CORRIGENDUM

In the article by M. SHINOHARA, K. SAKAI, A. SHINOHARA and D. K. BISHOP (GENETICS **163**: 1273–1286) entitled “Crossover Interference in *Saccharomyces cerevisiae* Requires a *TID1/RDH54* and *DMC1*-Dependent Pathway,” errors were made in the statistical analysis of the data in Table 2.¹ Although these errors resulted in incorrect assignments of statistical significance to certain differences, correction of these errors does not alter any of the main conclusions of the original article. A corrected version of Table 2 is provided below. The two rightmost columns in the original table are replaced by three columns in the corrected table.

The errors in the original version of Table 2 were twofold. First, incorrect *P*-values were reported for results of contingency chi-square tests used to compare PD, NPD, and T frequencies between strains (the rightmost column of Table 2, Deviation from wild type, in the original article). The properly determined *P*-values suggest that the two strains used as controls for the *tid1* mutant strain, the congenic *TID1*⁺, and the isogenic *tid1-pTID1*⁺, displayed significantly different frequencies of PD, NPD, and T tetrads for 2 of 3 intervals examined. The *TID1*⁺ strain may carry a hidden modifier(s) of recombination, or the *tid1-pTID1*⁺ strain may express at a different level of Tid1 protein than the *TID1*⁺ strain.

The second problem with Table 2 of the original article was that the method used to evaluate the significance of differences between *NPD_{ob}/NPD_{ex}* ratios failed to account for the accuracies of the ratios themselves. In the revised version of Table 2 presented here, the method of R. LANDE (<http://www.groik.com/stahl/EquationsMapDistance.html>) was used to estimate the error associated with calculation of *NPD_{ob}/NPD_{ex}* ratios. To determine if differences between mutant and control data were significant, the *Z*-statistic and corresponding *P*-values were determined for each comparison. In the case of the chromosome V experiments, the comparison to the congenic control indicates significant differences in the *CAN1-URA3* and *URA3-HOM3* intervals but not the

HOM3-TRP2 interval, while comparison to the isogenic control indicates significant differences in the *URA3-HOM3* and *HOM3-TRP2* intervals but not the *CAN1-URA3* interval. In the case of the chromosome III experiments, all three intervals showed significant differences when *TID1*⁺ was compared to *tid1*. Overall, the *tid1* mutant showed significant reductions in interference for five of six intervals when compared to isogenic controls. No evidence for interference was found in the *dmc1 p2μ-RAD54* strain, although a low residual level of interference in the *URA3-LEU2* interval might have gone undetected.

We also reevaluated the data in Table 3 of the original article using a more rigorous statistical method (Foss, E. J., and F. W. STAHL, 1995, A test of a counting model for chiasma interference. GENETICS **139**: 1201–1209). The evaluation of data using this method did not alter the conclusions from the original analysis: both the *tid1* and the *dmc1 p2μ-RAD54* strains displayed coefficients of coincidence that were not significantly different from unity, in contrast to controls (not shown).

Finally, in our discussion of prior data supporting the hypothesis that crossover sites are chosen prior to synapsis, we erroneously stated on page 1281 that “late” recombination nodules had been observed prior to synapsis. The relevant observation is that, in *Sordaria*, synaptic initiation sites have a distribution similar to late nodules and chiasmata suggesting that sites of crossovers are determined prior to synapsis (ZICKLER, D., P. J. MOREAU, A. D. HUYNH and A. M. SLEZEC, 1992, Correlation between pairing initiation sites, recombination nodules and meiotic recombination in *Sordaria macrospora*. GENETICS **132**: 135–148). This and several other observations (reviewed by BISHOP, D. K., and D. ZICKLER, 2004, Early decision: meiotic crossover interference prior to stable strand exchange and synapsis. CELL **117**: 9–15) are consistent with our conclusion in the original article that “interference acts at the strand invasion stage of recombination” (p. 1273).

¹Correction received March 21, 2004.

