SPECIFIC EFFECTS OF TEMPERATURE ON RECOMBINATION IN *SCHIZOPHYLLUM COMMUNE*

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1. INTRODUCTION

THE effect of temperature on recombination frequency and chiasma formation has been investigated in numerous organisms, with notoriously inconsistent results. The first investigations were made by Plough (1917), who observed that *Drosophila melanogaster* females raised at 13 or 31° C. had higher frequencies of crossing-over than those raised at 22°. The effect of temperature was not constant from one linkage group to another, however, or even from one region to another within a linkage group. The regions most sensitive to temperature were those near the centromeres. Lawrence (1963) found, however, that in crosses between inbred lines of *Drosophila* the effect of temperature was not confined to regions near the centromere.

The first report of a temperature effect on recombination in fungi was that of Hüttig (1931), who showed that in *Ustilago hordei*, studied at temperatures from 4 to 30°, the highest proportion of first-division segregation occurred at 8°. Olive (1956) studied recombination in *Sordaria fimicola* over a temperature range of 7-31° and found that the temperature of maximum crossing-over was different for the three loci examined. Recombination in yeast was studied by Johnston and Mortimer (1967), who found that recombination in most regions did not vary from temperature to temperature; those regions that were affected by temperature had a lower frequency of recombination at the low temperature than at the high. Such regions were not always located near the centromeres.

The fungus in which the influence of temperature on recombination has been most fully studied is *Neurospora crassa*. Rifaat (1959) found that recombination in regions adjacent to the centromere increased with increasing temperatures, for the range 17-30° C. Towe and Stadler (1964), however, reported that the frequency of crossing-over was higher at 18° than at 25°. Regions close to the centromere exhibited a stronger response to temperature than did distal regions. Experiments of McNelly-Ingle, Lamb and Frost (1966) gave results compatible with those of Towe and Stadler: a U-shaped temperature-response curve, with the minimum recombination frequency at 25°. Nakamura (1966) found that the effect of temperature on crossing-over differed not only for different chromosomes but even for the same chromosome region when tested in different crosses. Obviously, no clear understanding of the effect of temperature on recombination has yet emerged from these studies. Several workers (Rees and Thompson, 1958; Rifaat, 1959; McNelly-Ingle *et al.*, 1966; Nakamura, 1966; Johnston and Mortimer,

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1967) have suggested that the temperature sensitivity of crossing-over is under genetic control; segregation of genetic factors could explain the heterogeneity observed.

In the fungus *Schizophyllum commune* mating competence is controlled by two unlinked factors, *A* and *B*, each of which consists of two linked loci, α and β . Frequencies of recombination between the component loci of each factor were found to vary, for some crosses, at different temperatures (Raper, Baxter and Middleton, 1958; Stamberg, 1968). In the course of our studies on the genetic control of recombination in *Schizophyllum*, we found that the effects of temperature on recombination are highly region-specific. We have therefore been able to use temperature as a tool to distinguish various elements of the system controlling recombination.

2. MATERIALS AND METHODS

The strains of *S. commune* originated from the collection of Professor J. R. Raper at Harvard University. Most were derived from isolates of this collection as segregants from crosses. The strains used are listed in table 1.

TABLE 1
Details of the main strains used

Strain	Mating type	Other markers	Origin
14	A4B4		Harvard (fruit body from Ill.)
699	A41B41		Harvard (fruit body from Mass.)
5219	A43B43	nic2- ura1-	Birmingham (derived)
991	A97B97		Harvard (fruit body from Costa Rica)
MS-82	A41B41	arg7-	Harvard (derived)
HS-55	A α 1- β 6B41	pab- ade5-	Harvard (derived)
C40	A43B43	comp2 pab-	Birmingham (derived)
C41	A43B43	comp2 pab-	Birmingham (derived)
ES-1374	A43B43	x15-	Birmingham (derived)

Abbreviations: nic = nicotinic acid.

ura = uracil.

arg = arginine.

pab = para-amino benzoic acid.

ade = adenine.

comp = "compact" morphology.

x15 = unknown nutritional requirement.

Note: Linkage relationships are given by Simchen and Stamberg (1969a).

