Etude théorique des conséquences démographiques et évolutives des processus s'exprimant à faible densité dans les systèmes hôte-parasitoïde

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Theoretical study of demographic and evolutionary consequences of processes expressed at low density in host-parasitoid systems

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RÉSUMÉ

Dans une population de faible densité, la diversité génétique est réduite et les partenaires sexuels sont plus difficiles à trouver, ce qui augmente le risque d'extinction si la démographie est simple. Dans ces travaux, j'étudie par la modélisation l'impact de tels phénomènes sur les populations de parasitoïdes, chez qui la démographie est plus complexe en raison de l'interaction avec l'hôte. Les parasitoïdes sont souvent utilisés en lutte biologique. A cette occasion ils sont introduits dans un nouvel environnement en faibles densités, ce qui justifie particulièrement l'étude du devenir des petites populations de parasitoïdes. Ma thèse est sur articles et composée de trois parties : i) une synthèse de la bibliographie ; ii) mes travaux sur les conséquences de l'effet Allee comportemental sur la démographie des parasitoïdes ; iii) mes travaux sur les conséquences du *Complementary Sex Determination* (CSD), un mécanisme qui induit un fardeau génétique propre aux hyménoptères, sur la démographie des parasitoïdes et l'évolution du choix de partenaire. La prise en compte des interactions interspécifiques est absolument indispensable pour prédire les conséquences des mécanismes s'exprimant à faible densité. La sensibilité des parasitoïdes aux effets Allee dépend de la dynamique de leur hôte. Contrairement à ce qui a été décrit chez les populations non parasitoïdes, le CSD n'induit pas de vortex d'extinction à proprement parler chez les parasitoïdes, mais au contraire il permet de limiter leur risque d'extinction. Ce résultat très novateur apporte un nouveau regard sur les conséquences démographiques et évolutives des fardeaux génétiques dans les populations en interaction.

ABSTRACT

In low-density populations, genetic diversity is decreased and mate-finding is increasingly difficult, two phenomena that may induce extinctions if the demography is simple. During my thesis, I used mathematical modeling to investigate the impact of such phenomena on parasitoids in which population dynamics are more complex (due to the close interaction between the parasitoid and its host. The study of the fate of small parasitoid populations is particularly justified in the context of biological control, in which parasitoids are widely used and often introduced in low densities. This manuscript is built on scientific papers and organized into three parts: i) a literature synthesis; ii) the theoretical impact of mate-finding Allee effect on the demography of parasitoids and iii) the theoretical impact of *Complementary Sex Determination* (CSD), which induces a genetic load specific to the Hymenoptera, on the demography of parasitoids on the evolution of mate-choice to avoid the deleterious consequences of this burden at the individual level. I conclude that considering the presence of an interspecific interaction, such as the host-parasitoid interaction, is crucial to predict the consequences of Allee effects and genetic load. We show that parasitoid survival can be very sensitive to mate-finding Allee effects, depending on the host dynamics. We also show that contrary to what has been described in non parasitoid populations, CSD does not induce an extinction vortex in parasitoids, but on the contrary limits their extinction risk. This highly innovative result provides a new look at the demographic and evolutionary consequences of genetic load in interacting populations.

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Introduction générale

Extinction ou colonisation ?

Ces dernières décennies ont été témoin d'une escalade effrayante dans le rythme des extinctions d'espèces et des invasions biologiques. Parmi les points communs de ces deux évènements on retrouve leur impact écologique, économique et culturel redoutable et l'implication des activités humaines, mais également le passage de la population par un épisode où sa taille est réduite à un faible nombre d'individus. Pour certaines populations, cet épisode est lié à la colonisation d'un nouvel environnement et c'est le prélude à une expansion parfois catastrophique. Pour d'autres c'est un point de non retour qui précède l'extinction.
Pourquoi, à partir d'un même nombre d'individus, certaines populations s'éteignent quand d'autres deviennent pléthoriques, malgré nos efforts intenses pour les conserver ou les limiter ? Même aujourd'hui, on ne connaît pas l'ensemble des facteurs qui font le succès d'une invasion biologique. En revanche, on connaît assez bien les facteurs qui contribuent à mener une population de petite taille à l'extinction. Parmi eux, on trouve :

- *les accidents* (ce qu'on appelle la stochasticité) qui peuvent être démographiques (les descendants d'une génération ne sont que d'un sexe, stériles, ou non viables) ou environnementaux (un hiver rude, la crue d'une rivière, etc.)
- *le fardeau génétique*. Lorsque la taille de la population se réduit, une partie de la diversité génétique est perdue par dérive génétique, et le nombre de familles distinctes diminue. La consanguinité touche la plupart des individus, qui expriment des mutations délétères accumulées dans la population. Ces mutations peuvent réduire les capacités de reproduction ou la longévité des individus, et la perte de diversité génétique limite les capacités d'adaptation de la population à des changements environnementaux.
- *un taux de reproduction corrélé à la densité de population* (ce qu'on appelle un effet Allee élémentaire). Pour une espèce de prédateurs qui chasse en meute, la réduction de la taille du groupe peut rendre la chasse plus difficile ; à l'inverse, certaines proies sont plus exposées à leur prédateur lorsqu'elles sont en petit groupe. Les interactions de coopérations,

directes ou indirectes, on souvent une place importante dans la survie et la reproduction des organismes. L'interaction de coopération la plus répandue est celle liée à la reproduction sexuée : lorsque la densité de la population est faible, il peut devenir difficile de trouver un partenaire sexuel.

L'ensemble des facteurs qui réduisent le nombre de descendants produits et/ou leurs capacités de reproduction (c.à.d, les facteurs réduisant la fitness individuelle) lorsque la taille ou la densité de population est faible sont appelés des effets Allee élémentaires. Le principal effet Allee élémentaire est l'augmentation des difficultés à trouver un partenaire sexuel à faible densité, qui diminue les capacités de reproduction : c'est ce qu'on appelle un effet Allee comportemental. Les effets Allee élémentaires peuvent induire une réduction du taux de croissance de la population, et donc de sa taille : c'est l'effet Allee démographique. Si cet effet est fort, la taille de la population décroît avec le temps et celle-ci est amenée à s'éteindre à plus ou moins court terme.

Certaines populations peuvent porter peu de mutations délétères, éviter les accouplements consanguins, ou développer des techniques pour repérer un partenaire sexuel sur de très longues distances. Elles sont cependant le plus souvent incapables de neutraliser complètement le fardeau génétique et les effets Allee comportementaux. Pourtant, il existe des populations qui passent régulièrement par des épisodes de faible densité avant de croître à nouveau dans un cycle constant. C'est le cas par exemple de certaines populations de parasitoïdes dont la dynamique oscille entre forte et faible densité. Comment ces populations persistent-elles, et pourquoi ne s'éteignent-elles pas à l'occasion d'un de ces cycles ?

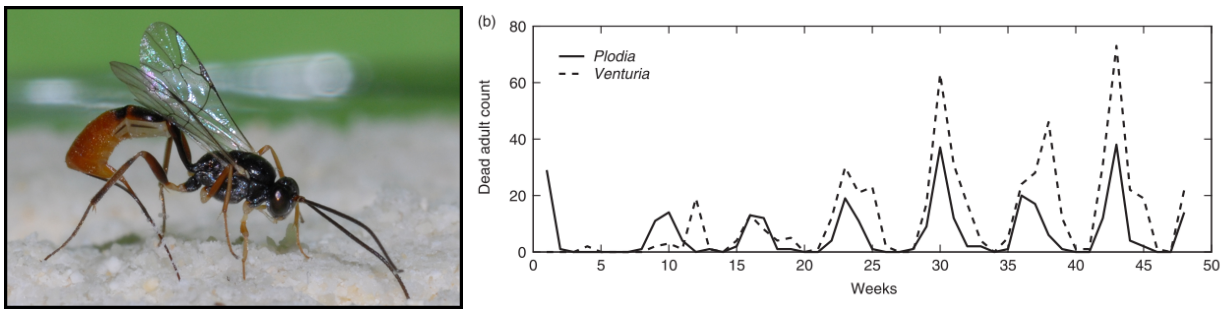

FIGURE 0.1 – Le parasitoïde *Venturia canescens* (crédit photo : François Debias) et l'évolution de la taille de sa population au cours du temps (depuis Wearing et al. (2004))

L'importance des interactions inter-spécifiques

La croissance ou le déclin d'une population n'est pas sans impact sur son environnement : il y a toujours une espèce proie, un parasite, un compétiteur, qui va bénéficier ou pâtir de votre absence. La plupart de ces interactions entre espèces dépendent de la taille de la population : on dit qu'elles sont densité dépendantes. Toutes les espèces interagissent d'une manière ou d'une autre avec une autre espèce, et ces interactions ont un rôle structurel majeur sur la démographie des populations qu'elles affectent.

L'interaction interspécifique peut parfois être exclusive et indispensable au développement d'une espèce : c'est le cas de la relation hôte-parasitoïde. Les insectes parasitoïdes ont la particularité de pondre et de se développer dans ou sur le corps d'un autre insecte, qu'ils tuent à un moment de leur développement. Très souvent, les parasitoïdes ne sont capables d'attaquer qu'une gamme restreinte d'espèces hôtes (voir une seule). Sans hôte dans lequel se développer, le parasitoïde est incapable d'atteindre l'âge adulte.

Lorsque le parasitoïde est en déclin, il exerce une pression moins forte sur l'hôte, qui peut croître. L'abondance des hôtes peut permettre au parasitoïde de retrouver une croissance positive : ce genre de phénomène produit les dynamiques cycliques présentées plus haut. Les relation inter-spécifiques peuvent donc affecter dramatiquement la relation taille de population - taux de croissance qui fait partie du vortex d'extinction présenté précédemment. Ceci nous amène à la question centrale de ma thèse :

Quelles sont les conséquences des phénomènes s'exprimant à faible densité (tels l'effet Allee comportemental ou les fardeaux génétiques) dans une population en intéraction comme les parasitoïdes ?

Pour répondre à cette question, je vais utiliser des modèles mathématiques théoriques qui décrivent ces deux phénomènes et les dynamiques de population qui en résultent. Bien que mes résultats soient basés sur la modélisation, j'argumenterai mon propos en fonction de la littérature existante sur les dynamiques, la physiologie et le comportement des populations naturelles.

Ce manuscrit composé sur articles s'articule en trois grandes parties :

1. L'introduction bibliographique, composée d'un article de synthèse de la littérature sur l'impact du fardeau génétique dans les populations en interaction (article 1, en préparation), d'une partie introductive plus détaillées sur les parasitoïdes et les différents objectifs du manuscrit,
2. L'impact théorique de l'effet Allee comportemental sur la démographie des parasitoïdes, composée de deux articles scientifiques (article 2, publié et article 3, en préparation), et
3. L'impact théorique d'un fardeau génétique (le *Complementary Sex Determination*, ou CSD) sur la démographie et l'évolution des parasitoïdes, composée de trois articles (articles 4, 5 et 6, en préparation).

Le manuscrit est conclu par une discussion générale cherchant à aller au-delà des discussions spécifiques de chaque article.

Mon but est d'améliorer la compréhension des mécanismes qui soutiennent les dynamiques des systèmes hôte-parasitoïde. Cette compréhension est essentielle pour pouvoir utiliser efficacement les parasitoïdes dans la lutte contre les ravageurs des cultures et les espèces invasives. Grace à ces travaux, j'ai également pu mettre en évidence à quel point il est important de tenir compte des interactions inter-spécifiques lorsqu'on cherche à prédire les risques d'extinction liés aux facteurs génétiques. Or, ces interactions sont le plus souvent ignorées des modèles théoriques incluant des facteurs génétiques. Mon travail pourrait donc permettre d'améliorer la description et la compréhension des risques d'extinction dans les petites populations, un point

crucial pour la mise en place de programmes de conservation et de lutte contre les invasions biologiques.

Première partie

Introduction bibliographique

CHAPITRE

1 Genetic load in interacting populations

Article 1

AUTEURS : A. Bompard, T. Spataro

Résumé

Le fardeau génétique est décrit comme l'ensemble des facteurs génétiques ayant une valeur sélective sous-optimale dans une population. Il est composé de deux parties : le fardeau de mutation, qui décrit l'accumulation des mutations délétères dans une population, et le fardeau de ségrégation, qui décrit la perte de diversité génétique. Les conséquences délétères du fardeau génétique sur le taux de survie et de reproduction des espèces sexuées sont étudiées depuis plus d'un siècle. L'interaction entre fardeau génétique et facteurs démographiques peut induire un vortex d'extinction, qui est une des principales causes d'extinction dans les petites populations. Cependant, les études théoriques combinant démographie et génétique sont encore relativement récentes et, jusqu'à présent, pratiquement toutes ont supposé la démographie d'une population sans interaction inter-spécifique. Pourtant, certains travaux ont montré la part très importante de la démographie de la population, et notamment des processus densité -dépendants, dans les conséquences démographiques d'un fardeau génétique. Or, les interactions inter-spécifiques induisent le plus souvent des processus de densité dépendance complexes, et sont très largement présentes dans les populations naturelles.

Dans cette étude, nous allons tenter d'adresser la question des conséquences d'un fardeau génétique dans une population en interaction par une synthèse de la bibliographie en trois points : i) à partir de la littérature existante, nous décrivons le fardeau génétique et ses principaux impacts sur les traits affectant la démographie d'une population sans interaction inter-spécifique, ii) à partir des impacts du fardeau génétique que nous avons pu isoler (c.à.d. une réduction de la fitness individuelle, une diminution de la variabilité des phénotypes, et une relation de densité dépendance positive entre fitness et taille de population) nous recherchons dans la littérature existante quels modèles théoriques ont évalué les conséquences de traits démographiques similaires dans des populations en interaction, et iii) à partir des deux études précédentes, nous mettons en évidence les possibles impacts

des fardeaux génétique dans une population en interaction, ainsi que les hypothèses clés qui semblent déterminer ces impacts.
Le rôle de cette synthèse bibliographique est de rassembler des pistes et d'identifier les points clés qui permettront à de futures études théoriques d'aborder la question de l'impact d'un fardeau génétique dans une population en interaction. A partir des indices rassemblés dans la littérature, nous mettons en évidence que ces conséquences démographiques pourraient varier fortement selon le type d'interaction considérée ainsi que selon le comportement et la biologie des individus qui la composent. A priori, ces conséquences pourraient être très différentes de celles d'une population supposée sans interaction. Nous discutons ces conclusions en regard de la conservation des espèces en déclin et de la lutte contre les impacts délétères des invasions biologiques.

Abstract

The feedback between genetic load and demographic factors may induce an extinction vortex, which is a major cause of extinction in small populations. This feedback has attracted much attention these last years, but theoretical studies rarely considered the demography of a population within an interspecific interaction. Nevertheless, some studies have shown the crucial role of population demography, including density -dependent processes, in determining the consequences of a genetic load. Interspecific interactions often induce complex density dependent processes, that have rarely been studied in combination with genetic load.
In this study, we will attempt to address the consequences of genetic load in an interacting population by a literature review in three points: i) from the existing literature, we describe the genetic load and its major impacts on traits affecting the demography of a population without interspecific interaction, ii) from the impacts of genetic load that we could isolate (*i.e.* a reduction of individual fitness, a reduction of phenotypic variance, and a positive density dependence relationship between fitness and population size) we look for theoretical models which have assessed the impact of similar demographic traits in populations in interaction from the existing literature, and iii) from the two previous studies, we highlight the potential impacts of genetic load in an interacting population, as well as the key assumptions that seem to determine these impacts.
The purpose of this literature review is to gather leads and identify the key points that would allow for future theoretical studies to address the impact of a genetic load in interacting populations. From the evidence gathered in the literature, we can already highlight that these demographic impacts vary greatly depending on the type of interactions, and are very different from those of a supposedly non-interacting population. We discuss these leads in relation to the conservation of declining species and the fight against biological invasions.

KEYWORDS: Inbreeding depression; extinction risk; modeling; competition; mutualism; predation; parasitism; Allee effects; phenotypic variation.

Introduction

The deleterious impact of genetic load on individual fitness is a well-recognized phenomenon that has been explored for more than a century (Darwin, 1876; Frankham, 2005; Lande, 1995; Lynch et al., 1995; Frankel and Soulé, 1981). Conversely, its impact on population dynamics and extinction risk has been disputed during a few decades, particularly in regard to the priority of demographic processes on extinction: Lande (1988) suggested that declining populations would go extinct due to demographic factors before genetic factors could impact. Since then, the piling number of examples of natural populations that went extinct due to genetic factors cut short the issue of their impact (Frankham, 2005), but the interaction between genetic and demographic factors remains poorly explored (Robert, 2011; Ho and Agrawal, 2012).

The expression *genetic load* gathers two genetic factors that may compromise fitness: the accumulation of deleterious mutations (*mutational load*) and the loss of genetic diversity (*segregational load*). Both reduce the population mean fitness and its ability to respond to environmental change (Lande, 1995; Lynch et al., 1995; Frankel and Soulé, 1981; Frankham, 2005). In small populations, genetic diversity is lost by drift or inbreeding and deleterious mutations are more likely to be fixed, so genetic load may have a significant impact on fitness (Lande, 1995; Lynch et al., 1995; Frankel and Soulé, 1981; Frankham, 2005). Threatened species are generally present in small populations: as a consequence, the demographic impacts of genetic load have been mainly studied in the scope of biological conservation (Frankham, 1995, 2005). Conservation biology is a mostly applied science and its issues are often population or specie focused. Thus, many detailed models including genetic factors and a variety of specific parameters have been developed to predict the extinction risk of each focus species. However, generic theoretical model including genetic load and relaxing the assumption of a small, isolated population with simple demography have conversely been scarce (Robert, 2011; Ho and Agrawal, 2012; Higgins and Lynch, 2001). In fine, there is little knowledge about the possible interplay between genetic load and complex mechanisms of density dependence such as generated by interspecific interactions (Ho and Agrawal, 2012).

Nevertheless, this question is of great interest. Most species are engaged in interspecific interactions that shape the demography of their populations. Interspecific interactions may induce density-dependent processes within and between species, and the demographic consequences of genetic load are affected by density dependence (Haldane, 1957; Turner and Williamson, 1968; Mather, 1969). Moreover, interspecific interactions may naturally produce situations in which population abundance is low, either due to cyclic dynamics or low equilibrium value, favoring the expression of genetic load. Genetic load may have different demographic consequences in populations that are engaged in interspecific interactions and to the author's knowledge, extremely few theoretical studies have investigated this question (Ho and Agrawal, 2012; Agrawal and Whitlock, 2012).

Due to this lack of theoretical studies, we can only infer hypothesis based on the existing literature and point out possible issues. The point of this review is not to solve the question, but to define it and to indicate which factors could be keys. First, we will review the known life

trait modifications generated by genetic load, and their associated demographic impact, in the situations that have been studied. Then, we will jump to the domain of deterministic modeling of two interacting populations, and look for the consequences of similar life trait modifications and demographic processes – a reduced mean fitness and increased mortality, a reduced variation in individual phenotypes, and positive density dependence (*i.e.* Allee effects) – on the demography of these populations. From these studies, we will infer the possible demographic consequences of genetic load in interacting populations, and sum up the model assumptions that appear to critically affect these consequences (Table 1.1). We will discuss these inferences in the light of the demography of natural threatened populations and the dynamics of biological invasions.

1. Genetic load

1. 1 Genetic load and individual fitness

Genetic load is present at different scales in all known taxa. The apparition of deleterious mutations is caused by replication errors and may happen in all natural populations. However, their fixation depends on various ecological characteristics (mating systems, strength of the selection, etc.). The loss of genetic diversity may be induced by the random fixation of alleles in a finite population (*i.e.* by genetic drift), by a drop in population size (a bottleneck) or by inbreeding (Wright, 1931; Frankham, 2005). The phenotypic expression of deleterious mutations produces sub-optimal genotypes for all aspects of fitness: sperm production, mating ability, female fecundity, juvenile survival, life expectancy, etc. (Frankham, 2002; Hughes et al., 2008). The loss of genetic diversity may also reduce the average fitness, because the alleles maintained are not necessarily the optimal ones, or because the optimal genotype is heterozygous (Richman and Kohn, 1996). Moreover, the loss of genetic diversity reduces the ability of the population to adapt to environmental change (Simberloff, 1988; Soule, 1980; Lacy, 1997; Hedrick, 2001). Homogeneous populations are less able to cope with biotic and abiotic stress, more vulnerable to pathogens and herbivores (Frankham, 2005).

In large populations, genetic load is rarely expressed. Natural selection purges the deleterious mutations that are not recessive and maintains the others at a low frequency, reducing the risk of expressing deleterious mutations when reproduction is panmictic. Mutations and migrations counter the genetic drift, and without inbreeding the population genetic diversity is not likely to lessen. Many taxa have acquired reproductive mechanisms that ensure panmictic reproduction and/or reduce inbreeding: among them, kin-mating avoidance, dispersion or gathering in swarms before reproduction, negative assortative mating and allogamy (behavioral, or through self incompatible alleles) have been observed in a wide range of sexually reproducing species (Pusey and Wolf, 1996). Conversely, positive assortative mating – mate-choice on a closely related phenotype - may be common when mate choice is costly (Fawcett and Johnstone, 2003), and inbreeding may occur more often than expected by chance due to geographical segregation. Both phenomena tend to reduce the mutational load in large populations by exposing deleterious alleles more quickly, which are then purged by natural selection (Knapp and Rice,

1998; Crow and Kimura, 1970; Agrawal, 2001; Ho and Agrawal, 2012; Whitlock, 2002).

In small populations, alleles are lost at random by genetic drift, and inbreeding is more likely because there is less outbreeding options: both phenomena combine in decreasing the genetic diversity. Wright (1931) described the link between population size and genetic diversity in a closed random mating population with fixed abundance:

$$\frac{H_t}{H_0} = \left(1 - \frac{1}{(2N_e)}\right)^t = 1 - F \tag{1.1}$$

Where H_t is the heterozygoty level at generation t, H_0 the initial heterozygoty level and F the inbreeding coefficient. N_e describes the genetically effective population size, *i.e.* the size of an ideal population that would have the same genetic response to random processes as the real population of size N (Wright, 1931; Ellstrand and Elam, 1993). The effective population size is on average an order of magnitude lower than the number of breeding adults (Frankham, 1995). This equation predicts an exponential decay of genetic diversity with generations that occurs at a faster rate at small than in large populations.

A low genetic diversity produces an excess of homozygotes, which triggers the expression of the recessive deleterious mutations that have not been previously purged: the average reproductive ability decreases and mortality increases (Charlesworth, 1987; Gilpin and Soule, 1976; Lynch et al., 1995). Natural selection is less effective in small populations, because most alleles are fixed by genetic drift: mildly deleterious mutations increase in frequencies and further decrease the average fitness (Lande, 1995; Lynch et al., 1995). The loss of genetic diversity on self-incompatible alleles also leads to an increased production of unviable homozygous individuals (Frankham, 2005; Zayed and Packer, 2005) that drastically increases mortality.

The decrease of individual fitness due to genetic factors in small populations has been documented since more than a century, for experimental populations (Darwin, 1876), wildlife in captivity (Charlesworth, 1987; Ralls and Ballou, 1983; Laikre and Ryman, 1991) and for wild species in natural habitat (Reed et al., 2003; Crnokrak and Roff, 1999; Keller and Waller, 2002). Darwin (1876), first demonstrated the inbreeding depression by comparing seed production: self-fertilized plants had a seed production on average 41% lower than cross-fertilized plants. Literature agrees on the importance of genetic load impact on individual fitness in small populations; however, its importance on the population extinction risk has been more polemical (Lande, 1988; Robert, 2011).

1. 2 Demographic consequences in a simple finite population.

Genetic load reduces reproduction and survival, so it is expected to increase the population extinction risk when all other factors are controlled or excluded (Frankham, 2005). Indeed, laboratory populations of various species are more prone to extinction when inbred (Jiménez et al., 1994; Madsen et al., 1996), suffering a low genetic diversity (Newman and Pilson, 1997), and/or a high mutational load (Saccheri et al., 1998). The actual occurrence of extinctions due to genetic factors in natural populations is more controversial (Ralls and Ballou, 1983; Frankham, 2005;

Lande, 1988; Spielman et al., 2004).

Gilpin and Soule (1976) first suggested that genetic load could, in theory, drive small natural populations to extinction by inducing an extinction vortex. The genetically-based extinction vortices they described are initiated by an external reduction of population size or genetic diversity and generally follow the sequence: reduction of genetic diversity by drift – reduction of individual fitness – reduction of population growth rate – reduction of population size – reduction genetic diversity by drift, etc. (Gilpin and Soule, 1976; Goodman, 1987). Genetic load induces a threshold population size below which the population enters the extinction vortex; above this threshold, the population growth rate decreases with decreasing population size, following a non-linear curve affected by the strength of genetic load and inbreeding. When density dependence is ignored, the population growth rate follows a similar slope.

Many conservationists have taken interest into this threshold population size, named minimum viable population size, which may rise due to a variety of demographic and ecological factors (Higgins and Lynch, 2001). Population Viability Analysis (PVA) evaluates a population probability of extinction over a specified time, and often revolves around the evaluation of the minimum viable population size. This field has much developed since the work of Gilpin and Soule (1976) (Frankham, 2002; Shaffer, 1981; Boyce, 1992). PVA is classically conducted through simulation models integrating various deterministic and stochastic threats to population viability: among them, genetic load is present in 66% of analyses (Traill and Bradshaw, 2007).

The reduction of genetic diversity and the expression of deleterious mutations have been identified in the extinction process of various natural populations (Saccheri et al., 1998; Keller and Waller, 2002; Newman and Pilson, 1997; Crnokrak and Roff, 1999; O Grady et al., 2006). However, the controversy about our ability to predict population extinction from genetic factors alone has taken various arguments along the years: 1) that in general, demographic factors could be the primary causes, driving populations to extinction before genetic factors could impact (Lande, 1988; Saccheri et al., 1998; Caro and Laurenson, 1994; Caughley, 1994), 2) that intraspecific competition could loosen the link between reduced individual fitness and reduced population growth rate (Ho and Agrawal, 2012; Agrawal and Whitlock, 2012) and 3) that rough assumptions on population demography lead to the ignorance of synergetic or antagonist factors and to poor predictions of the population extinction risks (Chapman et al., 2001; Robert, 2011; Traill and Bradshaw, 2007). To include these arguments and procure a more comprehensive description of the impact of genetic factors, several models with more detailed and complex assumptions on population demography have been developed these last years.

1. 3 Eco-genetic interactions

Theoretical literature agrees that demography may interact with genetic processes, and that the result of this eco-genetic interaction may greatly affect population viability (Robert, 2011; Higgins and Lynch, 2001; Tanaka, 2000; Lande, 1988; Liao and Reed, 2009; Ho and Agrawal, 2012). Demographic factors that are known to interact with genetic processes can be classified

in two groups whether their effect is synergetic (environmental stochasticity, fragmentation) or antagonistic (density dependent processes).

1. 3. a Synergetic factors

Besides genetic load, Gilpin and Soule (1976) presented demographic factors that may also lead a population to enter an extinction vortex: demographic and/or environmental stochasticity, and habitat fragmentation. Threatened populations are often subjected to several factors that can drive them into an extinction vortex (Lande, 1998; Reed et al., 2003). The first models considering genetic load and more complex demography investigated the synergetic impact of environmental stochasticity or habitat fragmentation with genetic load.

Theoretical studies show that environmental and demographic stochasticity clearly increase the frequency of extinctions induced by inbreeding and/or mutational load (Tanaka, 2000; Higgins and Lynch, 2001). Less intuitive consequences may also arise: Tanaka (1997) demonstrated that in theory, demographic disturbances combined with mutational load were more likely to produce extinctions in intermediate to large populations than in small ones, because large populations maintain a higher mutational load while deleterious mutations were already partially purged in small populations (Tanaka, 1997). This points out the importance of population history when considering the possible impacts of genetic load: a slow and progressive reduction of population size caused by genetic factors does not end up in populations as susceptible to extinction as a brutal reduction of population size caused by environmental stochasticity.

The combination of spatial segregation with genetic load also attracted an intense attention because anthropogenic extinction risks often go through habitat loss and habitat fragmentation (Lande, 1998; Reed et al., 2003). Theoretical studies show that habitat fragmentation has a strong synergetic effect on extinctions due to genetic factors (Higgins and Lynch, 2001; Theodorou et al., 2009; Robert, 2011). Higgins and Lynch (2001) investigated the consequences of mutational load within a the metapopulation and concluded that metapopulation structure greatly accelerates the accumulation of deleterious mutations, to such an extent that metapopulation need to be very large and well connected to escape extinction by genetic factors. Moreover, low levels of fragmentation optimize the viability of populations when genetic factors are concerned, but higher levels of fragmentation may be required when demographic factors - such as carrying capacity, dispersal rate, fecundity – are concerned. Consequently, population viability is generally lower when both genetic and demographic factors are considered because the optimal fragmentation is intermediate(Robert, 2011). First evidence of natural population extinction due to genetic factors was indeed observed in a fragmented butterfly metapopulation (Saccheri et al., 1998).

1. 3. b Antagonistic factors

The argument that the release of intraspecific competition may compensate for the mortality generated by genetic load has been used by many authors to argue that genetic load is irrelevant to demography (Agrawal and Whitlock, 2012, see): selective deaths (due to genetic

load) would only replace non-selective (ecological) deaths. However, the mechanisms behind selective and non-selective death make their combination more complex: if individuals that suffer a selective death have consumed resources before dying, then selective deaths do not completely release intraspecific competition and genetic load may affect population size (Agrawal and Whitlock, 2012). Thus, when genetic load is alleged to reduce fitness, the proximal component reduced is of importance: if genetic load reduces resource consumption, said resources might be freed to benefit other individuals; if it reduces resources assimilation, the resources are lost and the release of intraspecific competition cannot compensate for genetic load (Agrawal and Whitlock, 2012; Abrams, 2003). A few models investigated the combination of density dependence and genetic factors. They generally concluded on reduced - but not neutralized - extinction risks compared to populations without density dependence (Robert, 2011; Ho and Agrawal, 2012; Agrawal and Whitlock, 2012).

