Fig. 3).

Table 1. Cytological limits and relative lengths of duplications

Duplication	Original translocation	Cytological limits of duplication	Proportion polytene complement duplicated
Dp(3L;Y)66	T(Y;3)66	21A – 23A[a]	0.02
Dp(2R;Y)45	T(Y;2)45	18A – 20C	0.03
Dp(3R;Y)58	T(Y;3)58	37C – 40C[a]	0.03
Dp(6L;Y)13	T(Y;6)13	81A – 86C[a]	0.05
Dp(3R;Y)21	T(Y;3)21	34A – 40C	0.06
Dp(3R;Y)49	T(Y;3)49	34B – 40C	0.06
Dp(3L;Y)34	T(Y;3)34	26A – 30C[a]	0.06
Dp(6R;Y)63	T(Y;6)63	94C/95A – 100B[a]	0.06
Dp(6L;Y)56	T(Y;6)56	81A – 88A[a]	0.07
Dp(2R;Y)1	T(Y;2)1	12C/13A – 20C[a]	0.08
Dp(2R;Y)10	T(Y;2)10	13A – 20C	0.08
Dp(5L;5R;Y)39	T(Y;5)39	67A – 71A/B; 78A – 80D[a]	0.08
Dp(5L;Y)9	T(Y;5)9	61A – 69A[a]	0.08
Dp(4L;Y)35	T(Y;4)35	41A – 49C[a]	0.10
Dp(5L;Y)36	T(Y;5)36	61A – 70B[a]	0.10
Dp(5L;Y)46	T(Y;5)46	61A – 70B	0.10
Dp(5L;Y)28	T(Y;5)28	61A – 71A/B	0.10
Dp(6L;Y)62	T(Y;6)62	81A – 92B[a]	0.11
Dp(2L;Y)42	T(Y;2)42	1A – 12C	0.12
Dp(2L;Y)54	T(Y;2)54	1A – 12B/C[a]	0.12
Dp(2R;Y)31	T(Y;2;3)31	18A – 20C[a]	0.03
Dp(3R;Y)29	T(Y;2;3)29	36C – 40C	0.03
Dp(2R;Y)59	T(Y;2;6)59	18A – 20C	0.04
Dp(3R;Y)5	T(Y;2;3)5	34A – 40C[a]	0.05
Dp(5R;Y)44	T(Y;2;5)44	74A – 80D	0.07
Dp(5R;Y)26	T(Y;4;5)26	74A – 80D[a]	0.07
Dp(4L;5R;Y)22[b]	T(Y;4;5)22	46B – 47D/48A; 75B – 80D[a]	0.07
Dp(6R;Y)37	T(Y;3;6)37	92B/93A – 100B[a]	0.08
Dp(3L;Y)38	T(Y;3;5)38	21A – 29C[a]	0.10
Dp(5R^P;2R;Y)44	T(Y;2;5)44	74A – 76C; 13B – 20C	0.11

[a] See Foster et al. (1980b) for exact postition of break points
[b] Previously referred to as *Dp(5R;Y)22* (Foster et al. 1980b)

