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Rapid suppression of drive for a parasitic B chromosome

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Abstract. The persistence of parasitic B chromosomes in natural populations depends on both B ability to drive and host response to counteracting it. In the grasshopper *Eyprepocnemis plorans*, the B_{24} chromosome is the most widespread B chromosome variant in the Torrox area (Málaga, Spain). Its evolutionary success, replacing its ancestral neutralized B variant, B_2, was based on meiotic drive in females, as we showed in a sample caught in 1992. In females collected six years later, mean B_{24} transmission ratio (k_B) was 0.523, implying a very rapid decrease from the 0.696 observed in 1992. This shows that B_{24} neutralization is running very fast and suggests that it might most likely be based on a single gene of major effect.

Supernumerary or B chromosomes are dispensable chromosomes found in many plant and animal species (Jones and Rees, 1982). Most of them can be considered genome parasites that accumulate in the host germ line thus persisting in natural populations despite harmful effects on several aspects of host fitness (for review see Jones and Rees, 1982 and Camacho et al., 2000).

As other types of parasites, B chromosomes engage in an arms race with the host (A chromosomes) manifested by various A and B strategies all leading to get rid of each other (Shaw and Hewitt, 1990). The main B chromosome weapon is its ability to get transmission drive from a variety of mechanisms (see Jones, 1991), and a high frequency of mutation expanding its variability thus promoting its evolutionary chance. The main A chromosome strategy to counteract B chromosome attack is manifested by the control of B drive based on suppressor genes located on the A chromosomes (Shaw et al., 1985; Shaw and Hewitt, 1985; Nur and Brett, 1985, 1987, 1988; Romera et al., 1991; Cebriá et al., 1994; Jiménez et al., 1995; Herrera et al., 1996; Puertas et al., 2000; Chiavarino et al., 2001; González-Sánchez et al., 2003). For example, in the grasshopper *Myrmeleotettix maculatus*, a gene modifying B drive seems to be present as a polymorphism in all populations (Shaw and Hewitt, 1985). In the mealybug, *Pseudococcus affinis*, Nur and Brett (1987) showed that the high and low transmission lines, that they had previously obtained by artificial selection, differed at two unlinked loci with additive effects on B transmission rate. In maize, a single major gene controls preferential fertilisation of B-carrying male gametes (Chiavarino et al., 2001; González-Sánchez et al., 2003), and a dominant gene favours meiotic elimination of B univalents in females (González-Sánchez et al., 2003).

In some cases, the parasitic B chromosome seems to reach an equilibrium frequency because of a balance between the frequency gain derived from B drive and the frequency loss caused by its deleterious effects on the host (Nur, 1977). In others, B drive is suppressed completely and the B is condemned to stochastic loss (Camacho et al., 1997). In the first case, the A chromosomes will bear the B burden over generations, so that it is intriguing why these Bs are not neutralised. Two explanations are possible, namely, the absence of appropriate A chromosome genetic variation being able to drive suppression of B, or else that suppression is costly. Species where high variation for B transmission ratio among individuals has been reported, e.g. *Myrmeleotettix maculatus* (Hewitt, 1973), *Hypochoeris maculata* (Parker et al., 1982), rye (Romera et al., 1991) and maize (Rosato et al., 1996), are probable examples of costly suppression of B drive.

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The grasshopper *Eyprepocnemis plorans* probably illustrates a case with low cost of B drive suppression, since the A-B arms race, in this species, frequently leads to the neutralization of B drive (Camacho et al., 1997). This species has also illuminated a model for the long-term life cycle of a parasitic B chromosome (Camacho et al., 2003). In this model, the B is neutralized by the host genome through the evolution of host genes that suppress B drive. Because of not forming bivalents, such neutralized B chromosomes are destined to disappear after a long period of stochastic fluctuation, in the course of which the Bs can mutate into a new parasitic variant with high drive that restarts the cycle again (Camacho et al., 1997). The occurrence of B chromosome substitutions has been reported in a population collected at Torrox (Málaga, Spain), where a new B type, B_{24}, has just replaced the original one, B_2, because of its higher transmission ratio (Zurita et al., 1998). Therefore, it is possible to follow empirically the evolution of this newly parasitic B in this population, in order to test our model predicting that this new B should also be neutralized (Camacho et al., 1997). Here we show that the neutralization of this new parasitic B has taken place rapidly thus suggesting a suppression mechanism most likely based on a single gene of major effect.

