The effects of inversions and the *C(3)G* mutation on intragenic recombination in *Drosophila*

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SUMMARY

The effects of heterozygous inversions and of the *C(3)G* mutation on recombination between alleles of the rudimentary locus of *Drosophila melanogaster* were analysed. It is shown that the presence of either of these genetic alterations significantly changes the frequency and type of intragenic recombinants which are produced.

It has long been known in *Drosophila* that a heterozygous inversion in one region of the genome leads to an increase in intergenic recombination in the remainder of the genome (Sturtevant, 1919; Lucchesi & Suzuki, 1968). Also well documented is the fact that the *C(3)G* mutation, which lacks a morphologically recognizable synaptinemal complex when homozygous (Meyer, 1964; King, 1970) alters the frequency of intergenic recombination throughout the genome in both homozygous and heterozygous conditions. When homozygous, recombination is absent (Gowen & Gowen, 1922) and when heterozygous, recombination is increased in frequency above the normal level (Hinton, 1966). This work extends the analysis of these phenomena by examining the effects of heterozygous inversions and of the *C(3)G* mutation on intragenic recombination.

The genetic aspects of the *rudimentary* (*r*) cistron, as well as the general methodology used in this work has been presented in a previous publication (Carlson, 1971). The *r* cistron is located on the *X* chromosome of *Drosophila melanogaster* at a map position of 54·5. Crossing over between alleles of *r* occurs both by reciprocal and non-reciprocal mechanisms which generate both recombinant (*R*) and parental (*P*) classes of flanking markers associated with intragenic recombinants. The relative frequencies of the two *R* and the two *P* flanking marker classes are reproducible in crosses between alleles so that any deviation can be statistically analysed. The R_1 class of flanking markers can be accounted for by recombination between only the *r* alleles. The R_2 class can be explained by recombination between *r* alleles together with exchanges in both the proximal and distal flanking regions. The P_1 class can be accounted for by recombination between the *r* alleles and in the proximal flanking region, and the P_2 class by recombination between the alleles and in the distal flanking region. The important flanking markers used in this study are *tiny chaete* (*tc*) as the distal marker, and *forked* (*f*) as the proximal

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Table 1. *Flanking marker configurations associated with intragenic recombination at the r locus of* Drosophila melanogaster *in the presence and absence of heterozygous inversions and the C(3)G mutation*

Genotype of chromosome			Non-crossover for flanking markers		Crossover for flanking markers		Population size ($\times 10^{-5}$)	Frequency of wild-type recombinants ($\times 10^{-5}$)	Percentage increase
X	II	III	P_1	P_2	R_1	R_2			
$\dfrac{tc, r^{42}, f}{r9}$	$\dfrac{+}{+}$	$\dfrac{+}{+}$	14	5	23	1	1·6	27·1	—
$\dfrac{tc, r^{42}, f}{r18}$	$\dfrac{+}{+}$	$\dfrac{+}{+}$	9	4	13	1	1·2	21·7	—
$\dfrac{tc, r^{9}, f}{r21}$	$\dfrac{+}{+}$	$\dfrac{+}{+}$	11	2	18	0	3·2	9·6	—
$\dfrac{tc, r^{41}, f}{r27}$	$\dfrac{+}{+}$	$\dfrac{+}{+}$	13	3	17	1	4·7	7·2	—
$\dfrac{tc, r^{42}, f}{r9}$	$\dfrac{SM1}{+}$	$\dfrac{Ubx130}{+}$	6	4	19	0	0·9	31·8	17
$\dfrac{tc, r^{42}, f}{r18}$	$\dfrac{SM1}{+}$	$\dfrac{Ubx130}{+}$	4	3	21	1	1·2	24·9	15
$\dfrac{tc, r^{9}, f}{r21}$	$\dfrac{SM1}{+}$	$\dfrac{Ubx130}{+}$	24	13	63	3	8·2	12·6	31
$\dfrac{tc, r^{41}, f}{r27}$	$\dfrac{SM1}{+}$	$\dfrac{Ubx130}{+}$	17	7	51	2	7·9	9·7	34
$\dfrac{tc, r^{9}, f}{r21}$	$\dfrac{+}{+}$	$\dfrac{C(3)G}{+}$	10	3	41	1	3·8	14·5	51
$\dfrac{tc, r^{9}, f}{r21}$	$\dfrac{+}{+}$	$\dfrac{C(3)G}{C(3)G}$	0	0	0	0	—	—	—
$\dfrac{tc, r^{41}, f}{r27}$	$\dfrac{+}{+}$	$\dfrac{C(3)G}{+}$	10	2	28	0	4·0	9·9	37
$\dfrac{tc, r^{41}, f}{r27}$	$\dfrac{+}{+}$	$\dfrac{C(3)G}{C(3)G}$	0	0	0	0	—	—	—

marker (consult Linsley & Grell (1967) for details concerning the mutants and inversions used in this work).

