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Abstract. Extinctions have no simple determinism, but rather result from complex interplays between environmental factors and demographic-genetic feedback that occur at small population size. Inbreeding depression has been assumed to be a major trigger of extinction vortices, yet very few models have studied its consequences in dynamic populations with realistic population structure. Here we investigate the impact of Complementary Sex Determination (CSD) on extinction in parasitoid wasps and other insects of the order Hymenoptera. CSD is believed to induce enough inbreeding depression to doom simple small populations to extinction, but we suggest that in parasitoids CSD may have the opposite effect. Using a theoretical model combining the genetics of CSD and the population dynamics of host-parasitoid systems, we show that CSD can reduce the risk of parasitoid extinction by reducing fluctuations in population size. Our result suggests that inbreeding depression is not always a threat to population survival, and that considering trophic interactions may reverse some pervasive hypotheses on its demographic impact.

Key words: complementary sex determination; demo-genetic feedback; extinction vortex; host-parasitoid system; inbreeding depression; trophic interaction.

Introduction

Population genetics and population dynamics constitute the scientific foundations of modern population biology and have been fueling intense research activities for more than a century. Although complementary, these two disciplines have nonetheless followed parallel routes with only rare intersections. Genetic approaches of population structures, natural selection, genetic drift, and migration have generally assumed either infinite population size or very basic population growth (Wright 1922, Fisher 1930, Kimura 1983, Lynch et al. 1995). In contrast, researches in population dynamics have revealed complex patterns of abundance, as a consequence of interactions within or between species, but have generally ignored genetic variations. It is only recently that integrated approaches have emerged, yielding to novel and important insights on community ecology, evolution, and population genetics (Doebeli and de Jong 1999, Beissinger and McCullough 2002, Leimu et al. 2006, Hughes et al. 2008, Bell and Gonzalez 2011, Robert 2011, Lawrence et al. 2012). The present work contributes to this general perspective of bridging genetics and demography. With a demo-genetic model, we indicate that the consequences of inbreeding depression on demographic success depends on the nature of trophic interactions, and unveil some counter-intuitive situations where inbreeding depression can favor population persistence.

Bringing genetics and demography together is particularly justified when studying the extinction risk of small populations. Whether they are declining or introduced, small populations are so rarely at genetic and demographic equilibrium that analyzing them in the frame of classical models may yield erroneous results. It is therefore no surprise that admixing demographic and genetic approaches has often focused on applied perspectives such as conservation biology, invasion biology, or biological control, where in essence, populations are unstable (Caughley 1994, Lande 1998, Beissinger and McCullough 2002, Fauvergue et al. 2012). A major insight is that inbreeding and loss of genetic diversity in small populations may reduce individual fitness, and in turn, reduce population growth rate, which may push populations into an extinction vortex (Gilpin and Soule 1976, Lande 1988, Belovsky et al. 1999, Tanaka 2000, Schoener et al. 2003, Fagan and Holmes 2006, Kampichler et al. 2010, Palomares et al. 2012, Luque et al. 2016).

Most theoretical developments have focused on recessive or partially recessive deleterious mutations, but genetic incompatibility is another general and widespread mechanism potentially leading to reduced growth rate in small populations. Genetic incompatibilities that result from the genetics of sex determination of some

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parasitoid wasps provide an outstanding paradigm to approach the interplay between genetics and demography in systems where over-simplistic assumptions give misleading results. In these wasps, sex is determined by the complementarity of alleles at a complementary sex-determiner (*csd*) locus: normal haploid males develop from unfertilized eggs and are thus hemizygotes at the *csd* locus; diploid females develop from fertilized eggs that are heterozygotes at the *csd* locus (Whiting and April 1943). The problem rises from homozygosity at the *csd* locus, which causes the abnormal development of fertilized eggs into unviable or sterile diploid males (Cook and Crozier 1995, Van Wilgenburg et al. 2006, Heimpel and de Boer 2008). Complementary Sex Determination (CSD) is therefore at the origin of a particular type of inbreeding depression underpinned by overdominance.

CSD is not restricted to parasitoid wasps. It is the ancestral and probably most widespread genetic system of sex determination in insects of the order Hymenoptera, where it predominates in four of the five largest families (Crozier 1971, Bull 1981, Cook 1993, Cook and Crozier 1995, Zhou et al. 2006, Asplen et al. 2009, Schmieder 2012). The molecular genetics of CSD have been described and mostly studied in the honeybee (Beye et al. 2003, Hasselmann et al. 2008). Sex determination can be based on a single sex-determiner locus (*sl*-CSD), as described previously, or on multiple loci, (*ml*-CSD) in which case only homozygosity at all *csd* loci causes an abnormal development. With *ml*-CSD, diploid male production is less likely than with *sl*-CSD; however, in small and isolated populations, most *csd* loci are likely to become fixed due to genetic drift-particularly if one locus is highly diverse -, leading to the collapse of *ml*-CSD into *sl*-CSD (Asplen et al. 2009, de Boer et al. 2012).

At the population level, single-locus complementary sex determination yields a specific extinction vortex, where deleterious genetic and demographic processes amplify one-another according to the following scenario: decreased population size results in decreased genetic variability, including variability at the *csd* locus; diploid males become more frequent and population growth rate subsequently decreases, possibly causing a further decrease in population size. This demo-genetic feedback has been referred to as the diploid male vortex, potentially leading to extinction rates an order of magnitude higher than that caused by other forms of inbreeding depression in threatened diploid organisms (Zayed and Packer 2005). Hence, research on sex determination in parasitoid wasps challenges the pervasive idea that haplodiploids are less sensitive to inbreeding than other organisms (Henter 2003).

The diploid male vortex has been studied in the perspective of pollinator decline and biological conservation (Zayed et al. 2004, Zayed and Packer 2005, Zayed 2009). Although follow-up models have been enriched with processes such as intraspecific competition, sex ratio variations, or dispersal (Hein et al. 2009), the assumption of very simple population growth has not yet been relaxed.