Materials and methods

During autumn 1998, we caught adult males and nymph females in Torrox (Málaga) and started a number of controlled crosses between every female (when adult) and a 0B male (analysed by testis biopsy, see López-León et al., 1992a for details). In sixteen of these crosses, the female carried one or more B chromosomes and yielded enough ten-day-old embryos to analyze B transmission (Table 1).

After cross completion, the male was anaesthetized and dissected to fix the testes in 3:1 ethanol-acetic acid (to confirm absence of Bs), and the female was injected with 0.05% colchicine in insect saline solution for 6 h prior to fixation of ovarioles in 3:1 ethanol-acetic acid (to determine the number of Bs). The eggs were incubated for ten days at 28°C and then dissected to fix the embryos following the method described in Camacho et al. (1991). The number of B chromosomes in all fixed materials was analysed by the C-banding technique described in Camacho et al. (1991).

The two key parameters analysed in these crosses were B transmission ratio (k_B, the quotient between the mean number of Bs in the offspring and the number of B chromosomes in the parents, i.e. in the mother since all fathers carried no B chromosomes) and egg fertility (the proportion of eggs containing an apparently normal embryo after the ten days of incubation). The statistical methods employed included the Z test (approximation of binomial to normal distribution, to test k_B departing from the expected under the Mendelian segregation law, i.e. 0.5, suggested by López-León et al., 1992a), the Fisher exact test for a between-year comparison of the proportion of crosses showing B drive, the ANCOVA to test temporal variation in k_B and egg fertility, and its dependence on the number of B chromosomes, and the F-ratio test to analyse temporal changes in the variance of k_B.

We developed a Statistica® program to explore deterministically the dynamics of a drive-modifier gene (m) in a system with the following parameters: initial B chromosome frequency (from 0.1 to 1 B chromosome per host), B chromosome drive (0.8), proportion of B chromosome bivalents (0.2), initial frequency of the modifier (0.01–0.5), and dominance of the modifier (h = 0, 0.5, 1). We assumed a Mendelian transmission rate of B chromosomes through males (i.e., k_B = 0.5). Female fertilities were established from data in experimental crosses (Zurita et al., 1998): 1 for m^+m^+ females with 0B, 0.81 for m^+m^+ females with 1B, 0.68 for m^+m^+ females with 2B, 0.5 for m^+m^+ females with 3B, and 0 for females with 4 or more Bs. Fertilities for m^+m and mm females were parameterized depending on the m costs and h.

Results and discussion

Three females (nos. 7, 8 and 13 in Table 2) out of the 16 females analysed showed k_B significantly higher than 0.5, thus indicating B accumulation, but none showed k_B significantly

Table 1. Number of females analysed in Torrox (Málaga, Spain) in 1992 (Zurita et al., 1998) and 1998 (present paper)

Year	Number of B chromosomes in the female					Totals
	0	1	2	3	4	
1992	4	13	4	1	0	22
1998	8	10	4	1	1	24
Totals	12	23	8	2	1	46

Table 2. Results of 16 controlled crosses performed with grasshoppers collected at Torrox (Málaga, Spain) in 1998