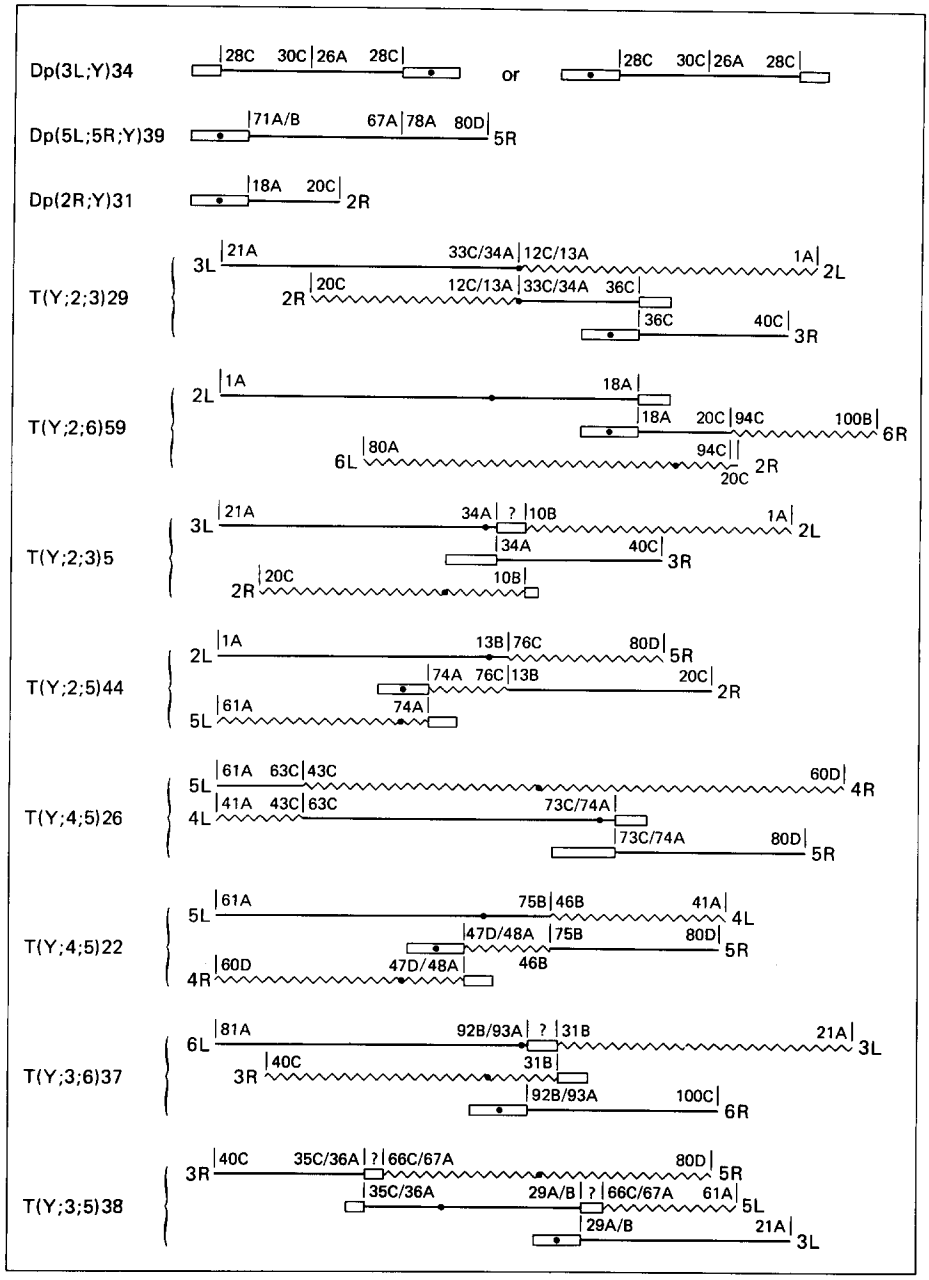


Fig. 3. Structures of complex duplications and translocations. Numbers followed by the letters *A*, *B*, *C* or *D* indicate the positions of the ends of chromosomes and the autosomal break points of translocations, with reference to the standard polytene chromosome map (Foster et al. 1980b). Numbers followed by *L* or *R* represent the left or right ends of the particular autosomes concerned

Of the 70 Y-autosome translocations examined, more than half yielded viable duplications. However, the frequency of duplication-generating translocations was not uniform for all chromosomes, being significantly lower for chromosome 4 than for the other autosomes (Table 2). Quantitative data on recovery of duplications from 29 of these translocations are presented in Table 3.

2 *Inviability of deficiencies*

Adjacent segregation in the type of cross outlined in Fig. 1 yields reciprocal aneuploid zygotes either duplicated or deficient for the portion of the autosome distal to the translocation break point. As noted earlier, recent experiments suggest that the male-determining factor is located in the centromere region of the Y chromosome. Thus, if the aneuploid products were viable, adjacent-1 segregation would lead to duplication-bearing mutant (a+) male and deficient (+b) female offspring. Adjacent-2 segregation would yield +b females bearing large duplications or a+ males with large deficiencies. In the initial screening of translocations for production of viable duplications (Fig. 1) a b females and + + males were always recovered, and occasionally a+ or +b males (but not both). No a+ or +b females were recovered in this type of cross.