About half of the species in the order Hymenoptera have a parasitoid lifestyle, with demographic features that are not captured by the few existing demo-genetic models. First, and most importantly, the host-parasitoid interaction implies strong density dependence, within and between populations, which induce complex dynamics (Nicholson and Bailey 1935, Hassell 2000). Cyclic dynamics with recurrent bottlenecks are typical emerging properties of theoretical models (Bernstein 2000), and have been observed in natural parasitoid populations (Wearing et al. 2004). Second, dramatic variations that typically disturb agroecosystems (cropping, pesticide use, habitat loss, etc.) constitute as many extrinsic causes of population reduction (Tscharntke and Brandl 2004, Rusch et al. 2011). Third, parasitoids are often introduced into new environments for the biological control of insect pests, and the consequent bottleneck these populations experience has been repeatedly hypothesized as an impediment for establishment (Fauvergue et al. 2012). Given these idiosyncratic features, it is straightforward to expect that parasitoid wasps should experience much more stringent consequences of CSD than what has been predicted to date.

In this paper, we investigate on the consequences of single locus Complementary Sex Determination on host-parasitoid dynamics via an original semi-stochastic model based on trophic interactions. We address three questions: (1) what are the consequences of *sl*-CSD on parasitoid extinction and the dynamics of host-parasitoid systems; (2) to what extent is the counter-intuitive effects of *sl*-CSD that we reveal an emerging property of the host-parasitoid interaction; and (3) does *sl*-CSD cause a diploid male vortex in parasitoids similar to that predicted for other species (Zayed and Packer 2005). We suggest that contrary to previous theoretical models and prior expectations, single-locus Complementary Sex Determination can stabilize the dynamics of host-parasitoids systems and promote parasitoid persistence.

Methods

We combined trophic interactions and genetic processes within a discrete-time, two-section population model. At each generation, the number of hosts and parasitoids was predicted by a deterministic host-parasitoid population dynamic model, whereas mating and allele transmission followed a stochastic sub-model. The deterministic section of the model describes the number of hosts (N) and the number of parasitoids developing from either unfertilized (M) or fertilized (D) eggs. Unfertilized eggs always produced normal, haploid males. In contrast, the fate of fertilized eggs depended on their genotype and the presence of *sl*-CSD, and was therefore governed by the stochastic section of the model. Here diploids developed into females (F) if heterozygous at the *csd* locus or if *sl*-CSD was absent, or into diploid males

(*DM*) otherwise. In cases where diploid males were assumed viable, the model also allowed for unviable triploid individuals (*T*) resulting from crosses between a female and a diploid male.

Mating structure and gene transmission

Initial allelic diversity at the *csd* locus was set to *w*, chosen within the range of naturally occurring genetic diversity for *csd* in Hymenopteran parasitoids (commonly 9–20 alleles, see Heimpel et al. 1999, Zayed and Packer 2005, Hedrick et al. 2006). Genotypes were then randomly attributed to males and females founding the parasitoid population. In the next generations, each newborn individual was assigned to a mother among the *F* females given by the population dynamic model, and, except for haploid males, to a father, by assigning to each of the *F* females a mate drawn randomly among the *M* (if *DM* are unviable) or the *M* + *DM* males (if *DM* are sterile; we assumed the same mating success for haploid and diploid males). By doing so, we assumed a monandrous–polygynous mating system, as observed in most parasitoid wasps (Godfray and Cook 1997, Quicke 1997). Offspring genotype at the *csd* locus resulted from classical Mendelian inheritance of parental alleles. When we assumed diploid males to be unviable, homozygous fertilized eggs (diploid males) died immediately after birth. When diploid males where assumed to be sterile, those homozygous eggs developed into diploid males and mated, but the females they mated produced only haploid sons and unviable triploid individuals. Mendelian inheritance yielded a form of demographic stochasticity: the number of offspring per female varied according to the random assignations of genotypes to newborn individuals. For simulations without *sl*-CSD, all fertilized eggs either homozygous or not developed into females. We ensured that the stochastic section of the model had no effect on the population dynamics without *sl*-CSD through a comparison with predictions from a purely deterministic version of the model described in Bompard et al. (2013).

Population dynamics

To predict the dynamics of host and parasitoid populations, we built from the host-parasitoid model from Bompard et al. (2013). At each generation, the abundance of hosts (*N*), fertilized (*D*) and unfertilized (*M*) parasitoid eggs were determined according to the abundance of parasitoid females (*F*) and hosts (*N*) at the previous generation (Eq. 1, deterministic section). We assumed that parasitoid males were not limiting so that all females were mated (no mate-finding Allee effect). To convert the real numbers provided by the deterministic section of the model into natural numbers suitable for the stochastic section, we used a stochastic rounding method: the decimal part of each real number gave a probability of producing a supplementary offspring (e.g., a predicted 2.3 offspring has 70% chance to give 2 offspring and 30% chance to give 3 offspring).

