POLARIZED INTRAGENIC RECOMBINATION IN CHROMOSOME REARRANGEMENTS OF NEUROSPORA¹

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PRONOUNCED inequalities have been observed between the two parentally marked classes of prototrophs selected from crosses between allelic auxotrophs (e.g. STADLER 1959; MURRAY 1960). STAHL (1961) and MURRAY (1961) both pointed out that this would be expected if one of the mutant sites was located close to the end of the "effectively paired region". Further analyses of recombination between methionine-2 (*me-2*) alleles of *Neurospora* showed that inequalities between the two parentally marked classes of prototrophs were correlated with inequalities between the two classes with marker genes recombined. The extent of these inequalities was determined by the positions of the mutant sites within the gene (MURRAY 1963). These findings were consistent with the notion that a region of pairing has fixed ends.

Similar studies using alleles at the *paba-1* locus of *Aspergillus nidulans* (SIDDIQI 1961, 1962) also demonstrated inequalities between the two parentally marked classes of prototrophs. These were described in terms of polarized negative interference but, because of an apparent correlation between the extent of the inequalities and the length of the selected region, the explanation offered was dependent on the gross organization of the chromosome rather than small fixed regions of pairing. It was suggested (SIDDIQI 1961; SIDDIQI and PUTRAMENT 1963) that some process essential for recombination was polarized with respect to the centromere from the distal towards the proximal end of the effectively paired region; effective pairing could be interrupted, preventing any further exchanges. In this connection it was of interest that in *Aspergillus* three genes, *ad-9* (CALEF 1957), *paba-1* (SIDDIQI and PUTRAMENT 1963) and *ad-8* (PRITCHARD 1960), all situated in the same arm of linkage group I, appeared to show polarity in the same direction. In *Neurospora* also the two examples of strong polarity, *cys-2* (STADLER and TOWE 1963) and *me-2* (MURRAY 1963), both showed polarity in the same direction with respect to the centromere as the *Aspergillus* genes.

RIZET, LISSOUBA and MOUSSEAU (1960) had earlier demonstrated polarized intragenic recombination for *Ascobolus immersus*, and they explained their findings on the assumption that replication, then postulated to be an essential condition for recombination, was polarized. They also invoked a discontinuous organization of the genetic material. Unfortunately, *Ascobolus* lacks the appro-

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priate markers to orient a map of intragenic sites with respect to a centromere.

The aim of the work reported here was to choose between two basic types of explanation of polarity—a model in which the discontinuous organization of the genetic material imposes fixed regions of pairing, and a model in which a component of the recombination process is polarized with respect to the gross organization of the chromosome. Experiments were designed in which chromosome rearrangements were used to transpose genes and flanking markers that could be used for analysing intragenic recombination. Reciprocal translocation $T(IV;VI)-45502$ was used to transpose *me-2* and its flanking markers from linkage group IV to linkage group VI, in the arm opposite the *cys-2* gene. Intragenic recombination was then analysed in crosses structurally homozygous for the rearrangement. The inversion *S1325* (ST. LAWRENCE and SINGLETON 1963) was used to change the orientation of *me-6* and its flanking markers with respect to centromere, and recombination was analysed in standard crosses and in crosses structurally homozygous for the inversion.

MATERIALS AND METHODS

1. *me-2 and the translocation experiment*: The details of the *me-2* strains, the unselected markers and appropriate methods were given by MURRAY (1963). The translocation strain $T(IV;VI)45502$ originally identified by HOULAHAN, BEADLE and CALHOUN (1949) has a break point in linkage group IV very close to a mutation induced simultaneously in *pyr-3* (pyrimidine-3; MITCHELL, PITTINGER and MITCHELL 1952). The translocation (T^-) strains with and without *pyr-3* were obtained from DR. D. D. PERKINS. *pyr-3* was used to mark the translocation while *me-2* alleles together with appropriate flanking markers were incorporated into translocation sequence by crossing over. The *pyr-3*⁻ marker (supplement: 1 mg uracil/ml medium) was subsequently removed by crossing over in a cross with a *pyr-3*⁺ T^- strain.

