3. The instability was due partly to a mosaic constitution in L_2, partly to incomplete differentiation between L_2 and L_3 such that the former sometimes displaced the latter.

4. Crossing experiments showed that defective plastids were maternally inherited but with, probably, a very low level of male transmission.

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GENETIC CONTROL OF RECOMBINATION IN *SCHIZOPHYLLUM COMMUNE*: SEPARATION OF THE CONTROLLED AND CONTROLLING LOCI

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Received 18.x.68

1. Introduction

MATING competence in the basidiomycetous fungus *Schizophyllum commune* is controlled by two unlinked factors, *A*, and *B*, each of which consists of two closely linked loci, α and β. Frequencies of recombination between the loci of each factor vary widely in crosses of wild-type strains (Raper, Baxter and Ellingboe, 1960; Koltin, Raper and Simchen, 1967). A series of studies has been undertaken to determine the basis for this variation. Stamberg (1968) ruled out structural causes—*e.g.* variations in physical distance between the loci—as the main source of variation in recombination frequencies in both the *A* and *B* factors, and concluded that the variation could best be explained by the presence of two independent gene systems, each of which regulated the frequency of recombination within one of the

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incompatibility factors. Direct proof that one such system exists was obtained by Simchen (1967), who selected strains for high and low frequencies of recombination in the *A* factor, and showed that the tendency for high or low recombination was separable from the *A* factor itself.

The experiment described here provides the first direct proof that the recombination frequency in the *B* factor, as well as in the *A* factor, is genetically controlled, by showing that the tendency for high or low recombination in the *B* factor is separable from the *B* factor itself.

2. Materials and methods

Strains 991 and 699 were obtained from the worldwide collection of *S. commune* assembled by Professor J. R. Raper of Harvard University. Strain E908, also obtained from Professor Raper, had previously been derived from a back-crossing programme to strain 699.

Procedures for performing crosses, isolation of spores and test matings have been described (Stamberg, 1968). Cultures were maintained at 18° C. for at least 48 hours prior to the collection of spores.

3. Results and discussion

The rationale for the experiment was the following: if there are three strains such that strain 1 × strain 2 gives high recombination for any region, *R*, and strain 3 × strain 2 gives low recombination for the same region, *R*, then—if the recombination frequency in *R* is genetically controlled—it should be possible to obtain, from a cross between strains 1 and 3, progeny carrying the *R* region of parent 1 associated with the *R*-control system of parent 3, and *vice versa*. When such progeny are crossed to the tester strain 2, the result should be the same low recombination for region *R* as given by parent 3 × tester strain 2.

The desired relationship was found in the strains 991 and E908, when 699 was used as the tester strain. 991, of mating type *A97 B97*, × 699 (*A41 B41*) gives low recombination in the *A* factor and high recombination in the *B* factor, whereas E908 (*A43 B43*) × 699 gives high recombination in the *A* factor and low recombination in the *B* factor (table 1). If recombination frequencies are regulated by elements external to the regions under control, it should be possible to find strains among the progeny of 991 × E908 that carry the *A* factor of 991 but which give high recombination in the *A* factor when crossed to 699; similarly, there should be strains among the progeny that carry the *A* factor of E908 and the control system of 991 and thus give low recombination in the *A* factor when crossed to 699. Similar expectations apply to the *B* factor. Thus, if any *A97 B97* strains among the progeny give high recombination in the *A* factor or low recombination in the *B* factor, or if any *A43 B43* strains among the progeny give low recombination in the *A* factor or high recombination in the *B* factor, when tested with 699, the controlled and controlling regions must be separable.

Seven of the progeny of 991 × E908 were tested for recombination frequencies in the *A* and *B* factors when crossed to 699 (table 1). The frequencies obtained were compared, by χ^2 tests, to both parental frequencies. In six of the seven crosses the frequency of *B* factor recombination was equivalent to that given by the parent of the same mating type. In one cross (No. 2), however, the frequency of recombination was equivalent to

that of the parent of opposite mating type. The strain involved in this cross must have received the *B* factor of parent 991 and the system controlling recombination of parent E908. That the recombination frequencies in the *B* factor of the progeny divided into two categories, high and low, equivalent to the parental frequencies, indicates the segregation of one gene (or one tightly linked group of genes), *i.e.* that the parental strains 991 and E908 differ by one allele in the system controlling recombination in the *B* factor.