The combination of deterministic and stochastic sections resulted in the following equations:

$$\left.\begin{aligned} N_{t+1} &= \lambda N_t g(N_t) f\left(F_t\right) \\ D_{t+1} &= (1-s_0) N_t g(N_t)\left[1-f\left(F_t\right)\right] \\ M_{t+1} &= s_0 N_t g(N_t)\left[1-f\left(F_t\right)\right] \end{aligned}\right\} \text{Deterministic section}$$

$$D_{t+1} \left| \begin{array}{l} \text{fathered by} M_t \left| \begin{array}{l} \text{heterozygotes at the } csd \text{ locus} \rightarrow F_{t+1} \\ \text{homozygotes at the } csd \text{ locus} \rightarrow \mathrm{DM}_{t+1} \end{array} \right. \\ \text{fathered by} \mathrm{DM}_t \rightarrow T_{t+1} \end{array} \right\} \text{Stochastic section} \tag{1}$$

where λ is the host finite rate of increase, and s_0 the proportion of unfertilized parasitoid eggs, that we assumed to be balanced (s_0 = 0.5), leading to a balanced distribution of sexes in the absence of diploid male production. Functions *f* and *g* describe the proportion of hosts escaping parasitism (May 1978) and the proportion of hosts surviving intraspecific competition (Maynard Smith and Slatkin 1973), respectively. The proportion of hosts escaping parasitism depends on the abundance of female parasitoids, their searching efficiency (*a*) and the aggregation of attacks (*k*), following the equation (May 1978):

$$f(F_t) = \left(1 + a\frac{F_t}{k}\right)^{-k}, \tag{2}$$

where *k* scales the aggregation of parasitoid attacks (May 1978). The distribution of parasitoid attacks is strongly aggregated when $k < 1$ and can be considered as random (Poisson) when $k > 5$. The probability of hosts surviving from intraspecific competition depends on population abundance (*N*), carrying capacity (*K*), finite rate of increase (λ) and the severity of competition (*b*), following the equation (Maynard Smith and Slatkin 1973):

$$g(N_t) = \left(1 + (\lambda - 1)\left(\frac{N_t}{K}\right)^b\right)^{-1}, \tag{3}$$

For high values of λ and when considered independently of parasitoids, host dynamics depend on

intraspecific competition via the parameter b. For $b < 1$, competition is undercompensated and the host population is asymptotically stable; for $1 < b < 2$, the host population is stable with damped oscillations; for $b > 2$, competition is overcompensated and the host population is cyclic (Maynard Smith and Slatkin 1973).

Simulations

Each set of parameters tested was iterated 10,000 times, and simulations ended either when the parasitoid went extinct (i.e., $F < 1$) or after 3,000 generations. Most extinctions nonetheless occurred within 200 generations. Unless otherwise mentioned, initial allelic diversity and the number of foundresses were high enough to prevent any early extinction that would be due to stochastic events (i.e., $w = 15$ and $F_0 = 50$).

The consequences of *sl*-CSD on host-parasitoid dynamics were quantified through the following measurements. For each set of parameters tested, both with and without *sl*-CSD, we calculated the probability of parasitoid extinction $P(E)$ as the proportion of simulations ending as a result of parasitoid extinction before 3,000 generations. When those extinctions occurred, we computed the mean time to extinction $T(E)$ for each parameter set; we discuss the median of these $T(E)$ for the range of tested parameter sets. In addition, we kept track of the evolution of population sizes and allelic richness across time. For parameter sets leading to cyclic dynamics, three additional features were computed for the parasitoid population: (1) the amplitude of parasitoid cycles, (2) the minimal population size during the cycles, and (3) the mean parasitoid abundance during the cycles. The effect of *sl*-CSD on cycling dynamics was appraised through the relative variation of these features between populations with and without *sl*-CSD. Finally, to appraise the effect of *sl*-CSD on host control, when parasitoid persisted with and without *sl*-CSD, we computed (1) the amplitude of host cycles (when present) and (2) the relative host abundance $q = N^*/K$ (where N^* is the mean host abundance over the last 2000 generations).

We first focused on the consequences of *sl*-CSD on parasitoid extinction. For this, we displayed the probability of parasitoid extinction $P(E)$ as a density plot on a λ-a diagram. This representation highlights two different extinction domains. The *cyclic extinction* domain, at the upper left side of the diagram, is characterized by high parasitoid searching efficiency and low host finite rate of increase; this λ-a combination produces high-amplitude cycles causing parasitoid extinction. The *asymptotic extinction* domain, at the lower right side of the diagram, is characterized by low parasitoid searching efficiency and high host rate of increase; this λ-a combination yields a progressive decrease in parasitoid abundance, until extinction.

To unravel the effect of host-parasitoid interactions on the demographic consequences of *sl*-CSD, we modified the core model by varying artificially the amount of parasitoid dependence on its host, but keeping the genetic processes unchanged. We assumed that only a constant fraction x of the parasitoid population was dependent on the host (Eq. 1). The complementary fraction $1-x$ was assumed to be independent and hence, only influenced by intraspecific competition as described for hosts (Eq. 3). The deterministic section of the model became:

$$\begin{cases} N_{t+1} = \lambda N_t g_h(N_t) f\left(xF_t\right) \\ D_{t+1} = (1-s_0)\left[(1-x)\lambda_2 (F_t + M_t) g_p\left((1-x)(F_t+M_t)\right)\right. \\ \qquad \left. + N_t g_h(N_t)\left(1-f\left(xF_t\right)\right)\right] \\ M_{t+1} = s_0\left[(1-x)\lambda_2 (F_t + M_t) g_p\left((1-x)(F_t+M_t)\right)\right. \\ \qquad \left. + N_t g_h(N_t)\left(1-f\left(xF_t\right)\right)\right] \end{cases} \quad (4)$$

where λ_2 is the finite rate of increase of the noninteracting fraction of the parasitoid population, g_h is the host intraspecific competition and g_p is the parasitoid intraspecific competition. Severity of the intraspecific competition (b) and carrying capacity (K) are distinct for the host and for the non-interacting fraction of the parasitoid population. For $x = 1$, the model is strictly identical to Eq. 1. For $x = 0$, the parasitoid population is completely independent from the host and grows as a free-living population (henceforth named *non-interacting population*); intermediate x values correspond to a parasitoid population growth with intermediate degrees of dependence on host species N, as representative of nonspecialist parasitoid species. We assessed the interaction between the presence of *sl*-CSD and x on the probability of extinction $P(E)$. For varying x values (from 0 to 1 with a 0.2 increment), we drew randomly 5,000 sets of parameter values from a large range of biologically meaningful values (see Table 1). For each of these sets of parameters, we estimated the effect of *sl*-CSD through the difference in extinction probability with and without *sl*-CSD. We call this difference $\Delta P(E)$. For a given set of parameter, a positive value for $\Delta P(E)$ indicates that *sl*-CSD increases the extinction probability whereas a negative value indicates that it reduces it. We plotted, according to x, the proportion of positive and negative $\Delta P(E)$ computed among those of the 5,000 $\Delta P(E)$ that are non-null.