A precise assertion of what is a population density-dependent process and what is an environmental disturbance can be crucial to predict accurately the extinction risk of a population (Chapman et al., 2001). The Vortex package developed by Lacy (1993) is a Population Viability Analysis model that accepts a form of density dependence, but it has not been used much in PVA studies. Chapman (2001) compared the accuracy of PVA models (including Vortex) with or without density dependence for the Soay sheep, a primitive domestic sheep known to undergo over-compensatory competition and wide population cycles. The projections offered by models without density dependence – that would interpret population cycles as environmental variation - were notably poor, yielding a probability of extinction of 60-80% over 50 years. Models including density dependence and correctly interpreting the cycles yielded a far more accurate and lower extinction risk (4.6% over 50 years). The mechanisms behind population demography, and particularly the density-dependent mechanisms, have demonstrated their importance when combined to genetic load for the prediction of population extinction risk (Agrawal and Whitlock, 2012; Chapman et al., 2001).

Chapman et al. (2001) 's cautionary tale about the importance of including the right origin for population demography also applies to the consideration of interspecific interactions. Interspecific interactions may generate strong density dependences (either negative or positive), and these mechanisms are not well captured by the description of single population demography. In this last chapter, we discovered that genetic load has three major impacts on non-interacting populations: the reduction of the average individual fitness, a decreased phenotype variance (through a decreased genetic diversity) and a positive, non-linear relationship between population size and individual fitness. These effects can be produced by various sources other than genetic load, and have sometime been studied within interacting populations. In the following chapter, we review the literature on the consequences these three features on the demography and persistence of populations engaged in interspecific interactions.

2. Interacting populations

Interspecific interactions can be sorted with regard to their benefit or cost for the species

involved: direct positive interactions (+,+) such as symbiosis, pollination, etc.; antagonist interactions (+,-), such as predation, parasitism, herbivory, etc.; and direct negative interactions (-,-), such as competition for resources or refuges, etc. Population size often affects the strength of the interaction, which lead to strong density dependent processes between species: plants population size is positively correlated to their pollinators' density, predators population size is positively correlated to their prey's density and prey population size is negatively correlated to their predators' density; and competitors abundance is negatively correlated with their competitor's density.

From these relationships, there is an intuitive answer to the question we are dealing with: as genetic load negatively affects the demography of the focus species, it should 1) negatively affect the demography of the other species that benefit from it (mutualistic or an upper-level antagonist) and 2) positively affect the demography of the other species that are negatively affected by it (competitor or lower level antagonist). Eventually, genetic load would induce a demographic feedback and would be particularly dramatic for species engaged in mutualistic or competitive interactions (negative feedback), but less in species engaged in antagonistic interactions (positive feedback) (Figure1.1). The reality of these interactions is more complex: over-exploitation of resources, stage-specific predation, indirect effects of interactions and temporal delay between phenomena may drastically affect the possible consequences of genetic load.

Natural populations are often engaged in more than one trophic interaction, leading to complex indirect effects and building community structure. In this paper, we consider simple, two-species direct interactions, as a first step to the consequences of genetic load within communities.

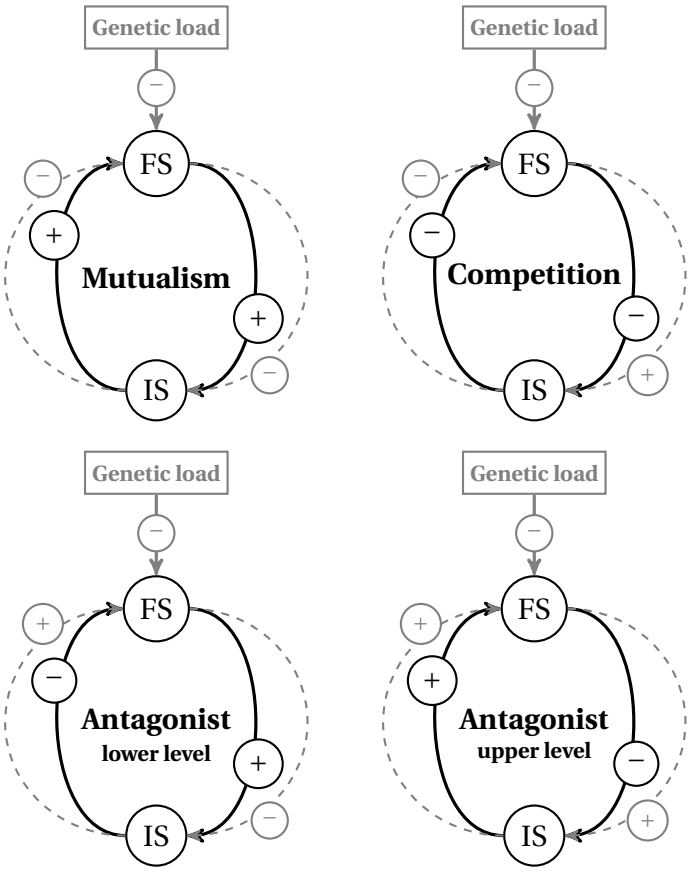

Figure 1.1: Classical types of two-species interactions (black lines and signs) and associated direct and indirect effects of genetic load (grey lines and signs). FS designates the focus species, which suffers the genetic load; IS designates the other (interacting) species.

2. 1 Reduction in fitness components and/or increase in mortality

The first observed consequence of genetic load is a reduction of all components of fitness due to inbreeding and to the expression of mildly to fully deleterious mutations. Various models considered the impact of the reduction of one or several fitness components on the demography of populations engaged in an interaction.

2. 1. a Competitive interactions

In a competition interaction, the demographic response to a variation in fitness components depends mostly if resource consumption is mechanistically or phenomenologically described (Abrams, 2003). With competitive Lotka-Volterra equation, reduction in the fitness components of one competitor species is always beneficial to the persistence and abundance of the other. In models of interference competition, variations in mortality or reproductive abilities of competitors lead to the same results (Jensen, 1987).

However, species engaged in exploitative competition may respond very differently to variation in their competitor's fitness depending on the variety and variability of resources involved and of their own resource requirement. Abrams (2003) studied a variation in species consumption rate in a mechanistic model with two resources and two competitors, each one having a major and a minor resource consumed with a saturating functional response. HE demonstrated that an increase in one competitor 's (species 1) attack rate on its major resource could have 3 different demographic responses, depending on resource requirement: i) (low resource requirement) species 1 declines while species 2 increases, because species 1 over-exploitation of species 2 major resource is relaxed; ii) (intermediate resource requirement) both species decline in abundances; iii) (high resource requirement) species 1 increases and species 2 decreases (Abrams, 2003). These results illustrate the variety of dynamical responses that may come from variation in one of the traits of a competitor species. Genetic load could traduce either as a decrease of one competitor attack rate or as an increase of its resource requirement - because reproductive abilities decrease, more resources are consumed to produce the same number of individuals. In this last assumption, genetic load may dramatically alter the dynamical response to competitor's variations in consumption preferences.

When one consumer already primes on the system because it is extremely efficient, an increase of its mortality may allow other competitors to coexist, because available resources become more abundant and more diverse (Abrams, 2001). In this frame, a reduction of the best competitors' s efficiency through genetic load could promote the species diversity in the ecosystem.

2. 1. b Antagonist interactions

Antagonist interactions encompass two trophic levels: a lower level, which suffers from the interaction (the prey or host) and a higher level, which benefits from the interaction (the predator or parasite). Both positions have different consequences in terms of demography, and both will be studied distinctly.

Within the lower level In phenomenological models of predator prey interaction, such as Lotka-Volterra 's or Rosenzweig and MacArthur (1963), a decrease in prey growth reduces the amplitude of predator prey cycles (Box 1). Prey mortality is thus considered as stabilizing for the system: Abrams (2003) interpreted this phenomenon as a form of Rosenzweig's paradox of enrichment, in which an increase in resource availability (here the prey) is destabilizing for the system.

Among the components of fitness, many are also associated with prey state, *i.e* its health and size. Prey state is known to directly affect the predation risk in a wide variety of systems, either because predators select low-state prey to minimize hunting costs (Christensen, 1996) or because they select high state prey to maximize energy income (Welton and Houston, 2001; Witter and Cuthill, 1993). A prey with a strong mutational load may have reduced quality and avoidance abilities, and is likely to be considered of low state. Subsequently, predation may act along or counter natural selection on deleterious mutations, whether predators select low state or high state prey. This indirect effect may impact the path of expression of deleterious mutations and their demographic consequences. Prey behavior can also vary with state and adapt to its predation risk, leading to adverse indirect effect of state on predation risk (Welton and Houston, 2001). Low state prey selection may yield another consequence: when the population becomes small and suffers inbreeding depression, prey state worsens, which may facilitate predation. These combined effects could produce a particular extinction vortex: as inbreeding depression decreases prey state, predation increases, prey population size decreases, which decrease genetic diversity and increases inbreeding depression so prey state decreases, etc.

Box 1. *Reduced components of fitness in prey*
(Rosenzweig-Mac Arthur's predator prey model)

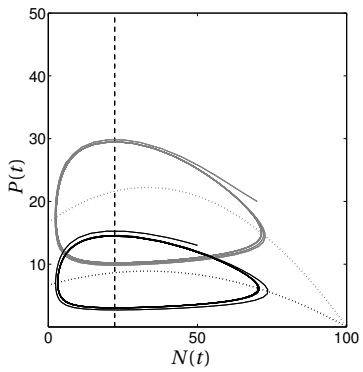

Figure 1. Isoclines (dashed and dotted lines) and trajectories (plain lines) for two parameters sets of the RMA: 1) a = 0.3, e = 0.15, K = 100, m = 0.6, h = 0.1, r = 5 (light grey) and 2) same values, r = 2 (black).

The Rosenzweig-Mac Arthur model (RMA) is a classical model of predator prey interaction where the prey (N) grows logistically when the predator is absent, and the predator (P) has a saturating functional response to prey density:

$$\begin{cases} \frac{dN}{dt} = rN(t)(1-\frac{N(t)}{K}) - \frac{aN(t)P(t)}{1+ahN(t)} \\ \frac{dP}{dt} = \frac{eaN(t)P(t)}{1+ahN(t)} - mP(t) \end{cases}$$

where r is the prey growth rate, K the prey carrying capacity, a the predator attack rate, h the predator handling time, e the predator conversation rate, and m the predator mortality rate. The model stability depends on the position of the predator isocline (fig. 1, dashed) before (cyclical) or after (stable) the maximum of the prey isocline (fig 1. dotted line)

A reduction in prey growth (r) decreases the prey isoclines and lead to cycles of smaller amplitude (see fig. 1).

Within the upper level Prey-predator models often exhibit wide population cycles due to overconsumption of the prey by its predator (Rosenzweig and MacArthur, 1963). In this situation, an increase in predator mortality rate is stabilizing for the system (see Box 2). When the predator exhibits a saturating functional response, an increase in predator mortality rate increases its abundance: this phenomenon is named the "hydra effect" (Abrams and Matsuda, 2005). For homogeneous prey, this effect usually appears when predator and prey populations are cyclic. The hydra effect becomes more common and may affect stable predator-prey systems if the prey is stage-structured and the predation stage-selective (Abrams and Quince, 2005) because stage selective predation creates temporal refuges that allow the prey to grow back. In this situation, a freed prey induces a particularly great increase in prey growth and population size, which in turn benefit the predator. Conversely, a strong density dependence in prey generally counters the hydra effect, because the freed prey succumbs to intraspecific competition and cannot benefit the predator (Abrams and Matsuda, 2005).

A decrease in the predator fitness may also be expressed as a decrease in prey conversion rate: the predator reproductive abilities are lessened, thus more prey are needed to produce an even number of predators. The impact of a decrease in prey conversion rate depends on its initial value, and on the topology of the predator prey system. When the predator is too efficient (high prey conversion rate, intense predation pressure that produce wide predator-prey cycles), a decrease in prey conversion rate reduces the cycle amplitude and may benefit the system. However, if predator prey system is stable, or if the predator has an already low prey conversion rate, a decrease of this parameter may drive the predator population to extinction (Box 2).

Box 2. *Reduced components of fitness in predator*
(Rosenzweig-Mac Arthur's predator prey model)

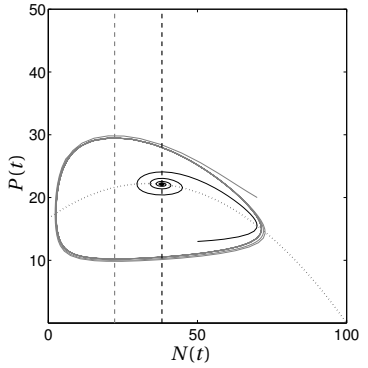

Figure 2. Isoclines and trajectories for two parameters sets of the RMA: 1) $a = 0.3$, $e = 0.15$, $K = 100$, $m = 0.6$, $h = 0.1$, $r = 5$ (light grey) and 2) same values, $m = 0.8$ (black).

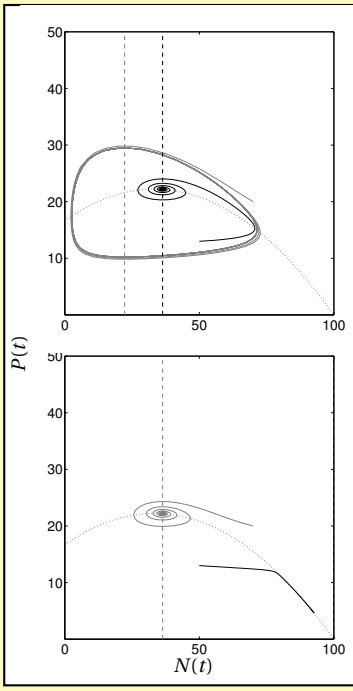

Figure 3. Isoclines and trajectories of the RMA for $a = 0.3$, $K = 100$, $m = 0.6$, $h = 0.1$, $r = 5$.
Fig 3a: $e = 0.15$ (light grey) and $e = 0.115$ (black)
Fig 3b: $e = 0.115$ (light grey) and $e = 0.8$ (black).

A increase of the predator mortality rate (m) stabilize the system (fig.2a)
A decrease of the predator prey conversion rate (e) stabilizes the system (fig 3a). Further increase excludes the predator population (fig 3b).

2. 2 Phenotypical variance

Small populations have a lower genetic diversity than large ones due to inbreeding and genetic drift. Consequently, phenotypic variance will generally be lower after a bottleneck (Frankham et al., 1999). Deterministic modeling of population interaction generally assumes a homogeneous population, but various authors investigated the consequences of including phenotypic variance on the predicted demography. These consequences are of two types: direct (the demographic consequences of considering stochastic model parameters), and indirect (through behavior, specialization and adaptivity) (Bolnick et al., 2011).

2. 2. a Mutualism

Mutualistic systems favor local adaption at a small scale. Genetic variance within a patch is low, as both species coevolve to gain a maximum benefit from their mutualistic species, but variability between patches is important, as mutualistic species tend to remain within their optimal patch (Packer et al., 2005; Müller et al., 2014). Thus, a reduction in genetic variability in a focus mutualistic species is likely to decrease the genetic variability of the other, as a result of natural selection to gain a maximum benefit from the focus species (Wernegreen and Wheeler, 2009). A reduction in genetic variability in a species engaged in mutualistic interactions is thus likely to decrease the genetic variability of the whole system, reducing its ability to cope with environmental change.

2. 2. b Competition

Trait variation is always beneficial to competitors, both directly and indirectly. Variability is a competitive advantage, because the maximum value of a trait can be more important than is mean value when only the best individuals are selected under sever interspecific competition (Lichstein et al., 2007).

In competitive systems, fitness and coexistence depend on the degree of dissimilarity between species: the more different the competitors are, the more likely they are to coexist and the better their fitness will be, because resources are more efficiently used (Macarthur and Levins, 1967; Vellend, 2006). In a changing environment, this force rests upon the ability of species to adapt to environmental change and to respond to the evolution of its competitor. A species with a reduced genetic diversity will be less able to adapt and will remain more similar to its competitor, reducing both competitor fitnesses and ability to coexist (Vellend, 2006; Wittmann et al., 2013). Coexistence with a competitor species tends to reduce the genetic diversity of a species because it reduces its population size and niche availability (Wittmann et al., 2013; Vellend, 2006). This ecological feedback may intensify the impact of genetic diversity on species involved in competitive interactions: a reduction in genetic diversity reduces niche availability, further reducing genetic diversity, until the competitive exclusion of the focus species (Wittmann et al., 2013).

2. 2. c Antagonist interactions

Many authors have investigated the demographic consequences of phenotypic variance and adaptivity in predator-prey and host-parasitoid systems (Okuyama, 2007; Saloniemi, 1993; Doebeli, 1997; Imura et al., 2003; Hartt and Haefner, 1998; Mougi and Kishida, 2009). Both in prey/host and in predator/parasitoid, phenotypic variance promotes stability and coexistence (Okuyama, 2007; Saloniemi, 1993; Doebeli, 1997; Imura et al., 2003). When the predator has a functional response that depend on prey phenotype frequency, phenotypic variance in prey may increase the prey mean realized fitness by decreasing the predatory pressure (Hartt and Haefner, 1998).

Plasticity and adaptivity in the traits associated with hunting (in predators) and avoidance (in prey) have also been considered as stabilizing factors of predator-prey systems (Doebeli, 1997; Mougi and Kishida, 2009). However, for populations that exhibit complex dynamics, such as cycles, and when switching between trait values is relatively slow (*i.e.* not behavioral, but adaptive) plasticity can be destabilizing. Abrams and Kawecki (1999) demonstrated that under these conditions, adaptivity in a parasitoid trait associated with host preference could be strongly destabilizing because parasitoids tend to over exploit a host type which is already at low frequencies.

Thus, reduction of phenotypic variance has direct consequences that are always deleterious in predator-prey systems, whether this reduction happens in the prey or in the predator (narrowed conditions for coexistence, decreased prey fitness, destabilized dynamics). The indirect consequence (*i.e.* loss in adaptivity) of this reduction when in the predator, may occasionally stabilizes the predator prey dynamics. However, in the constantly evolving phenotypic landscape - the prey adapts to avoid predation, the predator adapts to catch more prey – the loss of adaptivity is bound to ultimately drive a species to extinction (Van Valen, 1973).

2. 3 Allee effect

The Allee effect is described as a decrease of individual fitness when population size is low that often originates from the loss of direct or indirect positive interactions (sexual reproduction, group hunt, symbiosis, etc.). These phenomena may translate into demographic Allee effects, *i.e.* a reduction of the population growth rate when population size or density decrease. When strong, demographic Allee effects may drive a population to extinction if its abundance falls under a threshold (Courchamp et al., 2008). A density threshold and positive correlation between growth rate and size are close to the demographic consequences of a genetic load: for that matter, genetic load is often considered as a particular form of Allee effect, called genetic Allee effect (Vayssade et al., 2014). Conversely to an Allee effect that would originate from sexual reproduction or other cooperative behavior, the fitness once lost due to genetic Allee effects cannot easily be retrieved when the population grows back in size, because the genetic diversity remains low (Vayssade et al., 2014). While genetic Allee effects have not been much studied in the frame of interacting populations, other forms of Allee effects have, particularly these last decades. We will focus on Allee effects considered within one or several participants of an interspecific interaction, but not originating from the interspecific interaction.

2. 3. a Mutualism

Allee effects within mutualistic interactions induce strong extinction risk for both populations, which is a quite intuitive result. Mutualistic interactions are interspecific cooperative behaviors that create Allee effect of their own; thus, adding a supplementary Allee effect is always deleterious. Combined Allee effects can have additive or non-additive results on population extinction risk (Courchamp et al., 2008). Additive results suggest that both Allee effects do not interfere with each other, *i.e.* mutualism does not affect the rate at which a supplementary Allee effect reduces individual fitness. Conversely, non-additive results may come from a variation in the strength of the mutualistic interaction due to Allee effects: for instance, as plant population size declines, rates of pollination also decline (Knapp and Rice, 1998; Gates and Nason, 2012). Considering genetic load as a secondary Allee effect, it is difficult to predict whether the combination of mutualism and genetic load would produce additive result (*i.e.* mutualism and genetic load are independent) or non-additive result (*i.e.* mutualism and genetic load have synergetic effects).

Mutualistic interactions are *a priori* very susceptible to the deleterious impact of Allee effects on persistence. However, various indirect effects of mutualism may help to overcome these impacts: plant-pollinator interactions induce reproductive facilitation between plants through reproductive synchrony, which help plants to overcome the mate-finding Allee effect (Chen and Hsu, 2011). One might assume that the indirect effects of mutualism could also protect the organisms from genetic Allee effects through the facilitation of mechanisms that reduce the mutational load or maintain genetic diversity.

2. 3. b Competition

Within competitive interactions, Allee effects can have major impacts on the coexistence of populations and on the issue of the interaction (Courchamp et al., 2008). The competitor that suffers an Allee effect takes longer to reach equilibrium and has a lower equilibrium value; it goes extinct on a larger range of parameter combinations, and from a larger range of populations density; it may be excluded due to Allee effects even if it was the superior competitor without Allee effects (Munther, 2013; Courchamp et al., 2008).

More generally, Allee effects reduce the likelihood of competitive systems, because it reduces the range of parameters that allows the two competitors to coexist (Wang et al., 1999). When present in both competitors, Allee effect makes the invasion of one competitor impossible if its density is under a critical value (Ferdy and Molofsky, 2002; Courchamp et al., 2008). This process has been suggested as one of the main causes of invasion and introduction failures (Hopper, 1993; Simberloff, 2008; Fauvergue et al., 2012). A strong over-compensating intra-specific competition may reduce the impact of Allee effects and allow species coexistence (Kang, 2013).

2. 3. c Antagonist interactions

Within the lower level Many authors have investigated the demographic consequences of Allee effects in prey populations, and these consequences depend mainly on the temporal structure of the theoretical model they used. Continuous time models without delay conclude on an extremely destabilizing impact of Allee effects on the prey-predator system. Allee effects endanger the predator-prey system by increasing the range of parameter combinations for which prey and predator go extinct (Gao and Li, 2013; González-Olivares et al., 2011; Zu et al., 2010; Boukal et al., 2007), by transforming stable equilibrium into cycle and chaos (Wang et al., 2013; Morozov and Li, 2007), and by decreasing equilibrium values, which increases the system susceptibility to collapse due to environmental variations (Courchamp et al., 2008). Even if weak, Allee effects in prey increase the time needed to reach equilibrium and transform stable equilibria into unstable equilibria (Zhou et al., 2005; Merdan and Gümüs, 2012; Boukal et al., 2007). Kent et al. (2003) suggested that such extremely deleterious effects appear because Allee effects exaggerate the impact of the predator on its prey.

Introducing delay between predator uptake and the consequences of Allee effects on the prey neutralizes this phenomenon in continuous time models (Ferreira et al., 2013). With a continuous time model allowing for temporal delay, Allee effects can go from destabilizing phenomenon (no delay) to a stabilizing phenomenon (large delay) (Merdan and Gümüs, 2012). Authors whom investigated the consequences of Allee effects within discrete-time model - a structure that include an intrinsic time delay - all concluded on partially to fully stabilizing consequences of the Allee effect (Chen et al., 2013; Hadjiavgousti and Ichtiaroglou, 2008; Pal et al., 2011; Çelik and Duman, 2009). We suggest that the difference between both types of models may arise because time delay induces a temporal refuge for the prey, which prevent Allee effects from exaggerating the impact of the predator. Once this effect is canceled, the Allee effects may stabilize the system by decreasing prey growth, a phenomenon close to the "paradox of enrichment" (as presented in part 2.1.b).
The demographic consequences of Allee effects have also been investigated within continuous-time host-pathogen models. An Allee effect in the host within a host-pathogen interaction appears to be destabilizing, but beneficial to the host at first: the Allee effect reduces both the range of host densities for which an initially rare infection can spread and the disease prevalence when it spreads. However, once the infection established, Allee effects decrease the overall host density and widen the range of parasite species that can lead the host to extinction, increasing disease damage (Deredec and Courchamp, 2006; Courchamp et al., 2008).

In the upper level The demographic impact of an Allee effect on the predator (or parasitoid) has been much less explored than on the prey (or host). Two main factors appear to impact its consequences: the predator (or parasitoid) efficiency and dynamics, and the component of fitness that is affected by the Allee effect.

In prey-predator and host-parasitoid systems, the consequences of an Allee effect depend on predator efficiency and prey-predator dynamics. If the predator - or the parasitoid - has an attack rate so high that it may prompts cyclic dynamics, Allee effects are stabilizing and may

increase prey and predator densities (Bompard et al., 2013; Verdy, 2010; Zhou et al., 2005). However, when its attack rate is already low, Allee effects may cause predator (or parasitoid) extinction (Bompard et al., 2013).

The character affected by the Allee effect may be of crucial importance in the case of predator-prey interactions. Verdy (2010) investigated the consequences of Allee effects in predator-prey dynamics, considering that the Allee effect could affect either predation rate (as if the Allee effect originated from group hunting) or prey assimilation (as for a mate-finding Allee effect, or any other Allee effect exterior to predatory capacities). While an Allee effect affecting the predation rate could increase prey and predator equilibrium densities, an Allee effect affecting prey assimilation was always deleterious, decreasing predator abundance with an increased sensibility to the Allee effect parameter. When Allee effects impact prey assimilation, prey mass is lost and the mass budget unclosed, which reduces predator abundances. Conversely, when Allee effects impact the predation rate, there is no waste of prey and the mass budget remains closed. These results are particularly interesting because the expression of deleterious mutations is likely to affect reproductive abilities: in predatory species, this would eventually affect prey assimilation, driving the predator to extinction; in parasitoid species, this may rather impact parasitoid females' attack rate, which may stabilize the system.

3. What could be genetic load impact in interacting populations?

At the end of these investigations, it appears that genetic load may combine with indirect interaction in more ways than we expected, producing both direct and indirect impacts on species dynamics. We gathered the inferred impacts of genetic load in each interspecific competition in Table 1.1. Mutualistic species appear to be the most endangered by genetic load, but mutualism may facilitate biological or reproductive systems that would maintain genetic diversity (such as dispersal or panmictic mating) ahead of its expression. For competitive, prey or predatory species, the consequences of genetic load appear much more complex, and seem to relay on a variety of biological and ecological traits. In the following section, we focus on several critical consequences of genetic load that emerge from our investigations and confront them with literature cases of natural prey, predator, and competitive species.

	Mutualist	Competitive	Antagonist
Reduc. of phenotypic var. Reduction of fitness comp.	Increase of FS and IS extinction risk.	**Interference**⇒ Increased IS abundance, decreased FS abundance/persistence **Exploitation**⇒ Result depends on resource exclusivity & resource requirement If FS is a superior competitor, promotes IS persistence.	*In prey*: **Phenomenologic** description: promote stability in predator-prey system **Mechanistic** description: if the predator preference for low state prey= increased extinction risk *In predator*: **high attack rate**= promote stability, increase abundance of FS and IS; **low attack rate** = increases extinction risk, decreases abundance of FS
	Positive feedback: reduced genetic diversity in FS ⇒ reduced genetic diversity in IS.	Reduces FS competitive abilities (increases IS/ reduces FS abundance and persistence) Reduces the set of conditions for coexistence	*In prey*: Increases predation risk, decreases FS fitness, increases extinction risk *In predator*: If adaptive, freq. dependent predation: stabilizing. Else, reduces predator efficiency
Allee effect	Increased extinction risk of FS and IS	Increased extinction risk (up to exclusion) and reduced abundance of FS Reduced conditions for coexistence of FS and IS	*In prey*: **No refuges (temporal/spatial)** = increases predator impact on prey and risk of system collapse, strongly destabilizing; **refuges** = possibly stabilizing. *In predator*: Depend on predator efficiency (see Fitness components) **affects prey assimilation** = increases extinction risk; **affects prey capture rate** = possibly stabilizing
Genetic load	Fully deleterious impact for FS and IS survival.	Deleterious impact on the species coexistence in competitive systems Increases extinction risk and decreases abundance. See part 3.2	Controversial impact on extinction risk for prey and predator, depending multiple biological traits. See parts 3.1 and 3.3

Table 1.1: Table of the impact of the gathered impact of demographic features associated with genetic load (*i.e* a reduction of fitness components, reduction of phenotypic variance, and a component Allee effect) for species in various types of interspecific interactions. FS designates the focus species (in which the genetic load is assumed) and IS its interacting species.

3. 1 The two fold aspects of genetic load in prey: increasing predation risk or stabilizing ?

Along this study, we encountered completely adverse clues on the possible impact of genetic load in prey populations. On the one hand, genetic load seems dramatically deleterious for the prey: phenotype variance in prey is stabilizing and decrease predation risk, so its loss may have the adverse effects; the Allee effect may exaggerate the predator impact on its prey, and predator choice of high state preys may select deleterious mutations and accelerate the path of an extinction vortex (see Table 1.1). On the other hand, decreased prey growth and Allee effects can be stabilizing for the predator-prey system, particularly when the prey has temporal and/or spatial refuges; and predator choice of low state prey may act along with natural selection to exclude deleterious mutations. So, which aspect of genetic load will most likely affect the prey?

One possible answer is both, but at different scales. In large, healthy prey populations, when refuges are abundant and phenotypes are various, a low level of genetic load is likely to be a positive force for the prey population. Genetic load would translate on to prey demography after a bottleneck – *e.g.* a cycle – and may act as a failsafe, reducing population growth rate and stabilizing the system when prey enrichment endangers persistence. Conversely, a small, endangered prey population is likely to be driven to extinction at a dramatic pace compared to a non interacting population due to the synergetic effects of predation and genetic load. Small populations may loose their temporal and spatial refuges because their habitat shrinks and their generations become more synchronized, which reduce their chances to escape predation: in this scope, Allee effects would exaggerate the impact of the predator on its prey. The predator response can be frequency dependent, so predatory risk increases as the prey variability decrease. Genetic load will act along with predation in reducing the prey population, driving the prey into a fast extinction vortex.

In this catastrophic scenario, a factor remains that may tip the scales in favor of prey persistence or extinction: predator choice. When the predator tends to select low state prey, mildly deleterious mutations are purged both by natural selection and by predation, so the prey mutational load is likely to be lower. As the predator selection persists in small populations, the extinction vortex is likely to be slower. Conversely, when predators select high state prey, mildly deleterious mutation may be selected and the extinction vortex is likely to be faster.

