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Host recombination is dependent on the degree of parasitism

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Parasites and hosts are involved in a continuous coevolutionary process leading to genetic changes in both counterparts. To understand this process, it is necessary to track host responses, one of which could be an increase in sex and recombination, such as is proposed by the Red Queen hypothesis. In this theoretical framework, the inducible recombination hypothesis states that B-chromosomes (genome parasites that prosper in natural populations of many living beings) elicit an increase in host chiasma frequency that is favoured by natural selection because it increases the proportion of recombinant progeny, some of which could be resistant to both B-chromosome effects and B-accumulation in the germline. We have found a clear parallelism between host recombination and the evolutionary status of the B-chromosome polymorphism, which provides explicit evidence for inducible recombination and strong support for the Red Queen hypothesis.

Keywords: Red Queen hypothesis; recombination; coevolution; parasitism; B-chromosomes

1. INTRODUCTION

Host–parasite coevolution leads to continuous genetic changes due to prolonged selection for adaptation and counter-adaptation, with an increase in sex and recombination such as that predicted by the Red Queen hypothesis (Van Valen 1973; Jaenike 1978; Hamilton 1980). The coevolutionary interactions between parasites and hosts may select for an increase in host sexual reproduction, because sex and genetic recombination result in genetically different progeny that are expected to have a lower risk of being infected (Bell 1982). B-chromosomes are genome parasites that persist in natural populations of many living beings. Their accumulation (drive) through non-Mendelian mechanisms counteracts their elimination by selection due to harmful effects on carriers (for a recent review, see Camacho *et al.* 2000). These two forces (drive and selection) can reach an equilibrium, and thus maintain a constant B-frequency through time, as is assumed by the parasitic model of B-evolution. It is also possible that B-frequency changes through time as a result of a coevolutionary arms race between the parasitic B-chromosome and the host A-chromosomes, leading to an endless succession of evolutionary stages going from parasitic (i.e. driving) to neutralized (i.e. non-driving) and newly parasitic Bs (Camacho *et al.* 1997). It is therefore expected that B-effects can be dependent on the evolutionary status of the B-polymorphism.

One of the most reported B-chromosome effects is on chiasma frequency and, therefore, recombination, since chiasmata are the cytological visualization of crossing over, the physical phenomenon that produces intra-chromosome genetic recombination (Janssens 1909). It is well known that chiasma frequency depends on genetic and environmental factors (Nolte *et al.* 1969; Shaw 1971, 1972). In addition, B-chromosomes are frequently associated with increases in chiasma frequency, although cases of absence of effects, and even chiasma frequency decrease associated with B-presence have also been reported (for a review, see Jones & Rees 1982). These contradictory observations have been reported at both inter- and intraspecific levels, and thus no consensus has been reached regarding the ultimate causes of B-chromosome effects on chiasma frequency. A number of hypotheses have been proposed to explain chiasma effects of B-chromosomes:

(i) *Adaptation*: B-chromosome-induced chiasma effects are adaptive, so that an increase in chiasma frequency increases genetic variability and thus enables the population to evolve more rapidly (John & Hewitt 1965; Hewitt & John 1967), whereas a decrease conserves variability and helps to maintain adaptation (Fontana & Vickery 1973). This hypothesis has been strongly criticized by Bell & Burt (1990) because it implies that parasites are selected on the grounds of the favourable effects that they have on hosts.

(ii) *Passive effects*: chiasma effects represent some necessary mechanical or physiological effect of additional or exotic DNA (Bell & Burt 1990). It does not explain the contradictory results between cultivars or populations of a same species, e.g. rye (Jones & Rees 1967; Zečević & Paunović 1969), and the grasshoppers *Myrmeleotettix maculatus* (John & Hewitt 1965), *Phaulacridium vittatum* (John & Freeman 1975; Westerman & Dempsey 1977) and *Eyprepocnemis plorans* (Camacho *et al.* 1980; Henriques-Gil *et al.* 1982).

