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Genetics

Meiosis in haploid yeast

(sporulation/chromosome segregation/recombination)

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ABSTRACT **Haploid yeast cells normally contain either the *MATa* or *MATα* mating-type allele and cannot undergo meiosis and spore formation. If both mating-type alleles are present as a consequence of chromosome III disomy (*MATa/MATα*), haploids initiate meiosis but do not successfully form spores, probably because the haploid chromosome complement is irregularly partitioned during meiotic nuclear division. We have demonstrated that the ochre-suppressible mutation *spo13-1* enables haploid yeast cells disomic for chromosome III and heterozygous at the mating-type locus to complete meiosis and spore formation, yielding two haploid spores. Previous studies have shown that the absence of the wild-type *SPO13* gene function permits diploid cells to bypass homologous chromosome segregation at meiosis I and proceed directly to meiosis II. During *spo13-1* haploid meiosis, cells enter prophase of meiosis I. Genetic recombination, monitored on the chromosome III disome, occurs at levels similar to those seen in diploids, indicating that the level of exchange between homologs is an autonomous property of individual chromosomes and not dependent on exchange elsewhere in the genome. Exchange is then followed by a single meiosis II equational chromosome division. Recombination in *spo13-1* haploids is blocked by the *spo11-1* mutation, which also eliminates recombination between homologous chromosomes during conventional diploid meiosis. We conclude that Spo$^+$ haploids expressing both a and α mating-type information attempt a *SPO13*-dependent meiosis I division, and that this division, in the absence of paired homologous chromosomes, is responsible for the failure of such haploids to complete normal gametogenesis. Our observations support the conclusion that initiation and completion of meiosis II and spore formation are not dependent on either completion of meiosis I or the presence of a diploid chromosome complement.**

Diploid cells of the yeast *Saccharomyces cerevisiae* respond to glucose and nitrogen deprivation by undergoing a complex series of genetic and biochemical processes collectively referred to as sporulation (for review, see ref. 1). These processes include premeiotic DNA synthesis, commitment to high levels of genetic recombination, meiotic chromosome segregation, and formation of haploid ascospores. In most genetic backgrounds, sporulation requires heterozygosity at the mating-type locus on chromosome III (*MATa/MATα*). Neither diploids homozygous at the mating-type locus (*MATa/MATa* or *MATα/MATα*) nor haploids (*MATa* or *MATα*) are capable of completing even the earliest of the above landmark events, premeiotic DNA synthesis; subsequent events are also blocked.

The failure of haploids to complete meiosis, however, is not due solely to mating-type regulation of sporulation. Roth and Fogel (2) demonstrated that, although haploids disomic for chromosome III and heterozygous at *MAT* undergo premeiotic DNA synthesis and commitment to intragenic recombination at *leu2*, they nevertheless do not form mature asci. They suggested that nuclear division during this abortive meiosis produces grossly aneuploid genomes that cannot be packaged into ascospores.

We have used two meiotic mutations, *spo12-1* and *spo13-1*, to test the hypothesis that haploids expressing both mating-type alleles fail to complete meiosis and spore formation because they execute a meiosis I-like reductional division. During this division, unpaired undivided chromosomes might simply move randomly to the poles of the meiosis I spindle, forming highly aneuploid products; alternatively, nonhomologous chromosomes might pair and disjoin from one another. The *spo12-1* and *spo13-1* mutations cause *MATa/MATα* diploids to undergo a single predominantly meiosis II-like equational division and form asci containing two diploid spores (3, 4). If *MATa/MATα* disomic haploids are sporulation-defective as a result of attempted reductional division, then mutations, such as *spo12-1* and *spo13-1*, that cause cells to bypass the reductional division and proceed directly to a single equational division should permit these haploids to form asci containing two viable spores.