As genetic and demographic factors act in a synergetic way, they may be hard to differentiate. Will the prey go extinct due to genetic load or due to over-predation? This question comes back to Lande (1988) questioning on the priority of genetic and demographic factors. Many characteristics of the interaction are likely to be involved in the answer: the frequency dependence (or not) of predation, the availability of other sources of food for the predator - which may lead to apparent competition between prey, and drive the focus prey to extinction -, the state or stage dependence of predation, etc.

The woodland caribou (*Rangifer tarandus*) is an endangered species rapidly declining in north America, prey to various predators (mainly wolves (*Canis lupus*) and cougars (*Puma concolor*) (Wittmer et al., 2005). The caribou avoids its natural predators mainly by geographical

separation (Wittmer et al., 2005). Recently, the predator populations became more numerous and more present on caribou territories, because changes in forest structure have favored their secondary prey (the moose, *Alces alces*) (Rempel et al., 1997). Elevated mortality rates, and a large part of caribou decline can be attributed to predation, but a demographic Allee effect not imputable to predation was also identified in small sub-populations (Wittmer et al., 2005). Besides, anterior studies have showed that genetic diversity within wild sub-populations was extremely low, which suggest high levels of inbreeding (van Staaden et al., 1995). In this situation, genetic load and predation could interact at least in three ways:

1. the loss of genetic diversity prevents prey adaptivity to new predatory conditions;

2. predation can act as a barrier to gene flow between sub populations (because the caribou avoids its predators by geographical segregation), reducing the genetic diversity within patches;

3. spatial and temporal refuges are scarce, because the predators increased their geographical range and caribou sub populations are small: the demographic Allee effect observed in sub populations, which can be induced by genetic load, may exaggerate the impact of predation on the caribou.

The picture is quite dark for the woodland caribou, as well as for many other endangered species that suffer predation. Twenty years old studies on the caribou genetic diversity advised to promote connectivity between populations (van Staaden et al., 1995), while more recent studies focused on the avoidance of the predatory risk (Wittmer et al., 2005). Formal theoretical and empirical studies on the feedback between genetic load and ecological interactions would help to understand when conservation actions to promote genetic diversity also apply to predation avoidance (and vice versa), and which potential conservation program is the most beneficial for species persistence.

3. 2 Why so many biological invasions are successful, although genetic load seems catastrophic in competing species?

Biological invasion are likely to procure cases to study the combination of genetic load and interspecific interactions. While invasions may be facilitated by the absence of co-evolved predators or parasites (the *enemy release hypothesis* (Keane and Crawley, 2002)), invasive species are not freed from interspecific interaction in their new environment. The invasive species have to consume resources, so they will certainly find competitors among the native species. As the invader arrives, its abundance and genetic diversity are low, so our work suggests that because of genetic load, it should loose most of its competitive advantages, possibly become unable to coexist with a stronger competitor or fall below a density threshold, and go extinct. While this scenario may sometime apply, there is also numerous cases of successful biological invasions in natural populations, often driving native populations to extinction.

Wittmann et al. (2013) investigated the impact of the arrival of an invasive species on the genetic diversity of a native species competitor: this is one of the few demo-genetic model to date to address the question of the consequences of genetic load in interacting species. As the invader settles in its new environment, it reduces the native species population size - through competition - and thus its genetic variability (Wittmann et al., 2013). A few empirical studies have investigated such direct consequences of invasion on genetic load in natural populations (Kim et al., 2003; Krueger and May, 1991). The reduction of the native species genetic diversity raises its extinction rate by inducing the expression of its genetic load, thus reducing its competitive abilities and growth rate (direct effect) and its adaptivity to environmental change (indirect effect). The intensity of the competition between invasive and native species is a major determinant of invasive success and extinction risk of the native species: at low intensity, the invader will settle easily but will have little ecological (and thus genetic) effect on the native species, while at high intensity the invader effects will be strong once settled, but it will not likely settle due to competitive exclusion (Wittmann et al., 2013). Once the invader is settled, the native species may enter an eco-genetic extinction vortex whose speed is controlled by the species genetic diversity. The variations in this genetic diversity (because of habitat fragmentation and population genetic structure) lead to local disparities in native species extinction.

We suggest that the invader genetic diversity may also play a significant role on native species exclusion. After the strong bottleneck of biological invasion, the invader genetic diversity - and thus its adaptivity - is extremely low. In competitive interactions, adaptivity does not only benefit the species that possesses it but also its competitor: both species adapt toward more distinct ecological niches and lower intensities of competition. The invader both induces a reduction of the native species adaptivity (through the reduction of the native species genetic diversity) and arrives with an extremely low adaptivity itself, preventing a drift toward less intense competition. Counter-intuitively, the invasive species once settled could be more dangerous to its competitors when its genetic diversity is low than when it is high. Further investigations on the eco-genetic feedback between competing species could help to understand the dynamics and species extinction risks associated with biological invasions.

3. 3 Predator and parasitoid: a different tale

Our investigations also pointed out that predators and parasites could not be uniformly sorted as the “upper level of the antagonist interaction”, as they could respond very differently to genetic load. At the core of this difference is the tightness of the link between prey (or host) consumption and predator (or parasitoid) fitness.

In a predator, the link between consumption and fitness is relatively loose. Models predict that prey consumption directly translates into a number of predators, but for natural species prey consumption is only an energy income and predator reproduction depends on various other factors (such as its mating ability, individual fitness, age etc.). Genetic load may reduce predator fitness but not prey consumption: sterile predators would consume preys without producing offspring. Genetic load in predators would waste energy: the prey consumed can be lost for the predator, a situation that leads to destabilizing effects and decreased predator abun-

dance.

Conversely, parasitism is conservative of energy because the link between prey consumption and fitness is tighter. The parasitoid lays eggs and develops in the prey, rather than using it as energy: the prey is actually "replaced" by one or several offspring. Sterile parasitoid females do not consume a host, because they have no eggs to lay. Thus, genetic load tends to reduce parasitoid attack rate, which may benefit the parasitoid population if parasitism is already intense. Of course, energy can still be lost as some parasites also feed on their host, and genetic load may produce developmental defects and unviable eggs. However, these phenomena are likely to be of minor magnitude compared to the amount of energy lost by predators affected by deleterious mutations.

Predator and parasitoid cannot be considered as one when facing a genetic load, because their interactions include specificities that are likely to produce drastically different demographic consequences. To illustrate this hypothesis, let's take a general look on the rate of success of introduction in classical biological control. The rate of establishment of predatory Coccelinids is notably lower (10%) than that of parasitoids (about 34%) (Obrycki and Kring, 1998; Hopper, 1993). Most parasitoids are Hymenopteras, an order that possesses a low level of mutational load due to its haplodiploid sex determination system (Heimpel and de Boer, 2008), but coccelinids also have a lower rate of establishment than Tachninids (23.8%), the diploid fly parasitoid group most used in biological control (Hopper, 1993).

Conclusion and perspectives

In this review, we pointed out that genetic load could have an important role on species dynamics, a role that depends on the species ecological interaction. Whether in small, declining populations or in large, healthy populations, genetic load and interspecific interactions may combine.

In large interacting populations, genetic load can be beneficial: it may damp population cycles and acts as a failsafe against prey over-growth or predator over consumption, equilibrates competitive interactions and promotes competitors diversity. Interspecific interactions may also affect the strength of genetic load, particularly in predator-prey interactions: predator preference may select or exclude deleterious phenotypes, and frequency-dependent predation may maintain genetic diversity in the prey.

In small populations, interspecific interactions may dramatically affect the risk of entering an extinction vortex. First, because interspecific interactions can exaggerate the impact of genetic load on the species dynamics (in competitors, prey, mutualistic species), and genetic load may exaggerate the impact of interspecific interactions (particularly of predation). Second, because the dynamic of the interaction creates new ways to enter an extinction vortex, through the reduction of specific parameters such as a parasitoid attack rate, or a predator conversion rate. Finally, species extinction risk depends on the level of genetic load and thus on species

past history, which has been affected by interspecific interactions.

Both in large and in small populations, the impact of genetic load is determined by a complex eco-genetic feedback loop which strength and direction depend on the characteristics of the ecological interactions. In a world where each species has its network of interactions within a community, genetic load might have large, ecosystem-wide consequences. The field of community genetics has largely developed these last years, bringing back together small scale and large scale, and testifying how one species genetics may affect the whole structure of a community (Whitham et al., 2003, 2008; Ho and Agrawal, 2012; Wimp et al., 2005). The same attention could be given to that apparently sub-optimal part of the genetic structure that genetic load is to species: what is its role in the community?

To conclude, we must remind that all of this work is based on indirect, non-exhaustive clues from the combination of literature in various disciplines. A lot of work remains to be done in both theoretical and empirical studies to reach even a partial understanding of the place of genetic load in population dynamics in the context of community ecology. Nevertheless, such work would certainly help to understand the dynamics, extinction risks and chances of establishment of natural species.

Parasitoïdes et mécanismes à faible densité

1. Les parasitoïdes et la lutte biologique.

La lutte biologique consiste à limiter la prolifération des organismes ravageurs en employant leurs ennemis naturels (van Driesche et al., 2008). Elle passe soit par l'introduction d'ennemis naturels non-indigènes (c'est la lutte biologique classique), soit par la facilitation d'un ennemiindigène (c'est la lutte biologique conservatrice), soit par des lâchers réguliers d'un grand nombre d'ennemis naturels, une méthode souvent utilisée en serre (c'est la lutte biologiques inondative). La lutte biologique classique est utilisée depuis plus d'une centaine d'années, dans un grand nombre de pays à travers le monde (Clausen, 1936; van Driesche et al., 2008). L'augmentation de la fréquence des invasions biologiques ces dernières années fait de la lutte biologique classique un enjeu majeur non seulement pour protéger les cultures mais aussi les écosystèmes naturels (van Driesche et al., 2008).

Les parasitoïdes sont très souvent utilisés en lutte biologique classique contre les insectes ravageurs des cultures (van Driesche et al., 2008). En effet, ils sont généralement très efficaces contre leur population hôte (jusqu'à plus de 95% de réduction de l'abondance des hôtes) et plus spécifiques que la plupart des prédateurs, ce qui permet de limiter l'impact de l'introduction sur des espèces non-cibles (Bellows, 2001).

Cependant, environ deux tiers des introductions de parasitoïdes en lutte biologique se concluent par un échec d'acclimatation (Hopper, 1993). Comprendre les conditions qui permettent de maximiser les chances d'installation des parasitoïdes est donc un challenge majeur pour l'agronomie et l'écologie. Les fréquences d'établissement des parasitoïdes sont positivement corrélées avec le nombre d'individus introduits et le nombre d'individus initialement collectés (Hopper, 1993), ce qui suggère que lorsque les parasitoïdes échouent à s'installer des phénomènes tels que l'effet Allee ou le fardeau génétique sont au moins partiellement en cause (Hopper, 1993; Shea and Possingham, 2000; Fauvergue and Hopper, 2009).

Comprendre les conséquences démographiques des mécanismes s'exprimant à faible densité est donc particulièrement crucial lorsqu'il s'agit de populations de parasitoïdes. Or, la relation hôte-parasitoïde est une interaction antagoniste dont on ne peut ignorer l'impact démographique majeur, puisqu'elle affecte l'abondance, la stabilité et le risque d'extinction de l'hôte et du parasitoïde (Hassell, 2000).

2. Mécanismes de détermination du sexe

Paradoxalement, très peu de modèles théoriques se sont intéressés à l'impact démographique de l'effet Allee et du fardeau génétique dans un système hôte-parasitoïde (Hopper, 1993; Deredec and Courchamp, 2006). A ma connaissance, aucun ne s'était jusque là explicitement intéressé à l'impact de l'effet Allee ou du fardeau génétique chez un parasitoïde sur la démographie du système en tenant compte de l'interaction hôte-parasitoïde.

Cette absence d'étude est principalement due au principal système de reproduction rencontré chez les parasitoïdes, l'haplodiploïdie. En effet, la majeure partie des parasitoïdes (75%) fait partie des hyménoptères, un ordre intégralement constitué d'espèces haplodiploïdes. Les mâles sont haploïdes, généralement produits à partir de gamètes non fécondés, et les femelles sont diploïdes, produites à partir de gamètes fécondés. Ce système de reproduction est appelé la parthénogénèse arrhénotoque. Contrairement aux femelles diploïdes, les femelles arrhénotoques n'ont donc besoin de s'accoupler que pour produire des femelles (Heimpel and de Boer, 2008). Pour des raisons que nous allons exposer dans les prochaines sections, l'haplodiploïdie limite le risque d'apparition d'un effet Allee comportemental ou d'un fardeau génétique, et est donc à l'origine de l'idée répandue selon laquelle les parasitoïdes seraient relativement immuns aux effets Allee ().

Il existe également chez les hyménoptères des populations à parthénogenèse thélytoque, dans lesquelles les femelles peuvent être produites par parthénogénèse (soit par doublement du génome post-méiose, soit par clonage) (Heimpel and de Boer, 2008). La parthénogénèse thélytoque peut être génétique ou induite par un symbiote bactérien, et peut donc être rencontrée au sein d'espèces habituellement arrhénotoques. En pratique, ces populations ne sont composées que de femelles et ont abandonné la reproduction sexuée. Elles représentent une proportion mineure des hyménoptères (Heimpel and de Boer, 2008). C'est donc la parthénogénèse arrhénotoque, la plus commune, qui nous intéresse dans la suite de ce manuscrit et à laquelle nous ferons référence en parlant d'haplodiploïdie.

2. 1 Effet Allee et taux de fertilisation chez les hyménoptères

Chez les haplodiploïdes, des difficultés à trouver un partenaire sexuel peuvent subvenir à faible densité comme chez n'importe quelle autre espèce à reproduction sexuée, mais les femelles vierges restent capables de produire leurs descendants mâles. Chez une femelle vierge, la part de descendance qui aurait été fertilisée si la femelle s'était accouplée est soit complètement soit incomplètement produite en mâles haploïdes, selon l'existence et l'importance d'un

signal d'oviposition associé à l'accouplement (Fauvergue et al., 2008). Les femelles vierges augmentent la proportion et la densité de mâles dans la population, ce qui réduit les difficultés rencontrées par les femelles pour trouver un partenaire. L'haplodiploïdie est donc considérée comme un avantage majeur en cas de difficultés à trouver un partenaire. Or, lorsque ces difficultés apparaissent parce que les mâles deviennent rares, elles peuvent induire un effet Allee comportemental, *i.e.* une corrélation positive entre la fitness individuelle et la densité de population liée à la perte d'interactions de coopération (ici pour la reproduction sexuée) à faible densité. En théorie, l'haplodiploïdie limite donc le risque d'apparition et l'impact démographique d'un effet Allee comportemental. L'effet Allee comportemental est considéré comme l'effet Allee élémentaire le plus courant en raison de la large occurrence de la reproduction sexuée chez les plantes et les animaux, et des multiples indices de son apparition dans les populations naturelles (Dennis, 1989; Kramer et al., 2009). Prévenir l'effet Allee comportemental, c'est donc prévenir une grande partie des extinctions liées aux effets Allee.

La production de mâles par les femelles vierges n'est pas le seul avantage que l'haplodiploïdie procure aux hyménoptères face aux difficultés d'accouplement. Lorsque ces difficultés sont constantes, le sex ratio de la population à l'équilibre est biaisé en faveur des mâles et la production de femelle a donc une valeur sélective plus forte. Chez les femelles accouplées, le taux de fertilisation devrait évoluer vers des valeurs plus élevées, ce qui compense la production de mâles par les femelles vierges (Godfray, 1990). Cette hypothèse a été parfois avancée pour expliquer les taux de fertilisation élevés souvent observés dans les populations naturelles de parasitoïdes (Antolin and Strand, 1992; Heimpel, 1997; Ode et al., 1997). Godfray (1990) a également avancé que certains parasitoïdes pouvaient être capable d'ajuster leur taux de fertilisation lorsqu'ils sont accouplés en fonctions d'indices environnementaux sur la densité de mâles disponibles, comme par exemple le temps passé avant l'accouplement, le nombre de tentatives d'accouplement, etc. Chez les hyménoptères parasitoïdes, cette hypothèse est équivoquement supportée par les expériences empiriques, certaines montrant la présence d'une telle capacité à ajuster le taux de fertilisation (King, 2002) et d'autres démontrant son absence (Fauvergue et al., 1998).

Les taux de fertilisation élevés observés chez les parasitoïdes peuvent également être expliqués par la présence d'accouplements locaux et/ou consanguins. L'hypothèse du *Local Mate Competition* suggère que lors d'accouplements locaux des frères risquent d'entrer en compétition pour l'accès à l'accouplement. Il est donc plus efficace pour la mère de produire juste assez de mâles pour que ses filles soient accouplées (Hamilton, 1967; West et al., 2005). De plus, chez les haplodiploides, les mères sont davantage apparentées à leurs filles qu'à leur fils lorsque les accouplements sont consanguins, ce qui contribue à renforcer ce biais (Taylor and Bulmer, 1980). Ces hypothèses s'adaptent particulièrement bien aux parasitoïdes, où certaines espèces sont grégaires (c'est à dire qu'elles produisent plusieurs parasitoïdes par hôtes) et s'accouplent souvent à proximité du site d'émergence, voir strictement au sein d'une même fratrie. Chez les espèces de parasitoïdes où les accouplements frères-soeur sont courants, les taux de fertilisation sont généralement très élevés (Godfray, 1994).

Un taux de fertilisation élevé, quelle que soit son origine, permet de contrebalancer la production de mâles par les femelles restées vierges, et donc d'équilibrer le sexe ratio de la popu-

lation lorsque des difficultés d'accouplement apparaissent. En théorie, un taux de fertilisation élevé permet donc de limiter les impacts démographiques des difficultés d'accouplement et les risques d'apparition d'un effet Allee.

2. 2 Fardeau génétique et CSD chez les hyménoptères

Dans une large population de diploïdes, les mutations délétères récessives ne sont que rarement exprimées dans le phénotype des individus et s'accumulent au cours du temps (Frankham, 2005). En revanche, dans une population haplodiploïde, les mâles haploïdes expriment ces mutations qui peuvent donc être purgées par sélection naturelle (Henter, 2003; Hedrick and Parker, 1997; Goldstein, 1994). Le fardeau de mutation est donc beaucoup moins important chez les hyménoptères que chez d'autres insectes (Antolin, 1999). Toutes les mutations ne sont pas concernées: les mutations qui s'accumulent sur des caractères uniquement femelles ne sont pas exprimées par les mâles et s'accumulent donc normalement (Antolin, 1999).

Cependant, le principal mécanisme de détermination du sexe des hyménoptères, appelé *Complementary Sex Determination* (CSD), induit une dépression de consanguinité particulière, qui peut être plus forte que chez les diploïdes (Zayed and Packer, 2005). Lorsque le sexe est déterminé par CSD, le développement mâle ou femelle se fait en fonction de la complémentarité des allèles du gène *csd*. Les oeufs non fertilisés sont haploïdes et se développent en mâles, mais les oeufs fertilisés sont diploïdes et ne se développent en femelles que si ils sont hétérozygote à au moins un des loci du *csd*. Si ils sont homozygotes à tous les loci, ils se développent en mâles diploïdes, généralement non viables ou stériles (Heimpel and de Boer, 2008). Le gène *csd* peut être situé sur un seul locus (sl-CSD) ou sur plusieurs locus (ml-CSD) selon les espèces.

Dans les populations naturelles d'hyménoptères, la diversité génétique du CSD est généralement assez élevée (jusqu'à 86 allèles) (Cook and Crozier, 1995) et la production de mâles diploïdes est donc rare. Cependant, lorsque la population devient petite, sa diversité génétique se réduit et la production de mâles diploïdes augmente, réduisant la fitness des individus. La production de mâles diploïdes dans une population s'apparente à une dépression de consanguinité particulière liée à la super-dominance des allèles (Zayed and Packer, 2005; Hein et al., 2009).

Le CSD est un mécanisme de détermination du sexe qui est ancestral chez les hyménoptères (Asplen et al., 2009). Il est présent dans quatre super-familles sur cinq et sa présence a été vérifiée dans plus de 60 espèces (Heimpel and de Boer, 2008; van Wilgenburg et al., 2006). Cependant, on connait également des espèces qui ont abandonné le CSD, et utilisent des mécanismes de détermination du sexe alternatifs, généralement mal connus (van Wilgenburg et al., 2006). Pour la plupart des hyménoptères parasitoïdes chez lesquels la présence du CSD a été testée, la détermination du sexe se fait par sl- ou ml- CSD et la diversité génétique au locus du *csd* est relativement faible (de 9 à 20 allèles)(Hedrick et al., 2006; Zayed and Packer, 2005; Heimpel et al., 1999).

La dépression de consanguinité associée au CSD induit un vortex d'extinction chez les hyménoptères : lorsque la diversité génétique du *csd* est réduite, la production de mâles diploïdes réduit

le taux de croissance de la population, ce qui diminue sa taille et sa diversité génétique, et augmente la production de mâles diploïdes (figure 1.2). En fin de compte, la population s'éteint. Les mâles diploïdes stériles peuvent constituer un fardeau supplémentaire pour la population (par rapport aux mâles diploïdes non viables) parce que les femelles qu'ils accouplent sont incapables de produire des descendants fertilisés viables. L'impact théorique du CSD sur la démographie d'une population dépend également des traits d'histoire de vie de l'espèce. Particulièrement, le Diploid Male Vortex est contré par : 1) une forte densité dépendance négative, qui limite l'impact de la production de mâles diploïdes sur la croissance, 2) un taux de fertilisation élevé, qui permet de continuer à produire un nombre de femelles viables conséquent malgré la production de mâles diploïdes, 3) un renouvellement de la diversité génétique du *csd* par des mutations, et 4) le choix d'un partenaire non diploïde, qui permet de limiter le fardeau supplémentaire associé à la présence de mâles diploïdes stériles (Hein et al., 2009). Le modèle théorique du diploïde mâle vortex peine à être confirmé par des études empiriques chez les parasitoïdes(Elias et al., 2010b)(comm. personnelle C. Vayssade).

Les modèles théoriques associés au CSD considèrent généralement le sl-CSD (Zayed and Packer,

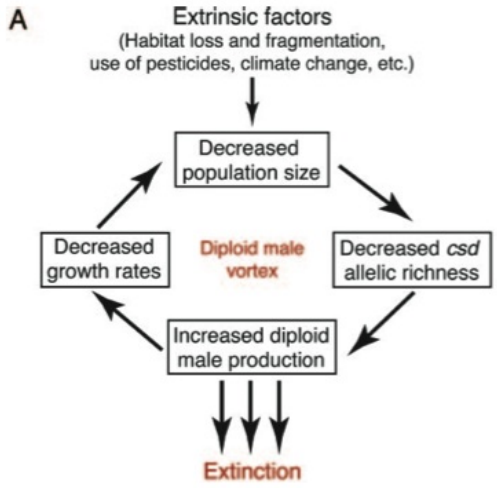

Figure 1.2: Diploid Male Vortex (depuis Zayed et al, 2005)

2005; Hein et al., 2009). Le risque de produire des mâles diploïdes est moins élevé avec le ml-CSD qu'avec le sl-CSD, puisque seuls les homozygotes à tous les loci se développent en mâles diploïdes. Cependant, le nombre de loci initialement impliqués dans le ml-CSD est souvent difficile à évaluer, et le ml-CSD devient sl-CSD lorsque la dépression de consanguinité est forte (Heimpel and de Boer, 2008). Dans la suite de ce manuscrit, nous nous intéresserons donc principalement au sl-CSD, auquel je ferai référence en parlant du CSD.

2. 3 Effet Allee et CSD: le cas des parasitoïdes

Chez les parasitoïdes, ni l'impact démographique de l'effet Allee comportemental ni celui du CSD n'ont été étudiés en tenant compte de l'interaction du parasitoïde avec son hôte. Pour-

tant, plusieurs caractéristiques démographiques des parasitoïdes, liées à leur intéraction avec l'hôte, pourraient affecter l'impact de l'effet Allee et du CSD:

1. La densité dépendance forte entre les populations hôte et parasitoïde. Les relations densité dépendantes ont un impact fort sur les conséquences démographiques des fardeaux génétiques, dont le CSD (Agrawal and Whitlock, 2012; Hein et al., 2009) et sur celles des effets Allee élémentaires (Courchamp et al., 2008). Les relations impliquant une densité dépendance négative, comme la compétition, ont tendance à atténuer l'impact démographique des fardeaux génétiques (Agrawal and Whitlock, 2012; Hein et al., 2009), tandis que les relations de densité dépendance positive, comme par exemple le mutualisme, peuvent avoir un effet synergique avec l'effet Allee et le fardeau génétique (Courchamp et al., 2008) (article1). Dans le cas de la relation hôte parasitoïde, la croissance de l'hôte est corrélée négativement avec la densité du parasitoïde, mais la croissance du parasitoïde est corrélée positivement avec la densité d'hôte. Cette relation domine la démographie des parasitoïdes dans les modèles théoriques classiques, et peut donner naissance à des dynamiques stables ou cycliques (Hassell, 2000). Des populations de parasitoïdes ayant des dynamiques cycliques ont été observées dans la nature (Wearing et al., 2004) mais ce type de dynamiques reste relativement rare comparé aux prédictions des modèles théoriques. Les populations cycliques passent régulièrement par des épisodes à faible densité, durant lesquels elles peuvent perdre une grande partie de leur diversité génétique et subir un effet Allee. Il est donc difficile de prévoir l'impact démographique du CSD et de l'effet Allee dans une population de parasitoïdes en extrapolant simplement leur impact dans une population sans interaction.
2. L'inégalité entre les rôles respectifs des deux sexes dans l'interaction. Chez les parasitoïdes, seules les femelles attaquent les hôtes, puisqu'elles sont responsables de l'oviposition. En conséquence, une variation de sexe ratio dans la population affecte l'interaction (Hassell et al., 1983). Un effet Allee comportemental peut entraîner de telles variations chez les parasitoïdes puisque les femelles vierges ne produisent que des mâles.

Organisation du manuscrit

Ce manuscrit de thèse est écrit sur articles et les articles qui le composent s'articulent en deux parties majeures, qui concernent respectivement l'effet Allee comportemental et le CSD. Elles sont précédées par l'introduction bibliographique, que nous venons de passer et qui contient la synthèse bibliographique (article 1). Ce premier article est en préparation et nous envisageons d'y associer d'autres collaborateurs avant la soumission.

1. Résultats (1) : Les effets Allee comportementaux

Dans la première partie, nous allons nous intéresser à l'impact démographique de l'effet Allee comportemental chez les parasitoïdes (article 2, 3). Dans un premier temps, nous nous focaliserons sur les dynamiques hôte-parasitoïdes qui résultent de la présence d'un effet Allee comportemental chez un parasitoïde hyménoptère, et sur leurs possibles conséquences pour la lutte biologique classique. Nous montrons que l'effet Allee peut avoir un impact particulièrement fort sur la démographie des parasitoïdes et qu'il peut induire des extinctions chez les populations stables et avoir un impact stabilisant ou déstabilisant sur les populations cycliques selon les caractéristiques des populations hôtes et parasitoïdes. L'efficacité de recherche du parasitoïde et l'intensité de la compétition intra-spécifique chez l'hôte sont apparues comme des facteurs d'une importance majeure pour déterminer l'impact démographique de l'effet Allee comportemental (article 2). Cet article a été publié dans la revue Plos One.

Dans un second temps, nous creuserons le rôle de l'haplodiploïde sur l'impact démographique de l'effet Allee chez les parasitoïde. Pour ce faire, nous comparerons l'impact de l'effet Allee sur les parasitoïdes diploïdes et haplodiploïdes, en considérant les différents traits d'histoire de vie autorisés par l'haplodiploïdie (notamment l'élévation ou l'ajustement du taux de fertilisation). Nous montrons que les principaux avantages gagnés par l'haplodiploïdie sur ce terrain s'expriment en terme de capacité d'invasion à faible densité et de robustesse à des variations environnementales ou à l'invasion d'un nouveau parasitoïde. L'ajustement ou l'élévation du taux de fertilisation n'offrent en contraste que des avantages minimes (article 3). Cet article est

encore en préparation, et sa soumission à la revue Oïkos est prévue pour l'été 2014.

2. Résultats (2) : Le *Complementary Sex Determination*

Dans la seconde partie de ce manuscrit, nous allons nous intéresser à l'impact du CSD sur la démographie et la valeur sélective du choix de partenaire chez les parasitoïdes (articles 4, 5, 6). Pour ce faire, nous avons développé un modèle éco-génétique original, contenant une partie déterministe et une partie stochastique, présenté en détail dans l'article 4.

Dans un premier temps, nous nous focaliserons sur l'impact du CSD sur le risque d'extinction et les dynamiques des populations de parasitoïdes, en considérant des mâles diploïdes non viables ou stériles. Nous montrons que contrairement à toute attente, le CSD réduit le risque d'extinction des parasitoïdes en amortissant les cycles de forte amplitude. Il entraîne également des extinctions chez certaines populations, dont les caractéristiques sont clairement différentes des extinctions induites par le Diploid Male Vortex ou les vortex d'extinction classiques (en effet, les extinctions induites par le CSD sont peu dépendantes de la diversité génétique ou de l'abondance des parasitoïdes). La présence de mâles diploïdes stériles augmente le risque d'extinction chez les parasitoïdes comme chez les populations libres (article 4). Cet article est à un stade avancé de préparation et devrait être soumis à la revue Ecology Letters d'ici la soutenance de la thèse (en mai-juin 2014).

Dans un second temps, nous avons élargi les hypothèses biologiques sur la population de parasitoïdes et étudié l'impact de six facteurs: le renouvellement de la diversité génétique à travers des mutations au locus du *csd* (1), la stochasticité environnementale et démographique (2 et 3), le taux de fertilisation du parasitoïde (4), le nombre de parasitoïdes produits par hôte (5) et la pseudo-virginité des femelles lors de l'accouplement avec un mâle diploïde (6, voir article 5 pour une explication détaillée). Nous montrons que les résultats qualitatifs du CSD sur les dynamiques des parasitoïdes sont très robustes à des variations sur les hypothèses concernant la biologie de la population. En revanche, l'impact stabilisant du CSD sur les dynamiques cycliques des parasitoïdes est atténué si le lien hôte attaqué-parasitoïde produit se relâche, et si la diversité génétique est renouvelée à l'apogée des cycles (article 5). Cet article est encore en préparation, et nous n'excluons pas de faire intervenir d'autres collaborateurs dans son élaboration ou de revoir sa structure avant de le soumettre.

