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Acknowledgements: We thank Kevin Poissenot, Fanny Husson for technical assistance, as well as Sylvie Nortier for medium preparation. Several lines used in this study were derived from flies collected by Jean R. David and Daniel Lachaise. We also thank Quentin Helleu and two anonymous reviewers for very helpful discussions. This work was funded by Centre National de la Recherche Scientifique and Université Paris-Saclay. HB was funded by a French ministry scholarship.

Authors contribution: H.B. conceived and designed the research; H.B., P.R.G. and D.O. performed research; P.R.G., H.B. and C.M.M. analyzed the data and wrote the paper.

Abstract

Sex-ratio (SR) meiotic drivers are X-linked selfish genetic elements that promote their own transmission by preventing the production of Y-bearing sperm, which usually lowers male fertility. The spread of SR drivers in populations is expected to trigger the evolution of unlinked drive suppressors, a theoretically predicted co-evolution that has been observed in nature. Once completely suppressed, the drivers are expected either to decline if they still affect the fitness of their carriers, or to evolve randomly and possibly get fixed if the suppressors eliminate their deleterious effects. To explore this issue, we used the Paris *sex-ratio* system of *Drosophila simulans* in which drive results from the joint effect of two elements on the X chromosome: a segmental duplication and a deficient allele of the *HP1D2* gene. We set up six experimental populations starting with 2/3 of X chromosomes carrying both elements (X^{SR}) in a fully suppressing background. We let them evolve independently during almost a hundred generations under strong sexual competition, a condition known to cause the rapid disappearance of unsuppressed Paris X^{SR} in previous experimental populations. In our study, the fate of X^{SR} chromosomes varied among populations, from extinction to their maintenance at a frequency close to the starting one. While the reasons for these variable outcomes are still to be explored, our results show that complete suppression can prevent the demise of an otherwise deleterious X^{SR} chromosome, turning a genetic conflict into cooperation between unlinked loci. Observations in natural populations suggest a contrasting fate of the two elements: disappearance of the duplication and maintenance of deficient *HP1D2* alleles.

Key words: meiotic drive, drive suppression, *Drosophila*, sex ratio, experimental evolution

Introduction

X-linked meiotic drivers favor the transmission of their carriers at the expense of the Y chromosome in heterogametic males, thus producing female-biased sex ratios. At the population level, they are predicted to rapidly spread and eventually lead to extinction if the driver is strong and males become too rare.

However, a stable polymorphism of X-linked drivers has been observed in natural populations of several *Drosophila* species (e.g., James and Jaenike 1990; Beckenbach 1996; Dyer 2012). Although this could be due to an observational bias, it reveals the existence of mechanisms that tend to balance the transmission advantage of the drivers (Jaenike 2001; Helleu et al. 2015; Price et al. 2020). First, in a number of species, it has been shown that the loss of Y-bearing sperm is not compensated by an overproduction of X-bearing sperm, which lowers male fertility (e.g., Policansky and Ellison 1970; Hauschteck-Jungen and Maurer 1976; Wu 1983b; Montchamp-Moreau and Joly 1997; Presgraves et al. 1997). This, and probably other pleiotropic effects, makes *Drosophila* s*ex-ratio* (SR) males poor competitors against standard males (e.g., Wilkinson et al. 2006; Angelard et al. 2008; Price et al. 2008a). Second, when the driver is located in low recombination regions or within inversions, linked deleterious mutations can hitchhike and lower the fitness of the carriers, as it has been documented in *Drosophila recens* (Dyer et al. 2007). Several adaptations have also been proposed as countervailing processes against drivers (Presgraves 2008), such as polyandry (e.g., Angelard et al. 2008; Price et al. 2008b 2010a; Pinzone and Dyer 2013; reviewed in Wedell 2013) or mate preference (e.g., Wilkinson et al. 1998). The spread of strong X-linked drivers can also be halted by the evolution of drive resistance on the Y chromosome and/or drive suppressors on the autosomes (Price et al. 2020). When a population is highly female-biased, any variant that produces more males will be favored through a frequency-dependent selective process towards a balanced sex ratio (Fisher 1930; Bull and Charnov 1988). In particular, Y-linked resistance should rapidly spread to fixation (Thomson and Feldman 1975; Clark 1987) but can be maintained at a stable equilibrium depending on frequency-dependent interactions between the driving X chromosome (X^{SR}) and the resistant Y (Carvalho et al. 1997) or cycle with the X^{SR} (Hall 2004). An autosomal suppressor of drive is also predicted to be selected and reach fixation (Hamilton 1967). It can also stay at an equilibrium frequency with the driver when there is overdominance in females (Wu 1983a) or when the driver is slightly deleterious (Vaz and Carvalho 2004). In the latter case, an autosomal suppressor should go to fixation only if the driver is neutral in fitness (Vaz and Carvalho 2004). A similar qualitative prediction can be made for a resistant Y chromosome, with an higher fixation probability even in the