MATERIALS AND METHODS

Strains. Sources of markers used in this study are as follows: A. Klar (Cold Spring Harbor Laboratory) provided a *mar1* haploid (K31); J. Haber (Brandeis University) provided a haploid strain marked with *his4*, *leu2*, *cry1*, and *thr4* (A418); and A. Hopper (Hershey Medical Center) provided a diploid homozygous for *csp1* (A169-αα). J. Strathern (Cold Spring Harbor Laboratory) provided a haploid strain disomic for chromosome III (DI3). Other strains used originated in this laboratory.

Chromosome III genotypes of strains used for recombination analysis in this work are shown below. Map distances (5) are given in parentheses.

$$\text{K284-10A: } \frac{\text{leu2 (5) } \circ \text{ (25) MATa}}{+ \qquad\qquad \text{MAT}\alpha}$$

$$\text{K355-13A: } \frac{+ \text{ (5) } \circ \text{ (25) MATa}}{\text{leu2} \qquad\qquad \text{MAT}\alpha}$$

$$\text{JW72-13B: } \frac{\text{his4 (17) leu2 (5) } \circ \text{ (21) cry1 (4) MAT}\alpha\text{ (22) thr4}}{+ \quad \text{leu2} \qquad + \quad \text{MATa} \quad +}$$

Relevant genotypes of other strains used are given in the text.

Media and Genetic Procedures. Growth and sporulation media have been described (6). Standard methods for crosses and other genetic procedures were used (7).

Photography of Sporulated Cells. Cells were grown to a density of $\approx 2 \times 10^7$ ml in liquid YPA-22 at 25°C, harvested, washed, and suspended in SPII-22 at a density of 4×10^7/ml. After vigorous shaking at 25°C for 72 hr in SPII-22, samples of the sporulated cultures were fixed in 70% ethanol for 10 min. The cells were then washed with deionized H_2O, stained with 4′,6-diamidino-2-phenylindole (0.25 μg/ml; Boehringer Mannheim) (8) for 10 min, and washed again with H_2O. They were stored in 50% glycerol and photographed with fluorescence and phase-contrast optics using a Leitz Orthoplan microscope.

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Table 1. Sporulation and spore viability of haploid strains

Genotype	Strain	Ploidy	Mature asci,%	Spore viability, %
SPO	K341-21B	*n* + 1	<0.5	—
	K341-8C	*n* + 1	<0.5	—
spo11-1	K341-8D	*n* + 1	<0.5	—
spo12-1	K285-5C	*n* + 1	0.5	43 (6/14)
	K285-7C	*n* + 1	1	—
spo13-1	K284-10A	*n* + 1	39	75 (60/80)
	JW72-2B	*n* + 1	24	59 (127/216)
spo13-1 mar1	K343-2B	*n*	28	40 (16/40)
	K343-3B	*n*	31	57 (24/42)
spo11-1 spo12-1	K341-15C	*n* + 1	<0.5	—
spo11-1 spo13-1	K286-2A	*n* + 1	32	83 (30/36)
	K286-4A	*n* + 1	40	97 (58/60)*
spo12-1 spo13-1	K341-22B	*n* + 1	21	—
spo11-1 spo12-1 spo13-1	K341-6C	*n* + 1	26	89 (34/38)
	K341-1C	*n* + 1	60	—

Sporulation was scored after 5 days on SPIII-22 plates at 25°C; results are means of two independent counts of at least 200 cells. Results in parentheses are viable spores/total spores. All *n* + 1 haploids are *MATa/MATα*. Diploid control values: *SPO/SPO* (K65-3D), 74% mature asci, 98% spore viability (39/40); *spo13-1/spo13-1* (K292), 67% mature asci, 52% spore viability (120/230).
* Data are from cells sporulated at 30°C.