2. *me-6 and the inversion experiment*: Two methionine mutations closely linked in IR were used: *me-6* (35809) and *mac* (65108). *mac* (DUBES 1953) is primarily a methionine mutant, but is stimulated by adenine and possibly cystine. Neither *me-6* nor *mac* responds to known methionine precursors, and although they complement one another in heterokaryons, they may be alleles. However, the symbols *me-6* and *mac* will be retained for convenience in this paper. The flanking marker genes were *thi-1* (56501; thiamine-1) and *ad-9* (Y154M37; adenine-9) (Figure 2).

The *nic-2* strain *S1325* (ST. LAWRENCE and SINGLETON 1963) has a long inversion which has not been separated from the niacin requirement of *nic-2*, and which includes the loci *thi-1 mac me-6 ad-9 al-2* (albino-2) and *aur* (aurescent) within the inverted segment.

The selective media were supplemented with thiamine (10 µg/ml), adenine sulfate (0.5 mg/ml) and, in crosses involving the inversion, nicotinamide (40 µg/ml). The methods used were as described for *me-2* analyses (MURRAY 1963), except that ascospores were plated at a reduced concentration (approximately 1,000 to 2,000 per plate) since the prototroph frequencies were higher and the methionine strains were slightly leaky.

In all crosses between the two methionine mutant strains, *me-6* entered in coupling with the linked albino marker *al-2* (15300), and *mac* entered with the complementing albino marker *aur* (34508; aurescent) Since *al-2* is very closely linked to *aur* (HUANG 1964), methionine prototrophs formed by recombination would be albino in phenotype whereas those resulting from nondisjunction would have wild-type pigmentation. Methionine prototrophs of wild-type pigmentation comprise only a small proportion of the total prototrophs isolated. Such isolates were backcrossed to determine whether they were pseudo-wild types or true wild types contaminated with methionine auxotrophs. The presence of methionine prototrophs in the backcross progeny indicated that the original isolate was a true wild type and a sample of such prototrophs was isolated and scored for flanking markers.

RESULTS

1. *Translocation experiment*: Figure 1 shows a genetic map based on data, some of which are given in Table 1, from crosses homozygous for $T(IV;VI)45502$. That part of linkage group IV including *pyr-3* and markers distal to this gene is transposed to the right arm of linkage group VI. The map distances, estimated for any interval from a minimum of 600 random isolates, are strictly comparable with those obtained in standard crosses. In particular the values for the intervals *tryp-4-me-2* (5 units) and *me-2-pan-1* (4.5 units) are very close to the respective values of 6 units and 4 units in standard crosses (MURRAY 1963).

In Table 2 the prototrophs from intragenic crosses are analysed with respect to flanking marker combinations. Three *me-2* alleles were used and the data from standard crosses, taken from MURRAY (1963), are included for comparison with results from crosses structurally homozygous for the translocation. Not only is the direction of polarity unchanged but the extent of the asymmetries is unaffected.

In intragenic crosses involving a few T^- strains the distribution of prototrophs differed in that the more frequent parentally marked class was considerably in excess over the more frequent class with flanking markers recombined. These atypical crosses are not included in Table 2 and differed from those listed only in the two majority classes. For example, in one cross of K5 by K44 the ratio of majority parental to majority recombinant class was 55:23 compared with 37:40 for the comparable cross in Table 2. Subsequent analyses were in agreement with the interpretation that one parent in such a cross carried a genetic factor affecting the pattern, but not the frequency, of recombination. Preliminary data suggest that this postulated factor is effective independent of the translocation. Since scoring for such a factor requires the isolation of large numbers of prototrophs from each cross, the investigation has not been continued.

FIGURE 1.—Gene order in $T(IV;VI)45502$. Centromeres are marked by a solid circle. The hollow line represents the translocated segment.

TABLE 1
Crosses homozygous for T(IV;VI)4502

Zygote genotype and percent recombination	Parental combinations (and percent germination)	Crossovers					Marker isolation numbers and linkage group
		Singles			Doubles		
		Region 1	Region 2	Region 3	(Regions in parentheses)		
$\frac{+ + + + T +}{ylo \quad pan-2 \quad try-p-2 \quad T \quad pyr-3}$ 8.4 11.6 6.3	76 69 (95%)	8 6	11 7	4 6	1 1 (1,2) (2,3)	0 0 (1,3)	Y30539Y (VI) Y153M96 (VI) 75001 (VI) 45502 (IV)
$\frac{+ + + T + try-p-4}{ylo \quad pan-2 \quad T \quad pyr-3} +$ 10.7 10.1 7.7	65 59 (84.5%)	9 8	10 4	8 3	0 1 (1,2) (2,3)	0 0 (1,3)	Y30539Y (VI) Y153M96 (VI) 45502 (IV) Y2198 (IV)
$\frac{T + try-p-4 + +}{T \quad pyr-3 + me-2 \quad pan-1}$ 7.0 7.5 7.0	83 65 (93.5%)	8 5	3 10	5 7	0 0 1,2) (2,3)	0 0 (1,3)	45502 (IV) Y2198 (IV) K44 (IV) 5531 (IV)

The left-hand member of each pair of complementary classes represents the genotype that contains the plus allele of the leftmost marker scored.