Cross	Bs in parents		Eggs	Embryos	Fertile eggs	Embryos analysed with					Total	Mean Bs	k_B	Z	P
	Mother	Father				0B	1B	2B	3B	4B					
1	1	0	71	56	0.789	30	24	2	0	0	56	0.500	0.500	0.000	1.000
2	1	0	149	55	0.369	26	27	0	0	0	53	0.509	0.509	0.137	0.891
3	1	0	41	27	0.659	29	17	0	0	0	46	0.370	0.370	–1.769	0.077
4	1	0	83	59	0.711	28	29	0	0	0	57	0.509	0.509	0.132	0.895
5	1	0	119	92	0.773	51	41	0	0	0	92	0.446	0.446	–1.043	0.297
6	1	0	127	97	0.764	53	44	0	0	0	97	0.454	0.454	–0.914	0.361
7	1	0	142	102	0.718	38	58	1	0	0	97	0.619	0.619	2.335	**0.020**
8	1	0	96	78	0.813	27	51	0	0	0	78	0.654	0.654	2.717	**0.007**
9	1	0	156	137	0.878	64	73	0	0	0	137	0.533	0.533	0.769	0.442
10	1	0	88	81	0.920	34	47	0	0	0	81	0.580	0.580	1.444	0.149
11	2	0	136	82	0.603	13	48	17	0	0	78	1.051	0.526	0.453	0.651
12	2	0	130	61	0.469	7	13	12	3	0	35	1.314	0.657	1.859	0.063
13	2	0	140	117	0.836	7	74	30	0	0	111	1.207	0.604	2.183	**0.029**
14	2	0	13	10	0.769	2	4	2	0	0	8	1.000	0.500	0.000	1.000
15	3	0	75	25	0.333	2	16	7	0	0	25	1.200	0.400	–1.000	0.317
16	4	0	92	30	0.326	3	8	14	4	1	30	1.733	0.433	–0.730	0.465

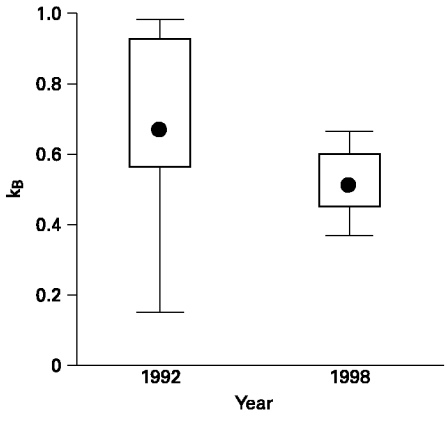


Fig. 1. Distribution of k_B values obtained from controlled crosses performed with grasshoppers collected at Torrox (Málaga, Spain) in 1998 and 1992 (Zurita et al., 1998). Error bars represent minimum and maximum values, box extremes coincide with the first and third quartiles, and the dot indicates the median value.

Table 3. Estimation of the load caused by B24 in the 1992 Torrox population, based on the B frequency and egg fertility data reported by Zurita et al. (1998)

	0B	1B	$2B^+$	Mean fitness	Load
Number of females	4	13	5		
Relative frequency	0.18	0.59	0.23		
Egg fertility	0.95	0.81	0.68		
Relative Fitness	1	0.86	0.71		
Post selection frequency	0.18	0.51	0.16	0.85	0.15

lower than 0.5. This 3/16 of crosses showing B accumulation is significantly lower than the 11/18 observed in 1992 in the same population (Zurita et al., 1998) (Fisher exact test: $P = 0.017$). On average, k_B in these 16 crosses (mean = 0.520, SEM = 0.021) did not differ significantly from the Mendelian one (t = 0.848, $P = 0.410$), which suggests that B_{24} is very close to neutralization.

In order to test the temporal evolution of B drive, we compared the k_B values reported by Zurita et al. (1998) with the present ones. An ANCOVA with k_B as dependent variable, year as fixed factor and number of Bs in the female as a covariate, showed that k_B is independent of the number of Bs ($F_{1,31} = 1.0344$; $P = 0.317$) but decreased significantly from 1992 ($k_B = 0.696$) to 1998 ($k_B = 0.523$) ($F_{1,31} = 8.4524$, $P = 0.007$). Variance in k_B has also decreased significantly from 1992 (0.046) to 1998 (0.008) (F ratio test = 6.08, $P = 0.001$). This decrease in variance could be indicative of a strong selection for $k_B = 0.5$, i.e., an increase in frequency of a putative drive modifier gene, thus forcing Bs to obey the Mendelian segregation law (Fig. 1). This result provides the first direct evidence for the neutralization of a parasitic B chromosome (B_{24}) through the suppression of its drive in the Torrox population. The suppression had practically been completed by 1998 implying a 26% decrease in k_B from 1992 to 1998 (an average of 4.3% per year).