(iii) *Inducible recombination*: considering B-chromosomes as parasites, they may select for increased host

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recombination (through chiasma effects), which would create recombinant progeny, some of which could be resistant to the accumulation of Bs in the germline (Bell & Burt 1990). Under this hypothesis, parasitized individuals should show greater rates of recombination than unparasitized individuals in the same population, as a result of selection for genes which increase the rate of recombination only when some stimuli associated with parasite activity are detected. Such a response has been called 'inducible recombination', and is a special case of the Red Queen hypothesis that predicts increased recombination when conditions are poor.

(iv) *Selfish purposes*: a variety of B-effects on chiasma frequency in different species may serve the selfish purposes of each specific B. Carlson has proposed three such selfish functions (Carlson 1994). First, effects of B-chromosomes on A-chromosome chiasma frequency in maize might be explained as a side-effect of increased pairing between Bs to reduce B meiotic loss. This explanation could be valid for B-chromosomes with accumulation mechanisms based on B-bivalent formation (e.g. in maize; see Carlson & Roseman (1992)), but not for B-chromosomes whose accumulation is achieved through B-univalents (e.g. meiotic drive during oogenesis in the grasshopper *Myrmeleotettix maculatus*; see Hewitt (1976)). This led Carlson to propose a second selfish function for chiasma effects, where chiasma frequency increase in A-chromosomes would result from the blocking of B-bivalent formation to best serve each univalent B-interest. The third selfish function for chiasma effects would be in restricting A–B pairing to facilitate B-evolution from an A-chromosome.

The grasshopper *Eyprepocnemis plorans* shows a very widespread B-chromosome polymorphism that is, presumably, extended to the entire Mediterranean region (Camacho *et al.* 1997; Bakkali *et al.* 1999) and reaches the Caucasus (Bugrov *et al.* 1999). B-chromosomes are highly mutable, as is indicated by the finding of more than 50 B-variants differing in morphology, size and C-banding pattern. These variants, however, seem to be composed of the same two types of repetitive DNA, i.e. a 180 bp tandem repeat DNA and ribosomal DNA (Cabrero *et al.* 1999). The most remarkable aspect of the *E. plorans* B-chromosome polymorphism is its population dynamics, with the existence of B-chromosomes passing through successive stages of parasitism (with drive) and neutralization (with no drive). In parallel, the load conferred by the B to the host genome (i.e. B's virulence) seems to vary depending on the existence of drive for the B-chromosome, as is indicated by the decrease in egg fertility shown by female hosts carrying driving Bs (Zurita *et al.* 1998). This is not apparent in hosts carrying neutralized Bs (Camacho *et al.* 1997). We analyse chiasma frequency on the A-chromosomes in the presence of three B-chromosome variants (B_1, B_2 and B_{24}) that are at different evolutionary stages: neutralized (B_2), partially neutralized (B_1) and parasitic (B_{24}). The results have shown that recombination in the host genome is dependent on the evolutionary status of the B-chromosome.

2. MATERIAL AND METHODS

A total of 481 males of the grasshopper *E. plorans* were collected from six Moroccan and three Spanish populations (table 1). Upon arrival in the laboratory, testes were immediately fixed in 3 : 1 ethanol–acetic acid and stored at 4 °C until study. The type of B-chromosome carried by each male was determined based on the C-banding pattern, using the procedure described in Camacho *et al.* (1991). Chiasmata were scored in preparations of two testis follicles squashed in acetic orcein. The number of chiasmata per cell were scored at 10 diplotene primary spermatocytes per male. The mean cell chiasma frequency was transformed to natural logarithms to improve adjustment to a normal distribution. In Morocco, the only three males with 2Bs were added to the 1B class for analysis. In Salobreña males, we measured several other parameters, including (i) the number (transformed to natural logarithm) of attached ectoparasitic mites (genus *Podapolipus*); (ii) the presence of a heterochromatic supernumerary chromosome segment located in the smallest autosome (which is composed of a tandem DNA repeat also present in the B chromosome); and (iii) the somatic and gonadal conditions (calculated as the standardized residuals of a regression of the somatic or gonadal weight on thorax length). We used the presence of the supernumerary chromosome segment to test proposition (iii) (passive B-effects), and somatic and gonadal conditions to test possible environmental effects. Statistical analyses were performed by means of parametric tests wherever possible (one-way and two-way ANOVA and ANCOVA), although non-parametric Kruskal–Wallis ANOVA was also employed.

