Proc. Natl. Acad. Sci. USA
Vol. 86, pp. 3694–3698, May 1989
Genetics

Enhanced meiotic recombination on the smallest chromosome of *Saccharomyces cerevisiae*

(yeast/genetic map/physical map)

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Communicated by David D. Perkins, December 30, 1988 (received for review October 4, 1988)

ABSTRACT **Chromosome I is the smallest chromosome in *Saccharomyces cerevisiae* and contains a DNA molecule that is only 250 kilobases (kb). Approximately 75% of this DNA molecule has been cloned. A restriction map for the entire DNA molecule from chromosome I was determined and most of its genetically mapped genes were located on this physical map. Based on the average rate of recombination (centimorgans/kb) found for other *S. cerevisiae* chromosomes, the outermost markers on the genetic map of chromosome I were expected to be close to the ends of the DNA molecule. While the rightmost genetic marker was 3 kb from the end, the leftmost marker, *CDC24*, was located near the middle of the left arm, suggesting that the genetic map would be much longer. To extend the genetic map, a copy of the *S. cerevisiae URA3* gene was integrated in the outermost cloned region located 32 kb centromere distal to *CDC24*, and the genetic map distance between these two genes was determined. The new marker substantially increased the genetic map length of chromosome I. In addition, we determined the relationship between physical and genetic map distance along most of the length of the chromosome. Consistent with the longer genetic map, the average rate of recombination between markers on chromosome I was >50% higher than the average found on other yeast chromosomes. Owing to its small size, it had been estimated that ≈5% of the chromosome I homologues failed to undergo meiotic recombination. New measurements of the zero-crossover class indicated that the enhanced rate of recombination ensures at least one genetic exchange between virtually every pair of chromosome I homologues.**

During meiosis I, homologous chromosomes pair, undergo recombination (crossing-over), and obligately move to opposite poles of the meiotic spindle (1). Mutants defective in meiotic recombination show high levels of meiotic nondisjunction, suggesting that crossing-over is obligatory for meiosis I chromosome segregation (1). Paradoxically, the amount of meiotic recombination previously observed on chromosome I, the smallest *Saccharomyces cerevisiae* chromosome (2), was not sufficient to ensure that it undergoes crossing-over in every meiotic cell. Based on its known genetic map length of 101 ± 5 centimorgans (cM) (3, 4), 5–6% of the chromosome I homologues were estimated to be nonrecombinant (ref. 5; D.B.K., unpublished results). Nevertheless, chromosome I homologues still segregate properly virtually all the time. Furthermore, multiplying the average cM per kilobase (kb) equivalent (rate of recombination) found for other yeast chromosomes (≈0.4 cM/kb; refs. 6 and 7) by the size of the chromosome I DNA molecule (250 kb; ref. 2) predicts that the total genetic map length of chromosome I would not exceed the 101 cM already found (250 kb × ≈0.4 cM/kb = ≈100 cM). However, since neither the recombination rate nor the total genetic map length of chromosome I had been determined directly, it was not certain that the genetic map length is only 101 cM and that nonrecombinant chromosomes occur in anywhere close to the 5–6% predicted.

In these studies, we constructed a restriction map of the entire chromosome I DNA molecule, determined the location of most of the genetically mapped genes on this physical map, and then compared the genetic and physical maps. The average rate of meiotic recombination was determined and found to be >50% higher than on other larger *S. cerevisiae* chromosomes and the total genetic map length of chromosome I was estimated to be ≈50% longer than before. These findings combined with additional tetrad analysis indicated that virtually all chromosome I homologues undergo recombination during meiosis.

MATERIALS AND METHODS

Genetic Techniques and Growth of *S. cerevisiae*. Strains of *S. cerevisiae* used are listed in Table 1. Genetic techniques and growth were as described (8–10).

Purification of DNA. Plasmid, bacteriophage, and *S. cerevisiae* DNA were prepared as described (8, 9, 11).