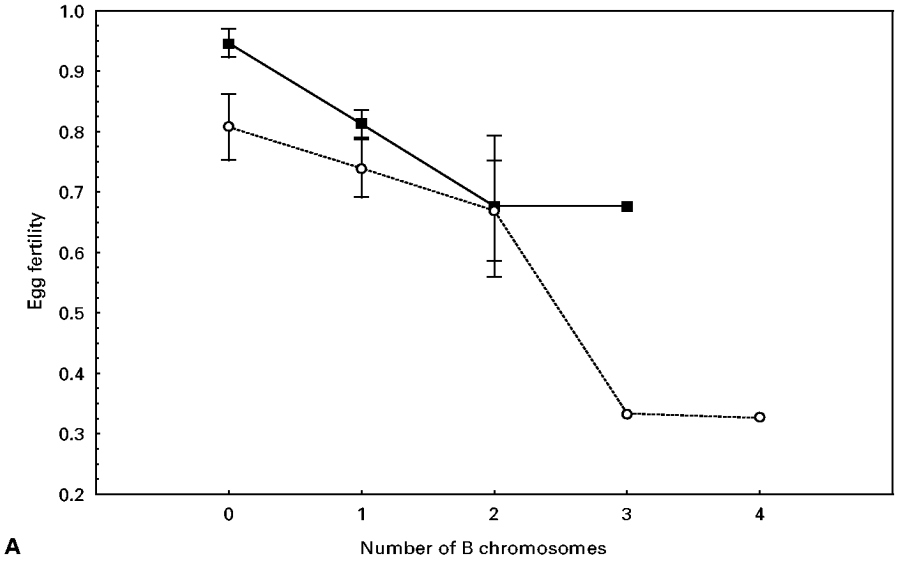
Our model of B chromosome evolution (Camacho et al., 1997) used polygenic modification of the drive rate, and predicted that for a B chromosome with the characteristics shown by B_{24} in Torrox, drive suppression should have taken place in about 40 generations. The process, however, has run much faster than expected, which raises several possible explanations:

i) Torrox *E. plorans* populations might produce more than one generation a year, resembling the laboratory non-diapause rearing. Although we have not yet made a detailed phenology of this population, the existence of six or more generations a year is, however, very unlikely.

ii) Perhaps the polygenic assumption is not valid and the suppression is based on a single gene, or a few genes of major effects. The drive data in 1992 appear to be bimodal (Fig. 3), suggesting the presence of a single modifier gene. Assuming that drive greater than 0.7 represents one homozygote (m^+m^+), drive of 0.55–0.7 the heterozygote (m^+m) and less than 0.55 the other homozygote (mm), the suppressor gene (m) frequency would have changed from 0.36 in 1992 (three females being homozygous, seven being heterozygous and eight lacking suppressor genes) to 0.75 in 1998 (11 homozygous and five heterozygous females). If this were attributable to selection, the estimate of the annual selection coefficient is ln(0.75/0.36)/6 = 0.12 (assuming a generation per year).

Previous analyses, trying to detect harmful effects of B chromosomes in natural populations of *E. plorans* have failed to find them at the level of mating frequency (López-León et al., 1992b), embryo-adult viability, post-mating selection and egg fertility (Camacho et al., 1997). But these analyses were performed for the B_2 chromosome in populations where it seemed to have been neutralized for a long time. In fact, laboratory crosses performed with specimens from these populations, manipulating mating frequency, showed that B chromosomes severely reduced egg fertility with mating scarcity, suggesting that this fitness component is sensitive to B presence in low-density populations (Muñoz et al., 1998). This was confirmed by the egg fertility decrease caused by the driving B_{24} variant in Torrox in 1992 (Zurita et al., 1998). An ANCOVA with egg fertility as dependent variable, year as fixed factor and number of Bs in the female as a covariate, showed that egg fertility decreased significantly from 1992 (0.806) to 1998 (0.712) ($F_{1,42} = 5.74$; $P = 0.021$) and also decreased significantly with increasing number of Bs in the female ($F_{1,42} = 27.06$; $P < 0.001$).