General procedures concerning fruiting, isolation of spores, and mating-type tests can be found in our previous publications (Simchen, 1967; Stamberg, 1968). With the exception of the experiment reported in section 3(a), in which time is a variable, crosses with sporulating fruiting bodies were incubated at the indicated meiotic temperature for at least 18 hours prior to the collection of spores for testing.

Strains were grown and crosses were performed on a complete medium (Koltin, Raper and Simchen, 1967); strains were scored for nutritional requirements on minimal or supplemented minimal medium (Raper and Miles, 1958). Morphological characteristics were scored macroscopically after several days' growth.

3. RESULTS

(a) *Temperature response in the incompatibility factors*

1. *Time of response to a change in temperature.* A dikaryon formed by strains 14×699 was maintained at 18°C . until it was shedding spores profusely. A sample of spores was obtained (this was designated the time = 0 sample) and the dikaryon was placed at 32° . Samples of from 100 to 350 spores were collected at various times up to 100 hours after the beginning of the incubation period; all were tested for recombination in the *A* and *B* factors (fig. 1).

FIG. 1.—Response to a change in temperature by recombination frequencies in the *A* and *B* factors of dikaryon 14×699 . The dikaryon was maintained at 18° prior to the start of the timed experiment. At $t=0$ the dikaryon was placed at 32° . After 14 hours, frequencies of recombination were stabilised at the values indicated. Open circles represent frequencies of recombination in the *A* factor; solid circles, frequencies of recombination in the *B* factor.

Recombination frequencies in both the *A* factor and the *B* factor changed after incubation at 32° . The 0-hour and 4-hour values were obtained from spores that underwent meiosis at 18° ; after 14 hours at 32° , the recombination frequencies were stabilised at new values that differed significantly from those at 18° (for the *A* factor, $\chi^2_{(1)} = 27.0$, $P < 0.001$; for the *B* factor, $\chi^2_{(1)} = 7.4$, $P < 0.01$); the 8-hour and 12-hour values were intermediate, probably because the spores released then included some that had recombined at 18° and some that had recombined later. That there was no change in recombination frequencies after 14 hours (for frequencies from 14 to 100 hours, $\chi^2_{(6)} = 7.6$, $P > 0.25$ for the *A* factor; $\chi^2_{(6)} = 8.2$, $P > 0.20$ for the *B* factor) indicates that events occurring after recombination but before spore release occupy less than 14 hours.

2. *Response to a range of temperatures.* Replicate plates of the dikaryon formed by 14×699 were maintained at 18° until they were shedding spores and were then placed at various temperatures for at least 36 hours before

spores were collected. (Dikaryons were incubated at the appropriate temperatures longer than the minimum 14 hours in the expectation that meiosis at lower temperatures would be somewhat slower than at 32°. The

TABLE 2

Effect of temperature on recombination in the A and B factors of dikaryon 14×699

Temperature (° C.)	Sample	% Rec. A	% Rec. B
15	140	2.1	7.9
18	363	3.3	6.3
21	139	2.2	10.8
23	188	2.1	8.5
25	157	1.9	7.0
28	267	2.6	9.7
30	205	4.9	2.9
32	1224	12.9	3.2
$\chi^2_{(7)}$ all temperatures		102.6***	46.0***
$\chi^2_{(8)}$ for 15°-30°		5.5	
$\chi^2_{(5)}$ for 15°-28°			4.1

Significance levels in this and the following tables indicated as:

unstarred: $P > 0.05$

*: $P = 0.05-0.01$

** : $P = 0.01-0.001$

***: $P < 0.001$

FIG. 2.—Temperature-response curve for recombination frequencies in the A and B factors of dikaryon 14×699. Recombination frequencies were constant between 15 and 30° in the A factor, and between 15 and 28° in the B factor. Solid circles represent frequencies of recombination in the A factor; open circles, frequencies of recombination in the B factor.

dikaryon placed at 15° was maintained at this temperature for 120 hours prior to the collection of a spore sample. No spores were released from fruiting bodies at temperatures higher than 32°.) Results are given in table 2 and fig. 2.