Host-parasitoid interaction can generate cyclic dynamics. To appraise the relative influence of cycles per se and cyclic dynamics typical of parasitoids on the demographical consequences of *sl*-CSD, we compared cyclic populations resulting either from host-parasitoid interactions ($x = 1$) or from strong intraspecific competition as it may be the case in non-interacting populations ($x = 0$). So, confining to parameter sets for which populations cycle without *sl*-CSD, we appraised for $x = 0$ or 1 the effect of *sl*-CSD on mean, amplitude and minima of cycles as previously explained.

The Diploid Male Vortex is characterized by an increased extinction risk when initial conditions get more restrictive (*e.g.*, lower parasitoid population size and allelic diversity). To appraise whether *sl*-CSD in parasitoids causes an extinction vortex similar to the Diploid

Male Vortex described in non-interacting populations (Zayed and Packer 2005) we estimated how restrictions in initial conditions affected extinctions induced by *sl*-CSD (positive $\Delta P(E)$). For this, we define the vortex index $V_{i,w}$ to characterize the impact of an initial number of females i and an initial allelic diversity w on the risk of extinction due to *sl*-CSD:

$$V_{i,w} = \frac{\Delta P(E)_{i,w} - \Delta P(E)_{\min}}{\Delta P(E)_{\min}} \quad (5)$$

where $\Delta P(E)_{i,w}$ is the difference in extinction probability with and without *sl*-CSD for initial conditions i and w, and $\Delta P(E)_{\min}$ is the minimal difference in extinction probability, *e.g.*, the effect of *sl*-CSD on extinction probability when using the least restrictive initial conditions, described in Table 1. Therefore, high values for the vortex index indicate a strong impact of initial conditions on extinction risk attributed to *sl*-CSD and reflect the occurrence of a Diploid Male Vortex. In contrast, a vortex index approaching zero suggests a dissimilar process in CSD-induced extinctions. We computed the vortex index over various initial conditions and a large range of demographic parameters, restricted to conditions where the parasitoid population persisted without *sl*-CSD, as described in Table 1. We compared the vortex index values for parasitoid ($x = 0$) and for non-interacting ($x = 1$) populations.

Results

CSD impact on parasitoid extinction and the dynamics of host-parasitoid systems

Overall *sl*-CSD reduced the probability of parasitoid extinction. Across 5000 randomly drawn parameter sets, we found a probability of parasitoid extinction of 71.6% without *sl*-CSD, 65.6% with *sl*-CSD and sterile diploid males, and 60.7% with *sl*-CSD and unviable diploid males (Fig. 1). The *cyclic extinction* domain, characterized by high parasitoid efficiency and low host growth rate, was smaller with *sl*-CSD than without. Conversely, the *asymptotic extinction* domain, characterized by low parasitoid efficiency and high host growth rate, was larger with *sl*-CSD than without (Fig. 1A vs. 1B, C).

A prominent effect of *sl*-CSD on host-parasitoid dynamics was to buffer parasitoid cycles. In the domain

Table 1. Parameters' descriptions and values.

	Symbol	Parameter	Range of value considered and **default value***	Key values
Demography	x	Fraction of the population interacting with the host	$0 < x < 1$ x **= 1**	$x = 1 \rightarrow$Parasitoid population $x = 0 \rightarrow$Non-interacting population
	λ	Host finite rate of increase	$0 < \lambda < 50$	
	λ_2	Finite rate of increase of the non-interacting fraction of the population	$0 < \lambda_2 < 50$	
	b	Severity of host intraspecific competition	$0.5 < b < 3$ b **= 1.5**	$b < 1 \rightarrow$contest $1 < b < 2 \rightarrow$mild $b > 2 \rightarrow$scramble
	b_2	Severity of the intraspecific competition for the non interacting fraction of the population	$0.5 < b < 3$	
	K	Host carrying capacity	$20 < K < 10{,}000$ K **= 5,000**	
	K_2	Carrying capacity for the non-interacting fraction of the population	$20 < K_2 < 10{,}000$	
	a	Parasitoid searching efficiency	$0 < a < 0.1$	
	k	Aggregation in parasitoid host search	$0.5 < k < 20$ k **= 10**	$k < 1 \rightarrow$aggregated $k > 5 \rightarrow$random
	s_0	Rate of unfertilized eggs	s_0 **= 0.5**	
Initialization	w	Initial allelic diversity for CSD	$2 < w_0 < 50$ w **= 15**	
	F_0	Initial number of females	F_0 = 20, 75 and 200 F_0 **= 50**	
	N_0	Initial number of hosts	N_0 **= 1,000**	
	Maximum duration of a simulation			3,000 generations
	Simulations/parameter set			10,000

*The value used for simulations, otherwise mentioned.