RESULTS

The *spo13-1* Mutation Permits Haploids to Sporulate. MATa/MATα disomic haploids of *SPO*, *spo12-1*, or *spo13-1* genotype were constructed and examined for sporulation efficiency and spore viability at 25°C (Table 1). As expected from previous studies (2), *SPO* disomic haploids exhibited negligible sporulation (<0.5%). Disomic haploids containing *spo12-1* also sporulated to only a very limited extent (0.5–1%). The presence of the *spo13-1* mutation, however, permitted 20–40% sporulation in MATa/MATα disomic haploids. Diploids homozygous for either *spo12-1* or *spo13-1*, by comparison, exhibit ≈75% sporulation (3). Double mutant *spo12-1 spo13-1* haploids showed sporulation efficiencies similar to *spo13-1* haploids, indicating that the *spo13-1* mutation is epistatic to *spo12-1* for the haploid sporulation phenotype.

The *spo13-1* mutation permitted efficient sporulation not only of chromosome III disomic haploids but also of haploids monosomic for chromosome III but mutant at the *mar1* locus (9). The *mar1* mutation results in expression of mating-type information carried in the normally silent mating-type loci, *HML* and *HMR*. It thereby renders *MATa* and *MATα* haploids incapable of mating and *MAT* homozygous diploids capable of sporulation. Another mutation, *csp1*, which permits *MATa/MATa* and *MATα/MATα* diploids to sporulate but does not affect mating behavior (10), did not allow appreciable levels of sporulation in *spo13-1 MATa* or *MATα* haploids (data not shown). This observation may merely be an artifact of the low efficiency of sporulation of strains homozygous for *csp1* and *MAT*, or it may reflect an actual dependence of haploid sporulation on the wild-type *CSP1* gene product.

Table 3. Nuclear division in *SPO13* and *spo13-1* haploids

Strain	Genotype	Frequency, % Mononucleate	Binucleate	Tri-/tetranucleate
JW72-3C	*SPO13*	87	6	7
JW72-20D	*SPO13*	70	15	14
JW72-2B	*spo13-1*	62	36	1
JW72-2C	*spo13-1*	62	36	1

4',6-Diamidino-2-phenylindole-stained cells (200–400 of each strain) were scored after 72 hr of sporulation.

Spore Death is Nonrandom in *spo13-1* Haploids. *MATa/MATα spo13-1* haploids, like *MATa/MATα spo13-1/spo13-1* diploids, exhibited 50–70% spore viability. However, spore death, which occurred at random in the diploids, was highly nonrandom in the haploids (Table 2). Dyads (two-spored asci) in which either both spores survived or neither spore survived occurred significantly more frequently in *spo13-1* disomic haploids than would be expected from random spore death, suggesting that much of the spore inviability in *spo13-1* haploid meiosis results from genetic events that have lethal effects on *both* spore products of a given meiosis (see *Discussion*).

***spo13-1* Haploids Undergo a Single Meiotic Nuclear Division.** Photographs of sporulated cells of a *MATa/MATα SPO13* haploid, a *MATa/MATα spo13-1* haploid, and a *MATa/MATα SPO13/SPO13* diploid are shown in Fig. 1. The photographs illustrate that (*i*) *SPO13* haploids fail to form mature asci, although they do form immature "incipient" asci, as noted by Roth and Fogel (2); (*ii*) *SPO13* haploids frequently undergo two nuclear divisions in sporulation medium to give tri- or tetranucleate cells; and (*iii*) tri- and tetranucleate cells (and therefore one of the meiotic nuclear divisions) are almost completely eliminated by *spo13-1*. Frequencies of mononucleate, binucleate, and tri- or tetranucleate cells in *SPO13* and *spo13-1* haploids are summarized in Table 3.