presence of a deleterious driver (Carvalho and Vaz 1999). The fate of the driver and suppressor will depend on several parameters, such as the fitness cost associated with drive and suppression in males and females, the time at which the suppressor arises during the spread of the driver, or the population structure (Carvalho and Vaz 1999; Hall 2004; Vaz and Carvalho 2004). Under complete suppression, the driver frequency is expected to decline if costly or to evolve stochastically if neutral. This process can generate evolutionary cycles of drive and suppression (Hall 2004), or cryptic meiotic drive systems like the *Drosophila simulans* Winters system (Tao et al. 2007a,b; Kingan et al. 2010; Helleu et al. 2015).

Resistant Y and autosomal suppressors have been detected in a number of *Drosophila* species exhibiting SR drive (e.g., Stalker 1961; Voelker 1972; Carvalho and Klaczko 1993; Carvalho et al. 1997; Cazemajor et al. 1997; Jaenike 1999; Montchamp-Moreau et al. 2001; Courret et al. 2019), but the dynamics of drive and suppression has rarely been assessed in natural populations. In *Drosophila mediopunctata*, X^{SR} frequency had not changed within 10 years in the 1990s, suggesting that the driver was kept at an equilibrium frequency reflecting a balance between partial suppression and natural selection (Carvalho and Vaz 1999).

The only documented example of ongoing SR drive/suppression co-evolution in natural populations is that of the Paris SR system of *D. simulans*. In this system, the drive is caused by the joint effect of two X-linked elements: a young segmental duplication, estimated less than 500 years old (Fouvry et al. 2011) and dysfunctional alleles of the *HP1D2* gene (Helleu et al. 2016). A first survey of natural populations 25 years ago revealed a sharp contrast between sub-Saharan Africa and Indian Ocean, where a complete drive suppression along with various frequencies of X^{SR} (up to 60%) were observed, and the rest of the world where X^{SR} were rare or absent with no or only a slight drive suppression (Atlan et al. 1997). Since then, X^{SR} have been found to steadily decrease in East Africa and Indian Ocean, whereas drive suppression has been persistent (Bastide et al. 2011; 2013). In contrast, the driver has been rising in frequency together with drive suppression around the Mediterranean sea, from Middle East to North Africa and Europe (Bastide et al. 2013; Helleu et al. 2019). In the two species *D. mediopunctata* and *D. simulans,* the rise of suppression when X^{SR} is fixed was demonstrated in experimental populations (Carvalho et al. 1998; Capillon and Atlan 1999). In contrast, the Paris driver of *D. simulans* was unable to invade laboratory populations when introduced at a 25% (or 67%) frequency and was even lost in very few generations in the absence of suppressors, suggesting the existence of strong deleterious effects (Capillon and Atlan 1999). Yet in nature, X^{SR} chromosomes are able to invade rapidly (Derome et al. 2008; Bastide et al. 2011). We can thus hypothesize that in the wild, Paris X^{SR} are much less

deleterious than in experimental populations where there is a strong competition between males, and/or that their detrimental effects are at least partly rescued by the coevolution of suppressors. This last assumption is supported by experimental data showing that X^{SR} chromosomes have no effect on cyst number or cyst length in the male testis (Montchamp-Moreau and Joly 1997).

Here, we have studied the effect of suppressors on the dynamics of the Paris drivers by following the evolution of X^{SR} chromosomes in experimental populations similar to those in Capillon and Atlan (1999) but set up with complete suppression. This means that the drivers did not benefit from any segregation advantage from the beginning. We obtained contrasting results depending on the replicates, which could partly reflect the variable dynamics observed in natural populations. Most importantly, we demonstrate that suppression slows down and can sometimes prevent the elimination of an otherwise deleterious X^{SR} chromosome.