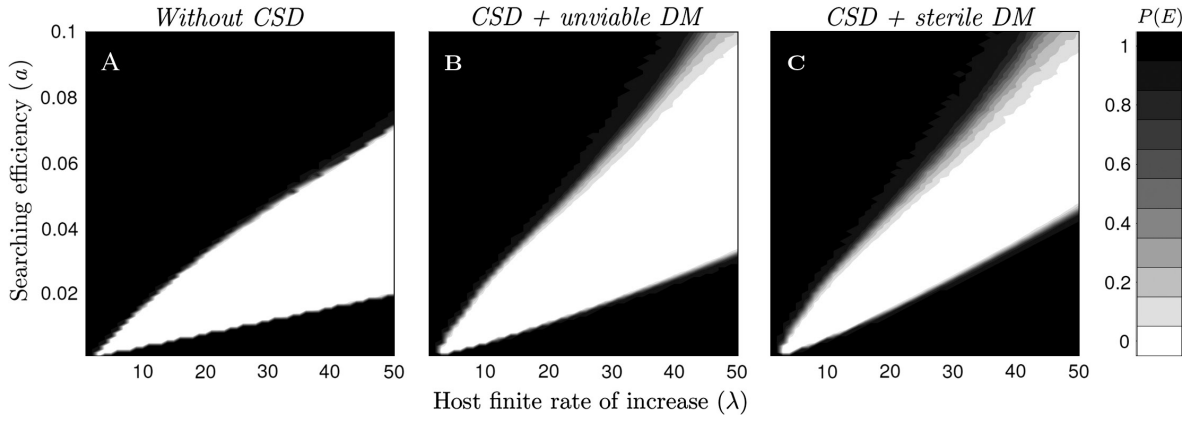

FIG 1. Probability of extinction ($P(E)$) for parasitoid populations without CSD (A), with CSD and unviable diploid males (B), or with CSD and sterile diploid males (C) as function of parasitoid searching efficiency (a) and host intrinsic growth rate (λ). For mild host competition (b = 1.5) and random attacks (k = 10). See Table 1 for other parameter values.

of parameters where parasitoid populations cycled, *sl*-CSD with unviable diploid males yielded in a 25.4% ± 2.7%, (mean ± SD; number of parameter sets = 1,296) decrease in the amplitude of cycles and a 253.5% ± 8.7% (n = 1,296) increase in the minima at the bottom of the cycles. With these two effects, some parasitoid populations driven into cyclic extinction without *sl*-CSD did persist with *sl*-CSD (Figs. 1 and 2A, B). Moreover, cyclic parasitoid maintained an average abundance 18.4% ± 5.4% higher with *sl*-CSD than without, driving a better control of the host population: the proportion of hosts remaining after parasitism was on average 16.3% ± 14.4% lower with *sl*-CSD than without. The amplitude of host cycles were also decreased by 28.1% ± 16%, driving lower maximum abundances for the host population. In contrast, for parasitoids that persisted close to the *asymptotic extinction* domain, *sl*-CSD with unviable diploid males yielded a decrease in parasitoid abundance, which ultimately drove populations to extinction (Fig. 2D vs. 2E with the median of average extinction times $T(E)$ being 116 ± 0.35 generations (n = 3,637)). When asymptotic populations persisted *sl*-CSD yielded a 6.4% ± 12.8% (n = 930) decrease in parasitoid abundance and a 46.6% ± 22.8% increase in the proportion of remaining hosts.

When diploid males are viable but sterile, both cyclic and asymptotic extinction domains slightly increased compare to the unviable scenario (Fig. 1B vs. 1C). For parasitoids that persisted close to the area of asymptotic extinction without *sl*-CSD, extinctions were faster with sterile than with unviable diploid males (Fig. 2E, F): for the same range of parameter sets, the median extinction times $T(E)$ with sterile diploid males was 38 ± 0.20 generations (n = 3,637), compared to 116 generations for unviable diploid males (see previous text).

In cyclic parasitoid populations, most of the initial allelic diversity at the *csd* gene was lost during the first cycle and populations persisted despite a low allelic richness (2–5 alleles; Fig. 2B). Conversely, in parasitoid populations close to the area of asymptotic extinction, the allelic diversity for the *csd* gene progressively decreased, following population abundance (Fig. 2E). When these populations persisted despite *sl*-CSD, their allelic diversity settled to moderate levels (4–10 alleles).

The impact of *sl*-CSD on parasitoid dynamics was qualitatively unaffected by host intraspecific competition (b) and parasitoid distribution of attacks (k) (see Appendix S1). Strongly aggregated attacks nonetheless damped parasitoid cycles even without *sl*-CSD, emphasizing the deleterious consequences of *sl*-CSD (see Appendix S1: Fig. S1D–F).

Is the observed effect of sl-CSD an emerging property of the host-parasitoid interaction?

The demographic impact of *sl*-CSD varied according to the parasitoid dependence on its host (x). In noninteracting populations (x = 0), *sl*-CSD much more frequently increased the probability of extinction (Fig. 3). The effect of *sl*-CSD was reversed as soon as the population growth partially depended on its host ($0.2 \leq x < 1$), with a beneficial effect at its maximum for strict parasitoid populations (x = 1; Fig. 3).

Our results revealed that the demographic impact of *sl*-CSD on cyclic populations also differed between noninteracting (x = 0) and parasitoid (x = 1) populations. The decrease in the amplitude of cycles induced by *sl*-CSD was smaller when cycles occurred in non-interacting populations than when they resulted from the host-parasitoid interaction (11.87% ± 0.47%, n = 19,989 vs. 25.39% ± 2.74%, n = 1,296). In addition, *sl*-CSD decreased by 12.59% ± 0.49% (n = 19,989) the minima of non-interacting population cycles, whereas it increased that of parasitoid cycles (by 253.5% ± 8.7%, see previous text).

Does sl-CSD cause an extinction vortex in parasitoids?