All these observations allow assuming that the main load caused by B_{24} is on egg fertility. An estimate of this load in Torrox, based on the 1992 data (Table 3), indicated a 15% load, which creates the conditions for a fast frequency increase of a drive-modifier gene that could both restore egg fertility and control B-chromosome drive. This might explain the easiness for drive suppression in *E. plorans*, manifested by the absence of drive for the principal B chromosomes in the Iberian Peninsula (López-León et al., 1992a). The average egg fertility in the 1998 sample (0.71 ± 0.03) was lower than that observed in 1992 (0.81 ± 0.03), implying a 9.4% decrease in the six years, equivalent to 2.07% per year. This is consistent with an overall cost for suppression of about 3%. Figure 2a suggests that the main change in egg fertility between 1992 and 1998 engaged 0B females, whose fertility had remarkably decreased in these six



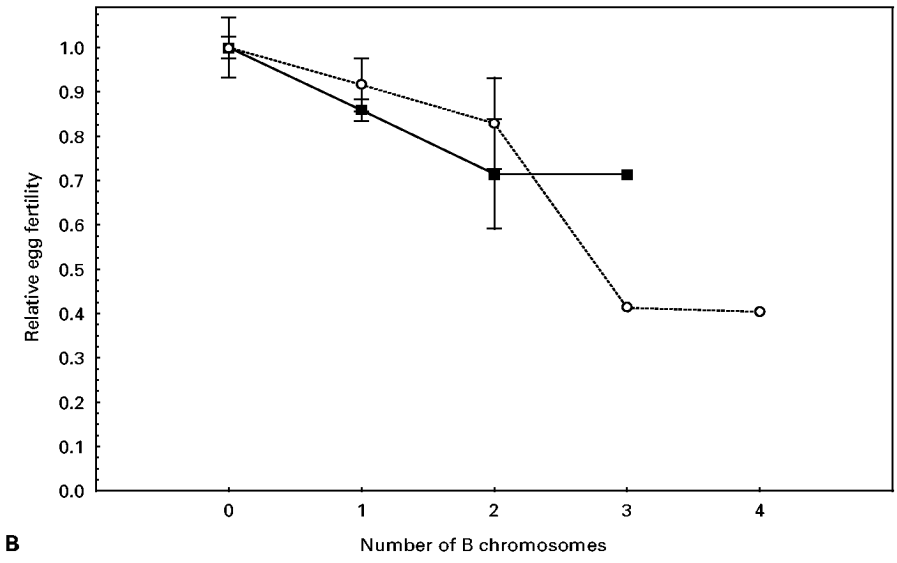


Fig. 2. Effects of B chromosomes on female fitness measured by the proportion of embryos containing an embryo (egg fertility). Solid squares and lines represent the 1992 sample and open circles and broken lines the 1998 sample. Values are means and error bars represent one standard error. (**A**) Actual data, (**B**) relative egg fertilities based on normalization with respect to 0B fertility.

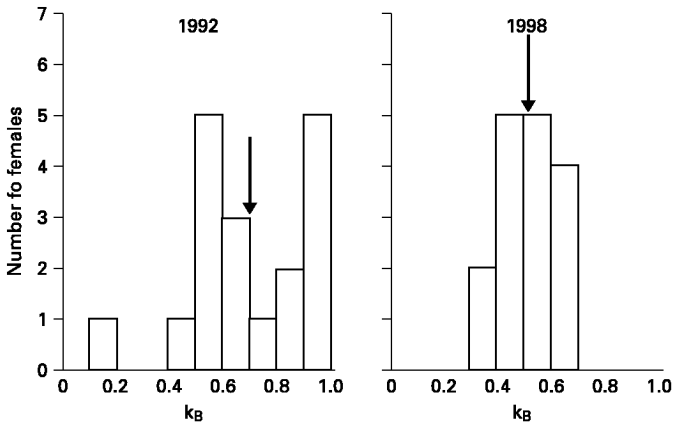


Fig. 3. Distribution of k_B values in the experimental samples of females. Arrow points to the distribution mean values.