Materials and Methods

Drosophila strains:

- SR is a reference strain for the Paris *sex-ratio* system. It originates from flies collected in the Seychelles archipelago in 1981 (Atlan et al. 1997). A *sex-ratio* X chromosome (X^{SR}) is fixed in the SR strain. It carries the two elements required to induce the drive: the tandem duplication Dp^{SR} and a distorter allele at the *HP1D2* locus ($HP1D2^{SR}$). It induces a strong female-biased sex ratio in a suppressor- and resistance-free background (90% of females in the progeny) but the SR strain contains a resistant Y chromosome and autosomal suppressors that together make X^{SR} fully suppressed (Cazemajor et al. 1997). The SR stock thus shows a 1:1 sex ratio.
- Seych1 and Seych3 are two isofemale lines with standard (i.e. non-driving) X chromosomes (X^{ST}). Each line was obtained from the progeny of a single female collected by D. Lachaise in the Seychelles (Mahé island) in 2003. The X chromosomes of Seych1 and Seych3 do not carry the Dp^{SR} duplication associated with Paris *sex-ratio* drive but display the same $HP1D2^{SR}$ allele than the one found in the SR strain (see molecular tests below). While both lines did not show any SR activity, they were found to completely suppress the *Paris* sex-ratio drive similarly to the SR strain (Table S1, S2 and S3, $\chi^2_{2\,df} = 1.06$; $P = 0.59$).

Experimental populations

Three replicate populations were set up by crossing 100 virgin females from the SR strain

with 100 males from the Seych1 line. The same procedure was used with the Seych3 line to set up three other replicate populations. In all six populations, the initial frequency of the X^{SR} chromosomes was then 2/3. Each population was made evolving with non-overlapping generations in a 500 mL bottle containing 100 mL axenic cornmeal-yeast medium (David 1962). At each generation, adults were allowed to emerge for a few days before being transferred into a new bottle for egg lying during half a day. At least 500 eggs were then randomly collected in each population to produce the next generation. All six populations were kept at 25°C during the whole experiment. The experimental procedure is described in Fig S1.

Molecular tests

- *Dp*^{SR}: DNA was extracted using a classical protocol and a PCR test was performed to detect the presence of *Dp*^{SR}, with primers specific to the junction region between the two paralogous segments (Bastide et al. 2011; Fouvry et al. 2011). Individual molecular tests of the presence of *Dp*^{SR} were performed on 20 males per population until G_{10}, and then on 20-60 males per population at generations G_{15}, G_{17}, G_{22}, G_{32}, G_{48}, G_{60}, G_{72}, G_{80}, G_{88} and G_{94}.

In addition, we checked for the *Trf2* gene copy number by real-time qPCR (Bastide et al. 2013) using subsamples of males with or without the duplication.

- *HP1D2*^{SR}: We sequenced two markers in the second drive element region from the three parental strains: the markers GA17 and GA19 spanning a total of 2 kb including the driving gene *HP1D2* (described in Table S7 in Helleu et al. 2016).

Sequences were aligned and edited with Geneious version R6 (Kearse et al. 2012).

Drive and suppression assays

Tests of drive and suppression abilities were conducted using the crossing procedures previously described (e.g. Montchamp-Moreau and Cazemajor 2002). In short, drive ability was assessed by measuring the sex ratio in the progeny of males carrying the X chromosome under study and a drive sensitive background (Y chromosome and autosomes). Suppression ability was assessed by measuring the sex ratio in the progeny of males carrying the autosomes and the Y chromosome under study and a reference X^{SR} chromosome.

Data analysis

We estimated the 95% confidence intervals on a binomial distribution of the X^{SR} chromosomes (i.e. that carry the SR duplication) with the Wilson's score method (Brown et al. 2001). We used Pearson's chi-squared tests or Kruskall-Wallis rank sum tests for comparing sex ratio and X^{SR} distributions.