When initial conditions became restrictive the deleterious effect of *sl*-CSD became stronger in noninteracting

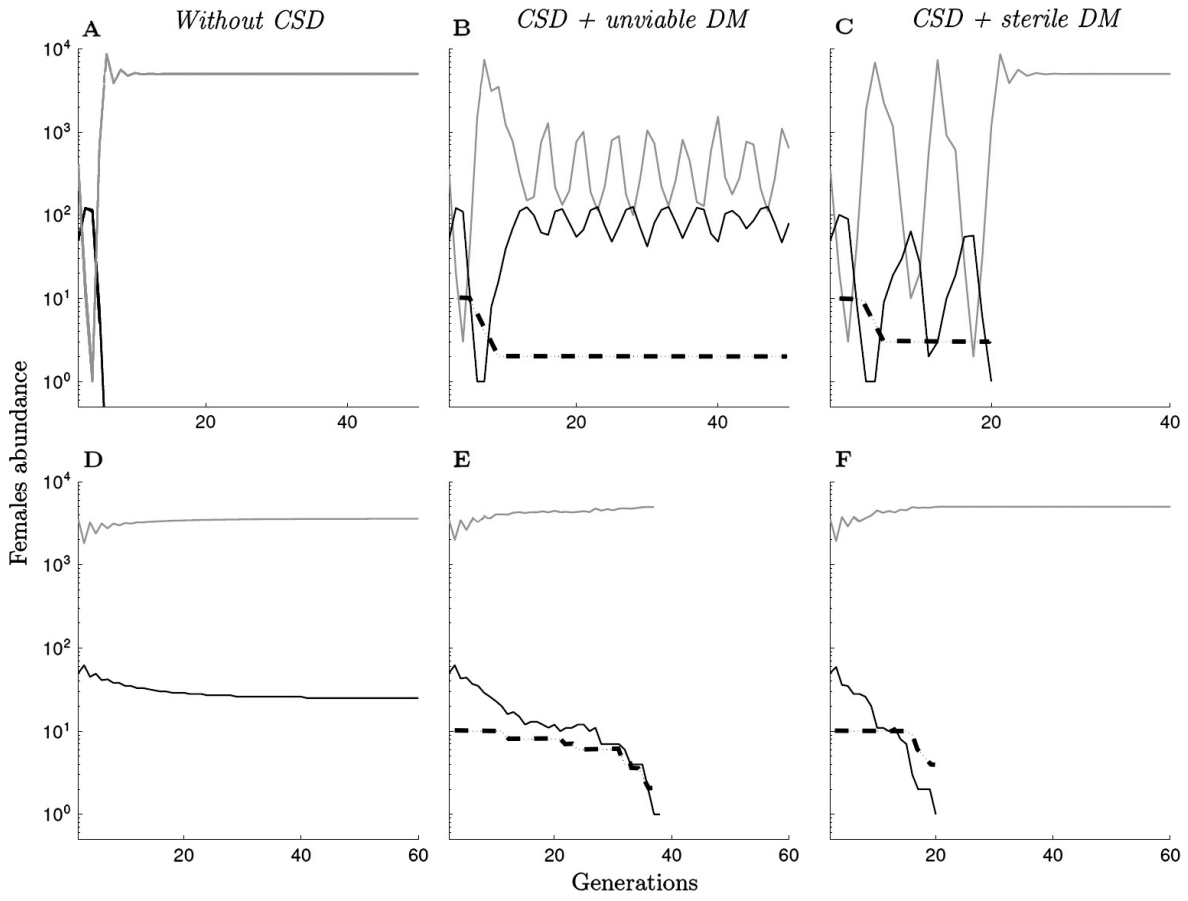

FIG 2. Host and parasitoid chronicles without CSD (A, D), with CSD and unviable diploid males (B, E) or with CSD and sterile diploid males (C, F), for two sets of parameters in high (A, B, C) and low (D, E, F) parasitoid efficiency. Black line: female parasitoid abundance; grey line: host abundance; dashed line: allelic diversity at the *csd* locus. Parameter combinations for high efficiency chronicles (A, B, C): $a = 0.049$, $\lambda = 9$; for low efficiency chronicles (D, E, F): $a = 0.01$, $\lambda = 6$. See Table 1 for other parameter values.

populations than in parasitoid populations, as revealed by the stronger increase in vortex index under reduced initial allelic diversity or number of foundresses (Fig. 4). The reduction of the initial allelic diversity (w) sharply increased the impact of *sl*-CSD on the extinction risk for non-interacting populations but had a much weaker impact in parasitoid populations. Moreover a decrease in the initial number of foundresses (F_0) caused a substantial increase in the *sl*-CSD impact in non-interacting populations but had no effect for parasitoids, which caused the three parasitoid lines in Fig. 4 to be superimposed. In summary, Fig. 4 shows that extinctions induced by *sl*-CSD are less dependent on initial conditions in parasitoids than in non-interacting populations.

DISCUSSION

Inbreeding depression is one of the most important drivers of extinction in small populations (Frankel and Soulé 1981, Caughley and Gunn 1996, Saccheri et al. 1998) and has been extensively studied in the context of conservation efforts (Caughley and Gunn 1996, Kampichler et al. 2010, Palomares et al. 2012). The extinction of small populations resulting from feedbacks between demographic and genetic processes is described as an extinction vortex (Gilpin and Soule 1976, Tanaka 2000). Previous modeling approaches of extinction vortices have generally assumed non-interacting populations with basic population dynamics. In more complex situations where, for instance, populations of several species interact with one another, the outcome of interplays between genetics and demography is far more difficult to predict. In this study, we focused on the demographic consequences of a particular form of inbreeding depression resulting from single-locus complementary sex determination (*sl*-CSD) that prevails in insects of the order Hymenoptera. Previous theoretical work indicated that *sl*-CSD can induce an extinction vortex in non-interacting populations (Hedrick and Parker 1997, Zayed and Packer 2005).