Table 2. Recovery of viable autosomal duplications from Y-autosome translocations

Chromosome	Number of translocations examined[a]	Number which yielded duplications
2	13	8
3	15	8
4	13	2*
5	16	10
6	13	9

[a] See Fig. 1

* Recovery of chromosome 4 duplications is significantly lower than for each of the other chromosomes, on the basis of a 2×5 contingency table test of independence ($\chi^2_4=9.72$, $0.01<p<0.05$). Removing the data for chromosome 4, $\chi^2_3=0.76$

In the crosses used to estimate viability (Fig. 2) both duplication-bearing (adjacent-1) and large-deficiency (Fig. 1) males would have the mutant marker phenotype (since the marker chosen for each of these crosses was *not* covered by the duplication), and deficient or large-duplication females would have been wild-type. No wild-type female offspring was recovered from any of the crosses performed (Table 3), suggesting that in each case the deficient zygotic products of adjacent-1 segregation, or duplicated products of adjacent-2 segregation (if they occurred), were unable to survive to adulthood. On the other hand, numerous mutant male offspring were recovered (Table 3). Although in theory these could be either deficient (adjacent-2 segregation) or duplication-bearing (adjacent-1 segregation), the inviability of the deficient female zygotes suggests that all

Table 3. Recovery of mutant and wild-type offspring from crosses involving translocation-male parents

Translocation no.[b]	Genetic marker used[a]	Number of single ♀ cultures	Total number of offspring			
			Mutant		Wild-type	
			♀♀	♂♂	♀♀	♀♀
66	*yw*	17	699	604	0	719
45	*bp*	16	623	537	0	667
58	*yw*	16	510	428	0	480
13	*y*	16	753	175	0	799
21	*yw*	15	454	173	0	582
49	*yw*	18	503	74	0	623
34	*yw*	6	153	20	0	172
63	*y*	16	650	297	0	663
56	*y*	19	850	155	0	868
1	*frag*	17	724	36	0	836
10	*frag*	20	984	34	0	1088
39	*mv*	17	597	319	0	552
9	*mv*	15	515	194	0	596
35	*gl*	15	607	31	0	702
36	*mv*	19	666	172	0	753
46	*mv*	15	493	101	0	466
28	*mv*	17	881	41	0	908
62	*st*	15	362	16	0	414
42	*bp*	21	895	78	0	959
54	*bp*	21	878	113	0	930
31	*bp*	19	453	351	0	461
29	*yw*	15	343	257	0	362
59	*bp*	17	335	304	0	364
5	*yw*	15	236	77	0	286
44	*mv*	15	248	66[c]	0	307
26	*mv*	18	491	77	0	530
22	*mv*	16	483	147	0	588
37	*y*	18	466	57	0	557[d]
38	*mv*	19	380	115	0	446

[a] Females homozygous for a single genetic marker not covered by the duplication were crossed to males heterozygous in repulsion for the translocation and that marker

[b] See Table 1

[c] Of the 66 mutant males, 28 had a coppery body-colour phenotype and 38 had wild-type body colour. See text for further details

[d] A portion of y^{+} ♂♂ offspring of *T(Y; 3; 6)37* were probably viable *Dp(3L)*-bearing individuals

the mutant male offspring recovered were duplication-bearing.

3 Fertility of duplication-bearing males

All 30 of the duplication-bearing genotypes detected in the present study were phenotypically male. All of those containing duplications of less than 8% of the polytene complement were fertile, but only 3 of the 11 which carried duplications of 8% or more of the genome were consistently fertile (Table 4).

Sterile duplication-bearing males fell into two categories. One class included very weak males which were usually small and physically abnormal. Such males died within a few days of eclosion, and were probably incapable of mating. *Dp(4L;Y)35, Dp(6L;Y)62, Dp(2L;Y)42* and *Dp(2L;Y)54* males were of this type. The second class of sterile duplications included all the large *5L* duplications. These males displayed a coppery-brown body colour (Foster et al. 1980b) and curled-down wings, and were longer-lived and capable of mating. The testes of such males contained normal-appearing sperm, but the spermathecae of females dissected after copulation with such males contained no sperm, suggesting some difficulty with sperm transfer. This class of male was not invariably sterile, however, as crosses involving *Dp(5L;Y)36* males occasionally produced offspring.

Table 4. Recovery of females and males from crosses[a] involving fertile duplication-bearing males

Duplication no.	Number of single ♀ cultures	Total number of offspring		χ_1^2[b]
		Mutant ♀♀[c]	Mutant ♂♂	
66	16	1037	911	0.05
45	18	1182	1138	2.36
58	17	955	817	0.06
13	18	1209	516	38.29
21	15	750	267	0.35
49	18	891	358	54.81
34	5	237	89	16.40
63	17	1086	515	0.18
56	15	979	311	26.34
1	17	745	288	158.41
10	16	859	204	128.99
39	19	1076	579	0.01
31	19	1264	1144	3.59
29	15	661	535	0.58
59	19	700	619	0.07
5	20	863	382	4.45
44 (5R)	18	494	183[d]	21.48
26	16	917	441	72.90
22	16	1039	434	8.29
37	16	572	93	2.53
38	19	872	325	2.79
44($5R^P$;2R)	5	203	97	41.65