Recombinant DNA Procedures. Routine procedures were used in all experiments (11). Enzymes were used according to manufacturer's specifications.

Physical Map of Chromosome I. Orthogonal field-alternation gel electrophoresis (OFAGE) (2) purified chromosome I DNA was subjected to restriction enzyme mapping. Intact chromosomes were electrophoresed for 24 hr at 300 V with 15-sec pulses as described (12). Gels were stained with ethidium bromide and the segment containing chromosome I DNA was excised and equilibrated with appropriate buffer. Restriction enzyme (20–40 units per 100 μg of gel) was added in a minimal volume of additional buffer and the mixture was incubated for 3–6 hr. Digested samples were placed in a second gel and electrophoresed with 10-sec pulses. Monomers and oligomers of bacteriophage λ DNA served as size markers. Smaller fragments were sized by conventional agarose gel electrophoresis.

Introduction of the p*URA3* Marker in Chromosome I. The 3.2-kb *Bam*HI/*Sal* I fragment from λR23d was subcloned into *Bam*HI/*Sal* I linearized integrating vector YIp31 (13) and the resultant plasmid pYY54 was transformed into *S. cerevisiae* strains W303-1B and MM1-1B as described (9).

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Abbreviations: cM, centimorgan(s); OFAGE, orthogonal field-alternation gel electrophoresis.

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Table 1. Yeast strains

Strain	Relevant genotype	Source
MM1-1B	*MATa adel ura3-1 trp1-1 leu1 met2 arg1 arg4*	This study
W303-1B	*MATα ade2 his3-11,15 leu2-3,112 ura3-1*	R. Rothstein
DK370-10C	*MATa adel spo7::LEU2 leu2-3,112*	This study
LK16t3-1C	*MATα spo7::LEU2 leu2-3,112 trp1*	This study
LK16t3-1D	*MATa spo7::LEU2 leu2-3,112 ura3-1*	This study
RW1769	*MATα adel mak16-1 leu2-3,112 trp1*	R. Wickner
26X16-4A	*MATa adel leu2-3,112*	This study
YS61	*MATα FLO1 leu2-3,112 ura3-52 trp1*	This study
GX329-3C	*MATa adel cdc15-1 pho11::LEU2 leu2-3,112*	This study
GM1-9B	*MATα pho11::LEU2 leu2-3,112 trp1-1*	V. Letts
DK329-10A	*MATa adel cdc15-1 leu2-3,112 ura3-1*	This study
DK382-3D	*MATα cdc24-5 ura3-1 leu1 argx metx*	This study
DK382-8D	*MATa cdc24-5 ura3-1 leu1 metx trp1-1*	This study
DK381-9A	*MATa pyk1-101 adel ura3 his3 trp1-1*	This study

argx, *arg1* or *arg4*; *metx*, *met1* or *met2*.

RESULTS

Additional Genetic Mapping. Tetrad analysis was required to map several genes with respect to other chromosome I markers (Table 2, Fig. 1). The *SPO7* gene was mapped 28.8 cM to the right of *MAK16*. *ADE1* and *CEN1* markers also were included in this analysis. Map positions of all four markers were consistent with published results (3, 4).

PHO11 (16) was mapped 40 cM to the right of *ADE1* (V. Letts and R. Kramer, personal communication). To determine *PHO11*'s position with respect to other genes, we measured its distance from *CDC15* and *FLO1*. Our results confirmed the location of *PHO11* and indicated that this gene was the outermost known marker on the right arm, mapping 3.8 cM from *FLO1* (Table 2, Fig. 1). These results extend the genetic map of chromosome I by ≈4%.

Alignment of the Genetic and Physical Maps. The cloning of half the DNA from chromosome I and the physical location of centromeric DNA (*CEN1*) and the majority of known genes on the cloned inserts has been described (8, 9, 17, 18). We cloned additional sequences by "chromosome walking" (19) with probes from YCp50(*MAK16*)2C (17), λH9a (8), pAP18 (16), or chromosome I DNA purified by OFAGE. To date, ≈75% of the DNA from chromosome I has been cloned. The physical locations of the *SPO7* and *PHO11* genes on these clones have been determined (D.B.K., unpublished results; V. Letts and R. Kramer, personal communication).