Enfin, nous avons considéré la valeur sélective et l'impact démographique d'un choix de partenaire actif visant à éviter la production de mâles diploïdes, dans lequel les femelles sélectionnent les individus non appariés au gène *csd*. Nous nous intéresserons particulièrement aux situations où l'évolution des traits associés au choix de partenaire - les capacités de discrimination des femelles et le coût associé au refus d'un partenaire - peut avoir un impact délétère sur la démographie de la population. Je montre que même si la discrimination des mâles appariés a généralement une valeur sélective forte, ce n'est pas toujours le cas lorsque la diversité génétique est faible et/ou que le coût associé aux refus d'un partenaire est important (article 6).

3. Modèle hôte-parasitoïde de référence et limites de l'étude

En amont de ce travail, j'ai développé un modèle hôte-parasitoïde déterministe en temps discret à partir de fonctions classiques de densité dépendance et de taux de survie au parasitisme. Sous sa forme la plus basique, il peut être étudié analytiquement (voir l'annexe de l'article 2) mais pas lorsqu'il intègre l'effet Allee ou le CSD. Ce modèle inclut une densité dépendance chez l'hôte (g) dont la sévérité peut être contrôlée (de sous-compensée à sur-compensée) (Maynard Smith and Slatkin, 1973) et qui affecte la population hôte avant l'impact du parasite. Il inclut également une fonction de survie au parasitisme qui autorise une distribution des attaques plus ou moins agrégée (May et al., 1981). Les populations d'hôtes (N) et de parasitoïdes (P) sont décrites par les équations suivantes :

$$\begin{cases} N_{t+1} = \lambda N_t g(N_t) f(P_t) \\ P_{t+1} = N_t g(N_t) \left(1 - f(P_t)\right) \end{cases} \tag{1.2}$$

Des variations sur ce modèle, incluant un effet Allee comportemental chez des haplodiploïdes ou chez des diploïdes (article 2, 3) ou une partie stochastique gérant les génotypes du *csd* (article 4, 5, 6) sont utilisées tout au long de ce manuscrit. Conceptuellement, ce modèle est proche de celui développé par Bernstein (1996). Il permet de caractériser l'interaction hôte-parasitoïde en autorisant l'apparition d'une grande variété de dynamiques, et intègre un grand nombre d'hypothèses biologiques.

Je n'ai pas intégré dans mes travaux de dimension spatiale explicite, principalement pour une question de temps. Nous n'étudierons pas non plus la présence d'hôtes alternatifs, bien qu'elle soit possible dans les populations naturelles de parasitoïdes, ni de manière plus générale d'interactions inter-spécifiques à plus de deux acteurs (excepté dans la seconde partie de l'article 3, où des parasitoïdes diploïdes et haplodiploïdes sont en compétition pour le même hôte). Un certain nombre d'autres hypothèses biologiques ont été ignorées dans l'effort de construire des modèles théoriques relativement simples et exploitables.

Malgré tout, j'estime que ce travail de thèse apporte des hypothèses nouvelles et très intéressantes à la fois sur le plan théorique et appliqué. D'un point de vue théorique, j'apporte un nouveau regard sur l'impact des fardeaux génétiques chez les populations en interaction - et particulièrement les parasitoïdes - et sur le rôle du CSD chez les parasitoïdes. J'apporte également une formalisation des hypothèses existantes sur l'impact démographique de l'effet Allee comportemental chez les parasitoïdes, et sur l'effet de l'haplodiploïdie sur cet impact. Sur le plan appliqué, mes travaux ne peuvent être transposés directement sans que les hypothèses n'aient été au préalable testées empiriquement. Cependant, si mes conclusions s'avèrent vérifiées, elles pourraient apporter des connaissances intéressantes pour la gestion des parasitoïdes en lutte biologique principalement, mais également pour la conservation des petites populations en général et la lutte contre les espèces invasives.

Part II

Conséquences démographiques d'un l'effet Allee comportemental chez les parasitoïdes

CHAPTER

2 Host-parasitoid dynamics and the success of biological control when parasitoids are prone to Allee effects.

Article 2

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Résumé

Lorsque la densité d'une population est faible, les organismes sexués peuvent avoir des difficultés à trouver un partenaire et à s'accoupler, ce qui diminue leur taux de fécondité : c'est ce qu'on appelle un effet Allee comportemental. Cet effet Allee comportemental peut induire une réduction du taux de croissance de la population à faible densité, c.à.d un effet Allee démographique, et donc une augmentation du risque d'extinction de la population. Pour les espèces qui sont en interaction avec une autre espèce, comme dans les systèmes hôtes - parasitoïdes, les dynamiques des différentes populations sont principalement pilotées par des interdépendances démographiques. Les conséquences des effets Allee comportementaux sont donc beaucoup plus complexes à prédire que chez une population isolée. Or, les parasitoïdes sont largement employés en lutte biologique classique, une utilisation qui amène à tenter d'acclimater des petites populations de parasitoïdes non-indigènes dans un nouvel environnement. Au cours de ces introductions, les parasitoïdes peuvent subir des faibles densités de populations et des difficultés d'accouplement.

L'objectif principal de cet article est d'étudier les conséquences démographiques des difficultés d'accouplement à faible densité chez les parasitoïdes, et ses possibles implications pour la lutte biologique. Pour cela, nous avons développé un modèle hôte - parasitoïde déterministe à deux sexes, avec un effet Allee comportemental. Nous considérons que le parasitoïde est un hyménoptère haplodiploïde, comme 78% des parasitoïdes (c.à.d. que les femelles vierges sont capables de produire des mâles). Nous abordons les interactions entre l'effet Allee et les déterminants clés de la démographie du système hôte – parasitoïde, tels que la distribution des attaques des parasitoïdes et la compétition intra-spécifique chez

l'hôte. Notre étude montre que les difficultés d'accouplement des parasitoïdes à faible densité induisent un seuil d'extinction et augmentent le domaine d'extinction déterministe du parasitoïde. Lorsqu'ils sont sensibles à des difficultés d'accouplement, les parasitoïdes ayant une faible efficacité de recherche ont tendance à s'éteindre, tandis que les parasitoïdes ayant une forte efficacité de recherche peuvent soit persister soit s'éteindre selon la sévérité de la compétition intra-spécifique de l'hôte. Nous montrons que les parasitoïdes les plus susceptibles d'être utilisés comme agents de lutte biologique (pour leur capacité à réduire les populations hôtes) sont également les plus susceptibles d'être amenés à s'éteindre à cause d'un effet Allee comportemental. Cette étude propose de nouvelles perspectives pour la compréhension des dynamiques observées dans les systèmes hôtes - parasitoïdes naturels, et remet en cause l'idée répandue selon laquelle les hyménoptères parasitoïdes seraient peu sensibles aux effets Allee. Nous espérons que les connaissances que notre étude apporte contribueront à améliorer les chances de réussite des programmes d'introduction de parasitoïdes en lutte biologique.

Abstract

In sexual organisms, low population density can result in mating failures and subsequently yields a low population growth rate and high chance of extinction. For species that are in tight interaction, as in host-parasitoid systems, population dynamics are primarily constrained by demographic interdependences, so that mating failures may have much more intricate consequences. Our main objective is to study the demographic consequences of parasitoid mating failures at low density and its consequences on the success of biological control. For this, we developed a deterministic host-parasitoid model with a mate-finding Allee effect, allowing to tackle interactions between the Allee effect and key determinants of host-parasitoid demography such as the distribution of parasitoid attacks and host competition. Our study shows that parasitoid mating failures at low density result in an extinction threshold and increase the domain of parasitoid deterministic extinction. When proned to mate finding difficulties, parasitoids with cyclic dynamics or low searching efficiency go extinct; parasitoids with high searching efficiency may either persist or go extinct, depending on host intraspecific competition. We show that parasitoids suitable as biocontrol agents for their ability to reduce host populations are particularly likely to suffer from mate-finding Allee effects. This study highlights novel perspectives for understanding of the dynamics observed in natural host-parasitoid systems and improving the success of parasitoid introductions.

KEYWORDS: Allee effects; host-parasitoid interaction; host-parasitoid model; parasitoid; haplodiploidy; density dependence; modeling; cyclic dynamics; stable dynamics; aggregation; arrhenotoky; biological control; biocontrol efficiency; pest; unmated females; mate-finding difficulties; mating failures, extinction; persistence

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Host-Parasitoid Dynamics and the Success of Biological Control When Parasitoids Are Prone to Allee Effects

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Abstract

In sexual organisms, low population density can result in mating failures and subsequently yields a low population growth rate and high chance of extinction. For species that are in tight interaction, as in host-parasitoid systems, population dynamics are primarily constrained by demographic interdependences, so that mating failures may have much more intricate consequences. Our main objective is to study the demographic consequences of parasitoid mating failures at low density and its consequences on the success of biological control. For this, we developed a deterministic host-parasitoid model with a mate-finding Allee effect, allowing to tackle interactions between the Allee effect and key determinants of host-parasitoid demography such as the distribution of parasitoid attacks and host competition. Our study shows that parasitoid mating failures at low density result in an extinction threshold and increase the domain of parasitoid deterministic extinction. When proned to mate finding difficulties, parasitoids with cyclic dynamics or low searching efficiency go extinct; parasitoids with high searching efficiency may either persist or go extinct, depending on host intraspecific competition. We show that parasitoids suitable as biocontrol agents for their ability to reduce host populations are particularly likely to suffer from mate-finding Allee effects. This study highlights novel perspectives for understanding of the dynamics observed in natural host-parasitoid systems and improving the success of parasitoid introductions.

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Introduction

Since the pioneering work of Nicholson and Bailey [1], many theoretical ecologists have been interested in modeling the tight interactions that occur between insect parasitoids and their hosts [2,3]. The importance of parasitoids for pest control programs undoubtedly accounts for this interest. Because parasitoids are more specific than predators and sometimes efficient enough to cause up to 95% reduction of the host population, they have been extensively deployed as biological control agents [4]. However parasitoids released into the field often fail to establish [5,6]. The most pervasive determinent of establishment failure is initial population size [7,8], so that the Allee effect has been hypothesized as a likely cause of these introduction failures [7,8,9,10,11].

Demographic Allee effects are defined as decreasing *per capita* population growth rates when abundance declines. In response to "strong" Allee effects, the population growth rate becomes negative below a critical density *i.e.* the population goes extinct if its abundance falls below this threshold [12,13,14]. Demographic Allee effects occur in most taxa [15], and play key roles in conservation biology, invasion biology, and biological control [9,16,17,18,19]. Demographic Allee effects are underpinned by component Allee effects, namely biological traits or trophic interactions that make individual fitness positively dependent on population size [17]. The most common and most often studied component Allee effect is the mate-finding Allee effect, which describes the difficulty that males and females experience in locating each other, and the consequent decline in reproduction at low population densities. Several theoretical studies have investigated how mating difficulties may translate into demographic effects [19,20]. Although firm evidence of causal relations between mate-finding Allee effects and establishment success in insect populations is still relatively rare [11], mating failure at low density is often considered to be an important cause of demographic Allee effects in invading species [21,22,23,24,25].

Parasitoids, in particular those introduced as biocontrol agents, are likely to experience low population densities, either because interactions with their hosts lead to cyclic dynamics, or as a consequence of external factors such as the introduction of small number of individuals in classical biological control, harvesting, crop rotation or chemical pest control [26,27,28]. Consequently, mate-finding difficulties may arise in parasitoid populations (the effect of number released is analyzed by Hopper and Roush [8]). Demographic Allee effects have been observed in field studies of introduced parasitoids [29,30], but could not be linked to mate-finding difficulties in particular [29,31].

The demographic consequences of a mating failures may be affected by other forms of density dependence in the population, such as those generated by intraspecific competition or by interactions between different trophic levels within a community [14]. The interaction between the host and the parasitoid is

particularly stringent; therefore, the dynamics of host-parasitoid systems strongly depend on the characteristics of both hosts and parasitoids populations - such as intraspecific competition within the host population, the searching efficiency of the parasitoid, and the spatial distribution of parasitoid attacks [32,33]. These biological characteristics should, in turn, combine with component Allee effects to produce idiosyncratic patterns in host-parasitoid population dynamics.

To date, Allee effects in host-parasitoid or host-parasite interactions have mainly been studied by assuming Allee effects on the host population, by considering either mate-finding [34] or demographic Allee effects [35,36]. These have revealed the existence of a critical host density below which the parasitoid or parasite goes extinct [35,36], but also a reduction of the chaotic domain of the dynamics [34]. Whether and how a mate-finding Allee effect in parasitoids impacts on the extinction propensity of the system remains to be investigated.

About 75% of the parasitoid species belong to the order Hymenoptera and are therefore haplodiploid (*i.e.* the males are haploid and females diploid [37,38]). This genetic feature is important in the context of mate-finding Allee effects because it allows females to reproduce even if they do not mate: virgin females can produce males parthenogenetically by laying unfertilized eggs. Consequently, whereas a mate-finding Allee effect decreases the mean fecundity in diploid species, it produces a sex ratio shift toward males in haplodiploid ones (assuming that the fecundities of mated and unmated females do not differ [39,40,41,42,43]). Hence, it is generally accepted that haplodiploidy prevents or alleviates the demographic consequences of mate-finding Allee effects for free-living organisms [8,44]. However, in host-parasitoid systems this assumption may not be valid because variable or density-dependent sex ratios also affect the dynamics of these systems [45,46,47].

In this study, we use mathematical models to tackle the dynamics of host-parasitoid systems when the parasitoid experiences mate-finding difficulties at low density. (1) We focus on the extinction propensity in relation to increasing difficulties in mating. (2) We explore the dynamics of parasitoid populations that appear persistent, and look at how the characteristics of the host (in various types of intraspecific competition) and those of the parasitoid (in various distributions of attacks) combine with mate-finding Allee effects to impact the system dynamics. (3) Finally, we adopt a more applied perspective by documenting the consequences of the mate-finding Allee effect on the minimum number of parasitoids insuring establishment and on the remanent host density.

Materials and Methods

Following most theoretical approaches concerning demographic consequences of the Allee effect [12,13,19,44,48] we used deterministic non-spatial models. Both demographic and environemental stochasticities are known to affect the risk of extinction of a population. However, our aim is not to estimate precisely the extinction probability of a given population but rather to capture the general features of host-parasitoid systems when parasitoids are prone to an Allee effect. In this perspective, stochastic processes are probably not liable to modify significantly our qualitative conclusions. Spatial components were implicitely included in our model through functions describing the distribution of parasitoid attacks and the proportion of unmated females.

We started out from a classical discrete-time host-parasitoid framework with a host population, N, and a parasitoid population, P. At each generation, a proportion $f(P_t)$ of hosts escape parasitism depending on parasitoid density. The host finite rate of increase is λ and its probability of surviving intraspecific competition is given by $g(N_t)$ (all parameters, their interpretation and the values used in this article can be found in Table 1). We assumed that parasitized and unparasitized hosts are equally likely to survive intraspecific competition, a situation that arises either if intraspecific competition precedes parasitism or if parasitism does not affect the competitive interactions among hosts. This gives us:

$$\begin{cases} N_{t+1} = \lambda N_t g(N_t) f(P_t) \\ P_{t+1} = N_t g(N_t)[1 - f(P_t)] \end{cases} \quad (1)$$

Males (M) and females (F) were distinguished within the parasitoid population in order to model a mate-finding Allee effect. We assumed females to produce offspring with a fixed fertilization rate $s_0 = 0.5$ (in haplodiploids, fertilized eggs will develop into females whereas unfertilized eggs develop into males).

A consequence of the mate-finding Allee effect is that a proportion of virgin females $p(M_t)$ occurs in the population as a decreasing function of male density:

$$p(M_t) = \exp\left(-\frac{M_t}{\alpha}\right) \quad (2)$$

[12]where α describes the intensity of the mate-finding Allee effect: for a given male density, the greater α, the greater the proportion of virgin females. If α approaches 0 (no Allee effect), then $p(M_t)$ approaches 0 as well.

Because we considered an haplodiploid reproductive system, both mated and virgin females attack the hosts; we assumed that virgin females attacks the host at the same rate and have the same fecundity as mated females, but produce only male offspring. The model becomes:

$$\begin{cases} N_{t+1} = \lambda N_t g(N_t) f(F_t) \\ F_{t+1} = (1 - s_0)[1 - p(M_t)] N_t g(N_t)[1 - f(F_t)] \\ M_{t+1} = [s_0(1 - p(M_t)) + p(M_t)] N_t g(N_t)[1 - f(F_t)] \end{cases} \quad (3)$$

Without Allee effects (α approaches 0), this model converges to the one-sex model (equation 1), which could be solved analytically (see Text S1 and Figure S1).

Probability of escaping parasitism

To describe the probability that a host escapes from parasitism, we chose a function that allowed us to consider various possible distributions of parasitoid attacks, which is known to affect the stability of the host-parasitoid system [49]. f is given by the zeroth term of the negative binomial distribution:

$$f(F_t) = \left(1 + a\frac{F_t}{k}\right)^{-k} \quad (4)$$

where a is the parasitoid's searching efficiency (the proportion of total hosts encountered by parasitoids *per* unit time), and k is the clumping parameter of the negative binomial. k determines the degree of contagion resulting from parasitoid attacks: as k decreases, attacks become more aggregated [49]. Decreasing k increases the stability domain: when the host alone undergoes

Table 1. Biological interpretation and numeric values for the parameters used in model simulations.

Symbol	Parameter	Values
λ	Host finite rate of increase	$1 \leq \lambda \leq 50$
a	Parasitoid searching efficiency	$0 \leq a \leq 1$
K	Environment carrying capacity for hosts	$K = 500$
		$b = 0.8$ (under-compensated)
b	Host intraspecific competition	$b = 1.5$ (mild compensation)
		$b = 3.2$ (over-compensated)
s_0	Proportion of males among parasitoid offspring	$s_0 = 0.5$
		$\alpha = 10^{-6}$ (no Allee effect)
α	Intensity of the mate-finding Allee effect	$\alpha = 5$ (mild Allee effect)
		$\alpha = 20$(strong Allee effect)
k	Distribution of parasitoid attacks	k = 0.8 (aggregated attacks)
		k = 10 (random attacks)

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exponential growth, the host-parasitoid model is completely stable when $k < 1$[49].

Host density dependence

The host's probability of surviving intraspecific competition is expressed by the Maynard Smith and Slatkin [50] density dependence function g:

$$g(N_t) = \left(1 + (\lambda - 1)\left(\frac{N_t}{K}\right)^b\right)^{-1} \quad (5)$$

where K is the carrying capacity of the environment for the host population, and b is the coefficient determining the severity of the density dependence, *i.e.* the degree to which mortality compensates for the increase in population size. The higher the value of b, the greater the compensation for host competition. The dynamics of the parasitoid-free host depends on the parameter $C = b\frac{\lambda-1}{\lambda}$. For $C < 1$, the host population is asymptotically stable (under-compensating competition); for $1 < C < 2$, the host population reaches stability through oscillatory damping; for $C > 2$, the host population is unstable (over-compensating competition). We were able to display the various host dynamics through b for $\lambda > 3$, by taking discrete values not too close from the borders ($b = 0.8$, $b = 1.5$, $b = 3.2$, Table 1) which contributed to the choice of this specific function. Compared to six other models of density dependence, the Maynard Smith and Slatkin function was found to provide the best description of a wide range of data [51].

Model analysis

We were not able to obtain analytical results from our model and we used numerical simulations to characterize the dynamics. Simulations were run with a program developed in C++ langage. Each simulation was stopped when an equilibrium (including extinction) or a periodic dynamic was reached. We considered the parasitoid population as extinct when female density decreased below 10^{-6}. Initial conditions for one simulation were taken as the coexistence equilibrium values of populations in the vicinity of its position on the a- λ diagram, and simulations were run for 10 000 generations.

The dynamics of the host-parasitoid system depends mainly on which party determines the dynamics: the parasitoids (through a high host parasitism rate) or the host (through strong intraspecific competition). The relative impact of the host and of the parasitoid on the dynamics can be captured by two parameters: the host's finite rate of increase, λ, and the product aK (the expected number of hosts a single parasitoid female with a searching efficiency of a would parasitize when encountering a new host population with a density of K). However, for simplicity, we fixed K and represented the parasitoid dynamics as a function of the parasitoid searching efficiency only, a, and of the host's finite rate of increase, λ. The higher a relative to λ, the higher the impact of parasitism on the dynamics of the system. Conversely, at low value of a relative to λ, competition between hosts is a prime driver of the dynamics.

The critical number of females

When the parasitoids undergo mate-finding difficulties, persistence of host-parasitoid systems may rest on the initial parasitoid density. We quantified the minimum number of female parasitoids F_{crit} that need to be introduced to allow parasitoid establishment. This is determined numerically by introducing an increasing number of parasitoid females into a host population with a stable density equal to its carrying capacity ($N_0 = K$). The number of hosts remaining is also of particular value for biological control: we quantified $N_{\max}$ as the number of hosts at equilibrium when the population is stable, or the maximum number of hosts when the host dynamics are cyclic.

Results

Allee effects and risk of extinction

When parasitoid mating success decreases with decreasing density, the domain of parameter values in which the parasitoid population goes extinct increases (figure 1). This general result appeared when we considered random attacks ($k = 10$) and medium compensation of the host competition ($b = 1.5$). In the absence of any Allee effect, the parasitoid went extinct in two distinct domains (figure 1.a): low searching efficiencies and high host finite rates of increase (the lower domain of extinction), and high searching efficiencies and low host finite rates of increase (the upper domain of extinction). An increase of the strength of the

Allee effect clearly induced an expansion of the lower domain of extinction, while the upper domain was reduced (figures 1.b, 1.c).

Allee effects, density dependence and aggregation

Parasitoid mating failures interacted with host density dependence to produce a complex pattern of extinction and persistence. In the absence of Allee effect, the domain of parameter values yielding parasitoid extinction was much larger for over-compensating than for under-compensating competition (figures 2.a, 2.d). The Allee effect led to an expansion of the lower domain of extinction whatever the severity of competition. However the consequences of the Allee effect on the upper domain of extinction and on the global area of parasitoid extinction were rather different depending on the competition. In a context of under-compensating host competition, the upper domain of extinction was progressively reduced, and the global area of extinction progressively increased when the Allee effect increased (figures 2.a, 2.b, 2.c). In a context of over-compensating competition, the upper domain of extinction and the global area of extinction increased when an Allee effect was introduced (figure 2.d and 2.e), but the upper domain of extinction was reduced and the global area of extinction remained equally large when the Allee effect increased from medium to strong intensities (figures 2.e, 2.f).

Aggregated attacks reduced the upper domain of extinction and stabilized the persistent parasitoid in the absence of any Allee effect (figures 3.a, 3.d). The Allee effect progressively expanded both upper and lower domains of extinction with aggregated attacks (figures 3.e, 3.f).

Qualitative dynamics and Allee effects

When the parasitoid persisted, the dynamics of the host-parasitoid system could be asymptotically stable, stable with oscillatory damping, or cyclic, from the lower domain of extinction to the upper one (figures 2 and 3).

Most cyclic parasitoid populations went extinct as soon as an Allee effect was introduced, regardless of the level of compensation of the host competition or the distribution of parasitoid attacks (figures 2 and 3). When attacks were aggregated, high parasitoid searching efficiency and low host finite rate of increase resulted in a stable equilibrium via damped oscillations (figure 3d, top left). Increasing strength of the Allee effect amplified these oscillations and produced either cyclic dynamics (if moderate) or extinctions (if strong) (figure 4c). This phenomenon was also observed when attacks occured at random, but with under-compensating competition only: persistent parasitoids in the vicinity of the upper domain of extinction could display cyclical dynamics when an Allee effect was introduced (figure 4.b).

Strong Allee effects induced a shift to higher searching efficiencies of the area corresponding to parasitoid persistence, and of all the areas corresponding to the various dynamics of persistent populations (cycles, oscillatory damping, and asymptotic stability) (figures 2.e, 2.f). This could enable a high searching efficiency parasitoid, which would not have settled without Allee effect, to become persistent (figure 4.a) and in this context Allee effects had a stabilizing impact on high searching efficiency parasitoids. For under-compensating host competition, destabilizing and stabilizing effects could appear for consecutive values of α, on persistent parasitoids in the vicinity of the upper domain of extinction (figure 4.b).

Quantitative effects and biological control

The intensity of the Allee effect affected the equilibrium density of female parasitoids (F^*). Male and female parasitoids densities at equilibrium were the highest when the population is close to the upper domain of extinction and the lowest when it was close to the lower domain of extinction. With increasing Allee effects, the population sex ratio at equilibrium became male-biased, and F^*decreased (figures 4.a, 4.b).

The critical number of females that have to be introduced for the parasitoid to persist (F_{crit}) expresses the value of the extinction threshold for the parasitoid population. F_{crit} increased with increasing Allee effect intensity (figures 4 and 5) and decreased with increasing parasitoid searching efficiency (figure 5). Compensation of the host competition did not qualitatively change this result, but F_{crit} was quantitatively higher with over-compensating competition in the host. Over the range of parameter values

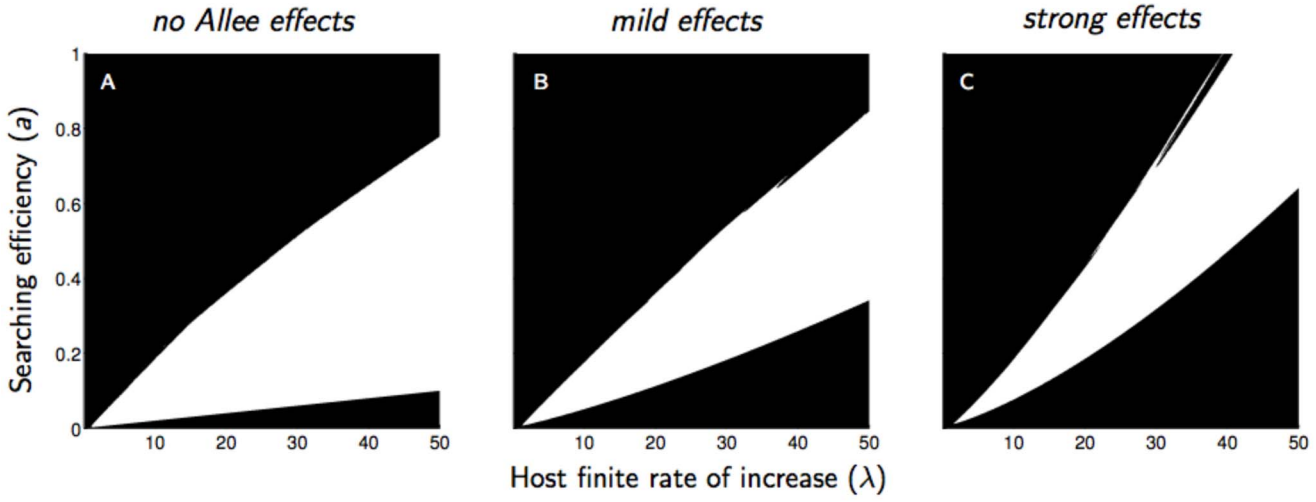

Figure 1. Domain of parasitoid extinction (black) and persistence (white) as a function of parasitoid searching efficiency (a) and host finite rate of increase (λ) for three intensities of a mate-finding Allee effect: no Allee effect ($\alpha = 0$), mild effects ($\alpha = 5$), strong effects ($\alpha = 20$). Other assumptions are a random distribution of parasitoid attacks ($k = 10$) and a medium intraspecific competition in the host population ($b = 1.5$). Two different domains of extinction can be distinguished: the upper domain corresponding to high parasitoid efficiency and low host finite rate of increase and the lower domain, corresponding to low parasitoid efficiency and high host finite rate of increase.
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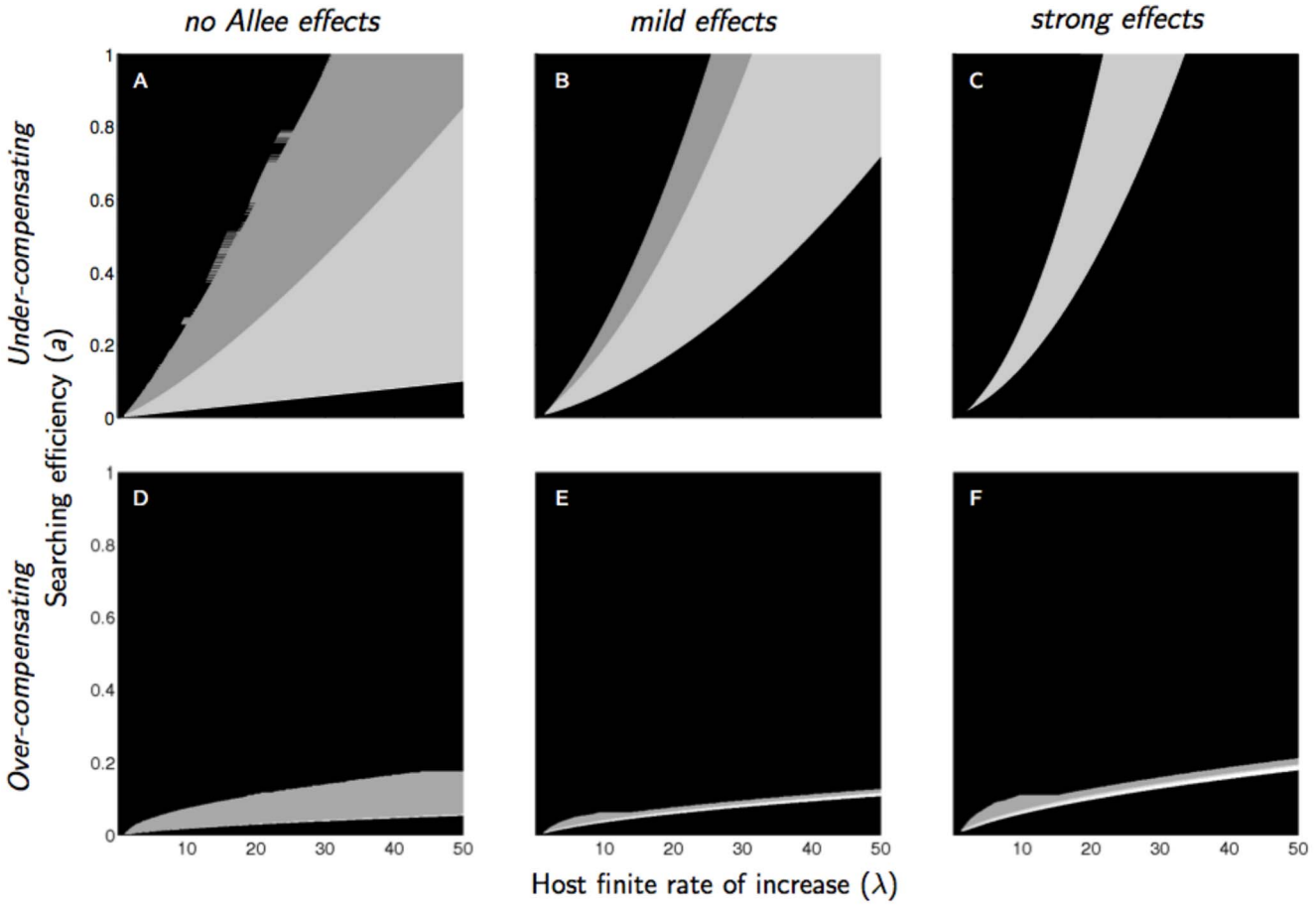

Figure 2. Domain of parasitoid extinction (black) and persistence (white and gray) as a function of parasitoid searching efficiency (a) and host finite rate of increase (λ) for three intensities of a mate-finding Allee effect (no Allee effect: $\alpha = 0$; mild effect: $\alpha = 5$; strong effect: $\alpha = 20$) and two levels of intraspecific competition in the host population (under-compensated competition: $b = 0.8$; over-compensated competition: $b = 3.2$). A random distribution of parasitoid attacks is assumed ($k = 10$). Parasitoid persistence is underpinned by qualitatively distinct dynamics; white: asymptotic stability; light gray: damped oscillations; dark gray: cycles or chaos.
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studied, F_{crit} reached its highest values for over-compensating host competition (for $\alpha = 20$, F_{crit} is about five times higher for $b = 3.2$ than for $b = 1.5$), and its lowest values for aggregated attacks (for $\alpha = 20$, F_{crit} is 80% lower for aggregated than for random attacks).