[a] See Fig. 3

[b] Chi-square values obtained from comparison of mutant ♀♀: mutant ♂♂ with those recovered from translocation parents (Table 3)

[c] See Table 3 for genetic markers used

[d] Does not include 60 male progeny *(Dp($5R^P$;2R;Y)44)* with coppery body colour phenotype

4 Recovery and relative viability of duplications

The recovery of duplication males relative to their euploid sisters in the crosses with translocation-male parents decreases with increasing duplication size (Fig. 4a), in remarkably close quantitative agreement with the data in Fig. 4 of Lindsley et al. (1972) for terminal hyperploids generated from translocation-bearing parents in *D. melanogaster* (assuming that each numbered salivary subdivision is approximately 1% of the *Drosophila* genome). Similarly, recovery of duplication males from duplication-bearing male parents also decreases with increasing duplication size (Fig. 4b). However, recovery of duplications from such parents is frequently higher than that from the corresponding translocation parents.

In 11 of the 22 cases where duplication-bearing males were fertile, the number of duplication-bearing offspring recovered in relation to females was significantly lower from translocation parents than from duplication parents (Table 4). Among the duplication male crosses, with the possible exception of *Dp($5R^P$;2R;Y)44*, there is no indication that the mutant ♂ : ♀ ratio from crosses which gave higher ratios than their corresponding translocations, differed significantly from crosses involving duplications of similar size which did not yield higher ratios than their corresponding translocations.

There is no suggestion from the data that recovery of females exceeds that of wild-type males in the translocation crosses. Thus the observed differences in duplication-male : female ratios probably represent decreased production of duplication zygotes by translocation parents rather than decreased production of female zygotes by duplication parents.

5 Effect of genetic background on viability of duplications

As noted by Lindsley et al. (1972) there are no apparent differences in viability of equivalent-sized duplications from different parts of the genome. However the data from duplication-male parents suggests a marked effect on viability of the mutations used to detect the duplications, with duplication males homozygous for *mv* being more viable than those homozygous for other markers. This effect causes the chromosome 5 duplications to appear more viable than the others. That this is not merely an effect of trisomy for chromosome 5 is suggested by the high viability of *Dp(3L;Y)38;mv/mv* and

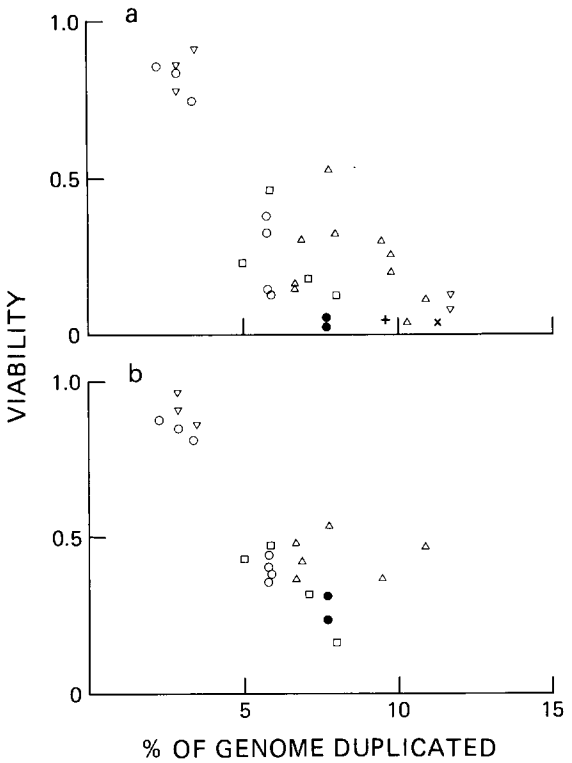


Fig. 4a, b. Relative recovery of duplications plotted against duplication size: **a** from translocation parents, **b** from duplication parents. Mutation symbols: ○ *yw;* □ *y;* △ *mv;* ▽ *bp;* ● *frag;* + *gl;* × *st.* Viability was calculated by dividing the number of mutant male offspring by the number of female offspring in each cross

Dp(5R^P;2R;Y)44;mv/mv males. *Dp(3L;Y)38* is 1.6 times the size of and includes nearly all of the largest other 3L duplication, *Dp(3L;Y)34,* but *Dp(3L;Y)38; mv/mv* males appear to have the same viability as *Dp(3L;Y)34;yw/yw* males (as measured by mutant ♂ : ♀ ratios). Similarly, *Dp(5R^P; 2R; Y)44* is 1.4 times the size of and includes virtually all of the genetic material contained in *Dp(2R;Y)1* and *Dp(2R;Y)10,* yet it appears to be twice as viable in duplication-male crosses.