TABLE 1

Segregation of recombination-controlled and recombination-controlling regions

Cross†	Sample	% Rec. *A*	$\chi^2_{(1)}$ for deviation from 4·5%	$\chi^2_{(1)}$ for deviation from 13·8%	% Rec. *B*	$\chi^2_{(1)}$ for deviation from 7·6%	$\chi^2_{(1)}$ for deviation from 2·2%
Parents							
991 (*A97 B97*)	157	4·5	—	8·6**	7·6	—	5·5*
E908 (*A43 B43*)	181	13·8	8·6**	—	2·2	5·5*	—
Progeny of 991 × E908							
1. (*A97 B97*)	127	0·8	3·5	16·4***	11·8	1·4	11·9***
2. (*A97 B97*)	122	7·4	1·1	3·0	0·8	7·2**	0·9
3. (*A97 B97*)	128	1·6	1·9	14·1***	9·4	0·0	7·8**
4. (*A97 B97*)	180	3·9	0·1	11·0***	10·6	0·9	10·5**
5. (*A43 B43*)	174	16·7	12·7***	0·6	2·9	3·9*	0·2
6. (*A43 B43*)	180	9·4	3·2	1·7	2·8	4·1*	0·1
7. (*A43 B43*)	200	5·5	0·2	7·7**	2·0	6·5*	0·0

† The common mate in all crosses was strain 699 (*A41 B41*).

Significance levels indicated as:

unstarred: P greater than 0·05
*: P = 0·05-0·01
**: P = 0·01-0·001
***: P less than 0·001

The situation with respect to the *A* factor is more complex, as the progeny fell into more than two groups on the basis of recombination frequencies in this region. In four crosses (Nos. 1, 3, 4, and 5) the frequency of *A* factor recombination was equivalent to that of the parent of the same mating type; in two crosses (Nos. 2 and 6) the frequency obtained was intermediate between the parental values, and did not differ significantly from either value; in one cross (No. 7) the frequency of recombination was equivalent to that of the parent of opposite mating type—*i.e.* the strain involved in this cross had the *A* factor of E908 associated with the *A* factor's control system of 991. That intermediate values were obtained suggests that two control genes were segregating; 991 and E908 must differ by at least two alleles in the system controlling recombination in the *A* factor.

It has already been shown in several ways that recombination frequencies in the *A* factor and in the *B* factor are independent of each other (Stamberg, 1968). The present data provide additional evidence that this is so: the factors that control recombination in the *A* factor and in the *B* factor segregated independently. One of the progeny tested, for example, carried the *A* factor control system of 991 and the *B* factor control system of E908 (No. 7).

Thus, the elements responsible for variation in recombination frequencies segregate like genes and are indeed separable from the regions whose recombination they affect. We conclude that the elements are genes or gene systems.

4. Summary

1. Frequencies of recombination between the component loci of the *A* and *B* incompatibility factors were determined for two monokaryotic strains and seven of their progeny after crossing to a common tester strain.

2. The tendencies for high and low recombination within each incompatibility factor are separable from the factors themselves and segregate like genes.

3. The elements controlling recombination within the *A* and *B* factors segregate independently.

Acknowledgments.—Thanks are due to Professor J. L. Jinks and Dr G. Simchen for generously providing laboratory facilities and for reading the manuscript, and to Miss J. Hay for excellent technical assistance. The study was supported by a Public Health Service fellowship from the U.S. National Institutes of Health. Financial support during the preparation of the manuscript was kindly made available by Professor J. R. Raper from N.I.H. grant AI06124.

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AN EXCESS OF L^ML^N HETEROZYGOTES IN A SOUTH EUROPEAN POPULATION

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Comparing theoretical and observed MN blood groups frequencies among the offspring of various parental combinations, Wiener (1943) noted that there was a significant excess of MN children in the mating MN × MN. He concluded that it was probably due to illegitimacies or errors in technique. Wiener gave the same explanation for the excess of L^ML^N heterozygotes which was found by Lattes and Garrasi (1932) in a sample of Italian population. It seems unlikely that such an explanation is correct for every case of overabundance of L^ML^N heterozygotes.

The present investigation is based on the records of the medico-legal analyses concerning 616 cases of disputed paternity examined by the courts