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Genetics

RED1: A yeast gene required for the segregation of chromosomes during the reductional division of meiosis

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ABSTRACT **A mutation at the *RED1* locus was identified in a search for sporulation-proficient, meiotic-lethal yeast mutants. The few viable spores produced in the *red1-1* mutant are highly aneuploid, suggesting that the spore lethality results from a high frequency of chromosome nondisjunction. Disomic spores produced by the *red1-1* mutant contain nonsister chromatids and the *red1-1* spore inviability phenotype is alleviated in *red1-1 spo13* double mutants; these results indicate that nondisjunction occurs at the first meiotic division. The *red1-1* mutant is recombination-proficient. The *RED1* gene was cloned by complementation of the meiotic lethal phenotype; strains carrying a disruption of the gene are mitotically viable. We propose that the *RED1* gene product is involved in meiosis I chromosome disjunction, perhaps by maintaining the connections between homologous chromosomes through metaphase I.**

Meiosis is a special form of cell division responsible for the sexual phase of an organism's life cycle. Meiosis consists of two chromosome segregations generating haploid products (gametes or spores) whose genomic content arises from an independent assortment of the recombined parental chromosomes. During meiosis I, paired homologous chromosomes segregate from each other, reducing the diploid ($2n$) number of chromosomes to a haploid ($1n$) set. It is this reductional division that distinguishes meiosis from mitosis. In the meiosis II equational division, sister chromatids segregate from each other as in mitosis.

Little is known about the mechanism of reductional chromosome segregation (ref. 1, for review). In chiasmate organisms, the proper segregation of chromosomes during meiosis appears to require the pairing of homologous chromosomes (synapsis) and at least one genetic exchange per chromosome (2). Asynaptic mutants in plants (reviewed in ref. 3) have a reduced frequency of paired homologous chromosomes at pachytene and display high levels of meiosis I nondisjunction. Mutants at the *HOP1* locus of *Saccharomyces cerevisiae* (N. Hollingsworth and B. Byers, personal communication) are defective in the pairing of homologous chromosomes; these mutants display reduced levels of recombination and produce inviable spores. Yeast mutants that completely abolish recombination, such as *spo11* (4) and *rad50* (5), also produce inviable meiotic products, presumably due to high levels of aneuploidy. These observations imply that meiotic recombination is required for the reductional segregation of chromosomes.

Synapsis and recombination do not ensure proper segregation at meiosis I. Meiotic mutants have been identified that are recombination-proficient but defective in chromosome segregation. For example, desynaptic mutants in maize undergo normal levels of exchange, yet homologs segregate aberrantly at meiosis I anaphase (6). In addition, the *Drosophila* mutants *ord, meiS332*, and *G67* (3, 7–9) and the yeast mutant *DIS1* (10) are recombination-proficient but undergo nondisjunction at both meiotic divisions. The yeast mutants *spo12* and *spo13* are recombination-proficient and bypass the first meiotic division (11).

To further analyze meiotic chromosome segregation in yeast, we have devised a screen to identify meiotic-lethal mutants. Here we describe *RED1*, a gene required for chromosome segregation during the first meiotic (*red*uctional) division.

MATERIALS AND METHODS

Yeast Strains. Yeast strains are listed in Table 1. The two alleles *arg4-8* and *arg4-9* display temperature-sensitive, intragenic complementation (10). The *arg4-8* allele is temperature-sensitive and displays a dosage effect; *arg4-8* haploids are prototrophic up to 27°C, whereas strains disomic for *arg4-8* are prototrophic up to 30°C. Strains carrying the *arg4-9* allele are auxotrophic for arginine at all temperatures. Diploids or disomes heteroallelic for *arg4-8* and *arg4-9* are prototrophic up to 35°C. The *HIS4* alleles *his4-280* and *his4-290* also display intragenic complementation. The *cyh10-100* allele was UV-induced and maps <0.5 centimorgan (cM) from *CEN2*.