In a separate experiment the possible influence of genotype on the viability of duplication-bearing males was illustrated by the following results. From the cross *pb bp gla/pb bp gla* ♀♀ × *pb bp gla/+bp+/ Dp(2R;Y)10(bp$^+$gla$^+$)* ♂♂ (mass mating), the following progeny were obtained: *178 pb bp gla* ♀♀, *186 +bp+* ♀♀, *15 pb+ +* ♂♂, *96 + + +* ♂♂, suggesting that the phenotypically wild-type duplication-bearing males were six times more viable than those homozygous for *pb.* Note also that the ratio of *+ + +* ♂♂, to *+bp+* ♀♀ progeny in this cross is more than twice that of *frag* ♂♂ to *frag* ♀♀ in the cross involving the same duplication reported in Table 3. Similar results have been obtained in other crosses involving Y-linked duplications in which genetic markers have been segregating.

Discussion

The failure in the present study to detect any viable heterozygous deficiencies, coupled with occasional survival of duplications of up to 12% of the autosome complement, is in accord with the conclusion of Lindsley et al. (1972) in *Drosophila* and Sandler and Hecht (1973) in man, that viability is much more severely affected by segmental monosomy than by trisomy. Similarly, the present observation that recovery of duplications decreases with increasing duplication size is also consistent with the observations and conclusions of these authors.

The observation that some mutations may markedly reduce the survival of duplications, suggests a need for caution in design and interpretation of experiments aimed at the study of aneuploidy. It is conceivable, for example, that some of the deficiencies and duplications adjudged by Lindsley et al. (1972) to be inviable (or duplications classed as inviable in the present study), could have survived in different (especially non-mutant) genotypes. However this does not affect the generality of their overall conclusions.

For the present study, translocations were saved and examined cytologically only if during initial screening they yielded viable duplications. Thus no data bearing directly on the question of triplo-lethal loci (Lindsley et al. 1972) are available. However the low recovery of duplications from translocations involving chromosome 4 may reflect the presence of such a locus on chromosome 4 (probably 4R). With the exception of small regions near the centromeres of all the chromosomes, chromosome 4R of *L. cuprina* is the only region in which no viable duplications have so far been detected (Foster et al. 1980b). Note that the only triplo-lethal locus in *D. melanogaster* is on 3R (Lindsley et al. 1972; Denell 1976), and this arm appears to be homologous with the *L. cuprina* chromosome 4 (Foster et al. 1981). However, as noted below there are other possible explanations for failure of recovery of duplications from particular translocations.

The lower recovery of certain duplications from translocation parents compared to duplication parents could result from any of several causes. The most likely possibilities appear to be: (1) an excess of alternate over adjacent segregation, (2) the occurrence of adjacent-2 segregation at the expense of adjacent-1 segregation, or (3) some "meiotic-drive" mechanism. Insufficient data are available to distinguish between these or other possibilities.

As noted earlier, a Y-linked duplication containing an insecticide resistance mutation could in theory be used in female-killing systems in a manner analogous to the use of sex-linked translocations in *L. cuprina* and other species (briefly reviewed by Smith et al. 1981).

However, it is likely that even the smallest of the duplications described in the present study would not be fully viable or competitive under field conditions. In the only relevant field trial conducted so far, the capture rate of released wild-type *L. cuprina* males bearing a duplication for the distal half of 3L (approximately 8% of the polytene complement), was only 21% of that of their euploid siblings (R. Mahon unpublished). Nevertheless it is possible that individuals bearing much smaller duplications, say those involving only a few polytene chromosome bands, may not be severely debilitated in the field. If such a strain can be developed it should be fully fertile, as deficient aneuploid segregation products would not be produced. This would be an advantage over most existing or proposed systems which use reciprocal Y-autosome translocations. Moreover, heterologous interchange between the Y and other chromosomes in such a strain may be less likely to yield more-fertile karyotypes with consequent strain breakdown, as observed by Foster et al. (1980a) in a *T(Y;5;3)* strain.

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