A map of OFAGE purified chromosome I DNA was constructed with infrequent cutting restriction endonucleases *Not* I, *Sfi* I, and *Sma* I. Locations of corresponding restriction sites on the cloned sequences were determined and the sequences aligned (Fig. 1). Orientation of the cloned segments were consistent with the genetic map. The cloned 17-kb segment containing *PHO11* did not overlap with another cloned region and could not be localized by restriction mapping because it did not contain any *Not* I, *Sfi* I, or *Sma* I sites. This region was mapped physically to the outermost 21 kb on the right arm (unpublished data) using the chromosome breakage technique of Vollrath *et al* (20). *PHO11* was 3–4 kb from the right end of the chromosomal DNA molecule. Positioning *CEN1* on the physical map of the entire chromosome indicated that chromosome I is submetacentric with ≈160 kb on the left arm and ≈90 kb on the right arm.

Map alignment allowed an assessment of the size and location of sequences that had not been cloned (Fig. 1). One of these regions was located between *MAK16* and *PYK1*. The size of this uncloned segment was determined by Southern blotting (21). Distal probes from YCp50(*MAK16*)2C and λD142a hybridized to like-sized *S. cerevisiae* genomic restriction fragments using *Bam*HI, *Bgl* II, or *Xho* I. Mapping the corresponding sites on the cloned inserts indicated the uncloned segment is 2.5 kb long (data not shown).

We originally thought *CDC24* would be close to the left end of the DNA molecule. Since the genetic map distance between *CDC24* and *PHO11*, the two most distal genes

Table 2. Genetic mapping

Gene pair	Cross	Number of asci PD/NPD/T	FD/SD	cM
*MAK16-SPO7**	LK16t3-1D × RW1769	28:1:26		29.0
	DK370-10C × RW1769	11:0:14		28.0
	Total	39:1:40		28.8
SPO7-CEN1*	LK16t3-1D × RW1769		53:1	0.9
	DK370-10C × RW1769		23:3	5.8
	26X16-4A × LK16t3-1C		36:2	2.6
			Total 112:6	2.5
SPO7-ADE1*	LK16t3-1D × RW1769	40:0:9		9.2
	26X16-4A × LK16t3-1C	33:0:6		7.7
	Total	73:0:15		8.5
MAK16-ADE1	LK16t3-1D × RW1769	21:2:34		40.4
CDC15-FLO1	YS61 × GX329-3C	52:3:116		39.2
*CDC15-PHO11**	YS61 × GX329-3C	46:7:118		46.8
	GM1-9B × DK329-10A	48:1:108		36.3
	Total	94:8:226		41.8
*FLO1-PHO11**	YS61 × GX329-3C	160:0:13†		3.8
pURA3-CDC24‡*	W303-1B × DK382-8D	25:0:20		22.0
	MM1-1B × DK382-3D	50:0:37		21.3
	Total	75:0:57		21.6
pURA3-PYK1‡*	W303-1B × DK381-9A	25:2:35		37.9

Mapping was by tetrad analysis (3, 4, 10). PD, parental ditype; NPD, nonparental ditype; T, tetratype; FD, first division segregation; SD, second division segregation. Map distances were calculated by Perkins' formula: cM = (6 NPD + T)100/2(PD + NPD + T) or (SD)100/2(FD + SD) (14).

*Mapping based on *spo7::HIS3* and *pho11::LEU2* mutations made by one-step gene replacement (15). LK16t3-1D and 1C and 26X16-4A are derived from the same *spo7::LEU2* transformant. DK370-10C is from a different transformed parent. *CEN1* was scored by assuming the heterozygous *TRP1* marker always segregated at the first meiotic division.