Recombination in the *A* factor was constant and low from 15 to 30°, inclusive. An abrupt transition must occur between 30 and 32°, for at the latter temperature recombination was significantly more frequent than at the other temperatures. In the *B* factor, the effect of temperature was the reverse. Recombination in this region was constant and high from 15 to 28°, inclusive. Between 28 and 30° a transition occurs; at 30 and 32° recombination was significantly less frequent than at the lower temperatures. The critical temperature is thus slightly different for recombination in the two regions.

(b) *Temperature response in a third linkage group.*

The genes *nic2* and *ura1* are linked to each other but unlinked to the *A* or *B* factor (Raper and Miles, 1958); the use of a strain carrying *nic2* and *ura1* thus made it possible to study recombination on three linkage groups simultaneously.

TABLE 3
Effect of temperature on recombination in three unlinked regions

Strain †	Sample		% Rec. <i>A</i>		$\chi^2_{(1)}$ for temperature variation	% Rec. <i>B</i>		$\chi^2_{(1)}$ for temperature variation	% Rec. <i>nic2-ura1</i>		$\chi^2_{(1)}$ for temperature variation
	18°	32°	18°	32°		18°	32°		18°	32°	
<i>Parent</i> 5219	182	179	3.9	11.7	7.8**	3.9	4.5	0.1	22.5	12.3	6.6*
<i>Progeny of</i> <i>5219</i> × <i>699</i>											
1	129	152	14.0	12.5	0.1	3.9	5.3	0.3	34.9	17.1	11.7***
2	189	140	1.6	18.6	28.9***	9.0	2.1	5.5*	22.8	18.6	0.9
3	166	174	1.2	14.4	20.1***	4.8	2.3	1.6	25.3	19.0	2.0
4	175	186	4.0	9.1	3.8	3.4	0.5	2.6	16.0	12.9	0.7
5	174	140	2.3	12.1	12.1***	4.0	5.7	0.5	15.5	22.9	2.7
$\chi^2_{(4)}$ for genotype variation among progeny											
	40.0***		6.6			7.7		10.6*		21.4***	

† All strains were crossed × tester strain 14.

The doubly auxotrophic strain 5219 was crossed to wild-type strain 699. Five doubly auxotrophic progeny carrying the *A* and *B* factors of 699 were taken at random; these and the auxotrophic parent (5219) were crossed to tester strain 14. Recombination in the *A* factor, the *B* factor, and the *nic2-ura1* region was measured after meiosis at both 18 and 32° (table 3).

From the results at 18° it has been previously concluded that recombination in the *nic2-ura1* region is independent of that in the *B* factor, for the former exhibited variation from genotype to genotype whereas recombination in the latter was constant (Simchen and Stamberg, 1969a). Furthermore, the pattern of response to the change in temperature, given here, shows that recombination in all three regions must vary independently of each other. Of the five progeny crosses, one varied only in *nic2-ura1* recombination, two varied in recombination in the *A* factor only, one varied

in both the *A* factor and the *B* factor, and one showed no variation in any region, whereas the cross involving parent 5219 showed variation in both *nic2-ura1* and *A* factor recombination at the two temperatures. Thus, recombination in the *A* factor may or may not vary from temperature to temperature; if it does vary it may be the only one of the three regions to do so, or it may vary at the same time as either the *B* factor or the *nic2-ura1* region.

(c) *Temperature response in regions linked to the incompatibility factors*

Markers linked to the incompatibility factors have been utilised to determine whether recombination throughout an entire linkage group is co-ordinately regulated. Although the linkage group on which the *B* factor is located is not entirely satisfactory for the purpose (very few markers have been located on this linkage group and none is very close to the *B* factor), nevertheless one marker was used; the results obtained support the conclusions reached from a study of regions linked to the *A* factor.

TABLE 4

Effect of temperature on recombination in the B linkage group and in the A factor

Strain†	Sample		% Rec. <i>A</i>		$\chi^2_{(1)}$ for temperature variation	% Rec. <i>B</i>		$\chi^2_{(1)}$ for temperature variation	% Rec. <i>B-arg7</i>		$\chi^2_{(1)}$ for temperature variation
	18°	32°	18°	32°		18°	32°		18°	32°	
1	161	140	5.0	9.3	2.2	5.0	2.9	0.9	29.8	48.6	11.1***
2	179	144	5.0	22.2	21.3***	7.3	6.3	0.1	46.9	44.4	0.2
3	172	173	1.7	20.2	30.1***	6.4	1.7	3.7	47.1	50.9	0.5
4	189	182	6.9	11.5	2.4	3.7	2.2	0.7	54.5	56.6	0.2

† The four strains were derived from a cross between MS-82 and 699. All had the genotype *A41 B41 arg-* and were crossed $\times$ tester strain 14.