Genetic Exchange Occurs at Diploid Levels During Haploid Meiosis. During meiosis in diploid yeast, at least 16 pairs of homologous chromosomes pair and recombine (6). Analysis of genetic exchange on chromosome III in the *spo13-1* haploid meiosis system, in which only one pair of homologs is available for recombination, is complicated by the fact that at least three types of chromosome segregation are possible. These three types of segregation, all of which have been observed in *spo13-1/spo13-1* diploid meiosis (4), are *reductional* (as in meiosis I), *equational* (as in meiosis II), and *aberrant* (yielding one monosomic spore and one trisomic spore). For a heterozygous cen-

Table 2. Spore viability patterns of *spo13-1* haploids and diploids

Strain	Ploidy	Dyad type (viable/inviable spores, no.) 2:0 Obs.	2:0 Exp.	1:1 Obs.	1:1 Exp.	0:2 Obs.	0:2 Exp.	χ^2
K215	2*n*	60	58	40	45	11	9	1.07 ($0.5 < P < 0.7$)
K292	2*n*	31	31	58	57	26	26	0.02 ($P > 0.99$)
K355-13A	*n* + 1	129	111	50	86	35	17	37.0 ($P < 0.01$)
JW72-13B	*n* + 1	136	109	122	178	102	73	35.8 ($P < 0.01$)

Expected dyad frequencies were calculated assuming the observed level of spore viability and random spore death. Cells were sporulated on SPIII-22 plates at 25°C, except for K215, which was sporulated at 30°C (4). Obs., observed; Exp., expected.

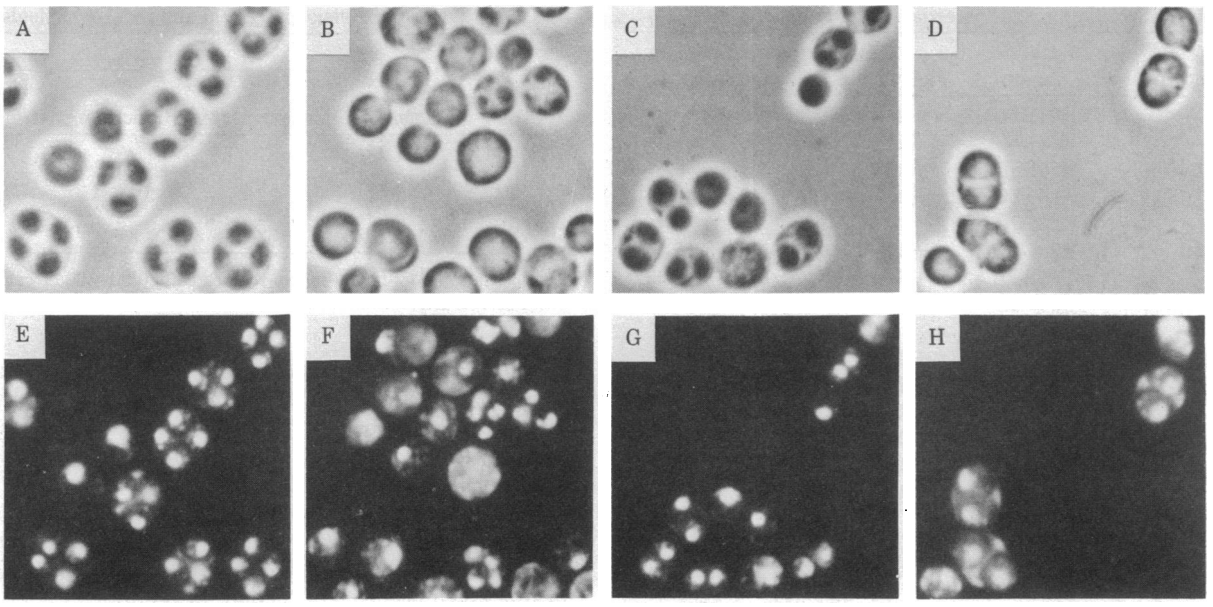

FIG. 1. Photographs of 4',6-diamidino-2-phenylindole-stained sporulated cells. (*Upper*) Phase-contrast images. (*Lower*) Fluorescence images. (*A* and *B*) K65-3D, 2*n SPO13*; (*C* and *D*) JW72-20D, *n* + 1 *SPO13*; (*E* and *F*) JW72-2B, *n* + 1 *spo13-1*; (*G* and *H*) K286-2A, *n* + 1 *spo11-1 spo13-1*. (×1450.)

tromere marker (*M/m*), in the absence of gene–centromere exchange, reductional division (followed by centromere division without cell division) should yield two homozygous spores (*M/M* and *m/m*), equational division should yield two heterozygous spores (*M/m*), and aberrant division should yield either one *M* spore and one *M/m/m* spore, or one *m* spore and one *M/M/m* spore.