†*PHO11* was shown to be centromere distal to *FLO1* by examining 13 asci containing crossovers between these two markers in a 5-factor cross containing heterozygous *CEN1*, *ADE1*, *CDC15*, *FLO1*, and *PHO11* markers. *CEN1* was scored by using the heterozygous *TRP1* marker. Twelve of the 13 asci could be explained by the minimal number of crossover events only if the gene order was *CEN1-ADE1-CDC15-FLO1-PHO11*.

‡Sums from three independent W303-1B (pYY54) transformants or six independent MM1-1B (pYY54) transformants.

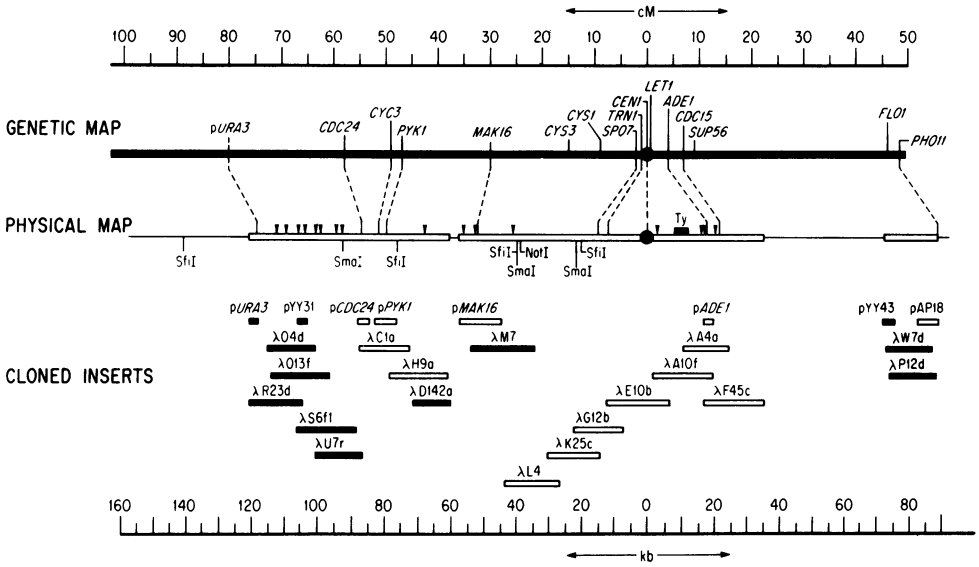

FIG. 1. Alignment of the genetic and physical maps of chromosome I in *S. cerevisiae*. Genetic map derived from published values and data in Table 2. Physical map: ▭, cloned regions; ——, uncloned regions defined by restriction mapping. *Not* I, *Sfi* I, and *Sma* I sites are noted, as are *Bam*HI sites (▼) in the cloned regions. Chromosome I DNA inserts from plasmids and λ bacteriophages: ■, inserts not described previously; ▭, inserts described previously. Plasmid notations: p*URA3*, pYY54 (see text); p*CDC24*, YRp7(*CDC24*)1 (8); p*PYK1*, pBR322-(*PYK1*)1 (8); p*MAK16*, YCp50(*MAK16*)2C (17); p*ADE1*, YEp13(*ADE1*)1 (9); pAP18 (16); pYY31 and pYY43, clones derived from OFAGE purified chromosome I DNA digested with *Bam*HI and *Bgl* II, respectively.

known, is 105 cM (Fig. 1) and there are, on average, ≈2.5 kb/cM for other *S. cerevisiae* chromosomes (6, 7), these two genes should have been ≈260 kb apart (105 cM × 2.5 kb/cM = 263 kb), putting each gene very close to an end of this molecule. Surprisingly, the physical map revealed that *CDC24* was near the middle of the left arm, making it likely that the genetic map of chromosome I would be substantially longer.