Here, we show that when CSD occurs in parasitoid wasps that interact with their hosts, it can promote

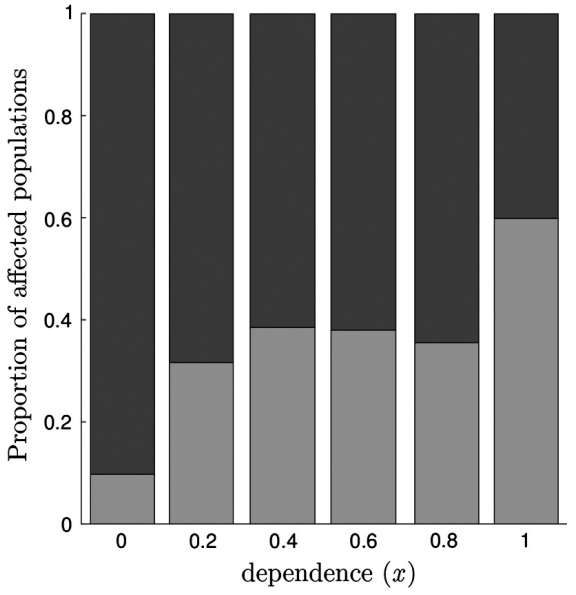

FIG 3. Proportion of populations where CSD increased (dark grey) or decreased (light grey) the probability of extinction, depending on the dependence of the population to its host (x = 0: independent population, x = 1: dependent (parasitoid) population). We included all parameter sets where CSD affected the probability of extinction, among 10,000 simulations from randomly drawn parameter sets (see Table 1 for limits).

population persistence. By artificially manipulating the degree to which parasitoids depend on their hosts, we demonstrate that the host-parasitoid interaction is the essential feature that yields a positive effect of inbreeding depression on persistence. This counterintuitive result appears as a consequence of the interplay between CSD and parasitism on cycling dynamics. Moreover, contrary to expectations, the fate of parasitoid populations with CSD is less determined by initial population size and initial genetic diversity than by parasitoid searching efficiency and host dynamics. Hence, the diploid male vortex predicted in non-interacting species (Zayed and Packer 2005) may not exist in parasitoid wasps.

Damping the cycles

Complementary Sex Determination promotes the persistence of parasitoid populations when dynamics are cyclic, i.e., when the host mortality induced by parasitoids is so strong that it is not compensated by an alleviation of host competition. The production of diploid males due to CSD limits the impact of parasitoids on their hosts. Two opposite effects of CSD on cyclic dynamics follow one another according to whether or not the host availability limits parasitoid growth: (1) when hosts are sufficiently abundant, diploid male production limits the parasitoid population rate of increase; and (2) when hosts are rare because of parasitism, the parasitoid population growth constrained by diploid males mitigates parasitism pressure, so that CSD limits host (and thus parasitoid) decline. By limiting parasitoid growth rate when parasitoid abundance increases and increasing parasitoid growth rate when parasitoid abundance decreases, CSD damps parasitoid cycles. Moreover, by maintaining host availability and enhancing parasitoid growth when it is the most constrained—at the bottom of the cycles—CSD allows an increase in the mean abundance of parasitoids at equilibrium. This is possible without destabilizing the cycles because CSD keeps limiting parasitoid growth at the high end of the cycles. Because the parasitoid population is more abundant with CSD, host control is also improved, with a lower host abundance, and lower amplitudes and maxima of the host cycles.

Observations on natural parasitoid population generally indicate more stability than predicted by mathematical models. Key stabilizing processes—such as heterogeneity in parasitoid attacks or meta-population structure—have already been suggested to account for this discrepancy (Murdoch 1994, Bernstein 2000). Our findings suggest that CSD could be considered as one of them. Previous attempts to attribute stability to one particular phenomenon based on experimental data previously have raised vigorous debate (Murdoch and Stewart-Oaten 1989, Pacala et al. 1990, Murdoch 1994). Similarly, disentangling CSD effect from other stabilizing

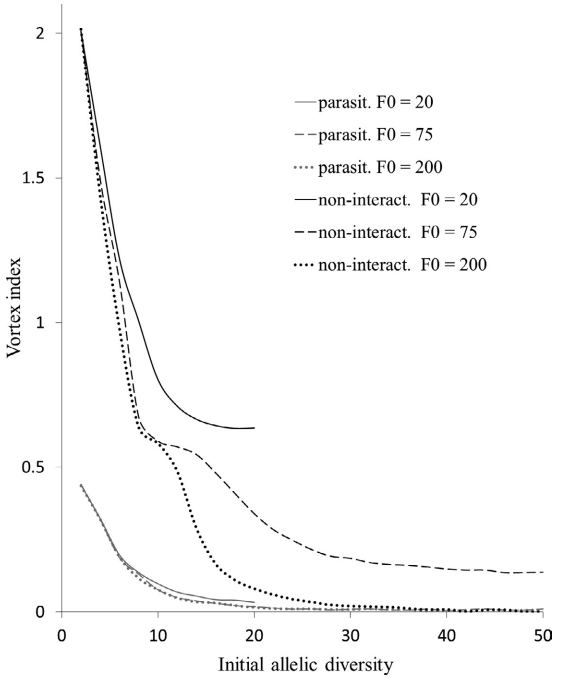

FIG 4. Vortex index (difference in CSD induced extinctions compared to optimal initial conditions) depending on the initial allelic diversity, for three different numbers of foundresses F_0 (plain line: F_0 = 20; dashed line: F_0 = 75; dotted line: F_0 = 200). High values indicate a strong effect of initial conditions on CSD propensity to cause extinctions, while low or superimposed lines suggest little impact of initial conditions. Parasitoid populations are figured by grey lines, and non-interacting populations are figured by black lines.

processes occurring in natural population might prove a difficult task. From an evolutionary perspective, population oscillations can be driven by a predator adaptation rate exceeding that of the prey (Mougi and Kishida 2009), a scenario that would position CSD as a safeguard, compensating for a fast evolution of parasitoid efficacy. A formal study of the eco-evolutionary dynamics of CSD and other key stabilizing processes in host-parasitoid systems could help untangle the effective impact of CSD on the stability of parasitoid populations.