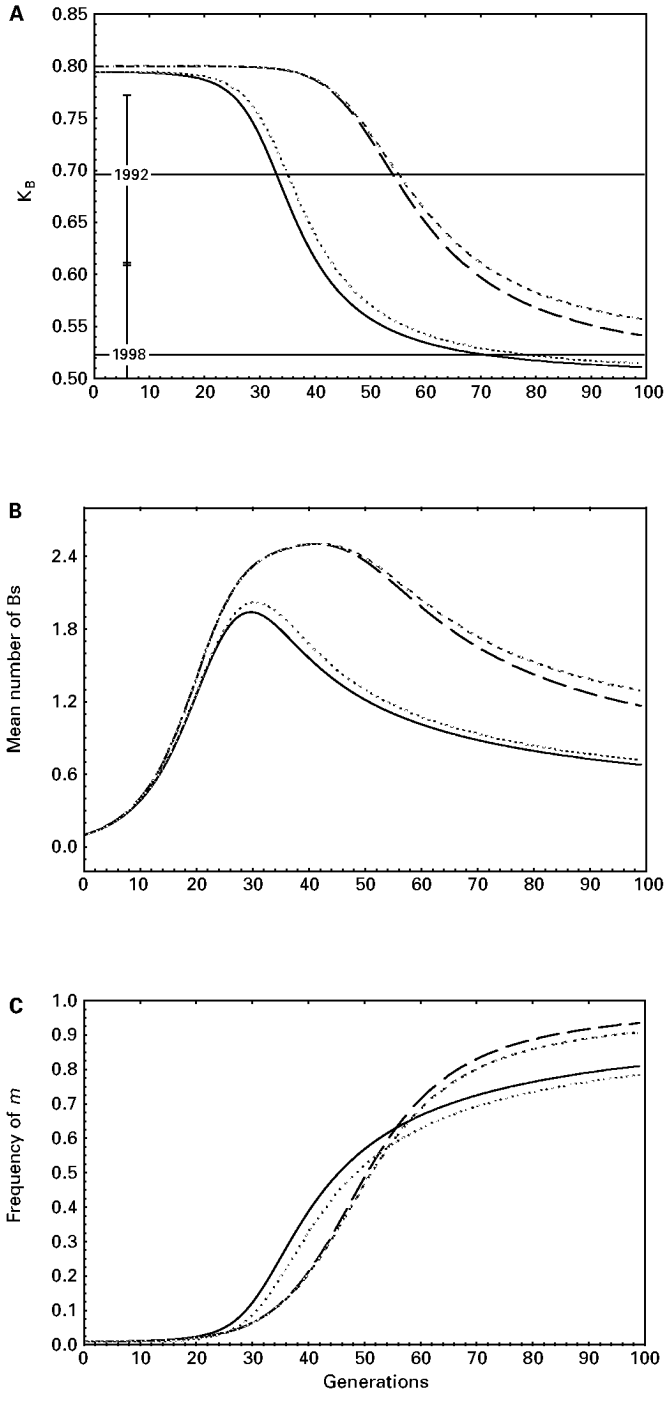


Fig. 4. Evolution of mean k_B (**A**), mean number of Bs (**B**) and frequency of a drive-modifier gene (**C**) in a simulated population with the following starting parameters: B chromosome drive = 0.8, B frequency = 0.1, drive-modifier frequency = 0.01, no drive through males, frequency of B chromosome bivalents at meiosis = 0.2, no reproduction of individuals with more than three B chromosomes. We assumed that the drive modifier allele (*m*) reduce both drive and virulence. B chromosomes reduced egg fertility in m^+m^+ females according to this distribution: 0B = 1.0, 1B = 0.81, 2B = 0.68, 3B = 0.5. Females of *mm* genotype showed complete fertility (1). Fertility of heterozygous females depends on *h*, the dominance of the drive-modifier allele. Continuous lines represent $h = 1$ (dominance) with no resistance cost, broken lines $h = 0$ and no cost, dot lines $h = 1$ with a 3% cost, and broken dot lines $h = 0$ with 3% cost. In A, confidence intervals for actual 1992 and 1998 k_B means are represented by min-max bars.