Genetic Procedures. Media were prepared and yeast manipulations were carried out according to Sherman *et al.* (12). YEPAD medium is YEPD medium supplemented with adenine. Copper-containing medium is synthetic complete medium (SC medium) made with 1.5% Phytagar (GIBCO); $CuSO_4$ was added after autoclaving. To select spore colonies resistant to copper, sporulated patches of cells were replicated to SC + cup (SC medium plus added $CuSO_4$ to 0.1 mM). UV mutagenesis (to 50% survival) was carried out on spores that had been treated with Zymolyase 100T, sonicated, and plated on solid YEPAD medium. Yeast transformations were carried out according to Sherman *et al.* (12); transformants were analyzed by Southern blot hybridization to confirm that substitution had occurred as expected.

Ether Test for Spore Viability. Sporulated patches of cells were replica-plated to YEPAD medium in glass Petri dishes. A 4 cm × 4 cm square of Whatman no. 1 filter paper, saturated with 0.5 ml of diethyl ether, was placed in the lid of each inverted dish. The Petri dishes were placed in an ether-resistant plastic box with a beaker containing several milliliters of ether. The box was sealed with a lid and incubated for 15 min; another 0.5 ml of ether was then added to each dish and the plates were incubated for another 15 min. The filter paper was then removed from each dish and the plates were set, with lids ajar, to dry in a hood for 45 min. Plates were scored after 24 hr.

RESULTS

Isolation of the *red1-1* Mutant. In designing a screen for mutants defective in meiotic chromosome transmission, we reasoned that such mutants would form spores but these

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Abbreviation: cM, centimorgan(s).

Table 1. Yeast strains

Strain	Genotype
BR1824-2B	*MATa his4-290 HO trp1-1 arg4-8 thr1-4 ura3-1* *MATα his4-290 HO trp1-1 arg4-8 thr1-4 ura3-1*
BR2140	*MATa leu2 his4-290 red1-1 arg4-8 THR1 trp1-1 ura3-1 cyh10-100* *MATα LEU2 his4-280 red1-1 arg4-9 thr1 trp1-289 URA3 CYH10*
BR2142	*MATa leu2 his4-290 RED1 arg4-8 THR1 trp1-1 ura3-1 cyh10-100* *MATα LEU2 his4-280 red1-1 arg4-9 thr1 trp1-289 URA3 CYH10*
BR2327	*MATa leu2-27 his4-260, 519 RED1 trp1-1 spo13::URA3 ura3-1* *MATα LEU2 his4-280 red1-1 trp1-289 spo13::URA3 ura3-1*
BR2328	*MATa leu2-27 his4-260, 519 red1-1 trp1-1 spo13::URA3 ura3-1* *MATα LEU2 his4-280 red1-1 trp1-289 spo13::URA3 ura3-1*
BR2344	*MATa leu2-27 his4-280 cyh10-100 red1-1 arg4-8 trp1-1 ura3-1* *MATα LEU2 his4-280 CYH10 red1::Tn20 arg4-9 trp1-289 ura3-1*
BR2354	*MATa leu2-27 HIS4 ura3-1 red1-1 trp1-1 spo13::URA3* *MATα LEU2 his4-280 ura3-1 red1-1 trp1-289 spo13::URA3*
BR2371	*MATa leu2-27 HIS4 RED1 trp1-1 spo13::URA3 ura3-1* *MATα LEU2 his4-280 red1-1 trp1-289 spo13::URA3 ura3-1*
BR2408	*MATa his4-280 arg4-8 trp1-1 cyh10-100 ura3-1* *MATα his4-290 arg4-9 TRP1 CYH10 ura3-1*

All genotypes are heterothallic (*ho*), *RED1*, and *CUP1*^{s} unless otherwise indicated. The allele *red1::Tn20* (strain BR2344) was constructed by transposon mutagenesis.

meiotic products would be highly aneuploid and therefore inviable. Thus, a screen for sporulation-proficient, meiotic-lethal mutants was employed. To recover recessive mutants in a diploid strain, spores from a homothallic (*HO*) strain were mutagenized (3). Homothallic haploid spores switch mating type within the first few cell divisions and then mate to form diploids homozygous for any induced mutations (13).