We also estimated selection coefficients in the six replicate experimental populations through a maximum likelihood procedure, in the same way as Bastide et al (2011). Briefly, the evolution of X^{SR} and X^{ST} frequencies under natural selection is modelled in a classical deterministic framework for a biallelic X-linked locus (Haldane and Jayakar 1964). We considered recessive deleterious effects of X^{SR} that lower the fitness of homozygous SR/SR females or SR males by *s*. The likelihood of *s* given the data from experimental populations is calculated by multiplying the binomial probabilities of obtaining the observed number of X^{SR} in males given the frequency predicted by the deterministic model at every generation sampled. 95% confidence intervals were computed with the values of *s* that correspond to a log-likelihood drop-off of 1.92 (half of a 1-df chi-squared random variable corresponding to a P-value of 5%) on each side of the maximum.

Results

Evolution of Dp^{SR} in a context of total suppression

Given that suppression is complete in the SR, Seych1 and Seych3 strains (Table S1, S2 and S3) and that all three lines carry the same $HP1D2^{SR}$ deficient allele, the Dp^{SR} duplication was used as a proxy to follow the evolution of X^{SR} chromosomes in each of the six populations. The results are shown in Figure 1 and Table S6, and a subsample of the data with confidence intervals is presented in Table S7. The final Dp^{SR} frequencies in G_{94} were significantly different within Seych1 replicates ($\chi^2_{2\,df} = 15.92$; $P < 10^{-3}$) and marginally significantly different within Seych3 replicates ($\chi^2_{2\,df} = 1.13$; $P = 0.047$). The distributions of Dp^{SR} frequencies were also marginally significantly different between the replicate populations from the two lines (Kruskal-Wallis $\chi^2_{1\,df} = 3.97$; $P = 0.046$).

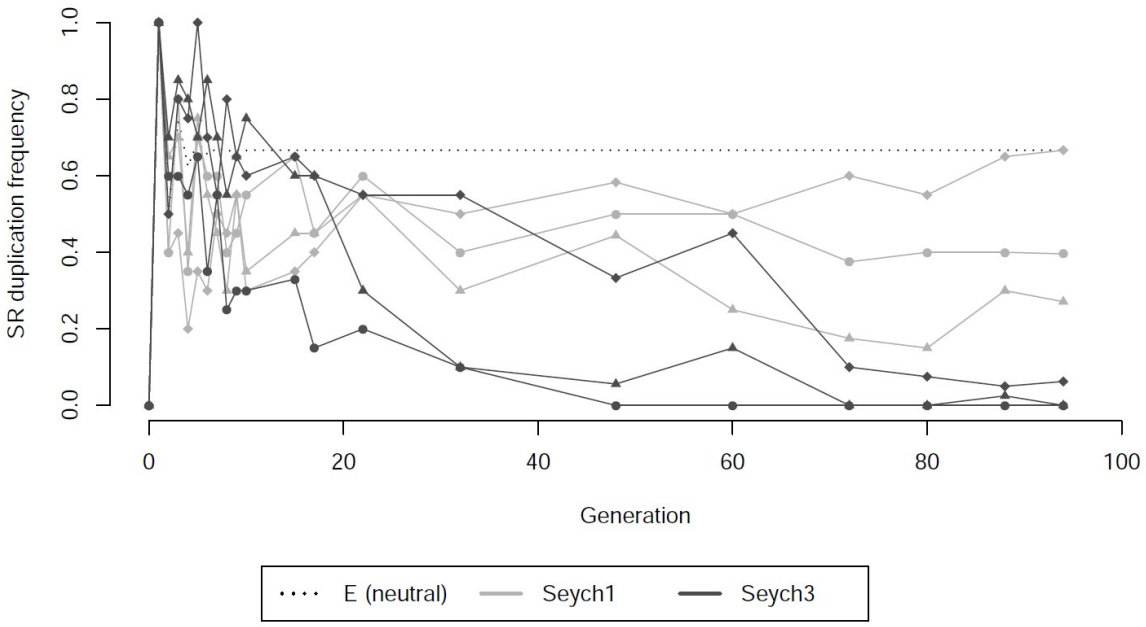

Figure 1: Evolution of the frequency of males carrying Dp^{SR} in the six experimental populations (E: expected frequency if neutral, also corresponding to the frequency of X^{SR} under a neutral Wright-Fisher model with infinite population size). Large fluctuations were expected during the first five generations, due to the exclusively maternal origin of the X^{SR} chromosome among the founders. Triangles represent the first replicates of each line (Seych1-1 and Seych3-1), diamonds the second replicates (Seych1-2 and Seych3-2), and circles the third replicates (Seych1-3 and Seych3-3).