3. RESULTS

(a) *Analysis per B-chromosome evolutionary status*

In the Spanish population of Torrox, a parasitic B-variant (B_{24}) emerged recently and has completely replaced the former B (the neutralized B_2) (Zurita *et al.* 1998). Mean cell chiasma frequency increased with the number of B_{24}-chromosomes ($F = 7.75$, d.f. = 2,51, $p = 0.001$). B_{24} showed an overall transmission ratio of almost 0.7 through females in 1992 (Zurita *et al.* 1998), although a recent analysis has shown this to be close to 0.5 in 1998 (J. M. Corral, J. A. Mesa, J. Cabrero, M. Bakkali, M. D. López-León, F. Perfectti and J. P. M. Camacho, unpublished data). In parallel with drive decrease, the effect of B_{24} on mean chiasma frequency decreased over years, being significant in 1992 ($F = 26.65$, d.f. = 2,9, $p = 0.000\,17$) but not in 1997 ($F = 3.23$, d.f. = 2,18, $p = 0.063$) or 1998 ($F = 1.38$, d.f. = 2,24, $p = 0.272$).

In Morocco, an analysis of B-transmission on individuals collected at 1997 indicated that B_1 was driving in some populations but not in others (Bakkali *et al.* 2002), which suggests that it is in the process of neutralization. A two-way ANOVA showed that mean cell chiasma frequency increased in the presence of B-chromosomes ($F = 26.80$, d.f. = 1,186, $p = 0.000\,001$) and varied among populations ($F = 4.21$, d.f. = 5,186, $p = 0.0012$).

In the Spanish population of Salobreña, where the predominant B-chromosome (B_2) is neutralized, as was shown by López-León *et al.* (1992) in females collected in 1990, mean cell chiasma frequency also increased with the number of B-chromosomes ($F = 5.16$, d.f. = 3,197, $p = 0.0019$), but the effect was apparent only in 2B and 3B males (figure 1). In this population, we also analysed the possible effects of *Podapolipus* mites, the presence of a supernumerary chromosome segment (made of a repeti-

Table 1. Materials analysed.

population	country	year	B-type	number of B-chromosomes 0	1	2	3	total
Smir	Morocco	1997	B1	12	20	2	0	34
Larache	Morocco	1997	B1	25	10	0	0	35
Ainlabid	Morocco	1997	B1	25	8	0	0	33
Tatouft	Morocco	1997	B1	20	9	1	0	30
Frain	Morocco	1997	B1	25	10	0	0	35
Sodea	Morocco	1997	B1	20	11	0	0	31
Claras	Spain	1999	—	22	0	0	0	22
Salobreña	Spain	1992	B2	72	66	54	9	201
Torrox	Spain	1992	B24	4	5	3	0	12
		1997	B24	4	14	3	0	21
		1998	B24	1	16	10	0	27
total				230	169	73	9	481

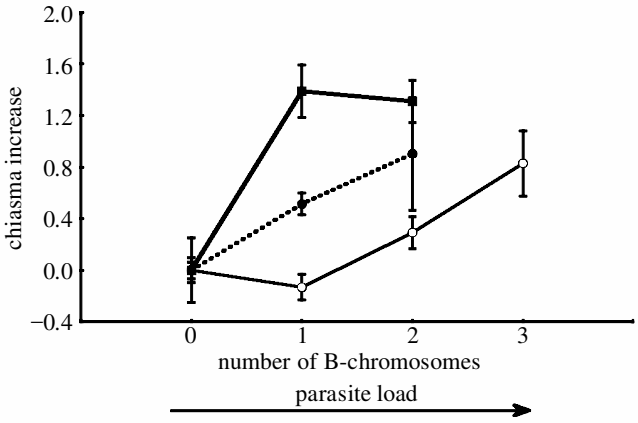

Figure 1. Chiasma increase (the difference between the mean cell chiasma frequency of B-carrying males and that of 0B males) with B-chromosome number (parasite load), for three B-chromosome types at different evolutionary stages, driving B_{24} (filled squares), neutralized B_2 (open circles) and partially neutralized B_1 (filled circles).

tive DNA also present in all the B-variants analysed), and somatic and gonadal conditions. Mean chiasma frequency only depended significantly on the number of B-chromosomes (table 2).