Because yeast does not sporulate with 100% efficiency, spore viability cannot be assessed simply by examining the growth of a sporulated culture. The growth of unsporulated cells would obscure the reduction in spore viability. To overcome this problem, we took advantage of the observation that vegetative cells are more sensitive than spores to killing by diethyl ether (14). Thus, the viability of spores is indicated by growth on rich medium after exposure to ether vapors (see *Materials and Methods*). Spores from the diploid BR1824-2B were mutagenized by UV, and diploid colonies incapable of growth after sporulation and exposure to ether were scored for the production of visible asci. Those colonies that sporulated were examined by tetrad dissection to confirm the spore inviability phenotype.

One mutant, *red1-1*, sporulates well (60–80%), yet only 1% of the spores produced are viable. The segregation of the *red1-1* mutation was followed in a cross between the *red1-1* isolate and a wild-type strain. Spores from the *red1-1* isolate were crossed to spores of a homothallic wild type and several individual diploids were selected. Half of these diploids produced dead spores, presumably because the spores from the mutant parent were frequently aneuploid, resulting in "diploids" that were also aneuploid. Upon sporulation, the segregation of extra chromosomes led to inviability (see below). A successful cross was dissected and the diploidized spore colonies were sporulated and scored for spore viability. Spore lethality segregated 2:2 in 24 four-spore-viable tetrads, indicating that *red1-1* represents a single mutation.

Viable Spores from *red1* Mutant Meioses Are Highly Aneuploid. If the spore inviability observed in the *red1-1* mutant is the consequence of an aberrant meiosis, then the few viable spores produced should be aneuploid. This was tested by constructing wild-type (*RED1/RED1* and *RED1/red1-1*) and *red1-1* strains and analyzing their meiotic products for evidence of aneuploidy. These strains were heterozygous for *cyh10-100*, a recessive, centromere-linked cycloheximide-resistance marker, to facilitate the identification of spores. Three pairs of codominant alleles permitted the distinction of monosomic and disomic spores. The diploids were marked on chromosome III with *MAT*a and *MATα* and by the complementing alleles *his4-280* and *his4-290*. Chromosome VIII was marked with the complementing alleles *arg4-8* and *arg4-9*. Physically isolated spores (15) were obtained from sporulated cultures and plated on cycloheximide-containing medium. The Red$^+$ diploid produced a low frequency of His$^+$ and Arg$^+$ colonies (Table 2) and these were haploids resulting from intragenic recombination (data not shown). The diploid homozygous for *red1-1* produced spores that were 38% histidine prototrophs, 33% arginine prototrophs, and 34% nonmaters (Table 2). These results demonstrate high levels of aneuploidy among the survivors of meiosis in the *red1-1* mutant. Thus, the spore inviability observed in the *red1-1* mutant is probably the result of meiotic nondisjunction.

Disomic Spores Are the Products of Meiosis I Nondisjunction. To determine whether the nondisjunction caused by a *red1* mutation occurs during meiosis I and/or meiosis II, chromosome VIII disomes were selected and analyzed for the presence of sister or nonsister chromosomes. Chromosome VIII disomes can be selected on the basis of copper resistance (10) mediated by the *CUP1* gene on chromosome VIII. The level of copper resistance is a function of gene dosage; thus, spores disomic for chromosome VIII can be selected independent of whether they contain sister or nonsister chromatids. Nondisjunction at meiosis I results in spores with nonsister chromatids, whereas nondisjunction at meiosis II generates spores with sister chromosomes (Fig. 1).