To determine the effects of heterozygous autosomal inversions on intragenic recombination, female flies were made heterozygous for $In(2LR)SM1$, $In(3LR)$-Ubx^{130}, and the appropriate r alleles and flanking markers. Control experiments utilized the same r alleles and flanking markers in the absence of inversions. The results, which are presented in Table 1, show that there is a significant difference in the classes of flanking markers which are associated with intragenic recombination in the presence or absence of heterozygous inversions ($\chi^2 = 12\cdot5$, 3 d.f., $P < 0\cdot01$). The differences in flanking markers specifically involve an increase in the R_1 class of flanking markers, a decrease in the P_1 class, and a slight increase in the P_2 class. The data indicate that the effect of inversions on intragenic recombination involves:

(1) an increase in the relative frequency of recombinant flanking markers associated with intragenic recombination, and

(2) a change in the polarity of the recovery of parental flanking markers associated with intragenic recombination.

The data also indicate a slight increase in the total frequency of intragenic recombination events in the presence of heterozygous inversions. The increase in intragenic recombination is, however, much less striking than the observed increases in intergenic recombination found in the same region of the X chromosome (Schultz & Redfield, 1951). These data suggest that the increases in intergenic recombination which are observed in the presence of heterozygous inversions are not due solely to an increase in the number of recombination events which are initiated, but rather to an alteration of the type of recombination event which is generated at a relatively stable number of initiation points. Such an explanation would account for the absence of a large increase in the number of intragenic events in the presence of heterozygous inversions, and for the marked increase in the R_1 class of flanking markers associated with intragenic recombinants. The data fit the hypothesis of crossover cancellation proposed by Ahmad, Bond & Whitehouse (1972). Their findings of the effects of inversion heterozygosity on intragenic recombination in *Sordaria brevicollis* are in agreement with the data presented here.

To examine the effects of the $C(3)G$ mutation on intragenic recombination, female flies heterozygous for the appropriate r alleles and flanking markers were also made homozygous or heterozygous for $C(3)G$. The results, which are presented in Table 1, demonstrate that $C(3)G$ in the homozygous condition eliminates intragenic recombination of both the reciprocal and non-reciprocal types. The $C(3)G$ mutation in heterozygous condition with a wild allele significantly alters the pattern of flanking markers recovered with an intragenic recombination event ($\chi^2 = 9\cdot9$, 3 d.f., $P < 0\cdot025$). The pattern of flanking markers generated in the presence of a heterozygous $C(3)G$ is distinct from that produced by heterozygous inversions. The effects of $C(3)G$ in heterozygous condition appear to be twofold: (1) an increase in the total frequency of recombination, and (2) an increase in the R_1 flanking marker class relative to the other marker classes.

An examination of the data shows that the effects of heterozygous $C(3)G$ can be explained by the assumption that it specifically increases the frequency of intragenic recombinants carrying the R_1 class of flanking markers, and demonstrates no marked alteration of the three remaining classes of flanking markers. Such an assumption will account for the fact that the total increase in intragenic recombination appears to be due to an increase in only the R_1 class. The data strongly suggest that heterozygous $C(3)G$ is involved in a process which generates the R_1 class, but which is not directly involved in the appearance of the remaining classes. Electron microscopy has demonstrated that homozygous $C(3)G$ females do not form a recognizable synaptinemal complex. The observation that homozygous $C(3)G$ females cannot undergo intragenic recombination demonstrates that a wild allele of $C(3)G$ is a necessary prerequisite for normal recombination, and circumstantially suggests that a synaptinemal complex is essential for recombination. The observation that heterozygous $C(3)G$ females display an altered pattern of intragenic recombination demonstrates that the $C(3)G$ mutation has a dominant effect upon recombination. The disturbed pattern of recombination in $C(3)G$ heterozygotes circumstantially suggests that some components of the synaptinemal complex are directly involved in the mechanism of intragenic recombination.

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