The net increase in number of chiasmata associated with the presence of B-chromosomes showed clearly different patterns in Torrox, Morocco and Salobreña, in parallel to the driving status (from driving to neutralized Bs) of the B-chromosome in each site (figure 1). The most parasitic B (B_{24}) showed the highest increase in chiasma frequency (more than one chiasma per cell), and a single B_{24} is enough to produce the effect. The neutralized B (B_2) does not produce any effect at a single dose, suggesting that drive neutralization has been accompanied by a weakening of chiasma effects. Finally, an intermediate situation is shown by the Moroccan B_1, which still seems to be in the process of neutralization (Bakkali *et al.* 2002). A single B in Morocco was associated with an increase of 0.5 chiasma per cell, and two Bs with almost one.

(b) *Comparison among Bs at different evolutionary stages*

An additional population lacking B-chromosomes (Claras, Albacete, Spain) was included for analysis. Mean chiasma frequency was higher in the populations harbouring parasitic Bs (B_1 and B_{24}), lower in the population with the neutralized B_2 and even lower in the non-B population (Kruskal–Wallis ANOVA $H = 23.98$, d.f. = 3, $n = 481$, $p < 0.0001$; figure 2).

4. DISCUSSION

The effects of *E. plorans* B-chromosomes on chiasma frequency were first reported by Camacho *et al.* (1980), who observed an increase in cell chiasma frequency in the five populations analysed. Later, it was shown that the effect of B-chromosomes depends on the genetic background of the individuals carrying them (Henriques-Gil *et al.* 1982). An analysis of chiasma frequency in males and females, from four natural populations containing B-chromosomes, showed the absence of B-effects on chiasma frequency in either sex (Cano *et al.* 1987). Our present results have shown that B-chromosomes increase chiasma frequency in A-chromosomes, although the intensity of the effect depends on the evolutionary status of the polymorphism; the more driving the B-chromosome, the more intense the increase in chiasma frequency (see figure 2). This provides an explanation for the interpopulation differences in chiasma effects of B-chromosomes previously reported in *E. plorans* (see above) and other species (see § 1). Such differences could be due to varying levels of drive (and probably virulence; see Zurita *et al.* (1998); Bakkali *et al.* (2002)) of B-chromosomes in different populations of the same species, due to the coevolutionary arms race between A- and B-chromosomes.

The effects observed with B_{24} in Torrox (B_{24} increasing mean cell chiasma frequency significantly only when it showed drive, i.e. in the 1992 sample) demonstrate a close relationship between B-accumulation and chiasma effects. A similar relationship can be deduced from our intra- and interpopulation analyses. The variation in intensity of chiasma effects correlated with B-chromosome drive contradicts the adaptive hypothesis, since, if chiasma effects were adaptive, there would be no reason to lose the benefit of increased recombination in the offspring in parallel to B-drive suppression.

Chiasma effects of B-chromosomes might not be based on DNA composition, because: (i) the three B-chromosomes analysed (showing variable effects) are made of the

Table 2. ANCOVA analysing mean cell chiasma frequency in the Salobreña population in respect to the number of B_2 chromosomes, the number of mites, somatic condition, gonadal condition and presence of a supernumerary chromosome segment (SCS).

variable	type	d.f.	mean squares	*F*	*p*
number of mites (ln)	covariate	1	0.0058	1.78	0.1845
somatic condition	covariate	1	0.0013	0.39	0.5322
gonadal condition	covariate	1	0.0025	0.77	0.3812
{1} number of Bs	fixed	3	0.0178	5.40	0.0014
{2} SCS	fixed	1	0.0020	0.60	0.4400
1 × 2	fixed	3	0.0027	0.81	0.4905
error		165	0.0033		