Aberrant division for chromosome III, signaled by dyads containing one mating spore (*MAT***a** or *MAT*α) and one nonmating spore (*MAT***a**/*MAT*α/*MAT*α or *MAT***a**/*MAT***a**/*MAT*α), occurs in 15–20% of *spo13-1/spo13-1* diploid meioses (4). For the three *spo13-1* haploids examined in detail in this study, 10–30% of dyads showed aberrant segregation for *MAT* (Table 4); in each of five aberrant dyads from haploid K355-13A examined, the mating-capable spore clone was shown to be monosomic for chromosome III as predicted, and mating-phenotype segregation after sporulation of the nonmating spore clone was consistent with the particular trisomy expected. In general, crosses of mating-capable spore clones from both aberrant and nonaberrant dyads to haploid testers gave ≥90% spore viability on sporulation and dissection, indicating that the spore clones were haploids lacking major chromosome rearrangements. Mating-capable spore clones from dyads containing one **a**-mating spore and one α-mating spore, when crossed to haploid testers, showed mating-phenotype segregation indicative of chromosome III disomy in the spore clones.

Among nonaberrant dyads, the frequency of equational chromosome III division versus reductional division can be inferred from the segregation patterns of heterozygous centromere-linked markers. As shown in Fig. 2, genetic recombination followed by equational division leads to a positive correlation between gene–centromere distance and frequency of +,− segregation (i.e., one spore phenotypically +, one spore phenotypically −) for a given marker; genetic recombination followed by reductional division, on the other hand, leads to a negative correlation. Assuming exchange frequencies similar to those observed in diploid meiosis, our data are inconsistent with either 100% equational or 100% reductional segregation. They are quite consistent, however, with ≈40% reductional segregation for chromosome III among nonaberrant dyads.

Two lines of evidence suggest that the frequency of intergenic exchange during haploid meiosis is, in fact, not significantly different from that in diploids. First, monosomic spores from aberrant dyads showed coupling changes for linked markers at frequencies close to those observed in random products of diploid meiosis (Table 5). Second, linked markers in nonaberrant dyads showed coupling changes as frequently as would be expected from diploid exchange frequencies and the 40% reductional division inferred from segregation of individual heterozygous markers (Table 6).

***spo11-1* Eliminates Genetic Exchange During Haploid Meiosis.** The *spo11-1* mutation reduces the frequency of both gene conversion and crossing-over between homologous chromosomes to <1% of wild-type levels during diploid meiosis (ref. 6; unpublished results). This mutation had striking effects on *spo13-1* haploid meiosis that closely resembled its effects on diploids homozygous for *spo13-1* (11). Spore viability increased from ≈60% to ≥90% in the presence of *spo11-1*, and the dyads produced (Fig. 1) showed no evidence of either gene–centromere exchange or reductional division, either of which should produce the **a**,α segregation pattern for mating phenotype (Table

Table 4. Segregation of mating phenotypes during haploid meiosis

Strain	Dyads, no.	Mating-phenotype segregation, no.				Aberrant, %
		N,N	**a**,α	**a**,*N*	α,*N*	
spo13-1						
K284-10A	70	48	14	4	4	11
K355-13A	118	58	27	16	17	28
JW72-13B	101	46	25	6	24	30
spo11-1 spo13-1						
K286-4A	34	33	0	0	1	3
spo11-1 spo12-1 spo13-1						
K355-9C	124	123	0	0	1	1
K355-24C	128	125	0	0	3	2

Dyad types: *N,N*, two nonmating spores; **a**,α, one **a**-mating and one α-mating spore; **a**,*N* or α,*N*, one **a**- or α-mating and one nonmating spore. Aberrant, dyads showing **a**,*N* or α,*N* segregation.