The host-parasitoid system had two local equilibria when an Allee effect was introduced: host-parasitoid coexistence and parasitoid extinction. In populations with under-compensating competition, close to the lower domain of extinction, F_{crit} increased with the Allee effect until it reached the coexistence equilibrium density: the conditions allowing the parasitoid to reach the equilibrium became more and more difficult to fulfill, and the parasitoid persistence became more and more vulnerable to a small decrease in density. Ultimately, the coexistence equilibrium became unstable and parasitoids were driven to extinction (figure 4.a). The ratio between F_{crit} and F^* provides a measure of the basins of attraction of coexistence and extinction equilibria: this ratio increased with the Allee effect, wich expresses a contraction of the basin of attraction for the coexistence equilibrium and an expansion of the basin of attraction for the parasitoid extinction.

Without Allee effect, the remaining host abundance after parasitism was naturally higher with over-compensating host competition, and reached its lowest levels - under 5% of the carrying capacity- with aggregated attacks (figure 6). Host abundance was generally increased by the Allee effect, even when it did not jeopardise the parasitoid persistence (figures 4 and 6). This increase was higher with under-compensating than with over-compensating host competition.

For the range of parameters considered in this study, an increase in α from 0 to 20 led to an increase of the host abundance between +6% and +116% (between +55% and +116% for under-compensating host competition, and between +6% and +33% for over-compensating host competition). An extreme increase in host abundance was observed for aggregated attacks and high searching efficiency parasitoids, as Allee effects lead the host to cycle at rates that could reach its carrying capacity (figures 4.c, 4.d, 6.c and 6.d).

Discussion

Despite the major economic and ecological importance of parasitoid insects, few studies have investigated the potential consequences of parasitoid mating failures on host-parasitoid population dynamics [6]. Here we demonstrate that (1) parasitoid populations can go extinct due to Allee effects, (2) extinctions are particularly expected for parasitoids with cyclic dynamics or stable dynamics and aggregated attacks, and (3) with Allee effects, severe intraspecific competition in the host population increases parasit-

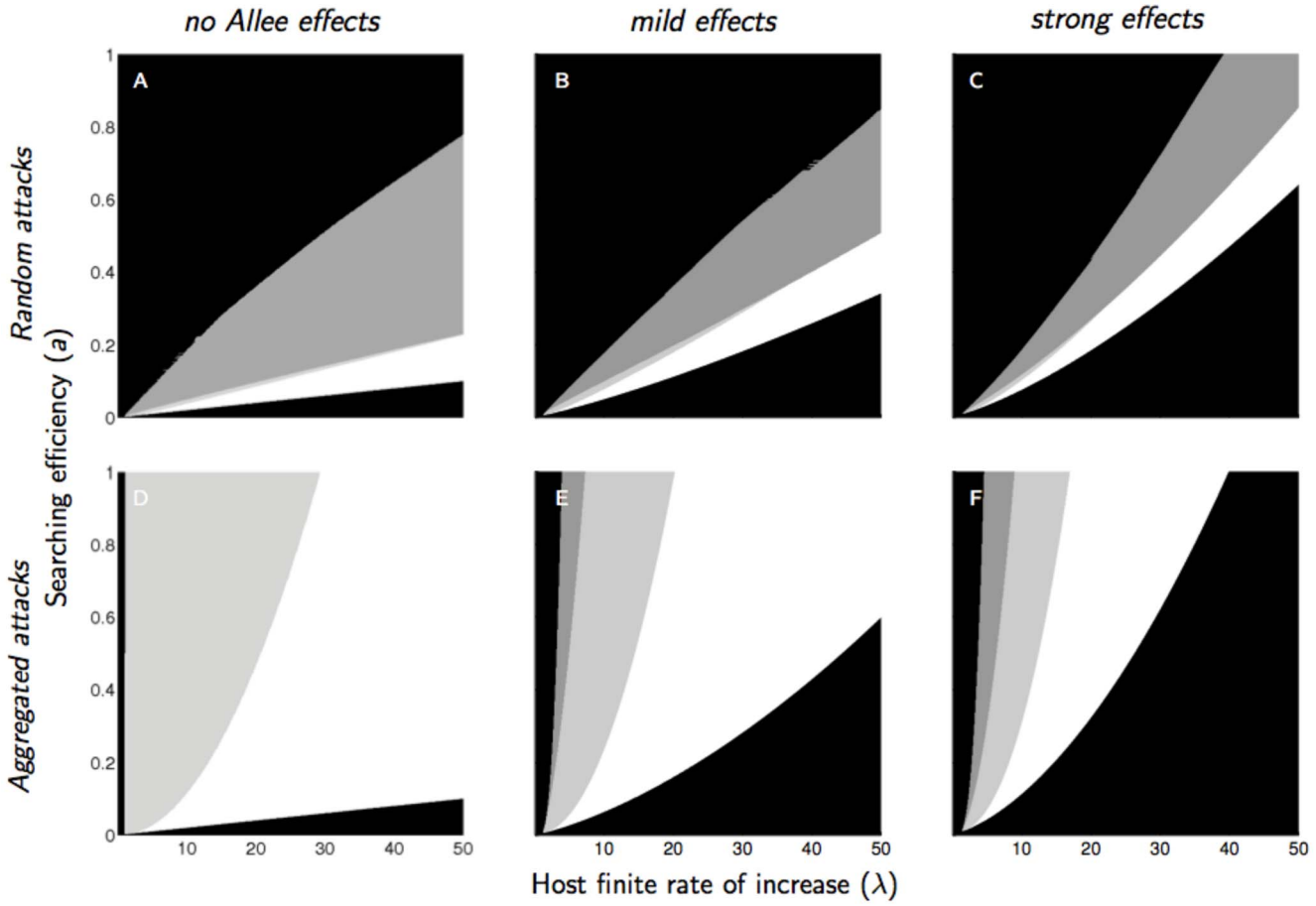

Figure 3. Domain of parasitoid extinction (black) and persistence (white and gray) as a function of parasitoid searching efficiency (a) and host finite rate of increase (λ) for three intensities of a mate-finding Allee effect (no Allee effect: $\alpha = 0$; mild effect: $\alpha = 5$; strong effect: $\alpha = 20$) and two distributions of parasitoid attack (random: $k = 10$; aggregated: $k = 0.8$). Intraspecific competition in the host population is assumed moderate ($b = 1.5$). Parasitoid persistence is underpinned by qualitatively distinct dynamics; white: asymptotic stability; light gray: damped oscillations; dark gray: cycles or chaos.
doi:10.1371/journal.pone.0076768.g003

oid risk of extinction due to external factors decreasing population densities. We also show that parasitoid control of the host population is reduced by Allee effects, and that parasitoids producing the highest levels of control are particularly susceptible to extinction. Hence, our theoretical results show that taking mate-finding Allee effects into consideration is crucial for predicting parasitoid establishment and the consequences of Allee effects on host-parasitoid dynamics depend, to a great extent, on other characteristics of the host-parasitoid interaction.

Parasitoid extinction and host-parasitoid dynamics with Allee effects

The Allee effect substantially increases the range of conditions prohibiting parasitoid persistence, and because it produces an extinction threshold, the Allee effect also increases the risk of parasitoid extinction by any external factor decreasing population density. An extinction threshold is commonly found in single-population models of Allee effects [8,14], as well as in models of interacting populations [35,36]. Our results suggest that in parasitoids, a mate-finding Allee effect at the individual level produces a strong demographic Allee effect at the population level. This implies that if the initial population size upon introduction is too small, or if once established population size decreases below a threshold, the population is doomed to extinction. The extinction threshold depends on the characteristics of both host and parasitoid populations: intrinsically stable host populations (through an under-compensating density dependence), high searching efficiency and aggregated parasitoid attacks lead to lower extinction thresholds, that is, higher resistance to external factor reducing density.

The conditions that allow parasitoids to persist and how they are modified by Allee effects are both linked to the host-parasitoid interaction. Introducing even moderate Allee effects into a host-parasitoid system with cyclical dynamics generally drives parasitoids to extinction. In the wild, very low densities should always produce mating failures, so that parasitoids with cyclical dynamics may be uncommon. Constantly, and in contrast with simple classical theoretical models, field observations suggest that cyclical parasitoid populations are rarely observed [2].

Much of our findings can be discussed from the perspective of parasitoid impact, that is, the relative influence of parasitism and intraspecific competition on the host dynamics. High-impact parasitism reflects a combination of parameters resulting in parasitoids driving most the host-parasitoid dynamics: (1) a large proportion of hosts parasitized, resulting from a high parasitoid seaching efficiency and/or a high parasitoid abundance, and (2) a moderate host intraspecific competition resulting from a high

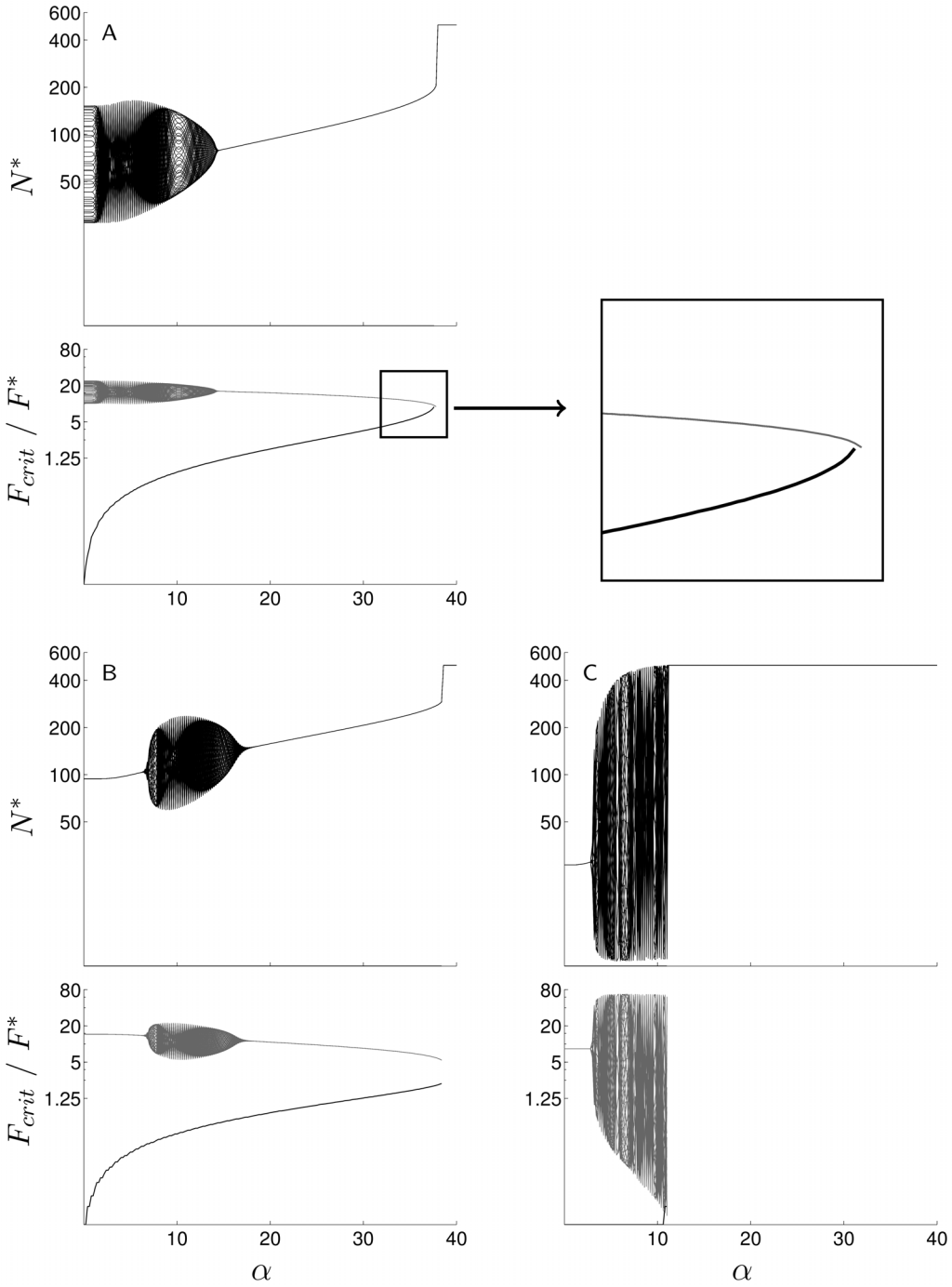

Figure 4. Bifurcation diagrams for hosts (black) and parasitoids (gray) populations, as a function of the intensity of a mate-finding Allee effects (α). Population characteristics: a) $k=10$, $b=1.5$, $a=0.14$, $\lambda=12.2$; b) $k=10$, $b=0.8$, $a=0.058$, $\lambda=4$; c) $k=0.8$, $b=1.5$, $a=0.25$, $\lambda=2.85$. The black curve on parasitoid graphics represents F_{crit}.
doi:10.1371/journal.pone.0076768.g004

carrying capacity, and/or a low host finite rate of increase, and/or a low compensation from intraspecific competition. Host and parasitoid coexist only for relatively moderated parasitism impacts: high-impact parasitism leads to cycles and parasitoid extinction (upper domain of extinction) while low-impact parasitism leads to asymptotic extinction of the parasitoid (lower domain of extinction).

Mating failure reduces parasitism impact because it reduces the number of females produced each generation and female density at equilibrium. The "effective" parasitism impact decreases with Allee effects but the conditions for parasitism impact allowing host and parasitoid coexistence remain the same, which results in the domain of parasitoid persistence shifting toward higher searching efficiency and lower host finite rate of increase (*i.e.*, to higher parasitism impacts). For a given Allee effect intensity, the proportion of unmated females is large at low male density, a situation which occurs mainly when hosts drive the system's dynamics and parasitism impact is low. Thus, the Allee effect

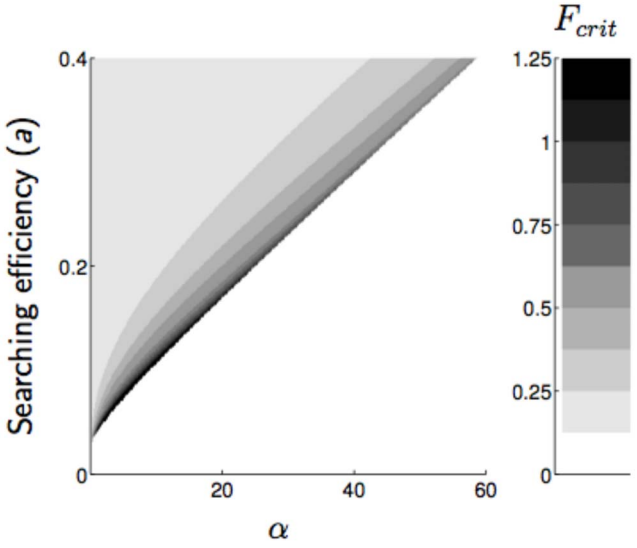

Figure 5. Minimum initial population density triggering parasitoid establishment (F_{crit}) as a function of parasitoid searching efficiency (a) and the intensity of Allee effects (α). Parameter values: $b = 1.5$, $k = 0.8$, $\lambda = 20$, $K = 500$.
doi:10.1371/journal.pone.0076768.g005

induces more reduction of the parasitism impact when it is already low, which in turn reduces the domain of parasitoid persistence. This mechanism shares some analogies with other processes leading to density-dependent reduction of the parasitism impact, such as parasitoid competition, autoparasitism, or host feeding on parasitized conspecifics [52,53,54]. While the mechanisms involved were different, these studies all pointed to a stabilizing effect on host-parasitoid dynamics. In his predator-resource model, Verdy [55] also observed stabilizing impact of the Allee effect at high predator growth rates, due to the effective reduction of the reproduction rate.

A different process may drive the parasitoid populations to extinction when attacks are aggregated or host competition is under-compensating, *i.e.*, when parasitoid populations persists despite high proportions of parasitized hosts. Without Allee effect, these populations reach stability through oscillatory damping; a mate-finding Allee effect reduces the production of female parasitoids at the bottom of the cycle (due to lower parasitoid density), which causes a release of parasitism pressure and an increase in host abundance. With a weak host competition, increased host abundance leads to increased production of female parasitoids at the top of the cycle; the existing oscillations are amplified and the parasitoid population eventually goes extinct. Severe host competition may conteract this destabilizing effect since it compensates for the increase in host abundance: the existing oscillations are not amplified and populations remain stable. This destabilizing effect is due to particular components of parasitoid growth rate (host abundance, which depends on parasitoid abundance). Similar process can also be found in predator-prey systems: destabilizing effects were indeed observed when Allee effects were introduced into the predator population in Zhou's predator-prey model [56] and in Verdy's predator-resource model [55].

Previous theoretical studies showed sex ratio variations, be they density dependent or not, affect the dynamics of haplodiploid parasitoids and may cause extinctions [45,46,57]. In haplodiploid populations, contrary to what occurs in diploids, mating failures induce sex ratio variations. Here, the model was developed for parasitoid wasps and we assumed haplodiploidy only. Nonetheless, comparisons between diploid and haplodiploid parasitoid systems may help to tackle the combined role of mating failures and sex ratio variations on the dynamics of host–parasitoid systems. Such comparisons are the scope of a forthcoming article.

The intensity of Allee effects

One prime ingredient of mate-finding Allee effects is the intensity of the mate-finding difficulties, represented in our study via a single parameter, α. Biologically, $\frac{1}{\alpha}$ represents the mate-searching efficiency of the species [11,24]. Various adaptations including volatile sex-pheromones and other signals generally emitted by females, as well as male cognitive and locomotory abilities, tend to increase mate-seaching efficiency; in contrast, stringent mate-choice may actually increase the time taken to find a suitable mate, and thus act as a reduction of $\frac{1}{\alpha}$ [24]. Some of the parasitoid adaptations for attracting mates (pheromone marking, reproductive aggregation) may be less efficient at low densities [24,58], making α density dependent. Moreover, female parasitoids sometimes display mate choice, tending to select non-kin individuals [59]. In small groups, males are more likely to be kin, and females may choose not to mate at all if they only encounter kin males [60,61]; in our model this would be seen as a relative increase in α.

In natural populations, the proportion of parasitoid females found to be virgin or sperm-depleted ranges from 1% to 50% [31,39,62,63,64,65]. With the parameter values considered in this study, and the subsequent male densities at equilibrium (around 5 to 40), this proportion of virgin females is equivalent to an α value ranging from 1 to 30.

The framework of biological control

The ability of parasitoids to reduce host abundance, a proxy of which is the proportion of parasitized hosts, is a major criterion for biological control. Successful biological control, leaving less than 5% of the original host population, has been observed following field parasitoid introductions [4,66]. Our model suggests that stability at this range of reduction can be obtained by considering aggregated attacks, as in the Beddington et al. model [66]. Aggregated attacks have been documented repeatedly in parasitoids, with values of k ranging from 0.5 to 1.6 [48,67,68,69,70]. Indeed, successful biological control has been achieved with highly aggregating species such as *Cyzenis albicans* and *Anagrus spp.*[67,68,71].

Our model suggests that parasitoids with aggregated attacks and a high proportion of parasitized host will be destabilized by Allee effects: a small reduction in parasitoid mate-searching ability may lead to either uncontrolled host abundances or to parasitoid extinction. If, in addition, hosts suffer strong intraspecific competition, the threshold for parasitoid extinction should be higher: parasitoids would have to be introduced at higher initial densities, and should then be more vulnerable to extrinsic factors decreasing population abundance; such factors are obviously common in agricultural landscapes.

As a conclusion, good candidates for biological control should be particularly sensitive to Allee effects. As suggested by Stiling [72], the establishment of a parasitoid population with Allee effects may depend on the characteristics of the host population. However, to fully link the results of our model to biological control, new empirical studies and meta-analyses of parasitoid introductions are necessary. If our interpretations hold true, special care with the following aspects should be taken when

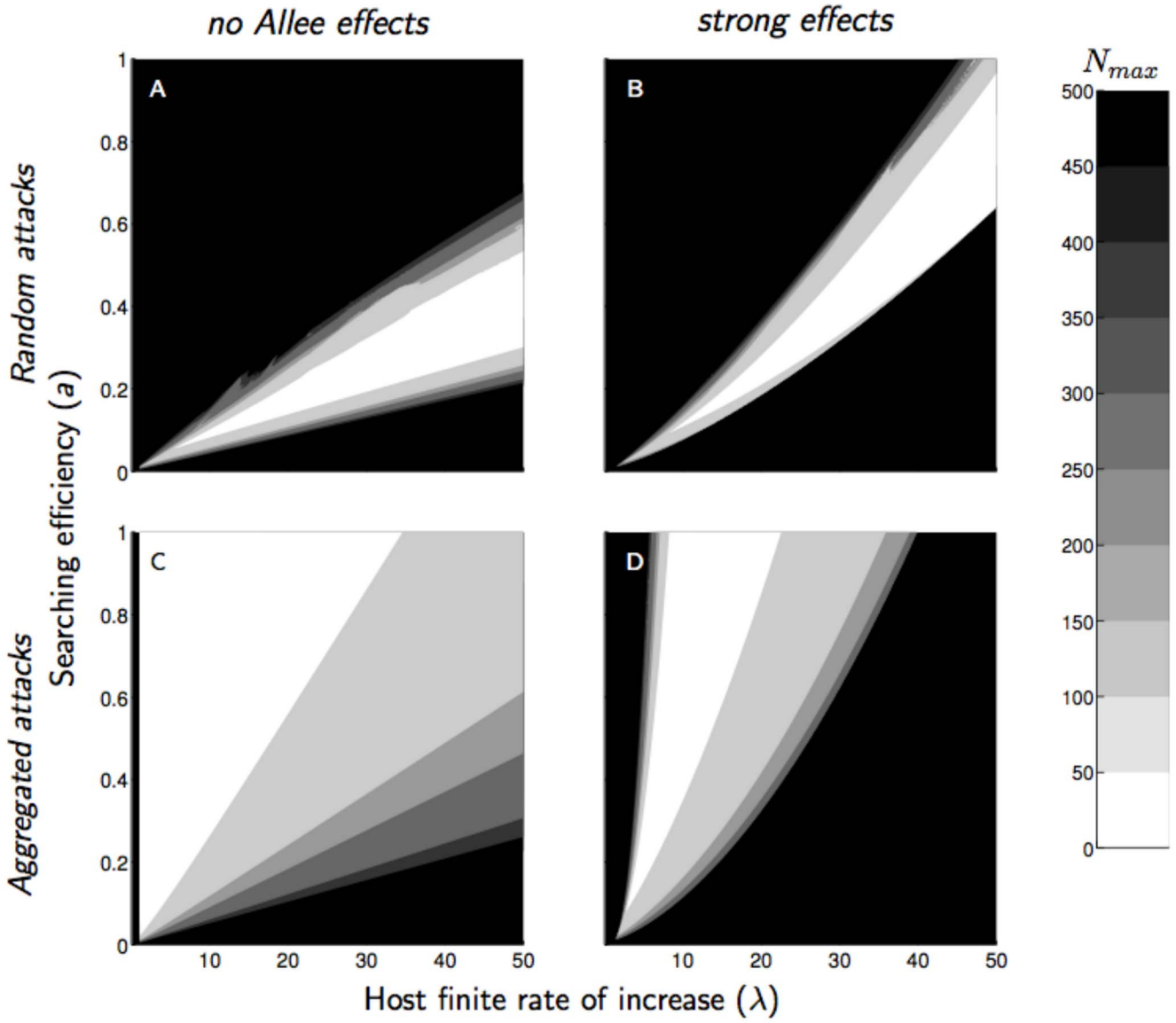

Figure 6. Residual host abundance after parasitism (N_{max}) without ($\alpha = 0$) and with ($\alpha = 20$) a strong mate-finding Allee effect and either a random ($\kappa = 10$) or aggregated ($\kappa = 0.8$) distribution of attacks, as a function of parasitoid searching efficiency (a) and host finite rate of increase (λ). Intraspecific competition in the host population was assumed medium ($b = 1.5$)
doi:10.1371/journal.pone.0076768.g006

parasitoids are introduced: 1) parasitoid mating strategies (will their mate-searching ability be modified in a new environment?), 2) the characteristics of parasitoid dynamics and parasitoid attacks (what would be the consequences of a small decrease in the parasitoid mate-searching ability?), and 3) the nature of the host density dependence, in relation to the density of parasitoids that needs to be introduced and the environmental changes to which parasitoids may be subjected (will their density remain above the extinction threshold?).

Supporting Information

Figure S1 Analytical results for the stability of the one-sex host-parasitoid model. The proportion of host abundance remaining with parasitism ($q = \frac{N^*}{K}$) and the host growth rate (r) are plotted for three levels of intraspecific competition in the host population and three distribution of parasitoid attacks. From left to right: the host is stable with exponential damping ($b = 0.8$), stable with asymptotic damping ($b = 1.5$), and unstable ($b = 3.2$); from top to bottom: random attacks ($k = 10$), medium aggregation ($k = 1.5$), and strong aggregation ($k = 0.8$). Some key values of q are highlighted: plain line: 0.8, dashed line: 0.5, dashed-dotted line: 0.3, dotted line: 0.05.
(TIF)

Text S1 Analytical results.
(DOC)

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Author Contributions

Conceived and designed the experiments: AB IA TS. Performed the experiments: AB. Analyzed the data: AB. Wrote the paper: AB IA XF TS.