The diploids used for this analysis were heteroallelic for the *ARG4* complementing alleles *arg4-8* and *arg4-9*. Disregarding the effects of recombination, all disomes originating from the first meiotic division should be *arg4-8/arg4-9* (i.e., heteroallelic). If the normal map distance between *ARG4* and *CEN8*

Table 2. Chromosome III and VIII aneuploidy in spores

Genotype	Strain	% His$^+$	% nm	% His$^+$ nm	% Arg$^+$	Total
RED1/RED1	BR2408	0.2	0	0	0.7	431
RED1/red1-1	BR2142	0.7	0	0	2.6	267
red1-1/red1-1	BR2140	38	34	64	33	466

Physically isolated spores were plated on cycloheximide-containing medium and scored for histidine and arginine prototrophy (complementation) and mating type. The percentage of nonmaters (nm) that were histidine prototrophs is also presented.

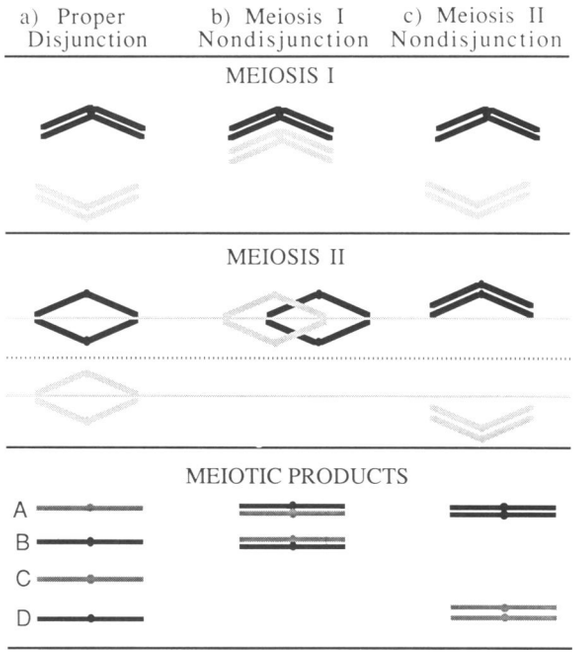

FIG. 1. Chromosome segregation during meiosis. A single pair of chromosomes is depicted undergoing normal meiosis (*a*), nondisjunction at meiosis I (*b*), and nondisjunction at meiosis II (*c*).

(15.5 cM) is taken into account, 84% heteroallelic disomes are expected. Chromosome VIII disomic spores were selected by replica-printing sporulated patches of cells to medium containing copper. Of 279 copper-resistant outgrowths tested, 231, or 83%, were heteroallelic disomes (Table 3). This value closely approximates the 84% heteroallelic disomes expected from aberrant reductional segregations. Thus, most, if not all, of the aberrant segregation in a *red1* mutant occurs at the reductional division.

The *red1* Spore Inviability Phenotype Is Rescued by a *spo13* Mutation. Diploids homozygous for a *spo13* mutation undergo homolog pairing and meiotic recombination but then proceed directly to an equational (meiosis II) chromosome disjunction (11). Thus, *spo13* mutants skip the reductional segregation and form dyads containing diploid spores. Meiotic lethal mutants defective in early steps in meiotic recombination, such as *spo11* and *rad50*, make viable spores in a *spo13* background, presumably because the step at which recombinant chromosomes are required is skipped (4, 5). If the *red1-1* mutation causes nondisjunction exclusively at the first meiotic division, the *spo13* mutation might be expected to restore spore viability to *red1-1* diploids.