Control of the strict association of the drive phenotype with Dp^{SR}

At G_{88}, we checked for the distortion phenotype associated with the X chromosomes carrying Dp^{SR} in the two populations where it was at its highest frequencies (i.e. the two replicates Seych1-2 and Seych1-3). According to the crossing procedure described above, 26 X chromosomes from population Seych1-2 and 24 X chromosomes from population Seych1-3 carrying Dp^{SR} were tested for drive ability and all of them produced significantly female-biased progenies. A sub-sample of X chromosomes that were used as control and were devoid of Dp^{SR} at G_{88}, did not show any sex ratio bias (Tables 1 and 2).

Whether an X chromosome is SR or not, recombination occurs freely between the two loci involved in drive (they are located about 1cM apart). Consequently, the complete association

observed between Dp^{SR} and the SR phenotype after 88 generations of experimental evolution in populations Seych1-2 and Seych1-3 is consistent with our observation that the Seych1 line carries an $HP1D2^{SR}$ allele.

Control of the persistence of complete suppression

We also checked for the persistence of total suppression in populations Seych1-2 and Seych1-3 by measuring the sex ratio in the progeny of males sampled at G_{93} (Tables S4 and S5). Among 61 males from population Seych1-2, 44 (72%) were found to carry Dp^{SR}, and thus a putative X^{SR} chromosome. The mean percentage of females in progenies was not different between Dp^{SR} and non-Dp^{SR} males (51.2% and 51.5% females respectively, Kruskal-Wallis $\chi^2_{1\ df} = 0.92$; $P = 0.34$). Similarly we did not find any significant difference between 27 males carrying Dp^{SR} and 25 males devoid of Dp^{SR} that were sampled in population Seych1-3 (52.7 % and 51,1% females respectively, Kruskal-Wallis $\chi^2_{1\ df} = 1.40$; $P = 0.24$).

Are suppressed X^{SR} neutral or deleterious?

The evolution of Dp^{SR} was very different across populations, especially after the 20th generation. It showed a marked decline in all three Seych3 replicates: Dp^{SR} disappeared or nearly disappeared from Seych3-1 and Seych3-3 around G_{48}, and became at very low frequency in Seych3-2 around G_{70}. In contrast, Dp^{SR} was maintained at a moderate to high frequency in the three Seych1 replicates, particularly in Seych1-2 where its frequency stood close to the expected value under a neutral Wright-Fisher model (0.66). The mean frequency of Dp^{SR} among the six populations headed steady at around 0.25 from G_{72} to G_{94}.

We assessed potential deleterious effects of X^{SR} by estimating selection coefficients from Dp^{SR} frequency data in every replicate. They are significantly different from 0 for all populations but Seych1-2 (Table 3). The estimates range from 0.021 to 0.053 for Seych1-1, Seych1-3 and Seych3-2, which correspond to moderate deleterious effects. The estimate for Seych3-2 is significantly different from the other two. The estimates for Seych3-1 and Seych3-3 are significantly higher (0.1 and 0.201 respectively). It is worth noting that we obtain similar values of $\hat{s}$ and of confidence intervals when considering dominant deleterious effects of X^{SR} in females (not shown).