References

1. Nicholson AJ, Bailey VA (1935) The balance of animal populations. P Zool Soc Lond 3: 551–593.
2. Hassell MP (2000) Host-parasitoid population dynamics. J Anim Ecol, 69: 543–566.
3. Bernstein C (2000) Host-parasitoid models: the story of a successful failure. *in* Hochberg ME and Ives AR Parasitoid population biology.Princeton University Press, Princeton. pp. 41–57.
4. Bellows T (2001) Restoring population balance through natural enemy introduction. Biol Control, 21: 199–205.
5. Hall RW, Ehler LE (1979) Rate of establishment of natural enemies in classical biological control. Bull Entomol Soc Am, 25: 280–283.
6. Van Lenteren JC (1983) The potential of entomophagous parasites for pest control. Agr Ecosyst Environ, 10: 143–158.
7. Stiling P (1993) Why do natural enemies fail in classical biological control programs? Am Entomol, 39: 31–37.
8. Hopper KR, Roush RT (1993) Mate finding, dispersal, number released, and the success of biological control introductions. Ecol Entomol, 18: 321–331.
9. Taylor CM, Hasting A (2005) Allee effects in biological invasions. Ecol Lett, 8: 895–908.
10. Liebhold AM, Tobin PC (2008). Population ecology of insect invasions and their management. Annu Rev Entomol, 53: 387–408.
11. Fauvergue X (2013) A review of mate-finding Allee effects in insects: from individual behavior to population management. Entomol Exp Appl, 146: 79–92.
12. Boukal DS, Berec L (2002) Single-species Models of the Allee Effect: Extinction Boundaries, Sex Ratios and Mate Encounters. J Theor Biol, 218: 375–394.
13. Dennis B (1989) Allee effects: population growth, critical density, and the chance of extinction. Nat Resour Model, 3: 481–538.
14. Courchamp F, Clutton-Brock T, Grenfell B (1999) Inverse density dependence and the Allee effect. Trends Ecol Evol, 14: 405–410.
15. Kramer AM, Dennis B, Liebhold AM, Drake JM (2009) The evidence for Allee effects. Popul Ecol, 51: 341–354.
16. Grevstad FS (1999) Experimental invasions using biological control introductions: the influence of release size on the chance of population establishment. Biol Invasions, 1: 313–323.
17. Stephens PA, Sutherland WJ (1999) Consequences of the Allee effect for behaviour, ecology and conservation. Trends Ecol Evol, 14: 401–405.
18. Tobin PC, Berec L, Liebhold AM (2011) Exploiting allee effects for managing biological invasions. Ecol Lett, 14: 615–624.
19. Courchamp F, Berec L, Gascoigne J (2008) Allee effects in ecology and conservation. Oxford University Press, Oxford.
20. Gascoigne J, Berec L, Gregory S, Courchamp F (2009) Dangerously few liaisons: a review of mate-finding Allee effects. Popul Ecol, 51: 355–372.
21. Contarini M, Onufrieva KS, Thorpe KW, Raffa KF, Tobin PC (2009) Mate-finding failure as an important cause of Allee effects along the leading edge of an invading insect population. Entomol Exp Appl, 133: 307–314.
22. Tobin PC, Robinet C, Johnson DM Whitmire SL, Bjornstad ON, et al. (2009) The role of Allee effects in gypsy moth, *Lymantria dispar* (L.), invasions. Popul Ecol, 51: 373–384.
23. Tobin PC, Whitmire SL, Johnson DM, Bjornstad ON, Liebhold AM (2007) Invasion speed is affected by geographical variation in the strength of Allee effects. Ecol Lett 10: 36–43.
24. Blackwood JC, Berec L, Yamanaka T, Epanchin-Niell RS, Hastings A, et al. (2012) Bioeconomic synergy between tactics for insect eradication in the presence of Allee effects. P Roy Soc B-Biol Sci, 279: 2807–2815.
25. Tobin PC, Onufrieva KS, Thorpe KW (2013) The relationship between male moth density and female mating success in invading populations of *Lymantria dispar*. Entomol Exp App 146: 103–111.
26. Beddington J, Cook J (1982) Harvesting from a prey-predator complex. Ecol Model, 14: 155–177.
27. Tscharntke T, Klein A, Kruess A, Steffan-Dewenter I, Thies C (2005) Landscape perspectives on agricultural intensification and biodiversity-ecosystem service management. Ecol Lett, 8: 857–874.
28. Rusch A, Valantin-Morison M, Sarthou JP, Roger-Estrade J (2011) Multi-scale effects of landscape complexity and crop management on pollen beetle parasitism rate. Landscape Ecol, 26: 473–486.
29. Fauvergue X, Hopper KR (2009) French wasps in the New World: experimental biological control introductions reveal a demographic Allee effect. Popul Ecol, 51: 385–397.
30. Engelkes T, Mills NJ (2011) A conceptual framework for understanding arthropod predator and parasitoid invasions. Biol Control, 56: 383–393.
31. Fauvergue X, Malausa JC, Giuge L, Courchamp F (2007) Invading parasitoids suffer no Allee effect: a manipulative field experiment. Ecology, 88: 2392–2403.
32. May RM, Hassell MP, Anderson RM, Tonkyn DW (1981) Density-dependence in host-parasitoid models. J Anim Ecol, 50: 855–865.
33. Beddington J, Free CA, Lawton JH (1976) Concepts of stability and resilience in predator-prey models. J Anim Ecol, 45: 791–816.
34. Liu H, Li Z, Gao M, Dai H, Liu Z (2009) Dynamics of a host-parasitoid model with Allee effect for the host and parasitoid aggregation. Ecol Complex, 6: 337–345.
35. Deredec A, Courchamp F (2006) Combined impacts of Allee effects and parasitism. Oikos, 112: 667–679.
36. Jang SRJ (2006) Allee effects in a discrete-time host-parasitoid model. J Differ Equ Appl, 12: 165–181.
37. Heimpel GE, de Boer JG (2008) Sex determination in the Hymenoptera. Annu Rev Entomol, 53: 209–230.
38. Eggleton P, Belshaw R (1992) Insect parasitoid: an evolutionary overview. Philos T Roy Soc B, 337: 1–20.
39. Ode PJ, Antolin MF, Strand MR (1997) Constrained oviposition and female-biased sex allocation in a parasitic wasp. Oecologia, 109: 547–555.
40. Fauvergue X, Hopper KR, Antolin MF, Kazmer DJ (1998) Does time until mating affect progeny sex ratio? A manipulative experiment with the parasitoid wasp *Aphelinus asychis*. J Evolution Biol, 11: 611–622.
41. Hardy ICW, Pedersen JB, Sejr MK, Linderoth UH (1999) Local mating, dispersal and sex ratio in a gregarious parasitoid wasp. Ethology, 105: 52–72.
42. Fauvergue X, Lo Genco A, Lo Pinto M (2008) Virgins in the wild: mating status affects the foraging behavior of a parasitoid foraging in the field. Oecologia, 156: 913–920.
43. Perez-Lachaud G (2010) Reproductive costs of mating with a sibling male: sperm depletion and multiple mating in *Cephalonomia hyalinipennis*. Entomol Exp Appl, 137: 62–72.
44. Boukal DS, Berec L (2009) Modelling mate-finding allee effects and populations dynamics, with applications in pest control. Popul Ecol, 51: 445–458.
45. Comins HN, Wellings PW (1985) Density-Related Parasitoid Sex-Ratio: Influence on Host-Parasitoid Dynamics. J Anim Ecol, 54: 583–594.
46. Hassell MP, Waage JK, May RM (1983) Variable parasitoid sex ratio and their effect on host-parasitoid dynamics. J Anim Ecol,52: 889–904.
47. Meunier J, Bernstein C (2002) The influence of local mate comptetition on host-parasitoid dynamics. Ecol Model, 152: 77–88.
48. Bessa-Gomes C, Legendre S, Clobert J (2004) Allee effects, mating systems and the extinction risk in populations with two sexes. Ecol Lett, 7: 802–812.
49. May RM (1978) Host-Parasitoid Systems in Patchy Environments: A Phenomenological Model. J Anim Ecol, 47: 833–843.
50. Maynard Smith J, Slatkin M (1973) The stability of predator-prey systems. Ecology, 54: 384–391.
51. Bellows T (1981) The descriptive properties of some models for density dependence. J Anim Ecol, 50: 139–156.
52. Briggs C (1993) Competition among parasitoid species on a stage-structured host and its effects on host suppression. Am Nat, 141: 372–397.
53. Briggs CJ, Collier TR (2001) Autoparasitism, interference, and parasitoid-pest population dynamics. Theor Popul Biol, 60: 33–57.
54. Collier T, Hunter M (2001) Lethal interference competition in the whitefly parasitoids, *Eretmocerus eremicus* and *Encarsia sophia*. Oecologia 129: 147–154.
55. Verdy A (2010) Modulation of predator-prey interactions by the Allee effect. Ecol Model, 221: 1098–1107.
56. Zhou SR, Liu YF, Wang G (2005) The stability of predator-prey systems subject to the Allee effects. Theor Popul Biol, 67: 23–31.
57. Wogin MJ, Gillespie DR, Haye T, Roitberg BD (2012) Female-biased sex ratio shifts in a solitary parasitoid and their effects on virginity, population dynamics, and biological control. Entomol Exp Appl 146: 165–176.
58. Fauvergue X, Hopper K, Antolin M (1995) Mate-finding via a trail sex pheromone by a parasitoid wasp. Proc Natl Acad Sci USA, 92: 900–904.
59. Metzger M, Bernstein C, Hoffmeister TS, Desouhant E (2010) Does kin recognition and sib-mating avoidance limit the risk of genetic incompatibility in a parasitic wasp? PLoS ONE, 5(10): e13505. doi:10.1371/journal.pone.0013505.
60. Moller AP, Legendre S (2001) Allee effect, sexual selection and demographic stochasticity. Oikos, 92: 27–34.
61. Kokko H, Rankin D (2006) Lonely hearts or sex in the city? Density-dependent effects in mating systems. Philos Trans R Soc Lond B Biol Sci, 361: 319–334.
62. Godfray HCJ (1988) Virginity in haplodiploid populations: a study on fig wasps. Ecol Entomol, 13: 283–291.
63. Hardy ICW, Mayhew P (1998) Partial local mating and the sex ratio: indirect comparative evidence. Trends Ecol Evol, 13: 431–432.
64. Kapranas A, Hardy ICW, Morse JG, Luck RF (2011) Parasitoid developmental mortality in the field: patterns, causes and consequences for sex ratio and virginity. J Anim Ecol, 80: 192–203.
65. Metzger M, Bernstein C, Desouhant E (2008) Does constrained oviposition influence offspring sex ratio in the solitary wasp *Venturia canescens*? Ecol Entomol, 33: 167–174.
66. Beddington J, Free CA, Lawton J (1978) Characteristics of successful natural ennemies in models of biological control of insect pests. Nature, 273: 513–519.

67. Parejaa M, Brown VK, Powell W (2008) Aggregation of parasitism risk in an aphid-parasitoid system: Effects of plant patch size and aphid density. Basic Appl Ecol, 9: 701–708.
68. Cronin JT (2009) Habitat edges, within-patch dispersion of hosts, and parasitoid oviposition behavior. Ecology, 90: 196–207.
69. Henne DC, Johnson SJ (2010) Laboratory evaluation of aggregation, direct mutual interference, and functional response characteristics of *Pseudacteon tricuspis Borgmeier* (Diptera: Phoridae). Biol Control, 55: 63–71.
70. Li J, Seal DR, Leibee GL, Liburd OE (2012) Seasonal abundance and spatial distribution of the leafminer, liriomyza trifolii (Diptera: Agromyzidae), and its parasitoid, opius dissitus (Hymenoptera: Braconidae), on bean in southern Florida. Fla Entomol, 95: 128–135.
71. Embree D (1966) The role of introduced parasites in the control of the winter moth in Nova Scotia. Can Entomol, 98: 1159–1168.
72. Stiling P (1990) Calculating the establishment rate of parasitoids in classical biological control. Am Entomol, 36: 225–230.

Text S1. Analytical Results.

The one-sex model

Conditions of stability

We defined the parameter q as the extent to which the host equilibrium (N^*) is depressed below its carrying capacity K [33].

$$q = \frac{N^*}{K} \quad (8)$$

This parameter decreases with the searching efficiency, a, as parasitoids with a high searching efficiency tend to reduce their host populations to a greater extent; it increased with b, $\frac{1}{K}$ and the host intrisic growth rate $r = (\lambda - 1)^{\frac{1}{b}}$, as a high level of density dependence compensated for the mortality due to the parasitoid. The equilibrium can be expressed as a function of the q parameter:

$$\begin{cases} P^* = \frac{k}{a}\left(\frac{1+(\lambda-1)q^b}{\lambda}\right)^{\frac{1}{k}-1} \\ N^* = \frac{P^*}{d\left(1+(\lambda-1)q^b\right)^{-1} - \frac{d}{\lambda}} \end{cases} \quad (9)$$

The conditions required for the stability of the model were explored through a linearized stability analysis using the method of May (1974).

The Jacobian matrix of this set of equations is as follows:

$$Jac = \begin{Bmatrix} 1-b-\frac{b}{1+(\lambda-1)q^b} & -\frac{c}{d}\frac{1-\left[\lambda^{-1}\left(1+(\lambda-1)q^b\right)\right]^{\frac{1}{c}}}{\left(1+(\lambda-1)q^b\right)^{-1}-\frac{1}{\lambda}} \\ \frac{d}{\lambda}\left[1-b-\frac{b}{1+(\lambda-1)q^b}\right]\left[\frac{\lambda}{1+(\lambda-1)q^b}-1\right] & \frac{d}{\lambda}\left[\frac{c}{d}\frac{1-\left[\lambda^{-1}\left(1+(\lambda-1)q^b\right)\right]^{\frac{1}{c}}}{\left(1+(\lambda-1)q^b\right)^{-1}-\frac{1}{\lambda}}\right] \end{Bmatrix}$$

(10)

We set $A = 1 - b - \frac{b}{1+(\lambda-1)q^b}$ and $B = -\frac{c}{d}\frac{1-\left[\lambda^{-1}\left(1+(\lambda-1)q^b\right)\right]^{\frac{1}{c}}}{\left(1+(\lambda-1)q^b\right)^{-1}-\frac{1}{\lambda}}$

Hosts and parasitoids reach a stable equilibrium if, and only if:

$$A < 2 - \sqrt{\frac{d}{\lambda}AB\left(\frac{d}{\lambda}AB + 4\lambda\left(1+(\lambda-1)q^b\right)^{-1} - 2\right)} - \frac{d}{\lambda}B \quad (11)$$

Results

With b=2, the Maynard Smith density dependent function of the host gives way to a Mann Ricker function. Our model then becomes very similar to May et al. (1981)'s model [32].

Four distinct regions can be described, as the equilibrium is either asymptotically stable (black), reached by oscillatory damping (dark gray), unstable with oscillations (light gray), or corresponds to asymptotic extinction of the parasitoid (white) (supplementary figure 1). Stability is reached for intermediate values of q, and decreases with r.

For $b < 1$, the competition is under-compensating and hosts alone are asymptotically stable. Thus, at low values of host depression, the host dynamics control the system, and are always asymptotically stable. For $1 < b < 2$, hosts become stable with oscillatory damping, and the dynamics of the model do not vary much. However, for $2 < b$, the competition is over-compensating, with hosts that are unstable alone: at low host depression, they result in asymptotic extinction of the parasitoid. The more unstable the host, the less likely the parasitoid is to reach a non-null equilibrium without strong depression of the host population. The b parameter controls the

stability of the model particularly at high values of q when the host population controls the dynamics.

Heterogeneity in parasitoid attacks (small values of k) increases the stability region at low values of q. For small values of q and of r, unstable oscillations give way to oscillatory damping, whereas oscillatory damping gives way to exponential damping. This finding parallels those reported in several previously published studies [32, 49]. For $k<1$, there is no unstable oscillations mode. When parasitoid attacks control the dynamics, the clamping parameter, k, controls the stability at low values of q.

The regions of action of parameters b and k do not coincide. For b and $k<1$, the host parasitoid system is always stable.

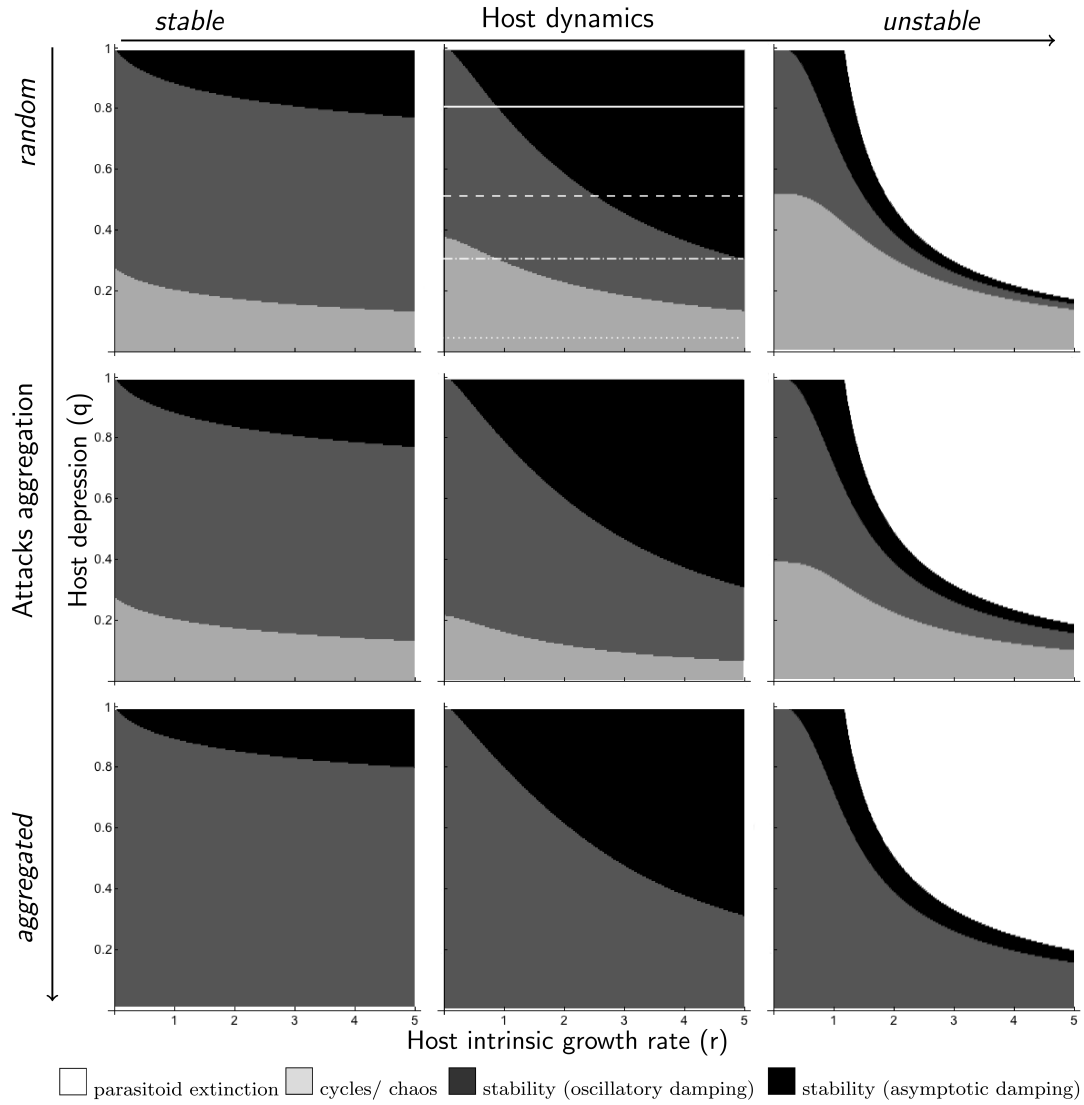

Figure 2.1: Analytical results for the stability of the one-sex host-parasitoid model. The proportion of host abundance remaining with parasitism ($q = \frac{N^*}{K}$) and the host growth rate (r) are plotted for three levels of intraspecific competition in the host population and three distribution of parasitoid attacks. From left to right: the host is stable with exponential damping ($b = 0.8$), stable with asymptotic damping ($b = 1.5$), and unstable ($b = 3.2$); from top to bottom: random attacks ($k = 10$), medium aggregation ($k = 1.5$), and strong aggregation ($k = 0.8$). Some key values of q are highlighted: plain line: 0.8, dashed line: 0.5, dashed-dotted line: 0.3, dotted line: 0.05.

CHAPTER

3 Extinction proneness of diploid *vs.* haplodiploid parasitoid populations face to mate finding difficulties

Article 3

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Résumé

L'haplodiploïdie est un système de reproduction dans lequel les mâles peuvent être produits par parthénogenèse. Cette caractéristique est supposée assurer une relative immunité face aux effets Allee comportementaux - c.à.d l'impact sur le taux de reproduction de difficultés d'accouplement apparaissant à faible densité -, puisque les femelles vierges restent capables de produire des mâles. L'étendue de cette immunité a rarement été quantifiée. Nos récents travaux sur l'impact théorique des effets Allee comportementaux chez les parasitoïdes haplodiploïdes montrent que les parasitoïdes sont très sujets à l'extinction quand de tels effets apparaissent. Ainsi, on peut se demander si l'haplodiploïdie apporte effectivement un bénéfice quelconque aux parasitoïdes dans cette situation.
Dans cet article, nous utilisons la modélisation afin d'établir une comparaison des risques d'extinction des parasitoïdes diploïdes et haplodiploïdes quand ils souffrent d'un effet Allee comportemental. Premièrement, nous examinons pour des parasitoïdes des deux modes de reproduction l'étendue du domaine d'extinction, la taille critique de la population lors d'une introduction et la résistance à la stochasticité environnementale. Nous envisageons la possibilité pour les femelles haplodiploïdes de biaiser le sex ratio de leur descendance lorsqu'elle sont accouplées, soit en réponse à la pression évolutive d'un effet Allee comportemental constant, soit en s'adaptant à la proportion de femelles vierges dans leur génération. Deuxièmement, nous considérons pour des parasitoïdes des deux types de reproduction leurs capacités d'invasion sur un système hôte - parasitoïde établit et leur résilience à l'invasion par un autre parasitoïde. Nous montrons que les caractéristiques de l'interaction hôte - parasite sont les principaux déterminants de l'étendue du domaine d'extinction du parasitoïde avec ou sans effet Allee. Cependant, les parasitoïdes haplodiploïdes ont une taille de population critique inférieure et une meilleure résistance à la stochasticité environnementale ; en outre, leurs capacités invasives et leur résilience aux invasions sont moins altérées

par les effets Allee que celles des parasitoïdes diploïdes. La capacité des femelles haplodiploïdes à biaiser le sex ratio de leur descendance n'a que très peu d'impact sur les conséquences démographiques d'un effet Allee, et devraient donc être principalement considérées comme des avantages en terme de fitness. Nous discutons de nos résultats en relation avec les introductions de parasitoïdes et les effets non - cibles en lutte biologique.

Abstract

Haplodiploidy is a reproductive system in which males can be produced by parthenogenesis. This feature is usually assumed to ensure a relative immunity to mate-finding Allee effects, but the extent of this immunity has rarely been quantified. In a previous work on the theoretical impact of mate-finding Allee effects in haplodiploid parasitoids, we have shown that parasitoids are highly extinction -prone when facing such Allee effects. Thus, one may wonder whether haplodiploidy is indeed beneficial in this case.
In this paper, we use theoretical modeling to set up a comparison of diploid and haplodiploid parasitoid extinction risks when they are prone to mate-finding difficulties. First, we investigate the extent of the sets of condition driving the two reproductive types to extinction, their critical size for introduction and their resistance to environmental stochasticity. Second, we consider for the two reproductive types the invasive abilities on an established host-parasitoid system and resilience to another parasitoid invader. We show that the host-parasitoid interaction mainly determines the extent of parasitoid extinction domain with or without Allee effect. However, haplodiploid parasitoids have a lower critical population size and a better resistance to environmental stochasticity; moreover, their invasive abilities and resilience to invasions are less impaired by Allee effects than that of diploid parasitoids. We discuss our results in regard with introductions and non-target effects in biological control.

KEYWORDS: Allee effects; host-parasitoid interaction; diploidy ; haplodiploidy; parasitoid competition; modeling; introduction; arrhenotoky; biological control; non-target effects; pest; mate-finding difficulties; extinction

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1. Introduction

Assessing the proneness of small populations to extinction (due to genetic and/or demographic processes) and characterizing the factors that may counter this phenomenon form the core of conservation biology. Insect parasitoid populations may dramatically decline or end up at small density *e.g.* due to the demographic constrain that hosts impose on them, when they are invasive or purposely introduced for pest management, or as threatened species. In either case, finding mate may prove difficult (23 % of females remain virgin in natural populations(Godfray, 1990)). Such difficulties in mate finding may give rise to a decrease of individual fitness when density decreases. This mate-finding Allee effect may lead to a demographic Allee effect, *i.e.* a decrease of population growth rate when population density decreases, even leading to the emergence of a critical density below witch growth rate becomes negative and populations turn out to be doomed to extinction (Courchamp et al., 2008).

Modes of sex determination may interfere with how mate-finding difficulties translate in demographical damages in parasitoid populations. The majority of parasitoid species (78%) belongs to the Hymenoptera order. While alternatives are possible (thelytoky or paternal genome elimination), the predominant mode of sex determination in Hymenoptera is arrhenotoky, in which haploid males are produced by parthenogenesis and diploid females develop from fertilized eggs (Heimpel and de Boer, 2008). When unmated, hymenopteran parasitoid females may remain able to lay most of their eggs, but not to fertilize them: all eggs develop as male (Godfray, 1994). About 20% of parasitoid species belong to the Diptera order. These are diploid organisms for which unmated females cannot produce offspring. While mate finding difficulties may strongly depress individual fitness of dipteran parasitoid females, they may (only) lead to male biased sex ratio in haplodiploid organisms.

Haplodiploid organisms are considered as rather immune to the mate-finding Allee effect because the production of males by virgin females reduces mate finding difficulties the following generation (Hopper, 1993). Moreover, Godfray (1990) suggested that when parasitoid face constant mate-finding difficulties, females with a higher fertilization rate should be selected and the population fertilization rate should increase to counter the sex ratio bias induced by virgin females. This fertilization bias has been observed in numerous parasitoid populations (Antolin and Strand, 1992; Heimpel, 1997; Ode and Hardy, 1995). Another suggestion is that mated haplodiploid females could adjust their fertilization rate to environmental clues (such as time delay between emergence and mating, number of encounters with a male, etc), immediately compensating for the sex ratio bias induced by virgin females (Godfray, 1990). This hypothesis is equivocally supported by empirical studies (King, 2002; Fauvergue et al., 2008). Consequently, with haplodiploidy and adjusted fertilization rate, the damages of the mate-finding Allee effect are believed to be insignificant. A theoretical investigation of the impact of mating difficulties on introduced hymenopteran parasitoids - the host population being fixed at its carrying capacity - showed that haplodiploidy decreases by 30% the critical female population size (Hopper, 1993) in comparison with diploid parasitoids.

The interaction the parasitoid shares with its host may impact the demographical conse-

quences of mate finding difficulties (article 2), and this impact may depend on the mode of sex determination. First, variations in parasitoid sex ratio resulting from mate-finding Allee effect in haplodiploids may affect parasitoid dynamics because only females interact with the host. Second, parasitoid growth is dependent of host availability and haplodiploid virgin females use up hosts to produce all-male clutches while virgin diploid females do not (as they have no eggs to lay). Consequently, the relative extinction propensity of haplodiploid and diploid parasitoid populations when faced with mate finding difficulties is less easy to foresee when the host parasitoid interaction is considered. Our study of the demography of haplodiploid parasitoids prone to mate finding Allee effects (article 2) revealed a complex pattern of extinction involving interactions between demographic parameters of both hosts and parasitoids. Unlike expectations for haplodiploid organisms, a general result is that mate-finding difficulties globally increase the risk of parasitoid extinction. However, two questions need to be get into in depth: is haplodiploidy notwithstanding beneficial in comparison with diploidy (*i.e.* by reducing extinction propensity or increasing invasive success) and does the flexibility in sex allocation enabled by haplodiploidy preserves haplodiploid populations from demographical damages due to mating failures.

A first aim of this study is to clarify the role of the mode of sex determination on extinction propensity of parasitoid populations coping mate-finding difficulties. For that aim, we seek to answer two main questions: 1) are haplodiploid populations less vulnerable than diploid populations when jeopardized by mate finding difficulties, and 2) does the ability of haplodiploid females to strongly bias or even adjust sex allocation decreases the extinction risk induced by mate finding difficulties. To address these questions, we investigate the impact of mode of sex determination on the extent of the parameter domain where populations are ineluctably driven to extinction, and on the robustness of the remaining populations to an introduction in small numbers and to external causes of mortality.

A second aim of this study is to determine the impact of the mate-finding Allee effect on the relative invasive success of haplodiploid and diploid parasitoids on an established host-parasitoid system. Through multiple invasion simulations, we investigate how the mate-finding Allee effect may affect diploid and haplodiploid ability to survive to an invading parasitoid and to invade an established system.

We conclude that when facing mate-finding difficulties, haplodiploid parasitoids can settle in smaller numbers, have a better resistance to external causes of mortality, are more resilient to invasions and more likely to invade an established host-parasitoid system than diploid parasitoids. Nevertheless, haplodiploidy did not make parasitoid immune to the consequences of the mate-finding Allee effect. Unlike expectations, an elevated fertilization rate or an adjusted sex ratio only yield a negligible impact on the features of robustness of parasitoid populations.

2. Methods

The deterministic two-sexe model of article 2 was adapted to account for both diploid and haplodiploid reproductions in order to compare the impact of mate finding Allee effect on the

extinction proneness according to the sex determination system. In this model we considered the host population (N), parasitoid females (F) and parasitoid males (M).

2. 1 Dynamics of diploid and haplodiploid parasitoids.

In haplodiploid parasitoids, all females attack the host at the same rate, whenever they are mated or not. However, the whole virgin female offspring consists in males.
The model equations are:

$$\begin{cases} N_{t+1} = \lambda N_t g(N_t) f(F_t) \\ F_{t+1} = (1-s_0)\left(1-p(M_t)\right) N_t g(N_t)\left(1-f(F_t)\right) \\ M_{t+1} = \left(p(M_t) + s_0\left(1-p(M_t)\right)\right) N_t g(N_t)\left(1-f(F_t)\right) \end{cases} \tag{3.1}$$

where $p(M_t)$ expresses the proportion of virgin females, s_0 the fraction of unfertilized eggs among offspring of mated female, λ stands for the host finite rate of increase and the functions f and g describe respectively the proportion of host surviving parasitism and the proportion of host surviving intraspecific competition.

In diploid parasitoids only mated females may attack the host. The previous model is thus modified as follow:

$$\begin{cases} N_{t+1} = \lambda N_t g(N_t) f\left((1-p(M_t))F_t\right) \\ F_{t+1} = (1-s_0) N_t g(N_t)\left(1-f\left((1-p(M_t))F_t\right)\right) \\ M_{t+1} = s_0 N_t g(N_t)\left(1-f\left((1-p(M_t))F_t\right)\right) \end{cases} \tag{3.2}$$

The proportion of virgin females p is a decreasing function of the number of males, and an increasing function of α, a parameter standing for the difficulty in finding a mate. For a given number of males: the greater is α, the greater is the proportion of virgin females. To be able to consider possible heterogeneities in mating (*e.g.* as a consequence of mate choice) we modeled p by the zero term of a negative binomial, following the equation:

$$p(M_t) = \left(1 + \frac{M_t}{\alpha k_1}\right)^{-k_1} \tag{3.3}$$

where k_1 describes the aggregation of mating: for large values of k_1, mating are randomly distributed among females (and p exponentially decays with M_t) while for small values of k_1 the distribution of mating among females is aggregated. For really low values of k_1 virgin females may occur even for very low value of α.

To describe host intraspecific competition (g function) and proportion of host surviving parasitism (f function) we used the classical functions of Maynard Smith and Slatkin (1973) andMay (1978):

$$g(N_t) = \left(1 + (\lambda - 1)\left(\frac{N_t}{K}\right)^b\right)^{-1} \tag{3.4}$$

$$f(F_t) = \left(1 + a\frac{F_t}{k}\right)^{-k} \tag{3.5}$$

The strength of the host intraspecific competition was affected by its finite rate of increase (λ), its carrying capacity (K) and the severity of the competition was given by the parameter b ($b < 1$: under compensating competition; $b > 2$: overcompensating competition). The probability to escape parasitism was a decaying function of parasitoid searching efficiency (a) and female abundance F, affected by a parameter k which describes the aggregation in parasitoid attacks. For small values of k, parasitoid attacks are aggregated (due to spatial aggregation, host choice, or variability in host immunity); for large values of k, parasitoid search for host at random and probability to escape parasitism is a decaying exponential of the number of females.