The *arg7* marker has been reported to map about 38 units from the *B* factor (Raper, 1966). Strain MS-82, carrying *arg7*, was crossed to strain 699 and four of the *arg-* progeny were taken at random and crossed to strain 14. Recombination frequencies in the *A* factor, the *B* factor, and the *B-arg7* region were assessed at 18 and 32° (table 4). None of the four crosses varied in *B* factor recombination at the two temperatures. Only one cross varied in the frequency of recombination between the *B* factor and *arg7* at the two temperatures; significantly, recombination within the *B* factor did not vary simultaneously. Thus, the effect of temperature on recombination appears to be independent in the two linked regions. Because of the great distance between the *B* factor and *arg7*, however, the frequency of double recombination in the region between them must be high; this would tend to obscure any correlation between recombination within the *B* factor and that in a neighbouring region.

The linkage group on which the *A* factor is located is well supplied with markers, several of which were used in studies to determine the extent of the region controlled jointly with the *A* factor. Some of the data obtained in these studies have been reported elsewhere (Simchen and Stamberg, 1969a); here, however, we are concerned with the temperature effects that were revealed.

The $x15$ marker maps about 28 units from $A\beta$ (Ellingboe and Raper, 1962). Strain ES-1374, of genotype $A43 B43 x15^-$, was crossed to 699 and five progeny of genotype $A41 B41 x15^-$ were taken at random, crossed to strain 14 and tested for frequencies of recombination at 18 and 32° (table 5).

TABLE 5

Effect of temperature on recombination in the $A\beta$ - $x15$ region, the A factor, and the B factor

Strain†	Sample		% Rec. A		$\chi^2_{(1)}$ for temperature variation	% Rec. $A\beta$ - $x15$		$\chi^2_{(1)}$ for temperature variation	% Rec. B		$\chi^2_{(1)}$ for temperature variation
	18°	32°	18°	32°		18°	32°		18°	32°	
1	192	200	1.6	3.5	1.5	43.8	35.0	3.1	3.7	3.5	0.0
2	193	198	2.6	4.6	1.1	37.3	39.4	0.2	6.7	4.6	0.9
3	193	194	2.6	8.8	6.9**	35.8	38.7	0.4	4.7	9.3	3.2
4	197	172	1.0	11.1	17.2***	29.4	34.9	1.3	3.6	2.3	0.5
5	191	194	2.1	10.8	12.1***	33.5	33.0	0.1	4.2	3.1	0.3
$\chi^2_{(4)}$ for genotype variation			1.9 13.5**			9.4 2.5			3.0 13.3**		

† The five strains were derived from a cross between ES-1374 and 699. All had the genotype $A41 B41 x15^-$ and were crossed $\times$ tester strain 14.

At 18° there was no variation in recombination frequency in any of the three regions among the strains. At 32°, recombination in both the A factor and the B factor varied from genotype to genotype. There was, however, no correlation between frequencies in the two regions; the correlation coefficient r is not significantly different from 0. Recombination in the $A\beta$ - $x15$ region did not vary; this is one indication that recombination frequencies in these two adjacent regions are independent of each other (additional proof is given by Simchen and Stamberg, 1969a). A comparison of recombination frequencies at 18 and 32° shows that, in three of the five crosses, recombination in the A factor was affected by the change in temperature while in the other two crosses there was no change in A factor recombination; but in all five crosses recombination between $A\beta$ and $x15$ was unaffected by the temperature change. Thus, temperature may or may not affect A factor recombination, depending on the genotype; but recombination in the adjacent $A\beta$ - $x15$ region is not simultaneously affected in these crosses.