Extension of the Genetic Map. To extend the map of chromosome I, a plasmid containing the *S. cerevisiae URA3* gene was integrated next to the most distal cloned sequence on the left arm and the genetic map distance between the integrated *URA3* gene (p*URA3*) and *CDC24* was determined. The chromosomal target was subcloned into plasmid YIp31 (13) yielding plasmid pYY54, which was transformed into *S. cerevisiae*. Ura$^+$ transformants with a single copy of the plasmid integrated on chromosome I were isolated. The structure of these integrants was confirmed by Southern blotting experiments (data not shown) and is shown in Fig. 2. The integrated *URA3* gene was only 300 base pairs behind the target sequence. Nine transformants were crossed to a *cdc24-5* strain. Tetrad analysis of resulting diploids (Table 2) indicated a genetic map distance 21.6 ± 5.1 cM between p*URA3* and *CDC24*. Consistent with this result, analysis of three of these transformants crossed to a *pyk1-101* strain gave a map distance of 37.9 ± 5.1 cM between p*URA3* and *PYK1*. As a control, distances between 2–4 other markers were monitored in each cross and found not to vary significantly from published values (data not shown). While we cannot completely eliminate the possibility that the integrated plasmid affects recombination, similar integration of plasmids at other locations had no significant effect on map distances that were this large (ref. 22; unpublished observations). The genetic map of chromosome I is now 127 cM, a total of 26% longer than before. Recombination in the 43-kb *CEN* distal to p*URA3* and in the 3 kb to the right of the *PHO11* gene should make the genetic map of chromosome I even longer.

Physical vs. Genetic Map Distances. We examined the relationship between physical and genetic map distances and calculated the rate of recombination (cM/kb) separating most gene pairs on chromosome I (Table 3). Similar to other chromosomes, we found a low recombination rate surrounding the centromere (6, 7, 23). However, beyond *SPO7* on the left and *CDC15* on the right, the rates went to >0.60 cM/kb between all markers. These regions include the previously described (8) recombination "hot spot" between *PYK1* and *CDC24* that recombines 2.5-fold more per kb than the average for this chromosome. Besides these deviations, the rates of recombination showed little variation along most of the DNA molecule (Fig. 3).

We estimate the average rate of recombination between all gene pairs on chromosome I to be 0.63 cM/kb (Table 3). This rate is >50% higher than found on other similarly analyzed large regions of *S. cerevisiae* chromosomal DNA (6, 7), and is more than twice the 0.28 cM/kb estimated for the entire genome by Mortimer and Schild (4) (Fig. 3). A rank sum test (24) indicated that recombination rates between intervals on chromosome I were significantly higher than those found on chromosomes III and V ($P < 0.01$ for being due to random fluctuation). In addition, variability studies (24) comparing recombination rates on these chromosomes indicated that it was extremely unlikely ($P < 0.0001$) that the clustering of

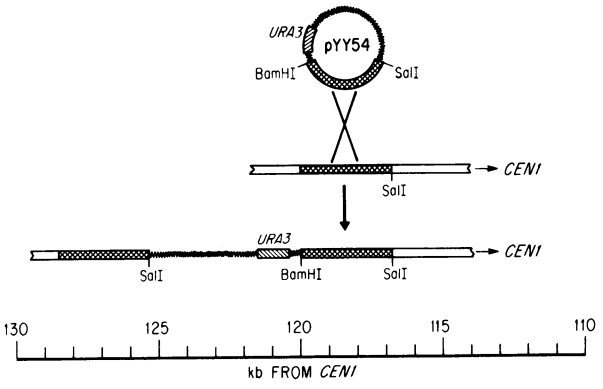

FIG. 2. Structure of the integrated *URA3* gene (p*URA3*) on chromosome I. Homologous integration of pYY54 gave the structure shown. The *Bam*HI site in pYY54 originated during cloning. ▭, *S. cerevisiae* DNA; ▨, *URA3* DNA; ▩, subcloned target sequence; ∿, pBR322 DNA.