2. 2 Modes of sex determination

Four categories of sex determination were considered: 1) haplodiploid mode of sex determination (model 3.1) with mated females fertilizing half of the eggs they laid ($s_0 = 0.5$ (article 2) henceforward named "equilibrated haplodiploidy"), 2) an equilibrated diploid mode of sex determination (model 3.2 with $s_0 = 0.5$), 3) a female-biased haplodiploid sex determination ($s_0 = 0.3$) and 4) haplodiploid sex determination with mated females adjusting sex allocation to the proportion of virgin females in the population as follow:

$$s_0 = \frac{1 - 2p(M_t)}{2\left(1 - p(M_t)\right)} \tag{3.6}$$

(Godfray, 1990) The diploid mode of sex determination was considered as a reference to which each of the three haplodiploidy variants was compared.

2. 3 Quantification of the extinction proneness

We quantified the extinction proneness for 11 α values ranging from 0 to 100 (on a log scale) and for randomly drawn parameter sets. This quantification lay on two sets of currencies. First, we determined the relative extent of the conditions of persistence of the three haplodiploidy variants compared to diploidy when coping with mate finding difficulties. For each haplodiploid variant and each α value, we counted the number of parameter sets which (1) went extinct with diploid and haplodiploid reproductive systems, (2) went extinct with diploid but not with haplodiploid reproductive system, (3) persisted with diploid and haplodiploid reproductive system and (4) went extinct with haplodiploid but not with diploid reproductive system. To avoid an overestimation of the extinction risks, initial abundances had to be chosen close to the equilibrium values. However we were not able to estimate the equilibrium value if all parameters are random. For this reason we proceeded in two steps. First we randomly drawn 1000 parameter sets for b, K, k, and k_1. Second, for each of these parameter sets, we screened the $\lambda - a$ plane considering 2500 combinations.

Second, for the parameter sets for which the population reachs a stable non-zero equilibrium despite mate finding difficulties we evaluate the robustness of this persistence. For this we

compute, for the different modes of sex determination: (i) the critical number of parasitoid necessary to establish (F_{crit}) and (ii) the resistance of the host-parasitoid system to external causes of mortality (F_{min}). The critical population size (F_{crit}) was computed by progressively increasing the initial number of parasitoid females in the system when the host is set to its carrying capacity. The resistance of the host-parasitoid system to external causes of mortality is determined by the minimum fraction of female equilibrium abundance (with hosts set at their equilibrium) below which the population becomes unable to recover (F_{min}). We address the impact of haplodiploidy on these two features by the representation of the ratio $\frac{F_{crit,HD}-F_{crit,DD}}{F_{crit,DD}}$ (resp. $\frac{F_{min,HD}-F_{min,DD}}{F_{min,DD}}$), *i.e.* the proportion of F_{crit} (resp. F_{min}) that can be reduced by haplodiploidy.
In order to investigate further the conditions in which haplodiploidy is beneficial, we set up an analysis of the variance of the difference $F_{crit,HD}-F_{crit,DD}$(resp. $F_{min,HD}-F_{min,DD}$) depending on α , the parameter b, k, and k_1, and their interactions with α, for each haplodiploidy variant. We used an ANOVA and considered each parameter as qualitative variables, with 11 levels going from 0 to 100 on a log scale for α, and respectively 2, 3 and 3 levels for b, k, and k_1. The levels were chosen delimited as following:

- $b < 2$: under-compensated and $b \geq 2$ overcompensated host competition

- $k \leq 1$ (resp. $k_1 \leq 1$) strongly aggregated, $1 < k \geq 5$ (resp. $1 < k_1 \geq 5$): middy aggregated, and $k > 5$ (resp. $k_1 > 5$) completely random host attacks (resp. parasitoid mate-choice)

As developed in White et al. (2013), we will not consider the results and p-values of the linear model, since mathematical modeling allows for the multiplication of "experiments" until any parameter inducing a variation is significant. We will rather consider the proportion of variance of the difference $F_{crit,HD}-F_{crit,DD}$ that can be explained by each parameter and its interaction, and compare these proportions to conclude which parameter has more effect on the impact of haplodiploidy.

2. 4 Relative invasibility of haplodiploids and diploids

In the second part of this article, we investigated how haplodiploidy (equilibrated) may affect parasitoid ability to invade an already established population or to resist invasion. We considered resident and invader as distinct populations sharing the same host but with no possible reproduction events between them. Both resident and invader may be haplodiploid or diploid, resulting in four invasion scenarios.
Dynamics of the resident population was iterated according to model 3.1 (or 3.2 depending on the resident reproductive system) for 1000 generations. If it persisted, the invader population was introduced and the joined resident / invader dynamic was followed for 1000 additional generations. Three issues were then possible: i) extinction of the whole host-parasitoid system, ii) extinction of the invader and maintenance of the resident or iii) successful invader excluding the resident (coexistence was tested but never lasted 1000 generations). Whenever resident and invader had different reproductive modes (one diploid, the other haplodiploid), their dynamics on the common host N were described by the following model :

$$\begin{cases} N_{t+1} = \lambda N_t g(N_t) f\left((1-p(M_{d,t}))F_{d,t} + F_{h,t}\right) \\ F_{d,t+1} = (1-s_0) N_t g(N_t) \frac{F_{d,t}(1-p(M_{d,t}))}{F_{d,t}(1-p(M_{d,t}))+F_{h,t}} \left(1 - f\left((1-p(M_{d,t}))F_{d,t} + F_{h,t}\right)\right) \\ M_{d,t+1} = s_0 N_t g(N_t) \frac{F_{d,t}(1-p(M_{d,t}))}{F_{d,t}(1-p(M_{d,t}))+F_{h,t}} \left(1 - f\left((1-p(M_{d,t}))F_{d,t} + F_{h,t}\right)\right) \\ F_{h,t+1} = (1-s_0)\left(1-p(M_t)\right) N_t g(N_t) \frac{F_{h,t}}{F_{d,t}(1-p(M_{d,t}))+F_{h,t}} \left(1 - f\left((1-p(M_t))F_{d,t} + F_{h,t}\right)\right) \\ M_{h,t+1} = \left(p(M_{d,t}) + s_0\left(1-p(M_{d,t})\right)\right) N_t g(N_t) \frac{F_{h,t}}{F_{d,t}(1-p(M_{d,t}))+F_{h,t}} \left(1 - f\left((1-p(M_{d,t}))F_{d,t} + F_{h,t}\right)\right) \end{cases} \tag{3.7}$$

where F_d and M_d describe the diploid males and females parasitoid, and F_h and M_h describe the haplodiploid males and females. When both resident and invaders were diploid (or haplodiploid), similar models were used, only considering two distinct diploid (or two haplodiploid) parasitoid populations.

3. Results

3. 1 Extinction proneness of single diploid and haplodiploid populations

We first appraised the impact of the mode of sex determination on extinction risk owing to mate-finding difficulties through pairwise comparisons of the proportion of parameter sets leading to extinction between diploidy and three variants of haplodiploidy (Figure 3.1). Unsurprisingly, extinction risk for both diploid and haplodiploid parasitoids increased with increasing difficulties in finding a mate (Figure 3.1). However the extent of this increase depended on the mode of sex determination.

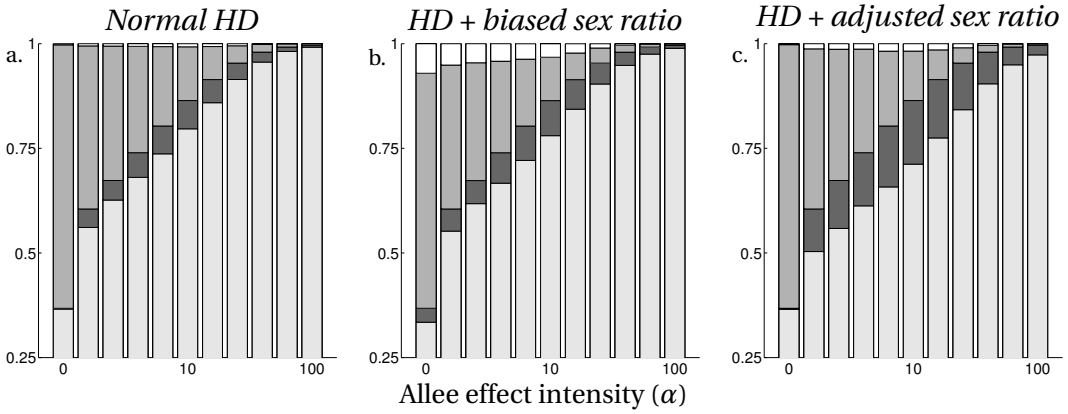

Figure 3.1: Proportion of parameter sets leading to extinction of diploid and haplodiploid populations (light grey), of diploids alone (dark grey), and of haplodiploids alone (white), or to survival of diploid and haplodiploid populations (mild grey) for the three haplodiploidy (HD) variants (normal, with biased sex ratio ($s_0 = 0.3$), and with adjusted sex ratio), as function of the intensity of Allee effects α (on a log scale from 0 to 100).

The increase in extinction risk with α is slower for equilibrated haplodiploid populations than for diploid populations. This can be viewed from the increasing proportion of parameter set for which only haplodiploid persist while the equivalent area for diploid organisms is almost nonexistent (Figure 3.1). If mated haplodiploid females biased their offspring sex ratio toward females ($s_0 = 0.3$), patterns of extinction of diploid *vs.* haplodiploid according to α slightly changes (Figure 3.1.b). Indeed, the proportion of parameter sets for which only diploids persist gets higher and the extinction risks of haplodiploid and diploid rebalance in comparison with equilibrated haplodiploidy (Figure 3.1. a). Extinction risks are roughly similar for both modes of sex determination, while for different parameter sets. Finally, the comparison of diploid *vs.* haploidiploid parasitoids adjusting fertilization rates to the proportion of virgin females in the population (Figure 3.1.c) reveals a pattern of extinction owing to mate finding difficulties similar to Figure 3.1.a, but with a strengthened advantage of haplodiploidy over diploidy.

Beyond the above described interactions between mode of sex determination and mate finding difficulties on extinction risk, it can be seen from Figure 3.1 that the majority of parameter sets for which parasitoids maintain is common to diploid and haplodiploid (medium grey areas on Figure 3.1). In this majority of sets, haplodiploidy has no impact on the existence of non-zero equilibrium, but may affect the difficulties encountered to reach it. Thus, for the parameter sets for which both modes of sex determination persist, we compared the impact of increasing difficulties to find a mate on two relevant features of their demography: F_{crit}, the critical number of parasitoids necessary to establish on a new host; and F_{min}, the minimum proportion of parasitoid populations that needs to remain for the parasitoid to persist (interpreted as the resistance of the population to external causes of mortality).

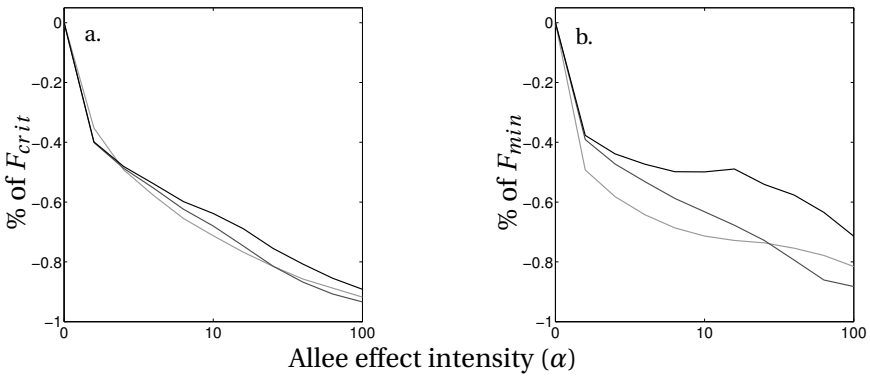

Figure 3.2: Proportion of the critical population size at introduction (F_{crit}) and minimum remaining population size (F_{min}) reduced by haplodiploidy as function of the intensity of Allee effects (α) for the three haplodiploidy (HD) variants: normal (black line), with biased sex ratio (grey line, $s_0 = 0.3$), and with adjusted sex ratio (light gray line).

Both F_{crit} and F_{min} undergo a monotonic increase with α in haplodiploid as in diploid parasitoids (result not shown). F_{crit} and F_{min} values are always lower for haplodiploid than for

diploid parasitoids, but the extent of this reduction may depend on various parameter, among which α has the most impact.
Figure 3.2 describes the percentage of reduction of F_{crit} (Figure 3.2.a) and F_{min} (Figure 3.2.b) by haplodiploidy, for the three diploid variants, as a function of the Allee effect intensity α. This percentage of reduction clearly increases with α for both F_{crit} and F_{min}, reaching 90% for extreme α values. As Allee effects increase, diploid parasitoids necessitate higher initial abundances to establish and become less resistant to external causes of mortality, while haplodiploid parasitoids are comparatively less perturbed. Biased or adjusted sex allocation do not seem to impact the proportion of F_{crit} reduced by haplodiploidy (Figure 3.2.a). Conversely, biased sex ratio and adjusted sex allocation allow a greater reduction of F_{min} at high values of α, up to a supplementary reduction of 18%. Thus, biased or adjusted sex allocation may improve the resistance of the population to external causes of mortality.

The parameters F_{crit} and F_{min} may depend on the characteristics of the system, notably the severity of host competition (b) the distribution of parasitoid attack (k) and of parasitoids mating (k_1). The impact of haplodiploidy on F_{crit} and F_{min} may also depend on such characteristics. To address this point, we analyzed the percentages of variance of the difference between haplodiploid and diploid F_{crit} (resp. F_{min}) explained by the parameters α, b, k and k_1 and their interactions with α for each haplodiploid variants. Table 3.1 lists these percentages of variance.

For both F_{crit} and F_{min} and for all haplodiploidy variants, the Allee effect intensity (α) explains most of the variance in the impact of haplodiploidy. As seen in Figure 3.2, this impact is more important for high values of α. The three variants of haplodiploidy had similar interaction with each of the parameters considered.

The analysis of variance shows that the distribution of parasitoid matings (k_1) never explains more than a negligible proportion of the variance of haplodiploidy impact, and neither does its interaction with α. Whether parasitoids carefully choses the best-looking mate or mate at

Parameter	F_{crit}			F_{min}		
	HD	HDaj	HDbiais	HD	HDaj	HDbiais
α	83.43	78.22	74.23	66.37	66.02	60.08
$\alpha : k$	9.78	13.04	10.80	4.47	6.35	9.67
$\alpha : b$	2.54	2.1	1.20	5.77	5.93	4.28
$\alpha : k_1$	< 1	1.36	1.85	< 1	< 1	< 1
k	3.35	4.42	8.16	5.73	6.17	13.80
b	< 1	< 1	3.42	16.02	14.80	10.45
$k1$	< 1	< 1	< 1	< 1	< 1	< 1

Table 3.1: Percentage of variance explained by the various model parameters and their interactions

complete random do not affect how much haplodiploidy may reduce F_{crit} and F_{min}.

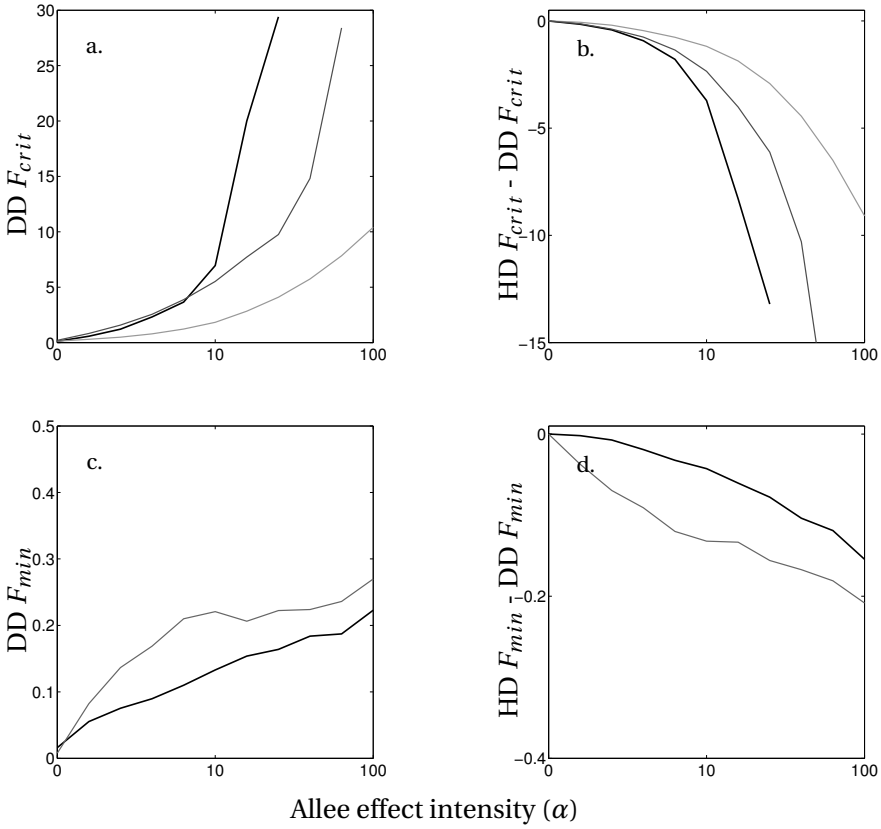

Figure 3.3: First row: impact of parasitoid distributions of attacks (black line: random; grey line: mildly aggregated; light grey line: strongly aggregated) on diploids critical population size at introduction (F_{crit}) and on the difference between haplodiploid and diploid critical populations sizes (resp. a and b) as function of the Allee effect intensity. Second row: impact of the severity of host's intraspecific competition (black line: over-compensate; grey line: under-compensated) on diploids minimum remaining population size (F_{min}) and on the difference between haplodiploid and diploid minimum remaining population size (resp. c and d) as function of the Allee effect intensity.

The interactions $\alpha : k$ and $\alpha : b$ explain a proportion of variance about an order of magnitude lower than α alone, but this proportion is not negligible. When parasitoid attacks are aggregated, the intensity of Allee effects does not affect F_{crit} and F_{min} as much as when attacks occur at random, for both diploid and haplodiploid parasitoid (Figure 3.3.a). Subsequently, haplodiploidy and its variants have a less sizable impact on F_{crit} and F_{min} when attacks are aggregated than when they occur at random(Figure 3.3.b). This result is congruent with the classical stabilizing

impact of aggregated attacks on host-parasitoid systems (May, 1978).

The severity of host competition in interaction with α explains a very small proportion of the variance of haplodiploidy impact on F_{crit}, and a larger proportion of its impact on F_{min}. Diploid parasitoid populations that interact with an under-compensated host are more susceptible to environmental stochasticity, particularly at high values of α (Figure 3.3.c). Haplodiploidy tampers the difference between populations that deal with over-compensated and under-compensated host competition, effectively reducing the F_{min} of populations that deal with under-compensated competition (Figure 3.3.d).

3. 2 Competition between diploid and haplodiploid parasitoids sharing a host

Without Allee effects, diploid and haplodiploid populations have the same demography and thus, the same invasive abilities (Figure 3.4, first bar in all subfigures). The invasion of a host-parasitoid system by a new parasitoid has 3 distinct issues: resident success (the resident persists and the invader dies, Figure 3.4, light grey) in 50% of the cases, invasion success (the resident dies and the invader persists, Figure 3.4, dark grey) in 40% of the cases, and death of both resident and invader (Figure 3.4, black) in 10% of the cases. Invader and resident were unable to coexist in all our simulations.

3. 2. a Resistance

The proportion of resident success decreases with the intensity of Allee effects when the resident was diploid, but not when the resident was haplodiploid (Figure 3.4, light grey). When the intensity or Allee effects increases, diploid resident becomes more susceptible to become extinct due to invasion, even if the invader does not successfully established (black area), and whether the invader is diploid or haplodiploid (Figure 3.4. a,b). Conversely, haplodiploid resident does not suffer such fate. The proportion of cases where the invader does not successfully establish but drive the resident to extinction remains the same for all Allee effect intensity (Figure 3.4. c, d, black area), such as the proportion of cases where haplodiploid residents are successful (Figure 3.4. d, light grey). When the invader is diploid, this proportion increases with Allee effects intensity as the proportion of invader success decreases (Figure 3.4. c, light grey and grey areas).

3. 2. b Invasive abilities

When the invader is diploid, its proportion of success decreases as soon as Allee effects appear, and further with higher Allee effects intensity when facing a haplodiploid resident (Figure 3.4. a, c, grey area). Conversely, haplodiploid invaders retain their proportion of success even at high Allee effect intensities, particularly when facing a diploid resident (Figure 3.4. b, d, grey area). However, the minimum population size necessary to invade increases with the intensity of the Allee effect whether the invader is diploid or haplodiploid. The resistance to invasion of haplodiploids increases with the Allee effect, while the invasive abilities and the resistance to invasion of diploid parasitoids decrease. As a result, when Allee effects are intense (α = 100),

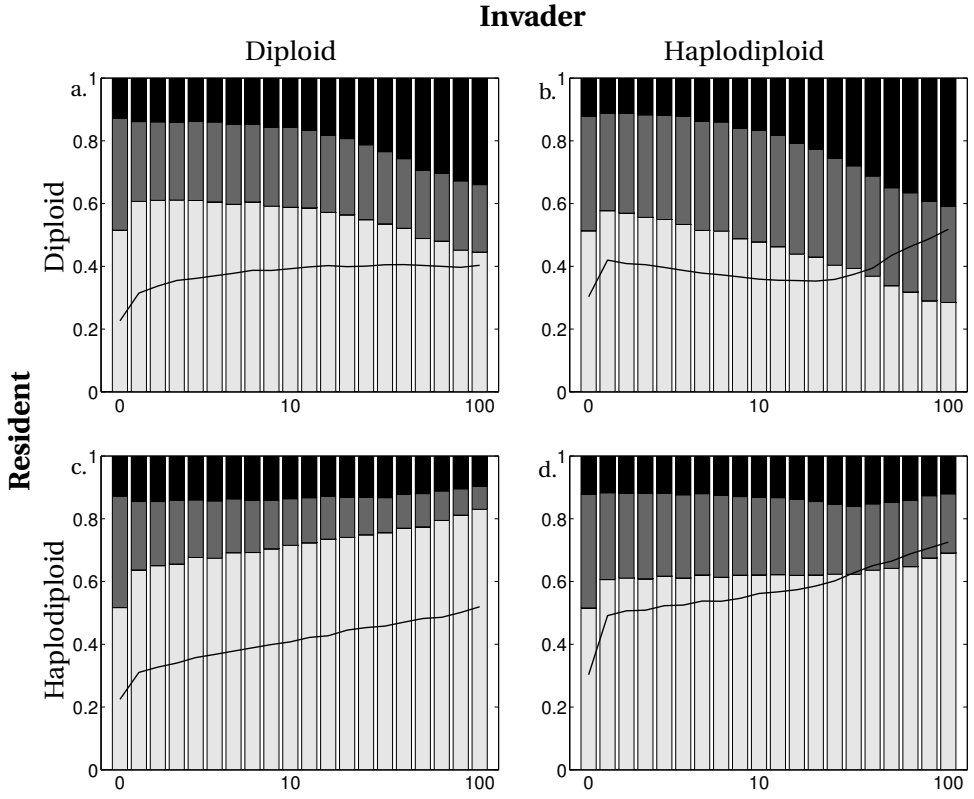

Figure 3.4: Proportion of invasions that end in 1) extinction of both parasitoids (black), 2) extinction of the resident parasitoid and persistence of the invader (grey) and 3) extinction of the invader and persistence of the resident (white) when the resident is diploid (a, b) or haplodiploid (c, d) and when the invader is diploid (a, c) or haplodiploid (b, d) . When the invader is successful, the minimum population size (in proportion of the resident population) to introduce for the invader to win the competition is featured by the black line.

diploid invasions of haplodiploid populations have little chances of success (less than 10%), and haplodiploid invasions of diploid populations are successful in 30% of the cases but drive a diploid resident to extinction in 75% of the cases. A strong Allee effect intensity clearly favors haplodiploid parasitoids in the competition for a common host.

4. Discussion

Recent work on the theoretical impact of mate-finding difficulties in parasitoids has exposed how such difficulties may dramatically increase parasitoid extinction risk, even while they were assumed to be haplodiploid (article 2). These conclusions challenged the common idea that haplodiploid organisms are relatively immune to mate-finding Allee effects, because virgin females remain able to produces males (Godfray, 1990; Hopper, 1993). Whether or not

haplodiploidy was beneficial at all to parasitoid that face mate-finding difficulties laid as an open question, to which we attempted to answer with this theoretical work. Considering haplodiploidy alone could have been short-sighed, because haplodiploidy may allow a variety of adaptations on sex allocation (*i.e.* female-biased sex allocation and fertilization rate adjusted to the proportion of virgin females) that can temper with the impact of mate-finding difficulties (Godfray, 1990).

Haplodiploidy appears to have little impact on the extent of the domain of parasitoid extinction, domain that dramatically increases with Allee effect intensity. A number of parasitoid populations could thrive with low mate-finding difficulties but ineluctably go extinct when these difficulties increases, and so whether they may be diploid or haplodiploid. On this aspect, the domain of parasitoid extinction when parasitoid faces mate-finding difficulties is mostly determined by the characteristics of the host parasitoid system, such as the searching efficiency and distribution of attacks of parasitoids and the strength and severity of the host competition, and little by parasitoid sex determination system.

Conversely, the invasive ability and resilience of parasitoids that may persist whilst facing mate-facing difficulties were clearly stronger with a haplodiploid sex determination system. Thus, haplodiploidy is beneficial only to parasitoids whose population characteristics allow persisting although facing mate-finding difficulties, but to these parasitoids it can be a valued advantage when invading a new environment.

4. 1 Host-parasitoid interaction and the impact of haplodiploidy

Haplodiploid parasitoids settle more easily and are remarkably more resilient once settled, both alone and when facing a diploid population. These results are in agreement with Hopper (1993), who found the critical parasitoid population size to be 30% higher when the parasitoid is diploid than haplodiploid. However we concluded on a dramatically stronger impact of haplodiploidy on the minimum and critical parasitoid population size, since we found the minimum and critical population sizes of parasitoids to be up to 90% lower with haplodiploidy for extreme Allee effect intensity, and up to 40% lower even with low Allee effect intensities ($\alpha = 4$, *i.e.* only 8% of virgin females with a population of 10 males). Compared to the work of Hopper (1993), we include the dynamical variations and demography associated with the host-parasitoid interactions and relaxed Hopper (1993) 's assumption of a fixed host abundance.

The host-parasitoid interaction thus appears to spring the beneficial impact of haplodiploidy on parasitoid critical population size for establishment and resistance to external causes of mortality once established. We suggest this result emerges because the host-parasitoid interaction makes parasitoids more sensible to extinctions due to low densities and mating difficulties than a classical, non-interacting population. Host-parasitoid dynamics tend to be mode complex, and less stable, than non-interacting populations dynamics (Hassell, 2000). As a consequence, parasitoids necessitate higher numbers to establish and have a lower resistance to external causes of mortality than expected in a non-interacting population. Because of this, haplodiploidy may reduce these features to a greater extent in parasitoids than in non-interacting populations. This theory is well supported by the interaction we could observe between ag-

gregated attacks or under-compensated host competition and haplodiploidy impact. Indeed, the more the population is intrinsically stable (due to aggregation and/or under-compensated host competition) the lower will be the critical and minimal abundances of parasitoids, and the weakest will be haplodiploidy impact on these features.

4. 2 Biased and adjusted sex ratio

Female-biased sex allocation and adjusted fertilization rates in mated females have sometime been described as an adaptation to an existing constraint upon fertilization in the population, among which mate-finding difficulties is the most commonly described (Godfray, 1990; King, 2002). Indeed, such adaptations allow mated mothers to produce a greater proportion of the lacking sex, which maximizes their fitness. However, individual benefit should not be confounded with a reduced extinction risk, as we show that these adaptations have little effect on the resilience of parasitoid populations to mate-finding difficulties. Female-biased or adjusted sex allocation significantly benefit parasitoid populations only by making parasitoid populations more resistant to external causes of mortality, such as environmental stochasticity. Populations with female-biased or adjusted sex allocation retain a naturally higher female abundance at equilibrium than equilibrated haplodiploid populations, a feature we did not presented in this paper, but which was also found by Kaitala and Getz (1992); Hassell et al. (1983). Such higher abundance classically protect populations from the impact of environmental stochasticity, particularly when an extinction threshold may appear at low densities(Dennis et al., 1991).

In our model, female-biased and adjusted sex allocation have little impact on the invasive abilities or resilience of parasitoids to invasions, and were subsequently ignored in their study. In contrast, Kaitala and Getz (1992) had found that populations with female-biased sex ratio had better competitiveness and invasive abilities than populations with equilibrated sex ratio. It appears that when mate-finding difficulties are considered, the competitive advantages of female biased sex ratio fade away face to the competitive advantages provided by haplodiploidy alone.

4. 3 Biological control applications

In classical biological control, the question of introducing multiple small populations (to minimize the effects of environmental stochasticity) or one large population (to minimize Allee effects and demographic stochasticity) often comes up. We do not hope to provide a definitive answer, but suggest that when mating difficulties are suspected, the introduction of Hymenopteran parasitoids is more likely to be successful with both choices because they can be successfully introduced in smaller populations and are naturally more resilient to environmental stochasticity. Meta-analysis on the success of biological introductions indeed suggest that Tachinid parasitoid flies introductions are less often successful (23.8%) than wasp parasitoid introductions (Chalcidoidea and Ichneumonidea confunded: 38%)(Hopper, 1993). However, the dispersal rate and spatial structure of parasitoids greatly affect the risk of mate-finding difficulties occurring in natural populations, and these characteristics may vary between species (Godfray, 1994). Thus, we do not advise to use the sex determination system alone to chose be-

tween to potential biocontrol agents.