Two markers, pab and $ade5$, map between the component loci of the A factor (Raper, 1966). Wild-type strain 14, and four progeny of a cross between 14 and 699, were crossed to the doubly auxotrophic strain HS-55. Each cross was tested for recombination frequencies at low (23°) and high (32°) meiotic temperatures (table 6). In each of the six crosses recombination in the pab - $ade5$ region varied at the two temperatures; in no cross was there variation in recombination in the $ade5$ - $A\beta$ region; in only one cross was there significant temperature-induced variation in the $A\alpha$ - pab region (although larger samples might well have revealed such variation in several other crosses, whose χ^2 values approached significance). Thus, temperature affects recombination primarily in the pab - $ade5$ region and sometimes between $A\alpha$ and pab as well, without affecting recombination noticeably in the $ade5$ - $A\beta$ region. From an analysis of the frequencies of recombination in the various subregions of the A factor at 32°, Simchen and Stamberg (1969a) concluded that genotype variation in recombination occurred in the $A\alpha$ - pab

TABLE 6
Effect of temperature on recombination in subregions of the A factor and in the B factor

Strain †	Sample		% Rec. <i>Aα-pab</i>		$\chi^2_{(1)}$ for temperature variation	% Rec. <i>pab-ade5</i>		$\chi^2_{(1)}$ for temperature variation	% Rec. <i>ade5-Aβ</i>		$\chi^2_{(1)}$ for temperature variation	% Rec. <i>B</i>		$\chi^2_{(1)}$ for temperature variation
	18°	32°	18°	32°		18°	32°		18°	32°		18°	32°	
Parent 14	433	379	0.2	2.1	4.9*	2.8	10.5	20.4***	1.4	2.6	1.6	7.2	2.4	9.9**
<i>Progeny of 14 × 699</i>														
1	187	120	0.0	0.8	1.6	3.2	9.2	5.0*	2.1	6.7	2.9	4.8	9.2	2.3
2	308	184	0.3	2.7	3.7	4.2	28.2	58.1***	1.6	4.4	3.3	5.2	4.4	0.2
3	162	216	1.2	5.6	3.7	6.2	15.3	7.6**	0.6	3.2	3.1	3.7	5.6	0.7
4	180	169	0.0	3.0	3.5	5.0	14.2	8.6**	1.1	3.0	1.5	2.8	1.8	0.4
5	201	187	1.0	4.3	3.0	1.0	8.6	12.5***	2.0	4.8	1.7	10.5	6.4	1.9

† Parent 14 and the five progeny were prototrophs with mating type A4 B4. All were crossed to strain HS-55, of genotype *Aα1-pab-ade5-β6 B41*.

and *pab-ade5* regions, and that such variation was not due to a common gene-control. They found no evidence for genetic control of recombination in the *ade5-A β* region. The pattern of temperature variation displayed here supports these conclusions.

The *comp2* marker is located near the *A α* locus (Simchen and Stamberg, 1969a). Five crosses between sibling strains C40 and C41, both of genotype *A43 B43 comp2 pab⁻*, and three wild-type strains were tested for recombination frequencies at low and high meiotic temperatures (table 7). Of the five crosses, one varied in recombination at the two temperatures in the three linked regions, three varied in recombination in the *comp2-A α* and *pab-A β* regions but had no significant change in recombination in the *A α -pab* region; and one cross varied only in the *pab-A β* region. In other words, the *pab-A β* region can vary in recombination from temperature to temperature while recombination in the other two regions is constant; it can vary along with the *comp2-A α* region; or all three regions can vary. In addition, the three linked regions do not exhibit genotypic variation at the same temperature: at 32° only the *A α -pab* region varies, whereas at 18° both the *comp2-A α* and *pab-A β* regions do so. Variation with respect to temperature (as well as to genotype, as shown by Simchen and Stamberg, 1969a), then, is independent in the three regions.