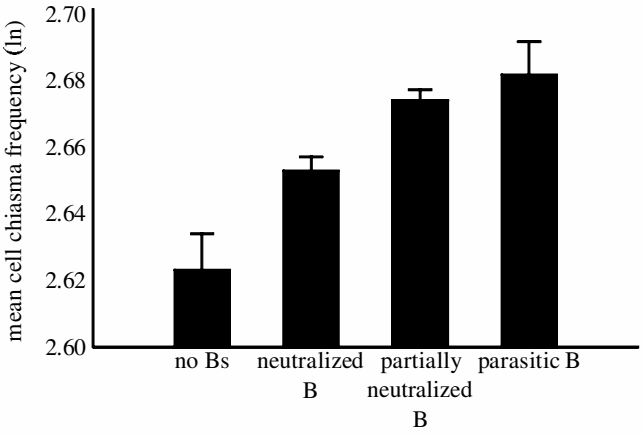

Figure 2. Mean cell chiasma frequency in four natural populations, one lacking B-chromosomes (no Bs) and three harbouring B-chromosome at different evolutionary stages: parasitic (B_{24} at Torrox), partially neutralized (B_1 at Morocco) and neutralized (B_2 at Salobreña), implying different degrees of drive and cost to the host (virulence). Significant differences were found among these populations.

same two types of repetitive DNAs (Cabrero *et al.* 1999); and (ii) the 180 bp tandem repeat is also contained in the supernumerary segment of the smallest autosome, which does not produce chiasma effects. This result undermines the hypothesis of passive effects (see § 1), although the, as yet undiscovered, possibility that the different effects produced by several B-variants are due to DNA sequence differences cannot be ruled out.

The selfish purposes proposal might be compatible with our present results, but it actually encompasses a rather heterogeneous collection of contradictory *ad hoc* explanations for differing effects of B-chromosomes on chiasma formation. The close association between the intensity of chiasma increase and the degree of parasitism is mostly in agreement with the inducible recombination hypothesis, since it suggests a host response that changes coevolutionarily depending on the degree of parasite attack.

We therefore think that the increase in chiasma frequency in males carrying parasitic B-chromosomes is best explained by inducible recombination, an adaptive response of the host to parasite presence that was envisioned by Bell & Burt (1990) in the light of the Red Queen hypothesis. The only previous attempt to test inducible recombination, in the endemic frog *Leiopelma hochstetteri*, led to inconclusive results owing to a low sample size (Chandler *et al.* 1993). In *E. plorans*, intrapopulation analyses have shown that the presence of different B-chromosome variants is correlated with an increase of chiasma frequency in the A-chromosomes, although the variants analysed showed differing effect intensities associated with the variable evolutionary status of the B-polymorphism in each population.

The absence of mite effects on recombination is also predicted by inducible recombination as the effect is expected as a response to vertically (but not to horizontally) transmitted parasites (Bell & Burt 1990). B-chromosomes are strictly vertically (i.e. intergenerational among relatives) transmitted parasites whereas mites are essentially horizontal (Muñoz *et al.* 1998). Our results are also consistent with Rice's hypothesis that pathogen transmission between parent and progeny (which is expected for B-chromosomes but not necessarily for mites) may strongly select for sexual recombination (Rice 1983).

The interpopulation analysis has also shown a clear relationship between the evolutionary status of the B-polymorphism in a population and the intensity of effects on chiasma frequency. The parasitism-dependent increase in host recombination meets all expectations of the Red Queen hypothesis, which predicts that populations with greater parasite loads will consist of individuals which produce higher numbers of recombinant offspring (Ebert & Hamilton 1996). In *E. plorans*, B-chromosomes follow an evolutionary life cycle that passes through parasitic (with drive), near-neutral (with no drive) and newly parasitic stages (Camacho *et al.* 1997). In parallel, the load conferred by the B to the host genome seems to change over its life cycle, linking drive with virulence, as is suggested by the harmful effects on egg fertility that are apparent for parasitic (Zurita *et al.* 1998) but not neutralized Bs (Camacho *et al.* 1997).