Table 3. Comparison of genetic and physical distances

Interval	Genetic distance, cM	Physical, distance kb	Recombination rate, cm/kb	Ref.
p*URA3–CDC24*	22	33	0.67	*
p*URA3–PYK1*	38	40.5	0.94	*
CDC24–PYK1	11	7.5	1.50	4
CDC24–MAK16	31	36	0.86	4
CDC24–ADE1	57	105	0.54	4
CYC3–PYK1	2.2	3.0	0.73	4
PYK1–MAK16	17	28	0.61	4
PYK1–ADE1	57	98	0.58	4
MAK16–SPO7	29	35	0.83	*
MAK16–CEN1	30	50	0.60	4
MAK16–ADE1	43	68	0.63	4
SPO7–CEN1	1.6	15	0.11	4, *
SPO7–ADE1	8	33	0.24	4, *
TRN1–CEN1	0.5	12	0.04	19
TRN1–ADE1	4	30	0.13	19
CEN1–ADE1	4	18	0.22	3, 4
CEN1–CDC15	7	22	0.32	3, 4
ADE1–CDC15	2.2	4	0.55	3, 4
ADE1–PHO11	41	64	0.63	†
CDC15–PHO11	41.8	60	0.70	*
p*URA3–PHO11*	127	202	0.63	*
Average	28.2	45.0	0.63	*
Chromosome I	157	250	0.63	*
Chromosome III‡	68	185	0.37	6
Chromosome V§	27	72	0.38	7
Yeast genome	4000	14,000	0.28	4

The average genetic distance was used where data from multiple crosses were available. Physical distances were from the center of a transcribed region or the *CEN1* consensus sequence.
*This study.
†V. Letts and R. Kramer, personal communication.
‡All intervals between *HML* and *MAT*.
§All intervals between *CYC7* and *CEN5*.

intervals with high recombination rates found on chromosome I could be due to random fluctuation (data not shown).

Based on the average recombination rate and a 250-kb DNA molecule, we believe the total genetic map length of chromosome I will eventually be as high as 157 cM (0.63 cM/kb × 250 kb = 157 cM). Thus, we predict ≈50% more recombination on chromosome I than had been observed previously.

Estimation of the Size of the Zero-Crossover Class for Chromosome I. As a result of the increased map length, the fraction of chromosomes that fail to undergo crossing-over (the zero-crossover or e^0 class) should be reduced substantially from previous estimates. A multiply marked 80-cM interval between p*URA3* and *CEN1* and a multiply marked 47-cM interval between *CEN1* and *PHO11* were examined by tetrad analysis. The fraction of asci in the e^0 class for each interval was determined and a new estimate for the size of the e^0 class for the entire chromosome was calculated (Table 4). Using the 157-cM map length, the results indicated the e^0 class for all of chromosome I would comprise only 0.17–0.44% of the asci, 10–30 times lower than the previous estimate based on an ≈100-cM chromsome. In the unlikely event that chromosome I is only 127 cM long, the genetic map length so far defined, the e^0 class would comprise only 0.8% of the asci.

DISCUSSION

A physical map of chromosome I was constructed showing the location of almost all genetically mapped genes. Positioning *CEN1* indicated that chromosome I was submetacentric with ≈160 kb on the left arm and ≈90 kb on the right arm.