Table 3. Chromatid constitution of disomic spores

%	*arg4-8*/*arg4-9*	*arg4-8*/*arg4-8*	*arg4-9*/*arg4-9*
Expected (reductional)	84	8	8
Expected (equational)	31	34	34
Observed (BR2344)	83 (253)	10 (30)	7 (21)

To determine whether disomic spores contain sister or nonsister chromosomes on the basis of *ARG4* segregation, recombination values must be taken into account. Nondisjunction at meiosis I produces heteroallelic spores when there has been no crossing over between *ARG4* and *CEN8* (69%) and half of the time when a crossover occurs (0.5 × 31%). Nondisjunction at meiosis II results in heteroallelic spores whenever there has been a crossover in the *ARG4-CEN8* interval (31%). Allelic types expected for nondisjunction events occurring entirely at the reductional division or entirely at the equational division are indicated. Numbers in parentheses indicate the numbers of copper-resistant outgrowths scored. The same distribution of allelic types was found among a subset of copper-resistant disomes that were cycloheximide-resistant (and therefore meiotic products that inherited a single copy of chromosome II).

Homozygous *red1-1 spo13::URA3* (a disruption of the *SPO13* gene with *URA3*; ref. 17) double mutants and Red$^+$ *spo13::URA3* strains were constructed and analyzed. Four hundred nine dyads from Red$^+$ *spo13::URA3* and 349 dyads from *red1-1 spo13::URA3* diploids were dissected; both crosses produced 82% viable spores and showed the same frequency of dyads containing two (70%), one (24%), and zero (6%) viable spores. These data indicate that the *spo13* mutation alleviates the *red1-1* inviability phenotype and, thus, is epistatic to the *red1-1* mutation.

***red1-1* Homozygotes Are Recombination-Proficient.** A possible cause for meiotic nondisjunction is an alteration in pairing and exchange. The competence of *red1-1* strains to undergo meiotic exchange was examined in two ways. (*i*) Intragenic recombination was assayed in Red$^+$ and Red$^-$ diploids. The frequency of Trp prototrophs from Red$^+$ (BR2142) and Red$^-$ (BR2140) strains heteroallelic at *TRP1* was 1.7×10^{-4} and 1.5×10^{-4} per spore, respectively. (*ii*) *spo13 red1-1* diploids were used to assay intergenic crossing-over by dyad dissection. Dyads were scored for genetic exchange in two intervals on chromosome III, *LEU2–MAT* and *HIS4–LEU2* (Table 4). The map distances for wild-type and *red1-1* homozygous diploids are 34 cM and 28 cM for the *LEU2–MAT* interval and 21 cM and 17 cM for the *LEU2–HIS4* interval, respectively. Thus, meiotic intra- and intergenic recombination in *red1-1* homozygous diploids occurs at normal levels.

Cloning and Disruption of the *RED1* Gene. The wild-type allele of *RED1* was cloned from a YCp50 library (19) by complementation of the *red1-1* spore inviability phenotype. The restriction map of a 4.8-kilobase-pair (kbp) *Eco*RI restriction fragment with *red1*-complementing activity is shown in Fig. 2.

To locate the *RED1* gene on the cloned DNA segment and to construct new alleles of the gene, m-Tn*3*(*URA3*) insertion mutations were generated according to the shuttle mutagenesis scheme of Seifert *et al.* (20). Plasmids containing insertion mutations were introduced into a homothallic *RED1 ura3-1* diploid by substitutive transformation. These Ura$^+$ transformants, heterozygous for the insertion mutation, were sporulated and tetrads were dissected. All transformants produced tetrads containing four viable spores, indicating that the *RED1* gene is not essential for mitotic growth. Fig. 2 shows the positions of various insertions obtained in and around the *RED1* gene. These insertion mutations delimit the gene to an interval ≈3 kbp in size. A deletion mutation that removes the 5′ end of the gene and about two-thirds of the protein coding sequence (sequence data, E. Thompson and G.S.R., unpublished results) was also constructed.

Strains homozygous for *red1::Tn3* alleles sporulate to about 30–45% efficiency, in contrast to 60–80% sporulation for the *red1-1* allele. These strains rarely form four-spored asci (1–10%). The viability of the spores from strains carrying insertions at the 5′ end of the gene (right end in Fig. 2) is about 10–15% and, therefore, higher than *red1-1* diploids. Strains carrying a disruption at the 3′ end of the gene or the deletion mutation produce only 2–4% viable spores. Strains carrying these m-Tn*3*(*URA*) insertions do not complement the *red1-1* mutant, indicating that the sequence delineated by these mutations is the *RED1* gene.