Discussion

Dynamics of drivers and suppressors in experimental populations

A few experimental evolution experiments have been carried out to assess the dynamics of SR drive in *Drosophila*. A very valuable piece of work has been made in *D. pseudoobscura*, a species devoid of drive suppressor where it has been shown that SR drive promotes countervailing mechanisms such as polyandry (Price et al. 2008b), which in turn prevents extinction of the populations by strong drive (Price et al. 2010a). In addition, non-SR males from SR populations evolve to suppress female remating when high rates of polyandry occur (Price et al. 2010b). In an earlier study in this species, Curtsinger and Feldman (1980) showed that SR drive disappeared very quickly from experimental populations set up with 70% of X^{SR} chromosomes: as early as 7 generations in two cases, the X^{SR} frequency being very low after 12 generations in all other cases. The estimated selection coefficients due to deleterious effects of the driving chromosomes were very high (0.3-0.4) and mainly associated with fertility defects. In similar experiments with the Paris system of *D. simulans*, Capillon and Atlan (1999) showed that unsuppressed X^{SR} almost disappeared from experimental populations after 17 generations when starting at a 25% frequency, even though it benefited from a strong transmission rate in the carrier males (90% on average). It is supposed to be also lost with an initial frequency of 67% (Capillon and Atlan 1999), but the data are not available. Besides, theory that has been developed to describe the population genetics of *sex-ratio* systems in *Drosophila* usually considered the drive suppressors as brakes that convert spreading unbalanced distorters into balanced ones (Carvalho and Vaz 1999). This view is based on the assumption that the frequency of X^{SR} chromosomes is primarily controlled by their segregation distortion rate, in other terms that the deleterious effects associated with X^{SR} are of a similar magnitude whether drive is suppressed or not. Our results show that it is clearly not the case for the Paris SR system. Here, starting at a frequency of 66%, a completely suppressed Paris X^{SR} still persists at a substantial frequency in half of the six experimental populations after nearly one hundred generations. We do not know how quickly it disappeared when unsuppressed and starting at the same frequency (2/3), but we can suppose that it happened earlier than in our experiment. The comparison with *D. pseudoobscura* is probably limited because of a much more ancient origin of the SR drive system in this species. Yet, our selection coefficient estimations seem to be always lower than those from Curtsinger and Feldman (1980), where there was no suppressor but supposedly a slight overdominance.

While deleterious effects associated with X^{SR} are much lower when fully suppressed, they still exist. All estimates of *s* are positive, and only one is not significantly different from 0 (in Seych1-2, for which the X^{SR} frequency is close to the starting one at G_{94}). In addition, under a neutral Wright-Fisher model the probability of fixation will depend on the initial frequency when we start the experiment. The time to fixation will depend on the effective population size, which in our case would have been moderate (less than 500 individuals). Then X^{SR} is expected to fix in 4 populations and X^{ST} in only 2. After almost a hundred generations, X^{SR} has never reached fixation and X^{ST} has fixed or nearly fixed in 3 populations. We can surely reject the hypothesis of complete neutrality, and suppose that X^{SR} has deleterious effects *per se*, even if suppression moderates these effects.

Comparison with natural populations

The Paris driver has been found to decrease in frequency and likely disappear in natural populations where suppression is complete (Bastide et al. 2011). In our experimental populations, estimates of the selection coefficient in 3 replicates are very close to the value estimated in Madagascar on data from a period of 8 years. Only one replicate shows a much lower estimate, and the two others show a much higher estimate. We cannot exclude the possibility that X^{SR} dynamics are very similar in natural and experimental populations, but the reasons why they are apparently variable in the latter are unclear. In at least some cases, the higher deleterious effects observed could be associated with a higher male competition in bottles (Atlan et al. 2004; Angelard et al. 2008), which could also reduce the effective population size.

Besides, we looked for the presence of Dp^{SR} and surveyed the *HP1D2* alleles in a sample of 22 F_1 males from 11 isofemale lines (2 F_1 males per line) collected in the Seychelles in 2011. None of them carried Dp^{SR} but we found the $HP1D2^{SR}$ allele in all tested males, suggesting a persistence of this potentially driving allele even when Dp^{SR} has long disappeared. A sample of X chromosomes from 72 males (collected in Mayotte in 2009 by CMM and François Wurmser) showed a similar trend, with 6 chromosomes (8%) carrying Dp^{SR} while 28 chromosomes (39%) carried a well-characterized $HP1D2^{SR}$ allele. This is in line with a spatiotemporal analysis of molecular data, prior to the characterization of *HP1D2* as the second driving element of the Paris system (Bastide et al. 2011). This analysis highlighted a discrepancy in the evolution of the two drivers, strongly suggesting that the counter-selection in natural populations acts mainly, if not exclusively, on Dp^{SR}.

Conclusion

We conclude that much of the deleterious effects associated with the Paris X^{SR} are a direct consequence of the drive, which is consistent with the high cost on male fertility observed when competition occurs (Atlan et al. 2004; Angelard et al. 2008). Thus, our present knowledge on the Paris system, which is of very recent origin (Fouvry et al. 2011) is consistent with the hypothesis proposed by Keais et al. (2020) about X^{SR} evolution, in which the dynamics of young X^{SR} are primarily governed by fitness consequences in males.