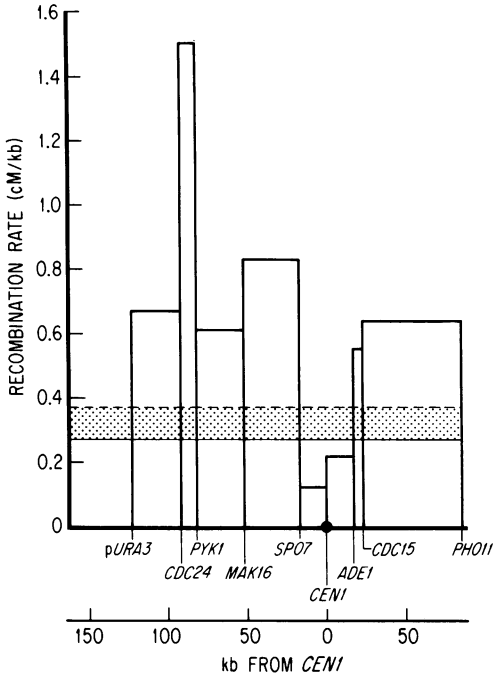

FIG. 3. Recombination rates between adjacent markers on chromosome I. Recombination rates (cM/kb) for adjacent genes that were genetically and physically mapped (see Table 3). The physical map is displayed on the abscissa. Average rate of recombination on chromosomes III and V (----) (6, 7) and for the entire genome (——) (4) are shown. Stippled area denotes that intermediate values were observed within this interval (4).

Genetic and physical distances were compared and found to be proportional over most of the chromosome. However, the recombination rate was higher at the previously described recombination hot spot near *CDC24* (8) and similar to two other *S. cerevisiae* chromosomes, lower, surrounding the centromere (6, 7, 23).

The average rate of recombination between marker pairs was 0.63 cM/kb, ≈50% higher than found for similarly analyzed regions on larger chromosomes (6, 7) and twice the value estimated for the whole genome (4). It is possible the higher recombination rate is due to several additional interspersed hot spots. In this case, the rate observed for each interval would represent an average of any heterogeneity. Further division of these intervals should reveal whether any heterogeneity exists. For now, we can conclude that genetic and physical distances are proportional over long (>7.5 kb) distances that are not near the centromere.

Using the average recombination rate found for chromosome I indicated that the genetic map length of this chromosome eventually should total 157 cM. This length is ≈50% more than before and is slightly larger than the total genetic map length predicted for chromosome III, which is 100 kb longer (350 kb × 0.37 cM/kb = 130 cM).

Using 101 cM as the total genetic map length, it was estimated that 5–6% of all asci contained nonrecombinant copies of chromosome I (ref. 5; D.B.K., unpublished results). It was suggested that a mechanism termed "distributive pairing" or "distributive disjunction" was responsible for the meiotic segregation of the nonrecombinant chromosomes (5). Distributive pairing or disjunction is defined by the meiotic segregation of either two nonhomologous chromosomes or two homologous chromosomes that have failed to undergo recombination (1, 26). This process was recently described in *S. cerevisiae* using synthetic chromosomes (5, 27, 28). However, the role of distributive pairing in segregating authentic *S. cerevisiae* chromosomes has not been established. With the longer genetic map length, the size of the

zero-crossover class for chromosome I is estimated as only 0.2–0.4% of the asci. This estimate is over an order of magnitude smaller than the previous estimate and suggests that virtually all chromosome I homologues recombine during meiosis. Indeed, preliminary results from 50 asci from a diploid strain multiply marked within the existing mapped region from p*URA3* to *PHO11* gave no zero-crossover copies of chromosome I. Thus, there may be little or no need for a distributive or nonexchange mechanism of homologue segregation for the smallest yeast chromosome. Furthermore, since chromosome I is the smallest, it should comprise the majority of the zero-crossover chromosomes. Therefore, the total number of meiotic cells with a nonrecombinant pair of any chromosome must be significantly lower than the previous estimate (5) of 10%. The four shortest chromosomes (I, III, VI, IX) should contribute most to the zero-crossover population. If the the zero-crossover class for each of these chromosomes is no larger than the size of the zero-crossover class for chromosome I, meiotic cells with a zero-crossover chromosome should total <0.8–1.6% (because of the low percentages, it can be estimated as <0.2–0.4% × 4 = <0.8–1.6%). Since map lengths for the other small chromosomes are probably underestimates, it is likely that the size of the zero-crossover class for all chromosomes will be reduced even further. Thus, while a mechanism for distributive pairing exists, its use in *S. cerevisiae* must be limited to a very small fraction of the meiotic cell population.