Map Position of *RED1*. The *spo11* mapping procedure (21) was used to locate *RED1* on chromosome XII. This result was confirmed by using the cloned *RED1* gene to probe yeast chromosomes that had been electrophoretically separated (22).

Table 4. Recombination in Red$^+$ and Red$^-$ *spo13* diploids

Diploid genotype	Two-spored viable dyads, no.		A	B	C	D	E	F	Map distance, cM
MAT–LEU2		*MAT*	2:0:0	0:1:1	2:0:0	0:1:1	1:0:1 or 1:1:0	1:0:1 or 1:1:0	
		LEU2	2:0	2:0	1:1	1:1	2:0	1:1	
						PD NPD			
RED1 / *red1-1*	262		172	32	43	5 2	3	5	34
red1-1 / *red1-1*	306		201	29	43	7 1	12	13	28
HIS4–LEU2		*LEU2*	2:0	1:1	2:0	1:1			
		HIS4	2:0	2:0	1:1	1:1			
RED1 / *red1-1*	115		75	11	13	10			21
red1-1 / *red1-1*	106		68	9	9	13			17

Two-spore viable dyads from *spo13*::*URA3* strains were scored for *MAT*, *LEU2*, and *HIS4* segregation. Combined data from BR2371 and BR2327, Red$^+$, and BR2328 and BR2354, Red$^-$, are shown for the *LEU2–MAT* interval; BR2354 and BR2371 were scored for the *HIS4–LEU2* interval. *MAT* segregation is indicated as the ratio of nonmaters (nm):a-maters:α-maters. *LEU2* and *HIS4* segregations are indicated as the ratio of prototrophs:auxotrophs (i.e., +:−). Type D segregations are divided into two classes: PD, in which the *LEU2* and *MAT* markers are in the parental configuration, and NPD, in which the markers are in the nonparental configuration. For the *MAT–LEU2* interval, the following dyad types were scored as single crossovers: type B (a Leu$^+$, α Leu$^+$) and type C (nm Leu$^-$, nm Leu$^+$). The dyads with the phenotype a Leu$^+$, α Leu$^-$ (type D-NPD) were scored as four-strand double crossovers. Single crossovers in the *LEU2–HIS4* interval produced dyads with the following phenotypes: Leu$^-$ His$^+$: Leu$^+$ His$^+$ and Leu$^+$ His$^-$: Leu$^+$ His$^+$. Dyads exhibiting a, nm or α, nm phenotypes (i.e., monosomes and trisomes, types E and F) were scored as nondisjunctants (11) and were not included in the calculation of map distances. Map distance was calculated by using a derivation of Perkin's formula (18) as follows: map distance = [single crossovers + 6(4-strand double crossovers)/total] × 100. Dyads in which *MAT* and *LEU2* have retained the parental configuration of markers (type D-PD) probably arose from a reductional division that had not undergone exchange in this interval. The alternative possibility is that these dyads arose from a two-strand double crossover followed by equational division. If this were true, then an equal number of dyads displaying four-strand double crossovers and equational division (i.e., type D-NPD) would be predicted. There were significantly fewer of these types (3 vs. 12). Thus, many of the type D-PD dyads are probably the products of reductional chromosome segregation.

DISCUSSION

We have identified a yeast gene, designated *RED1*, that is required for the proper segregation of chromosomes in meiosis. *red1* mutants undergo high levels of nondisjunction at the first meiotic division, yet they display wild-type levels of recombination. Thus, the *red1* mutant is unlike any previously described meiotic mutants of yeast.

***SPO13* Is Epistatic to *RED1*.** A *spo13* mutation restores spore viability to a *red1* mutant, indicating that *SPO13* is epistatic to *RED1*. This epistatic relationship suggests that the *RED1* gene product acts at a later step in meiosis I disjunction than does the *SPO13* gene product—i.e., that *spo13* mutants skip from meiosis I to meiosis II before the point of *RED1* action.

