

**Table 2** Mean frequencies of  $F_1$  sterility and second chromosome male recombination from reciprocal crosses between the MMIIa marker stock and five wild-type strains

Strain	Cross A*				Cross B†			
	$F_1$ female sterility %	$F_1$ male sterility %	Total progeny	Minimum (male) recombination %	$F_1$ female sterility %	$F_1$ male sterility %	Total progeny	Minimum (male) recombination %
Oregon R-C, 1925	6.7	0	1,825	0	0	0	1,710	0
Cranston, Rhode Island, 1964	46.0	57.8	3,020	0.83	0	0	4,449	0.07
Amherst, Massachusetts, 1966	ND	6.8	3,504	0.46	0	0	3,221	0
Harwich, Massachusetts, 1967	68.3	41.7	3,731	1.13	4.2	5.0	1,802	0.11
Weymouth, Rhode Island, 1974	20.0	9.1	3,451	0.32	0	2.0	3,024	0.07

\* MMIIa female  $\times$  wild-type male.

† Wild-type female  $\times$  MMIIa male.

ND, not determined.

case. Sterility is measured as the percentage of  $F_1$  individuals which produce no offspring when mated with three or more members of the opposite sex. This is a conservative estimate because many of the individuals which were classified as 'fertile' in cross A produced considerably fewer progeny than their counterparts in cross B.

Male recombination and sterility continued through at least two succeeding generations of suitable back-crossing, at levels undiminished from those in the  $F_1$ . In each of three generations, males heterozygous for Cranston and MMIIa, were back-crossed with MMIIa virgin females and their progeny tested as previously described. The results (Table 3) suggest that male recombination and sterility arise from interactions between chromosomes of some strains and the cytoplasm of others, each combination possibly exhibiting its own unique characteristics.

Reciprocal differences in both male recombination and sterility are not restricted to crosses with the MMIIa stock. Strains which produced high male recombination in the third chromosome (Table 1) showed greatly reduced frequencies in reciprocal crosses with the *rucuca* marker stock: the numbers of independent male recombination events were 0 in 1,848 progeny with Amherst, 1 in 5,678 (0.2%) with Cranston and 9 in 3,623 (0.25%) with Harwich.

Although *rucuca* crosses gave progeny with normal fertility irrespective of the direction of the cross, matings of the Cranston strain with several other, widely-used, balancer and marker stocks have yielded large reciprocal differences in sterility: for example, with the multiple balancer stock H-41 (ref. 11),  $F_1$  sterility was 35.8% in cross A and 0.8% in cross B; with the *Basc* marker stock, over 50% sterility was observed in  $F_1 \times F_1$  matings. Picard *et al.*<sup>12</sup> observed seemingly similar interactions between strains, resulting in female sterility.

**Table 3** Mean frequencies of sterility and male recombination in three generations of backcrossing Harwich 1967/MMIIa males to MMIIa females

Generation	Female sterility %	Male sterility %	Total progeny	Minimum male recombination %
1	68.3	41.7	3,731	1.13
2	57.1	51.2	1,870	1.07
3	47.5	49.2	3,270	1.01

In summary, we have observed non-trivial frequencies of male recombination in  $F_1$  males of crosses between second and third chromosome marker stocks and wild-type strains which had recently been collected from natural populations. Longer-established laboratory stocks did not exhibit this phenomenon to any significant extent. In susceptible strains, male recombination seems to be dependent on the direction of the initial parental cross. In at least three crosses, male recombination and sterility were highly correlated, both showing large reciprocal

differences in the same direction.

These results may have importance, not only for the understanding of the aetiology of these exceptional phenomena but may also have implications for reproductive isolation between sympatric populations. Moreover, if cytoplasm-chromosome interactions between laboratory marker and balancer stocks and other strains prove to be common, greater care will be necessary in the design and interpretation of experiments using such stocks.

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## Errata

In the article "Evidence for a Y chromosomal contribution to an aggressive phenotype in inbred mice" by M. K. Selmanoff, J. E. Jumonville, S. C. Maxson and B. E. Ginsburg (*Nature*, **253**, 529; 1975) the column headings in Table 1 should read DBA/1 Fathers and C57BL/10 Fathers respectively, in both studies.

In the article "Earthquake simulation by nuclear explosions" by O. C. Kolar and N. L. Pruvost (*Nature*, **253**, 242; 1975) the following corrections should be made. Under the heading 'Seismic monitoring' the first sentence of para. 3 should read, "P-wave trains from an earthquake are often extended in time whereas those from an explosion tend to be more compact." Under the heading 'Seismic evasion', para. 11, line 1 should read "... Fourier spectra of the composite of ...". Under the heading 'Practical considerations', para. 2, line 4 should read "operation; including the conceptual design of a ..." and para. 10, line 5 should read "... depth is in the range of 120W<sup>1/3</sup> m (ref. 5)."