use a plasmid carrying *URA3* and to select for uracil-independent transformants. It was then found that partial repair of the gap in *HIS3* sometimes occurred, probably resulting from enlargement of the gap beyond the region of homology to yeast DNA. Gene conversion was also observed in DNA flanking a gap. Thus, transformants were occasionally found that had a functional chromosomal *HIS3* gene but an unstable, that is, non-integrated, plasmid. This derivation of $HIS3^+$ without gap repair implied transfer from plasmid to chromosome of the normal allele of the chromosomal *his3* mutation, and was attributed to the formation of heteroduplex DNA and subsequent mismatch repair. On the other hand, several experiments indicated that linear plasmid DNA could be degraded and then the enlarged gap repaired. It was evident that conversion in regions flanking the original gap might occur by either mechanism.

The recombination model proposed by Szostak *et al.*[2] postulates that recombination is initiated by a duplex break, enlarged to form a gap. The gap is repaired by the process already outlined. This results in conversion for mutant sites within the gap and in heteroduplex DNA for mutant sites flanking the gap. Szostak *et al.*[2] propose that events in these heteroduplex regions follow the Meselson-Radding model[7]. So the new hypothesis can be visualised as a region of duplex repair flanked on each side by regions conforming to the predictions of the Meselson-Radding model.

As Szostak *et al.*[2] point out, their hypothesis can explain why yeast mutants, including large deletions and insertions, show parity in frequency of conversion to wild type and to mutant: they suggest that the duplex gap is much enlarged, so that most conversion in yeast results from gap repair rather than mismatch correction. Parity would arise if there was an equal chance of a chromatid of either parentage suffering a duplex break. The hypothesis also explains why recombination initiation sites function as a recipient of genetic information from the other recombining chromatid rather than as a donor of information to it. There is evidence for this from three sources: the M26 mutant in *Schizosaccharomyces pombe*[8], *cog* in *Neurospora crassa*[9] and YS17 in *Sordaria brevicollis*[10] (for review see ref. 11).

For many years Stahl[12] has believed that conversion in yeast arises by local DNA synthesis not triggered by mismatched bases. The duplex-break repair model[2], by combining this hypothesis with that of mismatch correction, surmounts the objections[13] to Stahl's hypothesis but at the price of a model that will be difficult to test. There are two reasons for this. In the first place, many of its predictions are the same as those of the Meselson-Radding model[7], some aspects of which already have substantial support[11,14]. Second, the patterns of polarity in recombination within eukaryotic genes have led to the conclusion that recombination is normally initiated outside the genes. The postulated duplex breaks are therefore expected to occur in regions lacking genetic markers. Szostak *et al.*[2] suggest that a direct search be made for double-strand breaks arising during the period of commitment to meiotic recombination. □

Harold L.K. Whitehouse is at the Botany School, University of Cambridge, Downing Street, Cambridge CB2 3GA.

1. Resnick, M. & Martin, P. *Molec. gen. Genet.* **143**, 119 (1976).
2. Szostak, J.W., Orr-Weaver, T.L., Rothstein, R.J. & Stahl, F.W. *Cell* **33**, 25 (1983).
3. Orr-Weaver, T.L., Szostak, J.W. & Rothstein, R.J. *Proc. natn. Acad. Sci. U.S.A.* **78**, 6354 (1981).
4. Prakash, S., Prakash, L., Burke, W. & Montelone, B. *Genetics* **91**, 31 (1980).
5. Game, J., Zamb, T., Braun, R., Resnick, M. & Roth, R. *Genetics* **91**, 51 (1980).
6. Orr-Weaver, T.L. & Szostak, J.W. *Proc. natn. Acad. Sci. U.S.A.* **80**, 4417 (1983).
7. Meselson, M. & Radding, C. *Proc. natn. Acad. Sci. U.S.A.* **72**, 358 (1975).
8. Gutz, H. *Genetics* **69**, 317 (1971).
9. Catcheside, D.G. *The Genetics of Recombination* (Arnold, London, 1977).
10. MacDonald, M.V. & Whitehouse, H.L.K. *Genet. Res.* **34**, 87 (1979); *Genet. Res.* 41, 155 (1983).
11. Whitehouse, H.L.K. *Genetic Recombination: understanding the mechanisms* (Wiley, Chichester & New York, 1982).
12. Stahl, F.W. *Genetics* **61**, Suppl. 1 (1969); *Genetic Recombination: thinking about it in Phage and Fungi* (Freeman, San Francisco, 1979).
13. Holliday, R. & Whitehouse, H.L.K. *Molec. gen. Genet* **107**, 85 (1970).
14. Paquette, N. & Rossignol, J.-L. *Molec. gen. Genet.* **163**, 313 (1978).