As discussed in the legend to Table 4, *spo13* mutants display a small amount of reductional chromosome segregation. *RED1 spo13* and *red1 spo13* diploids exhibit similar frequencies of those dyad types resulting from the reductional segregation of chromosome III. The ability of *red1* mutants to undergo reductional division in a *spo13* background demonstrates that the *spo13* mutation is epistatic to the *red1* mutation for this phenotype as well as for the spore inviability phenotype. The reductional segregations observed in *spo13* mutants are independent of *RED1* function.

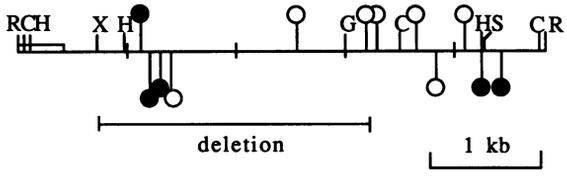

FIG. 2. Restriction map of the *RED1* gene. Open circles indicate m-Tn*3*(*URA3*) insertions that confer a Red$^-$ phenotype; filled circles indicate a Red$^+$ phenotype. Tn*3* insertions in one orientation are shown above the line; insertions in the opposite orientation are below the line. The end points of the *RED1* deletion are indicated. The open box at the left end of the fragment represents 375 base pairs (bp) of pBR322 DNA. R, *Eco*RI; G, *Bgl* II; B, *Bam*HI; C, *Cla* I; H, *Hin*dIII; Sc, *Sca* I; X, *Xba* I.

Possible Functions for the *RED1* Gene Product. The *RED1* gene product is required for chromosome segregation at meiosis I but not in meiosis II or mitosis. Several possibilities for the function of the *RED1* gene product are considered here. *RED1* may be involved in meiotic kinetochore function, the meiosis I spindle pole body or spindle, or chiasma terminalization or maintenance.

red1 mutants may be defective in kinetochore function such that chromosomes fail to attach to the meiosis I spindle. Alternatively, mutations at the *RED1* locus may allow the premature replication (differentiation) of kinetochores so that sister chromatids separate and segregate at the first meiotic division.

Another possibility is that the *RED1* gene product is required for the structure or function of the meiosis I spindle pole body or spindle. A defect in the meiotic spindle apparatus is suggested by the appearance of fragmented nuclei in Hoeschst-stained *red1* spores (unpublished results). These could have resulted from aberrant or multiple spindles producing micronuclei, as has been observed in certain meiotic mutants of plants (reviewed in ref. 3). However, fragmented spore nuclei are also generated by the recombination-defective yeast mutant *spo11* (4), suggesting that this phenotype may result from any perturbation of meiosis I disjunction. Furthermore, Nicklas (23) has presented evidence that a meiotic bivalent segregates reductionally when placed in a meiosis II spindle, suggesting that it is the structure of the chromosome, not the spindle, that distinguishes the reductional division.

Chiasmata move distally during late prophase I (diakinesis) in a process known as terminalization (16). If the *RED1* gene product is required for this process, then chiasmata might fail

to terminalize in *red1* mutants, causing bivalents to remain intact through anaphase I. As a result, both homologs might go to the same pole at meiosis I or the bivalent might break as is observed for dicentric chromosomes. Alternatively, the *RED1* gene product might be responsible for maintaining chiasmata until anaphase I. If chiasmata terminalize early in *red1* mutants, then univalents, instead of bivalents, would be present at metaphase I and these would probably segregate randomly.

The *red1* mutant is the only known yeast mutant that is recombination-proficient yet causes nondisjunction at meiosis I. By screening for other mutants with a *red1*-like phenotype and by isolating pseudorevertants of *red1* mutants, we hope to identify additional yeast genes whose products are required for reductional chromosome segregation.

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