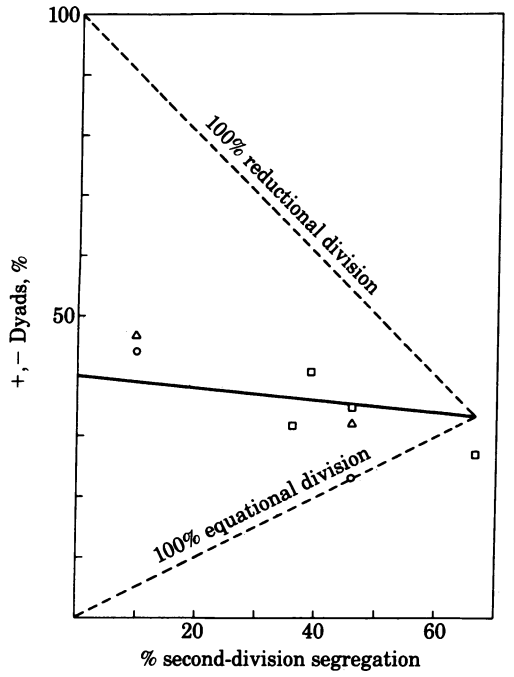

FIG. 2. Marker segregation in nonaberrant dyads from haploid meiosis. Abscissa, second-division segregation (SDS) frequencies during diploid meiosis (5) (markers used and their SDS frequencies are *leu2*, 10%; *his4*, 36%; *cry1*, 39%; *MAT*, 46%; *thr4*, 67%); ordinate, percent of nonaberrant dyads showing +,− segregation (i.e., one spore phenotypically +, one spore phenotypically −) for a marker. (For *MAT*, the equivalent of +,− segregation is **a**, α; for *cry1*, the equivalent is resistant, sensitive.) In general, $y = r(1 - x) + (1 - r)(0.5x)$, where x is SDS frequency during diploid meiosis, r is the fraction of divisions that are reductional, and y is the predicted value of +,− dyads/total dyads, assuming that crossover frequencies are equal in haploid and diploid meiosis. —, Predicted values for 100% reductional division ($y = 1 - x$) and 100% equational division ($y = 0.5x$); ——, predicted values for 40% reductional division, 60% equational division ($y = 0.4 - 0.1x$). Data are from the following *MATa*/*MAT*α *spo13-1* haploids: □, JW72-13B (71 dyads); ○, K284-10A (62 dyads); △, K355-13A (85 dyads).

4). No evidence for exchange between *leu2* and *MAT* was observed in 28 dyads of strains K286-2A and K341-6C, each heterozygous at both loci. In addition, *spo11-1* reduced the frequency of aberrant segregation of chromosome III by ≈90%. The identical sporulation phenotypes of *spo11-1 spo13-1* haploids and *spo11-1 spo12-1 spo13-1* haploids (Table 1) again indicate that *spo13-1* is epistatic to *spo12-1*.

DISCUSSION

The *spo13-1* mutation permits haploid yeast strains functionally heterozygous for mating type, by virtue of either disomy for chromosome III or expression of the silent copies of mating-type information in the presence of the *mar1* mutation, to complete meiosis and spore formation. The ability of *spo13-1* to overcome the sporulation defect of haploids is most easily understood as arising from its elimination of a meiosis I-like nuclear division. Autonomous reductional division of a single chromosome pair, however, is apparently not eliminated under these conditions. The phenotypes of *spo13-1* haploids and diploids indicate that (*i*) the wild-type *SPO13* gene product must be responsible for either promoting a meiosis I reductional division or preventing initiation of meiosis II directly after prophase of meiosis I and (*ii*) initiation of meiosis II is not obligatorily dependent on completion of meiosis I or on diploidy *per se*.