TABLE 2
Crossover frequency and crossover interference

Strain ^a	Interval ^b	No. PD	No. T	No. NPD	Total ^c	Map distance ^d	Fold increase ^e	NPD _{ob} /NPD _{ex} ±SE ^f	NPD _{ob} /NPD _{ex} differences (P) ^g	Contingency chi square (P) ^h
Chromosome V										
<i>TID1</i>	<i>CAN1-URA3</i>	409	923	34	1366	41	—	0.19 ± 0.02	—	—
	<i>URA3-HOM3</i>	455	859	52	1366	43	—	0.34 ± 0.05	—	—
	<i>HOM3-TRP2</i>	1094	268	4	1366	11	—	0.52 ± 0.26	—	—
<i>tid1</i>	<i>CAN1-URA3</i>	361	628	38	1027	42	1.0	0.37 ± 0.07	0.004/0.2	0.003/0.002
	<i>URA3-HOM3</i>	363	608	56	1027	46	1.1	0.62 ± 0.10	0.005/0.008	0.06/<0.0001
	<i>HOM3-TRP2</i>	617	386	24	1027	26	2.4*	0.95 ± 0.20	0.10/0.005	<0.0001/<0.0001
<i>tid1- YCpTID1</i>	<i>CAN1-URA3</i>	331	797	47	1175	46	1.1	0.31 ± 0.01	—	0.07
	<i>URA3-HOM3</i>	320	804	51	1175	47	1.1	0.38 ± 0.02	—	0.004
	<i>HOM3-TRP2</i>	828	342	5	1175	16	1.4	0.32 ± 0.14	—	<0.0001
Chromosome III										
<i>TID1</i>	<i>URA3-LEU2</i>	813	316	2	1131	14	—	0.14 ± 0.10	—	—
	<i>LEU2-MAT</i>	509	594	28	1131	34	—	0.42 ± 0.08	—	—
	<i>URA3-MAT</i>	346	728	57	1131	47	—	0.40 ± 0.06	—	—
<i>tid1</i>	<i>URA3-LEU2</i>	1002	186	5	1193	9.1	0.65	1.23 ± 0.55	<0.0001	<0.0001
	<i>LEU2-MAT</i>	462	670	61	1193	43	1.3	0.70 ± 0.10	0.0006	0.0002
	<i>URA3-MAT</i>	425	681	87	1193	50	1.1	0.94 ± 0.12	0.0003	0.0008
<i>DMC1- YEpRAD54</i>	<i>URA3-LEU2</i>	919	276	3	1198	12	—	0.32 ± 0.18	—	—
	<i>LEU2-MAT</i>	529	641	28	1198	34	—	0.37 ± 0.07	—	—
	<i>URA3-MAT</i>	379	772	47	1198	44	—	0.31 ± 0.05	—	—
<i>dmc1- YEpRAD54</i>	<i>URA3-LEU2</i>	1038	197	4	1239	8.9	0.74	0.91 ± 0.46	0.10	<0.0001
	<i>LEU2-MAT</i>	599	581	59	1239	38	1.1	1.10 ± 0.16	<0.0001	0.0001
	<i>URA3-MAT</i>	542	617	80	1239	44	1.0	1.27 ± 0.16	<0.0001	<0.0001

* Significant differences between mutant and wild type.

^a Strains used were as follows. Chromosome V: *TID1*, MSY1172 × MSY1174; *tid1*, MSY622 × MSY626; *tid1-YCpTID1*, MSY626 × MSY818. Chromosome III: *TID1*, NKY1543 × MSY1153; *tid1*, MSY962 × MSY964; *DMC1-YEpRAD54*, MSY1176 × MSY 1178; *dmc1-YEpRAD54* (MSY1068 × MSY 1072). *TID1* and *DMC1-YEpRAD54* controls carry the same number of functional copies of the amino acid biosynthetic gene used to mark the *tid1* and *dmc1* deletion alleles as do strains carrying the deletion alleles (e.g., if the mutant strain is *tid1Δ::LEU2/tid1Δ::LEU2 leu2/leu2*, the wild-type control is *TID1/TID1 LEU2/LEU2*). Controls were designed in this way because previous work showed amino acid auxotrophies can alter gene conversion frequency (ABDULLAH, M. F., and R. H. BORTS, 2001, Meiotic recombination frequencies are affected by nutritional states in *Saccharomyces cerevisiae*. Proc. Natl. Acad. Sci. USA **98**: 14524–14529). Additional control experiments showed that absence of the amino acid biosynthetic genes used as markers in this study altered conversion frequency slightly, but did not substantially alter crossover frequency or crossover interference (data not shown).

^b For chromosome V, *URA3* refers to the normal *URA3* locus. For chromosome III, *URA3* and *LEU2* refer to insertions of those genes at the *HIS4* locus (see Figure 1).

^c Only four-spore-viable tetrads that did not show non-Mendelian segregation were counted.

^d See MATERIALS AND METHODS for method of calculation.

^e Ratio of map distance for mutant over wild-type control. (Note that the *DMC1-YEpRAD54* strain is the control for *dmc1-YEpRAD54*.)

^f The ratio of NPD_{ob} to NPD_{ex} (see MATERIALS AND METHODS) is given with the standard error calculated by the method of R. LANDE (<http://www.groik.com/stahl/EquationsMapDistance.html>).

^g P-values from comparisons of NPD_{ob}/NPD_{ex} between mutant and control strains. These values were obtained from the Z statistic that was calculated from the values in the NPD_{ob}/NPD_{ex} ± SE column. In the case of the chromosome V *tid1* data, two numbers separated by a “/” are given; the first number corresponds to the result of the comparison of *tid1* to the congenic *TID1*⁺ strain, and the second corresponds to the result of comparison of *tid1*, the isogenic *tid1-pTID1* strain.

^h P-values from comparison of numbers of PD, T, and NPD frequencies between mutant and control strains using a contingency chi-square test. In the case of the chromosome V *tid1* data, two numbers separated by a “/” are given; the first corresponds to the result of the comparison of *tid1* to the congenic *TID1*⁺ strain, and the second corresponds to the result of comparison of *tid1*, the isogenic *tid1-pTID1* strain.