We have obtained here contrasting results depending on the replicates, which could partly reflect the variable dynamics observed in natural populations where complete suppression has evolved (Bastide et al. 2011). But importantly, we demonstrate that suppression slows down and could sometimes prevent the elimination of an otherwise deleterious X^{SR} chromosome. This is consistent with the concomitant rise of X^{SR} and drive suppressors observed in the wild (Bastide et al. 2013; Helleu et al. 2019). We can suppose that each of them takes advantage of the presence of the other to increase in frequency. The strongly biased transmission, associated with lower deleterious effects entailed by the suppressors, allows the driver to quickly rise in frequency. As for the suppressors, their frequency probably increases by frequency-dependent selection associated with drive. Thus, this genetic conflict appears to convert into a cooperation between alleles at unlinked loci.

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			Female %	
Dp^{SR}	chromosome	*n*	M	SE
-	2-7	5	49.4	1.8
	2-10	3	51.8	3.9
	2-25	5	53.4	7.0
	2-31	4	50.6	8.1
	2-43	3	54.1	7.7
	2-57	2	53.8	3.8
+	2-1	5	92.9	2.7
	2-5	3	88.3	8.5
	2-6	6	94.1	1.8
	2-9	5	95.8	1.9
	2-12	5	93.7	1.8
	2-14	3	89.4	6.3
	2-21	5	92.3	5.1
	2-23	5	94.2	3.0
	2-26	4	89.2	6.8
	2-28	4	92.5	3.5
	2-33	3	95.4	2.6
	2-35	3	93.9	3.4
	2-37	5	95.3	3.2
	2-38	4	93.3	3.6
	2-39	2	89.2	3.9
	2-41	5	89.8	5.8
	2-44	4	90.9	2.1
	2-47	3	92.3	5.7
	2-49	4	90.4	7.4
	2-51	3	89.4	6.7
	2-52	2	92.7	0.3
	2-53	5	94.8	1.1
	2-54	3	84.9	8.1
	2-58	5	92.5	0.9
	2-59	4	91.3	2.9
	2-60	5	93.7	5.4

Table 1: Assessment of the drive activity of X chromosomes with (+) or without (-) the SR duplication (Dp^{SR}) in Seych1-2 at G88. *n*: number of full-sib F1 males tested, M: mean percentage of females in the progeny, SE: standard error.

		Female %		
Dp^{SR}	chromosome	*n*	M	SE
-	3-6	3	51.5	3.3
	3-15	4	52.2	4.9
	3-25	4	50.7	4.4
	3-37	3	55.7	4.2
	3-45	3	50.3	4.5
	3-60	4	50.8	4.8
+	3-1	5	90.9	6.8
	3-3	4	94.6	2.2
	3-8	4	91.3	3.9
	3-9	4	89.8	5.7
	3-10	5	92.4	2.4
	3-16	3	90.9	4.4
	3-19	3	85.5	9.0
	3-20	5	85.9	9.2
	3-22	5	89.5	6.2
	3-24	3	81.0	6.8
	3-27	4	92.6	1.7
	3-28	5	91.5	7.5
	3-30	3	87.4	8.6
	3-31	3	93.5	3.0
	3-33	3	87.2	5.4
	3-34	5	94.4	1.9
	3-35	4	95.3	1.0
	3-36	5	90.6	7.4
	3-39	5	93.8	3.8
	3-43	3	94.7	1.4
	3-46	5	93.8	2.6
	3-49	3	93.1	3.3
	3-51	4	93.1	5.7
	3-56	3	95.2	1.4

Table 2: Assessment of the drive activity of X chromosomes with (+) or without (-) the SR duplication (Dp^{SR}) in Seych1-3 at G88. *n*: number of full-sib F1 males tested, M: mean percentage of females in the progeny, SE: standard error.

554

Strain	Replicate population	$\hat{s}$	$CI_{95\%}$
Seych1	1	0.039	[0.029 – 0.05]
	2	0.007	[0 – 0.014]
	3	0.021	[0.013 – 0.029]
Seych3	1	0.1	[0.077 – 0.13]
	2	0.053	[0.04 – 0.067]
	3	0.201	[0.149 – 0.266]

555 Table 3: Estimates of the selection coefficient ($\hat{s}$) in each of the six experimental populations,
556 and 95% confidence intervals (see text for details).