Spore viability in *spo13-1* haploids, as in *spo13-1/spo13-1* diploids, is appreciably lower than that in wild-type diploids. Inviability of the two spores in a dyad is highly correlated in *spo13-1* haploids whereas in *spo13-1/spo13-1* diploids spore death is nearly random. In both cases, this excess spore inviability is eliminated by the meiotic Rec⁻ mutation *spo11-1*. We propose that spore inviability in both *spo13-1* haploids and diploids is largely due to production of nullosomic or otherwise aneuploid genomes during meiosis and that the difference in observed patterns of spore death in haploids versus diploids is due to a difference in the predominant mechanisms by which inviable aneuploids are generated in the two systems. In diploids, nondisjunction of individual chromosome pairs during single-division meiosis, leading to monosomic:trisomic or nullosomic:tetrasomic spore pairs, is a plausible explanation for the lack of correlation between survival of the two spores of a dyad. This follows if nullosomics are invariably inviable and other aneuploids are only sometimes inviable, depending on the particular chromosomes involved (12). In *spo13-1* haploids, on the other hand, a likely candidate for the lethal event is pairing and "pseudoreductional" division of nonhomologous chromosomes, leading to the formation of two nullosomic spores. Such nonhomologous pairing during meiosis has been observed in a number of haploid plant varieties (13, 14). This proposal would explain the observation of high spore viability in *spo11-1 spo13-1* double mutants if the *SPO11* function is required for pairing of homologous chromosomes in diploids as well as for nonhomologous pairing in haploids. This hypothesis requires that a large portion of the meiotic nondisjunction that occurs in *spo13-1/spo13-1* diploids be dependent on chromosome pairing. An involvement of *SPO11* in chromosome pairing would, moreover, provide a

Table 5. Coupling changes (CC) between linked markers in monosomic spores from aberrant dyads

Marker pair*	Aberrant dyads, no.	Spores with CC, no.	Spores with CC, %	
			Observed	Expected†
MAT-his4	30	10	33	34
MAT-cry1	30	3	10	4
MAT-thr4	30	7	23	20
MAT-leu2	41	10	24	28

* *MAT-leu2* data are from K284-10A and K355-13A; other data are from JW72-13B.

† Calculated by using the formula $CC = (\frac{1}{2}T + N)/(P + N + T)$ with P, N, T data from Mortimer and Schild (5).

Table 6. Coupling changes (CC) between linked markers in dyads with two disomic spores

Marker pair*	Dyads, no.	Dyads with CC,† no.	Dyads with CC, %	
			Observed	Expected‡
MAT-his4	70	28	40	47 (32–70)
MAT-leu2	147	52	35	37 (26–54)

* *MAT-his4* data are from JW72-13B; *MAT-leu2* data are from K284-10A and K355-13A.

† Refers *only* to dyads in which one marker is homozygous in the two spores of a dyad and the other marker remains heterozygous (e.g., MN/Mn, mN/mn) or in which both markers are homozygous in the two spores of a dyad in coupling opposite that of the parental strain (e.g., Mn/Mn, mN/mN from a parent of genotype MN/mn).

‡ Calculated on the assumption that 40% of nonaberrant dyads are reductional. The first value given is $[0.4(N + T)/(P + N + T)] + [0.6(\frac{1}{2}T)/(P + N + T)]$; values given in parentheses are, first, $(\frac{1}{2}T)/(P + N + T)$, the predicted value for 100% equational division, and second, $(N + T)/(P + N + T)$, the predicted value for 100% reductional division. These predictions are valid *only* for markers on opposite sides of the centromere. Recombination data used to calculate predicted values are from Mortimer and Schild (5).

simple explanation of the Rec⁻ phenotype of *spo11-1* mutant strains.