Astronomy

IRAS circulars 4 & 5

The source name consists of four parts: (1) the letters 'IRAS' to indicate the origin; (2) the right ascension (RA) in hours and minutes, seconds omitted; (3) declination (Dec) in decimal degrees, multiplied by 10 and then truncated (thus +32° 42.3 becomes +327); (4) an appendix starting with 'P' and followed by the number of the circular; this appendix stresses that the data are preliminary. Position is given at Equinox 1950.0.

Source IRAS	RA (1950) h min s	Dec (1950) deg arc min	Flux density (Jy) 12 μm	24 μm	60 μm	100 μm
1608−185P04	16 08 38	−18 30.7	8.7	15	22	22
1623+030P04	16 23 33	+03 01.2	<0.2	0.66	3.8	5.5
1624+116P04	16 24 25	+11 41.5	<0.3	<0.4	2.6	7.2
1626+037P04	16 26 13	+03 43.4	<0.2	<0.3	2.2	3.6
1627+031P04	16 27 49	+03 07.4	<0.2	<0.3	1.7	2.5
1628+041P04	16 28 27	+04 11.4	<0.3	0.99	7.8	16
1639−096P04	16 39 56	−09 37.6	<0.4	1.1	8.7	16
1640−188P04	16 40 58	−18 51.7	<0.2	4.4	4.2	<3
1645+033P04	16 45 28	+03 23.5	<0.2	<0.3	2.2	3.5
1647−113P04	16 47 37	−11 22.9	1.6	5.2	2.7	<4

Source IRAS	RA (1950) h min s	Dec (1950) deg arc min	Flux density (Jy) 12 μm	25 μm	60 μm	100 μm
1650+024P04	16 50 28	+02 29.0	0.51	3.7	26	34
1651+305P04	16 51 41	+30 31.0	<0.2	<0.3	1.9	4.1
1700−234P04	17 00 40	−23 28.6	4.8	6.6	1.9	<3
1705−022P04	17 05 33	−02 16.5	6.6	6.1	1.2	<2
1709−165P04	17 09 22	−16 33.5	8.7	7.8	0.99	<3
1710−032P04	17 10 14	−03 12.5	<0.3	1.5	3.0	<2
1713−102P04	17 13 50	−10 17.5	0.57	2.2	19	31
1717−087P04	17 17 09	−08 44.0	38	34	6.0	<3
1718+113P04	17 18 02	+11 22.0	<0.2	0.40	2.3	3.7
1720+129P04	17 20 49	+12 57.1	<0.4	<0.2	1.9	3.2

Source IRAS	RA (1950) h min s	Dec (1950) deg arc min	Flux density (Jy) 12 μm	25 μm	60 μm	100 μm
0441+727P05	04 41 52	+72 46.2	<0.3	1.2	4.6	6.8
0449+781P05	04 49 44	+78 06.6	<0.6	0.64	6.6	12
0506+536P05	05 06 07	+53 38.7	0.34	1.7	9.4	16
0507+471P05	05 07 00	+47 07.0	0.58	3.0	17	38
0507+528P05	05 07 19	+52 48.9	200	290	69	32
0508+796P05	05 08 16	+79 36.7	<0.3	0.62	6.0	11
0512+531P05	05 12 52	+53 08.2	<0.4	0.67	3.3	6.6
0512+514P05	05 12 59	+51 28.7	<0.3	1.0	7.2	9.0
0513+581P05	05 13 28	+58 11.1	<0.3	0.47	5.2	13
0516+432P05	05 16 39	+43 15.3	0.33	0.79	6.3	11
0517+428P05	05 17 17	+42 49.8	0.58	0.73	4.5	14
0522+416P05	05 22 07	+41 39.2	2.6	18	140	190

Source IRAS	RA (1950) h min s	Dec (1950) deg arc min	Flux density (Jy) 12 μm	25 μm	60 μm	100 μm
0531−219P05	05 31 13	−21 58.8	0.42	0.77	9.7	32
0533+541P05	05 33 45	+54 08.0	<0.2	0.49	5.4	8.3
0536+467P05	05 36 09	+46 44.2	170	200	77	34
0538−220P05	05 38 06	−22 01.7	<0.2	<0.2	2.1	4.2
0540−240P05	05 40 57	−24 05.2	<0.3	0.52	2.8	4.4
0541+586P05	05 41 24	+58 40.8	0.60	0.87	16	40
0547−303P05	05 47 47	−30 18.7	<0.2	<0.3	3.7	8.3
0552−327P05	05 52 01	−32 45.1	<0.2	<0.4	1.8	4.2
0600+477P05	06 00 22	+47 47.9	34	31	5.1	<10
0610+668P05	06 10 39	+66 51.2	<0.3	<0.4	3.8	8.6
0623+744P05	06 23 57	+74 28.6	<0.2	0.88	5.4	8.3
0705+719P05	07 05 32	+71 55.0	<0.2	<0.3	2.4	6.1
0706+718P05	07 06 45	+71 50.0	<0.4	0.42	4.1	10

0028-0836/83/500646-01$01.00