It has been suggested that stressful environmental conditions can influence recombination rate (Hoffmann & Hercus 2000). For example, increased aridity stress is associated with an increase in chiasma frequency in the mole rat *Spalax ehrembergi* (Nevo *et al.* 1995). Parasitic B-chromosomes entail additional stress to the host genome whose response, increasing chiasma frequency, is favoured by natural selection. This constitutes the basis of inducible recombination and is explicit evidence for the Red Queen hypothesis.

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REFERENCES

Bakkali, M., Cabrero, J., López-León, M. D., Perfectti, F. & Camacho, J. P. M. 1999 The B-chromosome polymorphism of the grasshopper *Eyprepocnemis plorans* in North Africa. I. Types distribution and frequency evolution in Moroccan populations. *Heredity* **83**, 428–434.

Bakkali, M., Perfectti, F. & Camacho, J. P. M. 2002 The B-chromosome polymorphism of the grasshopper *Eyprepocnemis plorans* in North Africa. II. Parasitic and neutralized B_1 chromosomes. *Heredity* **88**, 14–18.

Bell, G. 1982 *The masterpiece of nature: the evolution and genetics of sexuality*. Berkeley, CA: University of California Press.

Bell, G. & Burt, A. 1990 B chromosomes: germ-line parasites which induce changes in host recombination. *Parasitology* **100**, S19–S26.

Bugrov, A., Warchalowska-Śliwa, E. & Vysotskaya, L. 1999 Karyotypic features of Eyprepocnemidinae grasshoppers from Russia and Central Asia with reference to the B chromosomes in *Eyprepocnemis plorans* (Charp.). *Folia Biol.* **47**, 97–104.

Cabrero, J., López-León, M. D., Bakkali, M. & Camacho, J. P. M. 1999 Common origin of B chromosome variants in the grasshopper *Eyprepocnemis plorans*. *Heredity* **83**, 435–439.

Camacho, J. P. M., Carballo, A. R. & Cabrero, J. 1980 The B-chromosome system of the grasshopper *Eyprepocnemis plorans* subsp. *plorans* (Charpentier). *Chromosoma* **80**, 163–166.

Camacho, J. P. M., Cabrero, J., Viseras, E., López-León, M. D., Navas-Castillo, J. & Alché, J. D. 1991 G banding in two species of grasshopper and its relationship to C, N and fluorescence banding techniques. *Genome* **34**, 638–643.

Camacho, J. P. M., Shaw, M. W., López-León, M. D., Pardo, M. C. & Cabrero, J. 1997 Population dynamics of a selfish B chromosome neutralized by the standard genome in the grasshopper *Eyprepocnemis plorans*. *Am. Nat.* **149**, 1030–1050.

Camacho, J. P. M., Sharbel, T. F. & Beukeboom, L. W. 2000 B-chromosome evolution. *Phil. Trans. R. Soc. Lond.* B **355**, 163–178. (DOI 10.1098/rstb.2000.0556.)

Cano, M. I., Jones, G. H. & Santos, J. L. 1987 Sex differences in chiasma frequency and distribution in natural populations of *Eyprepocnemis plorans* containing B-chromosomes. *Heredity* **59**, 237–243.

Carlson, W. R. 1994 Crossover effects of B chromosomes may be 'selfish'. *Heredity* **72**, 636–638.

Carlson, W. R. & Roseman, R. R. 1992 A new property of the maize B-chromosome. *Genetics* **131**, 211–223.

Chandler, M., Green, D. M., Taylor, A., Zeyl, C. W. & Sharbel, T. F. 1993 Chiasma frequency in frogs of the genus *Leiopelma* (Amphibia; Anura) and a test of the hypothesis of inducible recombination. *Hereditas* **118**, 205–210.

Ebert, D. & Hamilton, W. 1996 Sex against virulence: the coevolution of parasitic diseases. *Trends Ecol. Evol.* **11**, 79–82.

Fontana, P. G. & Vickery, V. R. 1973 Segregation distortion in the B chromosome system of *Tettigidea lateralis* (Say) (Orthoptera: Tetrigidae). *Chromosoma* **43**, 75–100.

Hamilton, W. D. 1980 Sex versus non-sex parasite. *Oikos* **35**, 282–290.