Our analysis of the segregation and exchange behavior of chromosome III during haploid meiosis indicates that chromosome III division is ≈30% reductional, 50% equational, and 20% aberrant and that crossover frequencies are similar during meiosis in diploids and disomic haploids. The occurrence of normal exchange frequencies on chromosome III clearly does not depend on the occurrence of exchange between any other pairs of homologs. Furthermore, chromosome III can divide reductionally or aberrantly with high probability during the same meiotic division in which all of the other chromosomes in a nucleus must have divided equationally to yield two viable spores. This high degree of autonomy in recombination and segregation is inconsistent with models for the control of these processes at the cellular level, such as the "limiting recombination enzyme" model proposed to explain interchromosomal effects in *Drosophila* (for review, see ref. 15). Such a model would predict the occurrence of elevated levels of exchange on the single disomic chromosome III.

In addition to its Rec⁻ phenotype and its striking enhancement of spore viability in *spo13-1* haploids, *spo11-1* reduces the frequency of chromosome III reductional division to <1% per meiosis and that of aberrant division to 1% to 2%. The marked effect of *spo11-1* on reductional division supports the hypothesis, discussed above, that the wild-type *SPO11* function is essential for establishment or maintenance of homologous chromosome pairing, which is in turn a prerequisite for reductional segregation.

The *spo12-1* mutation, which has an almost identical phenotype to *spo13-1* in otherwise wild-type diploids, fails to permit haploid meiosis. A similar distinction between *spo12-1* and *spo13-1* has been demonstrated in studies of multiple mutant diploid strains bearing either *spo11-1* or another meiotic Rec⁻ mutation, *rad50-1*. Diploids homozygous for *spo13-1* and either *spo11-1* (ref. 11; unpublished results) or *rad50-1* (16) can sporulate to give dyads containing two viable recombinationless spores; diploids homozygous for *spo12-1* and either of these Rec⁻ mutations cannot. Taken together, these results support the inference that the meiotic apparatus in *spo13-1* cells can use unpaired chromosomes, whether they result from haploidy or from lack of meiotic recombination between homologs in diploid cells. By contrast, *spo12-1* cells must require paired chromosomes to complete meiosis. The epistasis of *spo13-1* to *spo12-1* in both haploid meiosis and Rec⁻ diploid meiosis suggests that the *SPO13* gene product functions before the *SPO12* gene product in both haploids and diploids (1). Studies to determine the stage at which *spo12-1* haploids and Rec⁻ *spo12-1/spo12-1* diploids are blocked in meiosis should help to clarify the roles of the *SPO12* and *SPO13* gene products in normal meiosis.

The data presented here show that the *SPO13* gene product is responsible for the inability of meiotic products of wild-type haploid nuclei expressing both mating types to be packaged into mature viable ascospores. Paradoxically, however, a *SPO13* haploid nucleus present in a heterokaryon with a *SPO13* diploid nucleus of opposite mating type often undergoes a single equational meiosis II division like that observed in *spo13-1* haploids and forms haploid spores (17). The basis of this *spo13-1* "phenocopy" effect is not yet understood.

Preliminary studies indicate that the ability of mutations such as *spo13-1* to permit haploids to complete meiosis and spore formation will be of value in analysis of the genetic control of sporulation and meiotic recombination in several ways. First, the haploid meiosis system offers a straightforward approach to the isolation of recessive mutations defective in the hitherto "diploid-specific" functions of sporulation and meiotic recombination. Second, the system promises to simplify the analysis of meiotic sister-chromatid exchange (18) and meiotic intrachromosomal gene conversion (19) and to aid in the elucidation both of their genetic control and of the influence of homologous chromosome pairing on these processes. Third, the system should facilitate the isolation of recessive mutations that cause expression of the silent copies of mating-type information (like *mar1*) or remove sporulation from mating-type control. Finally, the haploid meiotic system may also provide a convenient way to isolate translocations following enhanced levels of recombination between univalent nonhomologous chromosomes.

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