TABLE 2

Comparison of crosses between me-2 alleles in standard and translocation homozygous crosses

Genotypes of parents <i>tryp⁻ me⁺ pan⁺ × tryp⁺ me⁺ pan⁻</i>	Methionine prototrophs		Classification of methionine prototrophs			
	No.	Frequency per 10 ⁵ viable spores	Parental		Recombinant	
			Pp*	Pd‡	R1‡	R2§
(a) Standard crosses¶						
— K44 + × + H98 —	114	15	13(11%)	50(44%)	41(36%)	10(9%)
— H98 + × + K44 —	91	45	6(7%)	39(43%)	40(44%)	6(7%)
— K44 + × + K5 —	114	19	11(10%)	50(44%)	41(36%)	12(11%)
— K5 + × + K44 —	211	17	17(8%)	94(45%)	82(39%)	18(9%)
(b) Translocation homozygous crosses						
— K44 + × + H98 —	148	12	15(10%)	64(43%)	58(39%)	11(8%)
— H98 + × + K44 —	668	19	61(9%)	281(42%)	258(39%)	68(10%)
— K44 + × + K5 —	316	15	36(11%)	119(38%)	128(41%)	33(10%)
— K5 + × + K44 —	139	15	12(10%)	61(44%)	56(40%)	10(7%)

* Parental combination of markers entering with proximal *me-2* allele (K44).‡ Parental combination of markers entering with distal *me-2* allele (H98 or K5).‡ R1 is more frequent combination of recombined markers always indicating the order *tryp*-K44-K5 (or H98)-*pan*.

§ R2 is less frequent combination of recombined markers.

¶ Results published in MURRAY (1963).

2. *Inversion experiment*: Figure 2a shows a genetic map of the right arm of linkage group I in a standard strain. The map is based on published information (e.g. PERKINS 1959, 1962; PERKINS and MURRAY 1963). ST. LAWRENCE and SINGLETON (1963) originally described S1325 as a paracentric inversion in the right arm of linkage group I with the proximal break point inseparable from *nic-2*. In crosses homozygous for S1325 and marked with group I markers (see the first two crosses listed in Table 3), *os* (osmotic) showed linkage to *hist-3* and therefore a paracentric inversion would require the second break point to be distal to *os*. However the absence of linkage between *al* and *os* accompanied by a high incidence of multiple exchanges cast doubt on this interpretation. Furthermore, ST. LAWRENCE (personal communication) has shown that the distal break point is very close to *lys-3* and this is compatible with data obtained by NEWMAYER (unpublished) from a cross of S1325 to a pericentric inversion (H4250) of linkage group I. NEWMAYER (personal communication) reconciled these findings with the suggestion that part of linkage group IR, from *nic-2* to around *lys-3*, is translocated and inserted into an unknown linkage group in a sequence inverted with respect to centromere. D. PERKINS (personal communication) identified the other linkage group involved as VR. The break point in group V is between *sp* (spray) and *inos* (inositol) and since *sp* is proximal to *inos* (STRICKLAND, PERKINS and VEATCH 1959) the data in Table 3 confirm that the group I segment is indeed inserted into linkage group V in inverted order. The rearrangement (shown in Figure 2b) is symbolized $T(IR \xrightarrow{In} VR)S1325$. For simplicity it is referred to in this paper as the inversion.

FIGURE 2.—Gene order in $T(IR \xrightarrow{In} VR)S1325$. The centromere is marked by a solid circle.
(a) Standard order. (b) Order in $T(IR \xrightarrow{In} VR)S1325$.