Henriques-Gil, N., Santos, J. L. & Giráldez, R. 1982 Genotype-dependent effect of B-chromosomes on chiasma frequency in *Eyprepocnemis plorans* (Acrididae: Orthoptera). *Genetica* **59**, 223–227.

Hewitt, G. M. 1976 Meiotic drive for B-chromosomes in the primary oocytes of *Myrmeleotettix maculatus*. *Chromosoma* **56**, 381–391.

Hewitt, G. M. & John, B. 1967 The B chromosome system of *Myrmeleotettix maculatus* (Thunb). III. The statistics. *Chromosoma* **21**, 140–162.

Hoffmann, A. A. & Hercus, M. J. 2000 Environmental stress as an evolutionary force. *BioScience* **50**, 217–226.

Jaenike, J. 1978 An hypothesis to account for the maintenance of sex within populations. *Evol. Theory* **3**, 191–194.

Janssens, F. A. 1909 La théorie de la chiasmatypie. *La Cellule* **25**, 387–411.

John, B. & Freeman, M. 1975 The cytogenetic structure of Tasmanian populations of *Phaulacridium vittatum*. *Chromosoma* **53**, 283–293.

John, B. & Hewitt, G. M. 1965 The B chromosome system of *Myrmeleotettix maculatus*. I. The mechanisms. *Chromosoma* **16**, 548–578.

Jones, R. N. & Rees, H. 1967 Genotypic control of chromosomal behaviour in rye. XI. The influence of B chromosomes on meiosis. *Heredity* **22**, 333–347.

Jones, R. N. & Rees, H. 1982 *B chromosomes*. New York: Academic Press.

López-León, M. D., Cabrero, J., Camacho, J. P. M., Cano, M. I. & Santos, J. L. 1992 A widespread B chromosome polymorphism maintained without apparent drive. *Evolution* **46**, 529–539.

Muñoz, E., Perfectti, F., Martín-Alganza, A. & Camacho, J. P. M. 1998 Parallel effect of a B chromosome and a mite decreasing female fitness in the grasshopper *Eyprepocnemis plorans*. *Proc. R. Soc. Lond.* B **265**, 1903–1909. (DOI 10.1098/rspb.1998.0518.)

Nevo, E., Ben-Schlomo, R., Beiles, A., Ronin, Y., Blum, S. & Hille, J. 1995 Genomic adaptive strategies: DNA fingerprinting reveals ecological correlates and genetic parallelism to allozyme, mitochondrial DNA and RAPDs diversities in the actively speciating blind mole rats in Israel. In *Gene families: structure, function, genetics and evolution* (ed. R. S. Holmes & H. A. Lim), pp. 55–70. River Edge, NJ: World Scientific.

Nolte, D. J., Dési, I. & Meyers, B. 1969 Genetic and environmental factors affecting chiasma formation in locusts. *Chromosoma* **27**, 145–155.

Rice, W. R. 1983 Parent–offspring pathogen transmission: a selective agent promoting sexual reproduction. *Am. Nat.* **121**, 187–203.

Shaw, D. D. 1971 The supernumerary segment system of *Stethophyma*. II. Heterochromatin polymorphism and chiasmata variation. *Chromosoma* **34**, 19–39.

Shaw, D. D. 1972 Genetic and environmental components of chiasma control. II. The response to selection in *Schistocerca*. *Chromosoma* **37**, 297–308.

Van Valen, L. 1973 A new evolutionary law. *Evol. Theory* **1**, 1–30.

Westerman, M. & Dempsey, J. 1977 Population cytology of the genus *Phaulacridium*. VI. Seasonal changes in the frequency of the B-chromosomes in a population of *P. vittatum*. *Aust. J. Biol. Sci.* **30**, 329–336.

Zečević, L. J. & Paunović, C. 1969 The effect of B-chromosomes on chiasma frequency in wild populations of rye. *Heredity* **33**, 173–180.

Zurita, S., Cabrero, J., López-León, M. D. & Camacho, J. P. M. 1998 Polymorphism regeneration for a neutralized selfish B chromosome. *Evolution* **52**, 274–277.