Table 4. Chromosome I homologues that fail to recombine

Interval	cM	Strain	Number of asci: Examined	Number of asci: e^0	Fraction e^0 asci	Estimated e^0 asci for all of chromosome I
p*URA3–CEN1*	80	DK387	58	2	0.035	
		DK391	46	2	0.043	
		Total	104	4	0.038	0.17%
CEN1–PHO11	47	DK386	147	30	0.204	
		DK388	121	23	0.190	
		Total	268	53	0.198	0.44%
p*URA3–PHO11*	127	—	—	—	0.008	0.80%

The size of the zero-crossover (e^0) class was predicted for chromosome I. e^0 asci were parental ditype and showed first-division segregation with respect to *trp1* for all markers in the interval. This number was corrected for two-strand double crossovers by subtracting the number of asci with four-strand doubles. Since there is no chromatid interference (ref. 25; D.B.K., unpublished observations), four-strand doubles equal two-strand doubles. An approximation for all of chromosome I can be obtained using joint probability. Expressed in percent, this number is calculated by raising the observed fraction of e^0 asci for the noted interval to the power of the total number of intervals of that size that are on the 157-cM-long chromosome I. This estimate must be considered maximal since it assumes that crossing-over in each interval is independent, whereas positive chiasma interference would lower the fraction of e^0 asci in most adjacent intervals even further. Fraction of asci in the e^0 class for p*URA3–PHO11* interval is the product of observed fractions of e^0 asci for intervals p*URA3–CEN1* and *CEN1–PHO11*. Percentage is 100 times this value. Intervals p*URA3–CEN1* and *CEN1–PHO11* are on opposite sides of the centromere and recombination in the two intervals is assumed to be independent. These latter calculations represent the maximum value in the unlikely event there is not recombination beyond p*URA3* and *PHO11*. Relevant genotypes of diploid strains analyzed are as follows:

DK387	DK381-9A	*MATa pyk1-102 0*	*trp1-1*	*ura3-1*
	W303-1B (pYY54)	*MATα PYK1* p*URA3*	*TRP1*	*ura3-1*
DK391	DK382-3D	*MATα cdc24-5 0*	*TRP1*	*ura3-1*
	MM1-1B (pYY54)	*MATa CDC24* p*URA3*	*trp1-1*	*ura3-1*
DK386	GX329-3C	*MATa ade1-1 cdc15-1 flo1 pho11::LEU2*	*TRP1*	*leu2-3,112*
	YS61	*MATα ADE1 CDC15 FLO1 PHO11*	*trp1-289*	*leu2-3,112*
DK388	GM1-9B	*MATα CDC15 pho11::LEU2*	*trp1-1*	*leu2-3,112*
	DK329-10A	*MATa cdc15-1 PHO11*	*TRP1*	*leu2-3,112*

Crossing-over on chromosome I during meiosis is ensured by a significantly enhanced rate of meiotic recombination. The factors that contribute to these enhanced rates are unknown. Enhancing the recombination rate on homologous chromosomes that are either small or have limited homology may serve as a general mechanism for ensuring their ability to segregate. Consistent with this idea, all *S. cerevisiae* chromosomes recombine more per kb than much larger mammalian chromosomes (29). In addition, human X and Y chromosomes, which must segregate from each other, recombine at a very high rate within the small homologous pseudoautosomal region (30). Elucidating the factors that control the rate of meiotic recombination should prove important for understanding mechanisms of pairing and segregation.

We thank Carol Newlon, Vincent Guacci, Frank Stahl, Joan Skurnick, and Bart Holland for stimulating discussions and/or valuable comments on the manuscript; Verity Letts and Rick Kramer for plasmid pAP18, the *pho11:LEU2* mutant in advance of publication, and their unpublished data that *PHO11* was located on the right arm of chromosome I; Diane Muhammadi, Brenda Lemon, and Linda Alegre for assistance in preparing the manuscript; and Dave Freschi for technical assistance. This work was supported by grants from the National Science Foundation and the North Atlantic Treaty Organization.

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