A major difference between CSD and other stabilizing processes is its impact on host control. The works of Luck (1990) and Murdoch (1994) describe the "biological control paradox" as the opposition between theoretical model predictions, which show that increasing stability tend to increase prey abundance, and empirical examples from successful biological control where the prey is stable at very low abundances. CSD, by buffering parasitoid cycles and yielding a higher parasitoid abundance, can increase both stability and host control. Obviously, CSD cannot be the universal response to the biological control paradox as it is specific to a number of Hymenopteran parasitoids, but our work gives a viable lead to understand how it could be bypassed by the genetic structure of the parasitoid or predator species.

The fact that CSD stabilizes host-parasitoid systems by damping demographic cycles does not imply that CSD will stabilize any cyclic dynamics. By comparing the effect of CSD on cycling populations outside of the host-parasitoid system we suggest that in non-interacting populations, CSD reduces cycles amplitude to a lesser extent and decreases minimal population size, exposing the population to higher extinction risks in case of stochastic events. Several authors already noted that strong density dependence and cycles could counteract the deleterious effects of genetic load on population persistence (Chapman et al. 2001, Agrawal and Whitlock 2012). What we highlight here is the importance of the biological processes sustaining the cycles when appraising the effects of genetic load.

As CSD provokes a reduction in female parasitoid production per attacked host, its demographic consequences resemble a decrease in parasitoid efficiency. In parasitoid wasps, other phenomenon leading to a decrease in parasitoid efficiency (such as interference) or more globally to a decrease in parasitism pressure (such as mating failure) have been largely examined by various theoretical works (Hassell and Varley 1969, Briggs and Collier 2001, Bompard et al. 2013, Wogin et al. 2013). However these mechanisms act differently from CSD in the sense that their impact on parasitism pressure depends on parasitoid density and is consequently under feedback control. Conversely, CSD impact on parasitism pressure primarily depends on allelic diversity. At an ecological time scale, demographic processes can make the density fluctuate up and down but the loss of allelic diversity is irreversible, resulting in a constant restriction of parasitoid growth. Frequency-dependent selection on *csd* alleles participates to this phenomenon by allowing the allelic diversity at the *csd* locus to settle to an equilibrium. This suggests an original effect of the synergy between demographical and genetic processes on extinction risks and highlights an important difference between genetic-based and density-based Allee effects (see also Luque et al. 2016).

Extinctions under CSD

Single-locus complementary sex determination can drive parasitoid populations to extinction, particularly at low parasitoid searching efficiency and high host rate of increase. However, these extinctions have underlying causes that differ from those predicted by the diploid male vortex described in free-living hymenoptera (Zayed and Packer 2005). We showed that parasitoid extinctions induced by CSD are almost independent of the initial population size and genetic diversity, two major triggers of the diploid male vortex (Zayed and Packer 2005). Hence, parasitoid populations could be, to some extent, immune to moderate erosions of effective population size (Henter 2003, Fauvergue et al. 2007). Rather, CSD results in parasitoid populations being more sensitive to declines in searching efficiency or host carrying capacity. Such decreases occur regularly in agro-ecosystems due to crop rotations or pesticide use (Tscharntke and Brandl 2004, Rusch et al. 2011); our modeling results suggest that these extrinsic causes of population decline could combine with genetic mechanisms and impact populations more severely than previously envisaged.

Diploid male sterility and stochasticity in interacting populations

In populations experiencing CSD, the fate of diploid males is not always early death. In some species, diploid males are viable but sterile (Van Wilgenburg et al. 2006). In this latter case demographic impact of CSD is more complex because the impact of diploid male production on realized fecundity extends to an extra generation (Fauvergue et al. 2015). As expected this additional reduction of female production further increases the domain of *asymptotic* extinction but its impact on cyclic dynamics is more intricate. When allelic diversity at the *csd* locus is low, the probability of mating with a diploid male and thus producing only unfertilized eggs increases and with it the variability in realized fecundity. This increased stochasticity may drive cyclic parasitoid populations to extinction, as classically observed when demographic stochasticity is introduced in host-parasitoid models (Wilson and Hassell 1997).

Natural parasitoid populations

Although demographic data on parasitoids with CSD are too rare to draw general conclusions, it is interesting that the two experimental studies that we are aware of failed to demonstrate a diploid male vortex and hence,

support our theoretical predictions (*Cotesia glomerata* (Elias et al. 2010) and *Venturia canescens* (Vayssade 2014)). In the wild, if parasitoid populations persist with a low number of alleles at the *csd* locus, as predicted by our model, inbreeding avoidance may not result in drastic changes in the production of diploid males. In such situation and if inbreeding avoidance is costly, sib-mating is expected to be selected for (Getz et al. 1992). Although there is evidence for inbreeding avoidance in parasitoids with CSD (Metzger et al. 2010) this is not a rule, and sib-mating exists in species with CSD (El Agoze et al. 1994, Cowan and Stahlhut 2004, Fauvergue et al. 2015). This has been considered to be a paradox for species with CSD, given that the Diploid Male Vortex should drive such inbred population to extinction (Heimpel and de Boer 2008). Our theoretical results suggest that there may be neither a Diploid Male Vortex, nor such paradox in parasitoids.

Conclusion

Our modeling study reveals how feedback between demography and genetics can follow different routes and reach distant endpoints. By assuming interacting populations in the form of a host-parasitoid system, we show that inbreeding depression can sometimes reduce population extinction risk, and therefore, yield an effect opposite to that expected in non-interacting populations. The demographic cycles caused by trophic interactions could have been envisaged as catalyzers of the erosion of allelic diversity; speeding up the drop in the extinction vortex. Rather, we identify conditions in which the reduction in allelic diversity buffers the cycles, increases parasitoid abundance and promotes both stability and host control. Conversely, CSD can drive stable parasitoid population to extinctions; but here again the underlying host-parasitoid interaction impacts the demographic process to the point that it becomes very different from a single-population extinction vortex. Combining trophic interactions and genetic diversity can yield drastically different demographic predictions than considering genetic diversity in a single population, which suggest we should exert caution when using demo-genetic models with over-simplified demography for predictions and conservation policies.

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