Crosses homozygous with respect to either *me-6* (35809) or *mac* (65108) were infertile; therefore conidia were screened to obtain estimates of the reversion rates. *me-6* gave one revertant and two partial revertants in approximately 6×10^7 conidia plated, whereas *mac* gave only one partial revertant in 5.5×10^8 conidia plated. Since prototrophic ascospores were formed from crosses of *me-6* $\times$ *mac* with frequencies of 4 to 6 in 10^4 ascospores, it seems likely that these prototrophs arose by recombination rather than reversion.

In Table 4 the methionine prototrophs from crosses of *me-6* by *mac* are analysed in terms of flanking marker combinations. In every cross the more frequent class with flanking markers recombined indicates the order *thi-1 mac me-6 ad-9*. Although it is not known whether *me-6* and *mac* are allelic, they are sufficiently closely linked that recombination between them appears to be associated with the high frequency of coincident exchanges characteristic of crosses between alleles. These apparent exchanges must be asymmetrically distributed to give unequal classes of parentally marked prototrophs.

The data in Table 4 show that, in any cross of *me-6* $\times$ *mac*, prototrophs having the flanking marker combination of the *mac* parent are the more frequent. This

TABLE 3
Crosses homozygous for *S1325*

Zygote genotype and percent recombination	Parental combinations and percent germination	Crossovers									
		Singles		Doubles		Triples		Quadruples		Region No.	Region No.
		Region No.	Region No.	Region No.	Region No.	Region No.	Region No.	Region No.	Region No.		
<i>A</i>	<i>his-3</i>	1	2	1,2	0	2,4	3	1,2,3	0	2,3,4	2
<i>a</i>	<i>os</i>	2	8	1,3	3	2,5	0	1,2,4	0	2,3,5	1
	<i>aur</i>	3	34	1,4	1	3,4	11	1,2,5	0	2,4,5	0
	<i>me-6</i>	4	6	1,5	0	3,5	1	1,3,4	0	3,4,5	0
	<i>thi-1</i>	5	1	2,3	8	4,5	2	1,3,5	1		
		Total	51	Total	30	Total	30	Total	4	Total	2
<i>+</i>	<i>his-3</i>	1	8	1,2	4	2,4	2	1,2,3	2		
<i>os</i>	<i>al</i>	2	29	1,3	3	3,4	0	1,2,4	1		
	<i>ad-9</i>	3	4	1,4	0			2,3,4	1		
	<i>me-6</i>	4	2	2,3	12						
		Total	43	Total	21	Total	21	Total	4	Total	0
<i>+</i>	<i>al</i>	1	3	1,2	1	2,4	1	1,2,3	0	2,3,4	0
<i>ad-9</i>	<i>me-6</i>	2	4	1,3	0	2,5	3	1,2,4	0	2,3,5	0
<i>thi-1</i>	<i>+</i>	3	3	1,4	0	3,4	0	1,2,5	0	2,4,5	0
<i>sp</i>	<i>+</i>	4	7	1,5	1	2	3	1,3,4	0	3,4,5	0
		5	17	2,3	0	4,5	0	1,3,5	0		
		Total	75	Total	13	Total	13	Total	0	Total	0

* Only *his1⁺* selected
The left-hand member of each pair of complementary classes represents the genotypes that contains the plus allele of the leftmost marker scored.

TABLE 4

Comparison of crosses of me-6 × mac in standard and inversion homozygous crosses

Genotypes of parents	Number of methionine prototrophs	Classification of methionine prototrophs			
		Parental		Recombinant	
		P <i>mac</i> *	P <i>me-6</i> †	R1‡	R2§
(a) Standard crosses					
<i>thi me-6 ad</i> × + <i>mac</i> +	282	78(27.7%)	30(10.6%)	173(61.3%)	1(0.4%)
<i>thi me-6</i> + × + <i>mac ad</i>	81	21(25.9%)	2(2.5%)	57(70.3%)	1(1.2%)
+ <i>me-6</i> + × <i>thi mac ad</i>	147	33(22.5%)	15(10.2%)	96(65.3%)	3(2.0%)
+ <i>me-6 ad</i> × <i>thi mac</i> +	220	81(36.8%)	13(5.9%)	125(56.8%)	1(0.4%)
(b) Inversion homozygous crosses					
<i>thi me-6 ad</i> × + <i>mac</i> +	116	22(19.0%)	5(4.3%)	88(76.0%)	1(0.9%)
+ <i>me-6 ad</i> × <i>thi mac</i> +	97	22(22.6%)	11(11.3%)	61(62.8%)	3(3.9%)

* Parental combination of markers entering with *mac*.† Parental combination of markers entering with *me-6*.‡ R1 is the more frequent combination of recombined markers always indicating the order *thi-mac-me-6-ad*.