4. DISCUSSION

In previous publications (Simchen, 1967; Simchen and Connolly, 1968; Stamberg, 1968; Simchen and Stamberg, 1969a) we have shown that recombination in *Schizophyllum* is under genetic control, and that recombination frequencies in adjacent chromosomal segments, as well as in unlinked segments, are independent of each other. The genes which individually affect recombination in specific, short segments, but which collectively control recombination throughout the genome have been termed the "fine control" of recombination (Stamberg, 1968; Simchen and Stamberg, 1969b). The data presented in the preceding sections support our conclusion that the genes comprising the fine control are highly region-specific. A change in temperature generally does not affect frequencies of recombination in all marked segments of the genome. In our experiments involving three unlinked regions (table 3) we found a temperature effect in none, one, or two of the regions, depending on the genotypes of the strains. Similarly, adjacent regions were not always co-ordinately affected by a change in temperature (e.g., cross 1, table 4; crosses 3, 4, 5, table 5; all crosses, table 6; crosses 1, 2, 3, table 7). Furthermore, the temperature-response curves need not be identical for recombination in any two regions. For the two regions investigated at a range of temperatures, it was found that in one region recombination was constant until some point between 30 and 32°, whereas in the other region the frequency of recombination changed between 28 and 30°. The direction of change is also different in different regions; in some crosses, one region increased in recombination frequency, one region decreased, and one region was constant in response to an increase in temperature (e.g., cross 2, table 3; the parental cross, table 6).

It is our belief that the complexity of the effect of temperature on recombination is a direct reflection of the complexity of the fine control of recombination. Gene products made by different loci comprising the fine control may well have different sensitivities to temperature; this would

TABLE 7
Effect of temperature on recombination in the comp-2 -Aa region, the A factor, and the B factor

Cross	Sample		% Rec. comp-2-Aa		$\chi^2_{(1)}$ for temperature variation		% Rec. Aa-pab		$\chi^2_{(1)}$ for temperature variation		% Rec. pab-A β		$\chi^2_{(1)}$ for temperature variation		% Rec. B		$\chi^2_{(1)}$ for temperature variation	
	18°	32°	18°	32°	18°	32°	18°	32°	18°	32°	18°	32°	18°	32°	18°	32°	18°	32°
1 C40 $\times$ 699	177	117	20.3	19.7	0.0	0.9	4.0	0.9	1.5	18.0	7.9	18.0	6.8**	0.0	1.7	0.0	2.0	2.0
2 C41 $\times$ 699	148	168	18.2	31.6	7.4**	1.2	2.0	1.2	0.4	17.3	8.1	17.3	5.8*	2.4	2.7	2.4	0.0	0.0
3 C40 $\times$ 14	181	190	2.2	19.5	28.1***	2.1	0.0	2.1	2.1	13.2	0.6	13.2	22.6***	3.2	5.5	3.2	1.3	1.3
4 C41 $\times$ 14	175	201	7.4	21.9	15.2***	7.0	1.1	7.0	6.4*	11.0	3.4	11.0	7.7**	3.5	1.1	3.5	2.2	2.2
5 C41 $\times$ 991	118	126	11.0	21.4	4.8*	6.4	0.9	6.4	3.8	19.1	2.5	19.1	16.9***	5.6	3.4	5.6	0.7	0.7
$\chi^2_{(4)}$ for genotype variation	38.0***		9.3		15.8**		17.3*		6.9		7.4		6.9					

result in different effects of temperature on recombination in various regions within one genome. Likewise, gene products made by alternate alleles at one fine-control locus may also have different temperature sensitivities, resulting in different effects of temperature on recombination within one region in various crosses. Both kinds of variation have been found in *Schizophyllum*, and in other organisms as well (see Introduction).

Thus, the still widely-held beliefs (in spite of the accumulating evidence to the contrary) that effects of temperature are confined to regions near the centromeres, or are constant throughout the genome, or that temperature-response curves are always U-shaped, are erroneous. It is a serious oversimplification to generalise from the effect of temperature on recombination in one region, or in one cross, to the effect of temperature on recombination in other regions, or in other crosses. Although this complexity may be a nuisance to geneticists, we believe it has a great adaptive advantage to the organisms in which it occurs: new environmental conditions, such as changes in temperature, can release genetic variability at different rates for different regions of the genome, thus allowing each region to produce that amount of variation that will be optimal for the species.

5. SUMMARY

1. The effect of temperature on recombination in adjacent and non-adjacent chromosomal regions has been studied in *Schizophyllum commune*.

2. A change in temperature need not affect frequencies of recombination throughout the genome; neither linked nor unlinked segments always respond co-ordinately to temperature.

3. The effect of temperature on recombination in any particular region can vary from cross to cross.

4. The effect of temperature on recombination is thus both region-specific and genotype-specific. We believe this specificity is a manifestation of the complex "fine control" of recombination and has adaptive significance.

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