years. This could reflect a certain cost of B drive suppression because some 0B females could harbour the suppressor genes. However, the reduction in fertility observed in 1998 could also be due to uncontrolled environmental factors acting in the *E. plorans* populations submitted to increased environmental stresses because of their natural environment being anthropically changed, which generally could produce a decrease in fertility and viability (Hoffmann and Parson, 1993). Relative fertilities (referred to that of 0B females) in the 1998 sample showed a typical (but not significant) tolerance response curve, indicating that tolerant genotypes would be able to carry one or two B chromosomes with less reduction in fitness (see Fig. 2b).

It is also remarkable that egg fertility at population level continues being significantly lower than that observed in populations with neutralized Bs since long, e.g. B_2 at Salobreña and Jete populations, where it was close to 100% (López-León et al., 1992a). Although tentatively, it might indicate that the *E. plorans* A genome seems to have ease in neutralizing B chromosome drive but is less prepared to get rid of deleterious effects caused by B chromosomes. This might simply be due to a different genetic architecture of both phenomena, i.e. single gene drive suppression but polygenic tolerance to Bs.

However, a rapid drive suppression is not compatible with a model where drive (measured by k_B) and virulence (measured by the reduction in fertility) are independently counteracted by the A genome. We have modelled the evolution of monogenic drive suppression by parameterizing it with data obtained from the 1992 sample (see Materials and methods and Fig. 4) and found that the condition promoting the faster reduction in drive is the coupling of drive-resistance with egg fertility, which could be conceivable if B-effects on fertility would be by-products of the B drive mechanism. For a dominant drive-modifier gene (*m*) causing a reduction of both B drive and egg fertility, it would take 35 generations to decrease mean k_B from 0.70 to 0.52 (the observed values in 1992 and 1998, respectively), and even more if *m* would be recessive (see Fig. 4 for details). The inclusion of a resistance cost of around 3% did not substantially modify the results. Although 35 generations is a high number even in the case of two generations per year, the experimental confidence intervals of estimations (see Fig. 4a) allow experimental data to fit in the model. Taking into account the inherent experimental sampling error, a few generations would be enough to produce a decrease in k_B similar to that observed in the Torrox population.

In addition to sampling error, other possibilities remain, at least at the theoretical level, to explain the rapid suppression of B accumulation. Fluctuations in population size that could randomly accelerate this process, other selective pressures acting against B chromosomes, such as the decrease in mating of B-carrying males reported by Martín et al. (1996), or epigenetic changes in the B chromosome producing trans-generational neutralization induced by the modifier gene, are some possibilities to explore.

It has been usual to attribute a cost to any allele producing resistance to a parasite (Combes, 2001), but this cost could be negligible (Brown, 2003) and not strictly necessary to maintain a polymorphism for resistant alleles in spatially structured populations (Thrall and Burdon, 2002). If a polymorphism for a

drive modifier gene exists in the host population, some individuals could be pre-adapted to a new B chromosome variant, and the increase in frequency of the modifier gene could actually be fast because it is not necessary to wait for a hopeful mutation.

B_{24} is not the first B that invaded the Torrox population since it has recently replaced a former B variant (B_2, a neutralized B lacking drive) (Henriques-Gil and Arana, 1990) by virtue of meiotic drive in females (Zurita et al., 1998). When B_{24} first appeared, the A genome in this population presumably contained a high frequency of suppressors against B_2, as a consequence of its previous suppression. The successful invasion by B_{24} suggests that these suppressors were not active against it, which might be due to some quantitative or qualitative difference in B_2 and B_{24} mechanisms of drive. Both B chromosome types are mainly made up of two types of repetitive DNA (ribosomal DNA and a 180-bp tandem repeat DNA) with B_{24} harboring less ribosomal DNA but a higher amount of the 180-bp repeat (Cabrero et al., 1999). The possibility remains that the specific mechanism for B_{24} is based on this repetitive DNA. Anyway, the A genome has again found its pathway to suppress the drive of this new parasitic B, and the arms race goes on.

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