§ R2 is the less frequent combination of recombined markers.

is true both in the control crosses where *mac* is proximal to *me-6* and in the inversion-homozygous crosses where *mac* is distal to *me-6*. The asymmetry is apparently a property of the inverted region itself.

In contrast to studies of other loci cited (MURRAY 1963; SIDDIQI and PUTRAMENT 1963; STADLER and TOWE 1963) the asymmetries are such that the parental combination of flanking markers entering with the proximal methionine marker (*mac*) is the more frequent. However, since *me-6* and *mac* could conceivably be at separate loci, the asymmetries may reflect differences in the relative frequencies of conversion at two sites within adjacent genes rather than within the same gene. More recently *me*(S2706), which does not complement *mac*, has been located between *mac* and *me-6*. *me*(S2706) and *mac* therefore are probably allelic and methionine prototrophs isolated from a cross of *mac* × *me*(S2706) show asymmetries very similar to those from a cross of *mac* × *me-6*. This finding and the fact that both *mac* and *me-6* revert, convert, are leaky, and complement in heterokaryons support the conclusion that the asymmetries do not reflect differences in the extent of the mutations *me-6* and *mac* but rather that they indicate a polarity of the type more adequately demonstrated for other loci. The experiment reported here shows that this polarity is not determined by orientation with respect to centromere.

DISCUSSION

The translocation experiment shows that the pattern of polarity in *me-2* is unaffected either by transposition to linkage group VI or by an increase in the centromere distance. It is also evident that the direction of polarity is in this case different on opposite sides of a centromere, since the gene cysteine-2 (*cys-2*) is in the opposite arm of linkage group VI and is polarized in the opposite direction (STADLER and TOWE 1963) to the translocated *me-2* gene. The inversion experi-

ment demonstrates that the direction of polarity is independent of the orientation with respect to the centromere. These findings show that polarity is not determined by the gross organization of the chromosomes but suggest that it is intrinsic to the region in which recombination is being selected. The critical test of this idea requires a very small inversion comprising only the gene in which recombination is being analysed; such an experiment seems technically impossible.

The present findings are consistent with any models that invoke fixed regions of pairing. These include not only the earlier modifications made to multiple-exchange hypotheses (STAHL 1961; MURRAY 1961), but more recent hybrid DNA models in which the primary breakage of nucleotide chains is restricted to fixed points (HOLLIDAY 1964; HASTINGS and WHITEHOUSE 1964). All these models, with the possible exception of HOLLIDAY's (1964), require a reversal of the direction of polarity within the paired region. Evidence is mounting in support of such reversal of polarity of recombination within the recombination unit (WHITEHOUSE and HASTINGS 1965; PEES 1965; RIZET and ROSSIGNOL 1966; MOUSSEAU 1966). Two examples of reversal of polarity in *Neurospora* (within *me-2* and *me-7*) will be described in a separate paper (MURRAY, in preparation). The fact that polarity of recombination is normally reversed for different locations within a gene supports the conclusion reached from the inversion experiment, that polarity is not a function of gross orientation within the chromosome.

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SUMMARY

Polarized recombination in the *me-2* (methionine-2) gene has been studied previously (MURRAY 1963). In the present work a reciprocal translocation was used to transpose *me-2* and its flanking markers from linkage group IV to linkage group VI in the arm opposite the *cys-2* gene. Recombination was analysed in crosses structurally homozygous for the rearrangement and the direction of polarity of recombination with respect to the centromere (and the flanking markers) was found to be unchanged but was in the opposite direction to that within *cys-2* (as reported by STADLER and TOWE 1963).—*Ab(S1325)*, previously thought to be a paracentric inversion, has been shown instead to be an insertional translocation with part of linkage group IR inserted in inverted sequence into group VR (Unpublished data of NEWMAYER, PERKINS, ST. LAWRENCE, and MURRAY). The rearrangement, symbolized $T(IR \xrightarrow{In} VR)S1325$, was used to change the orientation of *me-6* and flanking markers with respect to centromere. Recombination was analysed in standard crosses and in crosses structurally homozygous for the inversion. Polarity within the *me-6* region remained constant in

direction with respect to flanking markers, but was reversed with respect to centromere.

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