These last years, the non-target effects of biological introductions have attracted considerable attention (Rossi and Fowler, 2004). Non-target effects of parasitoids mostly concern the extension of their host range to native species, but may also include the exclusion of native parasitoids by newly introduced parasitoids (Simberloff and Stiling, 1996; Rossi and Fowler, 2004). Our simple theoretical model does not allow for parasitoid coexistence on the same host species, which can hardly be discussed as various cases of coexistence on the same host have been observed in natural populations (Godfray, 1994; Sato et al., 2002; Memmott et al., 1994). However, our results also suggest that with a mate-finding Allee effect, a diploid native parasitoid is more likely to be driven to extinction than a haplodiploid native parasitoid by the introduction of a new species, and that this effect is stronger when the introduced parasitoid is haplodiploid. The introduction of *Cotesia Flavipes* (Hymenoptera: Braconidae) to control the lepidoptera *Diatraea saccharilis* has been recently pointed as responsible for the exclusion of the native Tachinid parasitoid flies *Lydella minense*, *Paratheresia claripalpis* and *Lyxophaga diatraea* (Rossi and Fowler, 2004). Meta-analysis on the occurrence of such non-target effects could reveal whether diploid parasitoids are indeed more at risk than haplodiploids when facing an invading parasitoid.

To conclude, we must recommend caution against ignoring the risk of mate-finding Allee effects when introducing parasitoids. Clearly, haplodiploid parasitoids are not immune to the demographic consequences of mate-finding Allee effects. Moreover, the features that usually betray the presence of a mate-finding Allee effects may be relatively misleading in parasitoid, since haplodiploidy may reduce the consequences of a small population size but some haplodiploid parasitoid populations still ineluctably go extinct due to Allee effects. The host-parasitoid interaction is probably largely responsible for the difficulties to identify and understand the risk of extinction of introduced parasitoid populations.

Part III

Conséquences démographiques et évolutives du *Complementary Sex Determination* chez les parasitoïdes

CHAPTER

4 No diploid male vortex in parasitic wasps: how a genetic load may favor parasitoid persistence

Article 4

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Résumé

Le feedback entre les facteurs génétiques et démographiques est une cause d'extinction majeure dans les populations naturelles. De nombreux modèles de génétique des populations décrivent le l'accumulation et l'expression des mutations délétères, la perte de la diversité génétique, et leur impact sur la démographie de la population. Cependant, très peu ont choisit d'inclure dans la démographie de la population des facteurs écologiques tels que les interactions interspécifiques. Nous étudions l'impact du mécanisme *Complementary sex determination* (CSD) sur la démographie des populations de guêpes parasitoïdes dans un système hôte - parasitoïde. Le CSD est un mécanisme de détermination du sexe basé sur la compatibilité allélique, qui conduit à la production d'individus non viables ou stériles (des mâles diploïdes) lorsque la diversité génétique sur le gène *csd* est perdue. Le CSD est donc considérée comme une forme de fardeau génétique, qui produit un vortex d'extinction fort appelé *Diploid Male Vortex* dans les populations d'hyménoptères non parasitoïdes.
Nous étudions son impact sur les parasitoïdes en utilisant un modèle éco-génétique original, qui décrit la démographie de la population à travers un modèle déterministe, et les accouplement et génotypes à travers une partie stochastique.

Avec cette étude théorique , nous montrons que non seulement ce vortex d'extinction est absent des populations de parasitoïdes, mais également que le CSD stabilise largement la dynamique des parasitoïdes de forte efficacité de recherche, ce qui réduit leur risque d'extinction. Les parasitoïdes faible efficacité sont entraînés dans des extinctions déterministes, dont les caractéristiques diffèrent d'un vortex d'extinction classique : le risque d'extinction est indépendant de la taille de la population ou de sa diversité génétique. Lorsque les mâles

diploïdes sont stériles, les femelles qui s'accouplent cependant avec eux sont incapables de produire des descendants fertilisés. La stérilité des mâles diploïdes augmente le risque d'extinction pour les populations de parasitoïdes de forte et de faible efficacité de recherche par rapport à des mâles diploïdes non viables. Nous démontrons que nos résultats sont issus de l'interaction hôte – parasitoïde en manipulant la dépendance de la population de parasitoïde à son hôte. Les populations indépendantes de l'hôte sont entraînées par le CSD dans un *Diploid Male Vortex* similaire à celui précédemment décrit chez les populations d'hyménoptères non parasitoïdes.

Nous discutons ces résultats en regard des expériences récentes sur les populations de parasitoïdes naturelles, qui ne parviennent pas à démontrer la présence d'un *Diploid Male Vortex*. Ce résultats originaux contestent la l'impact supposément délétère du CSD chez les populations hyménoptères parasitoïdes, et remettent en question les impacts démographiques et évolutifs du fardeau génétique dans les populations en interaction. Ce nouveau regard pourrait remettre en cause les bases théoriques des programmes de conservation des espèces en interaction et de lutte biologique.

Abstract

The synergy of demographic and genetic factors is a major cause of extinction in natural populations. Numerous models of population genetics describe the flow of deleterious mutations, the loss of genetic diversity and their subsequent impact on population demography. However, very few have assumed that the population demography may include ecological factors such as interspecific interactions. We investigate the impact of Complementary sex determination (CSD) on the demography of parasitoid wasp populations within a host-parasitoid system. CSD is a sex determination mechanism based on allelic compatibility that leads to the production of unviable or sterile individuals (diploid males) when the genetic diversity on the *csd* gene is lost. CSD is thus considered as a form of genetic load, which produces a strong extinction vortex named Diploid Male Vortex in non-parasitoid Hymenoptera populations. With this theoretical study, we show that not only is this vortex absent form parasitoid populations, but CSD is largely stabilizing for the dynamics of high searching efficiency parasitoids, which reduces their extinction risk. Low efficiency parasitoids are driven into deterministic extinctions of which features differ from genetically-induced extinction vortex. Diploid males sterility increases the extinction risk in all parasitoid populations. We demonstrate that these results come from the host-parasitoid interaction, as populations without dependency to a host are driven to extinction by a diploid male vortex. We discuss these results in regard with recent experiments on natural parasitoid populations, which fail to demonstrate the presence of a diploid male vortex. This work challenges the view of CSD as a threat for parasitic Hymenoptera, and questions the demographic and evolutive impacts of genetic load in interacting populations. This new look might question the theoretical foundations of conservation and biological control programs that address interacting species.

KEYWORDS: Hymenoptera, *Complementary sex determination*; host-parasitoid interaction; genetic load; extinction; haplodiploid; Diploid Male Vortex; parasitoid, persistence, haplodiploidy, extinction vortex, interspecific interaction

1. Introduction

Population genetics and population dynamics constitute the scientific foundations of modern population biology, and as such, they 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; 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 (Doebeli and de Jong, 1999; Leimu et al., 2006; Frankham, 2002; Hughes et al., 2008; Robert, 2011) . The present work contributes to this general perspective of bridging genetics and demography. With a demo-genetic model, we demonstrate that the consequences of inbreeding depression on demographic success depend dramatically on the nature of trophic interactions, and unveil 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 reveal major flaws. 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 (Lande, 1988; Frankham, 2002; Caughley, 1994; 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, population growth rate, which may drive populations into an extinction vortex (Gilpin and Soule, 1976; Lande, 1988; Tanaka, 2000; Belovsky et al., 1999; Schoener et al., 2003; Fagan and Holmes, 2006; Palomares et al., 2012; Kampichler et al., 2010).
Genetic incompatibilities that result from the genetics of sex determination of some parasitoid wasps provide an outstanding paradigm to approach the interplay between genetics and demography in systems where over-simplistic assumptions do not hold. In these wasps, sex is determined by the complementarity of alleles at a single complementary sex-determiner (*csd*) locus: normal haploid males develop from unfertilized eggs, consequently hemizygotes at the *csd*) locus, and diploid females develop from fertilized eggs that are heterozygotes at the *csd*) locus (Whiting and April, 1943). The problem rise from homozygosity at the *csd*), 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) . Single-locus Complementary Sex Determination (CSD) is therefore at the origin of a particular type of inbreeding depression underpinned by overdominance.

Single-locus complementary sex determination 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 (Bull, 1981; Cook,

1993; Asplen et al., 2009; Crozier, 1971; Cook and Crozier, 1995; Zhou et al., 2005; Schmieder, 2012). The *csd* gene has been described and mostly studied in the honeybee (Beye et al., 2003; Hasselmann et al., 2008).

At the population level, CSD 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 as 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 frame of pollinator decline and biological conservation (Zayed and Packer, 2005; Zayed et al., 2004; 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 dependences within and between populations, which induces complex dynamics (Hassell, 2000). Cyclic dynamics with recurrent bottlenecks are typical emerging properties of these theoretical models (Bernstein, 1996, 2000) and have been observed in natural parasitoid populations (Wearing et al., 2004) . Second, dramatic variations that fatally disturb agro-ecosystems (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 will experience much more stringent consequences of CSD than what has been predicted to date.

In this paper, we investigate on the consequences of 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 CSD on parasitoid extinction and the dynamics of host-parasitoid systems; 2- to what extent is the observed effect of CSD an emerging property of the host-parasitoid interaction; 3- does CSD cause an extinction vortex in parasitoids. We consider that an extinction vortex appear when a reduction in population size or genetic diversity results in an increase of the extinction risk. We demonstrate 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.

2. Methods

We combined trophic interactions and genetic processes within a discrete-time, two-phase population model. At each generation, a deterministic host-parasitoid population dynamic model predicted the number of hosts and parasitoids, whereas mating and gene transmission followed a stochastic subroutine. The deterministic phase of the model was founded on equations describing 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 was therefore governed by the stochastic phase of the model: fertilized eggs developed into females (F) if heterozygous at the *csd* locus or diploid males (DM) otherwise. In cases where diploid males were assumed viable (and as fit as haploid males), the model also allowed for triploid individuals (T) resulting from crosses between a female and a diploid male.

2. 1 Mating structure and gene transmission

Initial allelic diversity at the *csd* locus was set to w, and genotypes were randomly attributed to the F_0 male and female parasitoids founding the population. In the following generations, each new individual was attributed 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 $M + DM$ males. For this, we assumed a monandrous – polygynous mating system, as observed in most parasitoid wasps (Godfray, 1994). Offspring genotype at the *csd* locus resulted from classical Mendelian transmission of parental alleles. When we assumed unviable diploid males, fertilized eggs homozygous at the *csd* locus died. When we assumed sterile diploid males, the females that they mated produced only haploid males and unviable triploid females. Without CSD, all fertilized eggs developed as females. This genetic phase of the model yielded a form of demographic stochasticity: the realized fecundity of females varied according to the random assignations of parents and genotypes to newborn individuals.

2. 2 Population dynamics

To describe the dynamics of host and parasitoid populations, we built from the host-parasitoid model from article 2. At each generation, the abundance of hosts (N), diploid (D) and haploid parasitoids (M) were determined according to the abundance of parasitoid females (F) and hosts (N) at the previous generation (equation 1, deterministic part) as well as a constant rate of egg fertilization (0.5). We assumed that parasitoid males were not limiting so that all females were mated (no mate-finding Allee effect). The abundance of diploid males (DM) and of triploid females (T) depended on a constant rate of fertilization, and respectively on their parents' genotypes and on the frequency of diploid males in the male population at the previous generation. Parasitoid females (F) developed from heterozygote diploid eggs (equation 1, stochastic part). These assumptions resulted in the following equations:
Deterministic phase:

$$\begin{cases} N_{t+1} = \lambda N_t g(N_t) f(F_t) \\ D_{t+1} = (1-s_0) N_t g(N_t) \left(1 - f(F_t)\right) \\ M_{t+1} = s_0 N_t g(N_t) \left(1 - f(F_t)\right) \end{cases} \tag{4.1}$$

Stochastic phase:

$$\begin{cases} DM_{t+1} & \text{function of } D_{t+1} \text{and parent's genotypes} \\ T_{t+1} & \text{function of } D_{t+1} \text{and } \frac{DM_t}{DM_t + M_t} \\ F_{t+1} & = \begin{cases} D_{t+1} - DM_{t+1} & \text{for unviable DM} \\ D_{t+1} - DM_{t+1} - T_{t+1} & \text{for sterile DM} \end{cases} \end{cases}$$

where f and g described respectively the proportion of host escaping parasitism (May, 1978) and the proportion of host surviving intraspecific competition (Maynard Smith and Slatkin, 1973) ; λ was the host finite rate of increase, and s_0 the proportion of unfertilized parasitoid eggs, that we assumed balanced ($s_0 = 0.5$).

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:

$$f(F_t) = \left(1 + a\frac{F_t}{k}\right)^{-k} \text{ (May, 1978)} \tag{4.2}$$

The smaller the k value, the more aggregated the parasitoid attacks(May, 1978) . The distribution of parasitoid attacks is strongly aggregated if $k < 1$ and can be considered as random (Poissonian) if $k > 5$.

For hosts, when considered independently of parasitoids, the probability to survive from intraspecific competition depends on population abundance (N), carrying capacity (K), intrinsic rate of increase (λ) and the severity of competition (b), following the equation:

$$g(N_t) = \left(1 + \left(r\frac{N_t}{K}\right)^b\right)^{-1} \text{ (Maynard Smith and Slatkin, 1973)} \tag{4.3}$$

For high values of λ, host dynamics depends on intraspecific competition via the parameter b. For $b < 1$, competition is under-compensated and the host population is asymptotically stable; for $1 < b < 2$, the host population is stable with damped oscillations; for $b > 2$, competition is over-compensated and the host population is cyclic (Maynard Smith and Slatkin, 1973)

2. 3 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 3000 generations, whichever happened first. Most extinction nonetheless occurred within 200 generations.

We first focused on the consequences of CSD on parasitoid extinction. For this, we estimated, for each set of parameters, the probability of parasitoid extinction $P(E)$ as the proportion of simulations ending as a result of parasitoid extinction, and the time to extinction ($T(E)$) as the mean time before extinction when extinctions occurred. We also kept track of the evolution of population sizes and allelic richness across time.

To better understand the specific consequences of host-parasitoid dynamics on the effect of CSD, we artificially varied the amount of parasitoid dependence on its host via a single parameter x. For this, a constant fraction x of the parasitoid population was considered demographically independent from the host; the demography for this fraction of the population was influenced by intraspecific competition, which is described by the previously mentioned function g (equation 3). The first three equations of the model (1) then became:

$$\begin{cases} N_{t+1} = \lambda N_t g(N_t) f\left((1-x)F_t\right) \\ D_{t+1} = (1-s_0)\left(x\lambda_1(F_t+M_t)g\left(x(F_t+M_t)\right) + N_t g(N_t)\left(1-f\left((1-x)F_t\right)\right)\right) \\ M_{t+1} = s_0\left(x\lambda_1(F_t+M_t)g\left(x(F_t+M_t)\right) + N_t g(N_t)\left(1-f\left((1-x)F_t\right)\right)\right) \end{cases} \tag{4.4}$$

where λ_1 is the finite rate of increase of the non-interacting fraction of the parasitoid population. The stochastic processes in the model remained unchanged. For $x = 1$, the parasitoid population is completely independent from the host and grows as a free-living population; for $x = 0$, the model is strictly identical to equation (1). In order to unravel the consequences of CSD underlined specifically by host-parasitoid dependence, we assessed the effect of x on the proportion of parameter sets for which the parasitoid population was promoted or endangered by CSD. We randomly drawn each parameters value on a large range of biologically meaningful values (see Table 1) and calculated the extinction probability $P(E)$ (calculated over 10000 replicates) of each parameter set with and without CSD. Among parameter sets for which $P(E)$ changed, we quantified those for which $P(E)$ increased and decreased.

A demographical consequence of host-parasitoid interaction is to generate cyclic dynamics. To disentangle the contribution of cycling dynamics and host-parasitoid interaction per se on demographical consequences of CSD, we compared the demographic impact of CSD on cyclic population when cycles were a consequence of the parasitoid interaction ($x = 0$) and when they were the consequence of a strong intraspecific competition in non-interacting populations ($x = 1, b > 2.5$). Two features of cyclic dynamics were considered: i) amplitude of the cycles and ii) minimal density of the cycles. We calculated the mean percentage of difference with and without CSD for these two features, in interacting and non-interacting populations. We restrained the comparisons to parameter combinations where populations are cyclic without CSD.

Finally, in order to derive predictions for parasitoid introductions in the frame of classical biological control, we assessed the effect of initial conditions (size and diversity of the released populations) on extinction probability with and without CSD. For this, we varied initial allelic diversity (w) and initial number of foundresses (F_0) and computed the mean difference of extinction probability with and without CSD as a function of these two variables. We restricted the analysis to parameter ranges for which CSD has been shown to potential drive parasitoids

to extinctions (see Table 1), and compared host- parasitoid systems ($x = 0$) to non-interacting populations ($x = 1$).

2. 4 Representation

We displayed the probability of parasitoid extinction as a density plot on a $\lambda - a$ diagram. Without CSD, the dynamics of the parasitoid population described by our model converged to the dynamics given by the deterministic model alone, as described in article 2 . Two different areas of parasitoid extinction were distinguished: i) the upper left side of the stability diagrams, characterized by high parasitoid searching efficiency and low host finite rate of increase, a combination of $\lambda - a$ values that generated high-amplitude cycles causing parasitoid extinction (henceforth named the area of cyclic extinction) and ii) the lower right side of the diagram characterized by low parasitoid searching efficiency and high host rate of increase, a combination of $\lambda - a$ values that lead to a progressive decrease of parasitoid abundance, until extinction (henceforth named area of asymptotic extinction).

3. Results

3. 1 CSD impact on parasitoid extinction and the dynamics of host-parasitoid systems

3. 1. a Parasitoid extinction risk

Overall, CSD reduced the probability of parasitoid extinction. Across the 5000 tested parameter sets, we found a probability of parasitoid extinction of 71.6% without CSD, 60.7% with CSD and unviable diploid males, and 65.6% with CSD and viable but sterile diploid males (Fig. 1), The area of cyclic extinction, characterized by high parasitoid efficiency and low host growth rate, was smaller with CSD than without. Conversely, the area of asymptotic extinction, characterized by low parasitoid efficiency and high host growth rate, was larger with CSD than without (Figures 4.1.a,4.1.b).

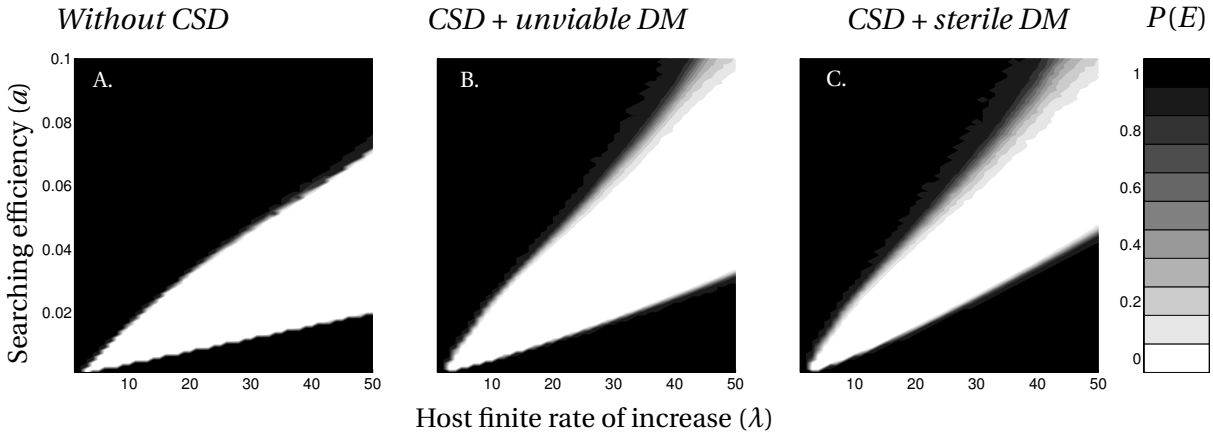

Figure 4.1: Probability of extinction (*P(E)*) for parasitoid populations without CSD (left), with CSD and unviable diploid males (middle), or with CSD and sterile diploid males (right) as function of parasitoid searching efficiency (a) and host intrinsic growth rate (λ). For mild host competition (b = 1.5) and random attacks (k = 10). Other parameter values: K = 5000, w = 10, $F_0 = 50$.

3. 1. b Parasitoid dynamics

A prominent effect of CSD on host-parasitoid dynamics was to buffer parasitoid cycles. In the domain of parameters where parasitoid populations cycled, CSD yielded in a $25.4 \pm 2.7\%$, (mean$\pm$ SD; number of parameter sets = 1296) decrease in the amplitude of cycles and a $253.5 \pm$ 8.7% (n = 1296) increase in the minima at the bottom of cycles. With these two effects, some parasitoid populations that were driven into cyclic extinction without CSD did persist with CSD (Figures 4.1, 4.2.a, 4.2.b). In contrast, for parasitoids that persisted close to the area of asymptotic extinction, CSD yielded a decrease in parasitoid abundance, which ultimately drove populations to extinction (Figures 4.2.d, 4.2.e). For the latter populations, extinctions occurred in a mean extinction time of 116 ± 0.35 generations (n = 3637). With sterile diploid males, extinctions occurred in cyclic populations that persisted with CSD and unviable diploid males (Figures 4.2.c, 4.2.f). These extinctions were attributed to the females mating only with diploid males, and failing to renew the female pool for the next generation. For parasitoid that persisted close to the area of asymptotic extinction, extinctions were faster with sterile rather than unviable diploid males: for the same parameter sets, the mean extinction time with sterile diploid males was 37.6525 ± 0.20 generations (n = 3637), compared to 116 generations for unviable diploid males (see above). The impact of CSD on parasitoids dynamics remained qualitatively similar whatever the type of host intraspecific competition (b) and parasitoid distribution of attacks (k) (Figure 4.5 in Appendix). However, strongly aggregated attacks damped parasitoid cycles even without CSD so that only the expression of the deleterious consequences of CSD for population persistence remained (Figure 4.5 D-F in Appendix).

3. 1. c Genetic diversity

In cyclic parasitoid populations, most of the initial allelic diversity on the *csd* gene was lost during the first cycle and populations persisted despite a low allelic richness (2-5 alleles) (Fig-

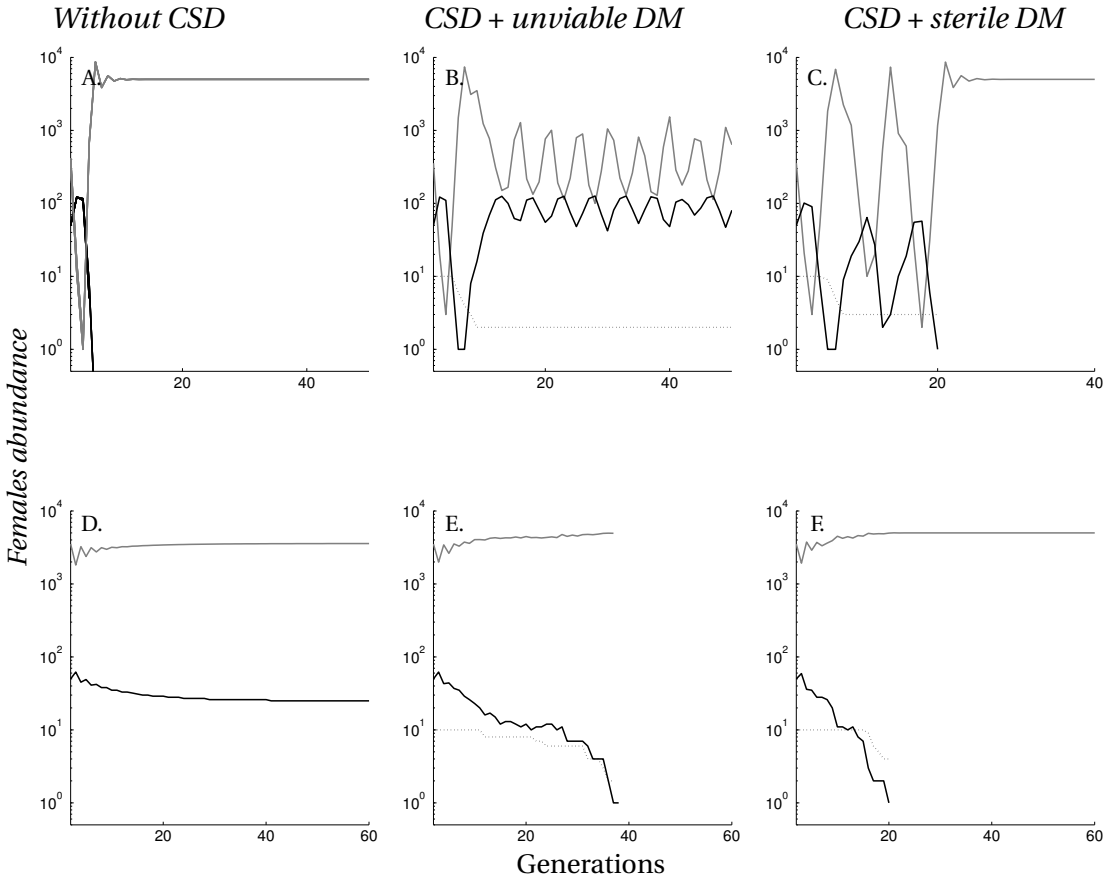

Figure 4.2: Host and parasitoid dynamics without CSD (A,D), with CSD and unviable diploid males (B,D) or with CSD and sterile diploid males (C, F), for two sets of parameters in high (A, B and C) and low parasitoid efficiency (D, E and F). Black line: parasitoid female abundance, grey line: host abundance, dotted line: allelic diversity on the *csd* locus. Parameter combinations for high efficiency parasitoids (A, B and C): $a = 0.049$, $\lambda = 9$; for low efficiency parasitoids(D, E and F): $a = 0.01$, $\lambda = 6$; for all subfigures : $K = 5000$, $b = 1.5$, $k = 10$, $w = 10$, $F_0 = 50$

ures 4.2.b). Conversely, in parasitoids populations close to the area of asymptotic extinction, the allelic diversity for the *csd* gene progressively decreased, following population abundance (Figures 4.2.e). When these populations persisted despite CSD, their allelic diversity settled to moderate levels (4-10 alleles).

3. 2 Is the observed effect of CSD an emerging property of the host-parasitoid interaction?

In order to understand the contrast between our results and that of (Zayed and Packer, 2005) we manipulated the fraction of parasitoids depending on hosts via a single parameter x, from x=0 (corresponding to complete independence, as assumed by (Zayed and Packer, 2005)) to $x = 1$ (corresponding to complete dependence, as assumed in the model above). The fraction of

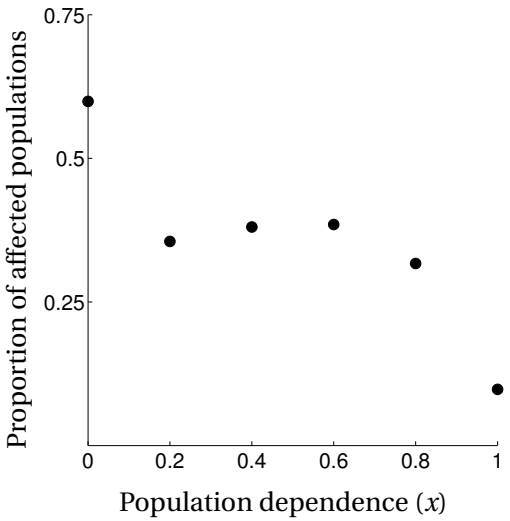

Figure 4.3: Proportion of parameter combinations for which CSD decreases the probability of the population extinction, among the parameter combinations for which the CSD causes a shift in the probability of the population extinction, as function of the independence of the population to the host N (x=1: independent population and $x = 0$: parasitoid population). Probability of extinction are computed over 10 000 simulations for each parameter combinations. Parameter values are randomly drawn in the following range: $a = 0-1$; $\lambda = 0.01-100$;$\lambda_1 = 0.01-100$; K = 20-5000; $K_1 = 20-5000$;$b = 0.5-2.5$; $b_1 = 0.5-2.5$; $k = 0.5-20$; $w = 10$; $F0 = 50$.

parasitoids depending on hosts did affect the demographic impact of CSD. CSD more frequently reduced than increased the probability of extinction of the population only when x was close to 0, *i.e.* with strict parasitoid growth (Figures 4.3). As a larger fraction of the population became able to grow independently of the host, a smaller proportion of the parameter combinations benefited from CSD in term of persistence (Figures 4.3).

When the population grew completely independently of the host ($x = 1$), CSD increased the risk of parasitoid extinction. Extinctions occurred in a medium time-span, with a mean time to extinction of 114.23 ± 0.34 generations (n = 3729). The reduction of the cycles amplitude induced by CSD was less important when cycles were caused by a severe intraspecific competition in non-interacting populations than when they arose from the interaction of the parasitoid with its host ($11.87 \pm 0.47\%$, n=19989) *v.s* $25.39 \pm 2.74\%$, n = 1296). Moreover, CSD decreased the minimum of cycles resulting from the competition of $12.59 \pm 0.49\%$(n = 19989) while it increased that of parasitoid cycles (see above).

3. 3 Does CSD cause an extinction vortex in parasitoids?

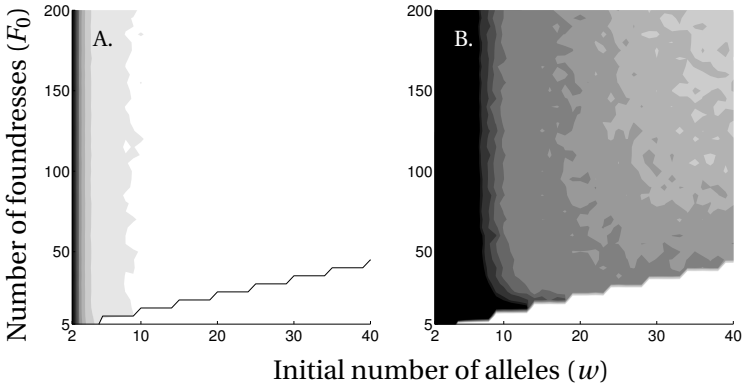

Figure 4.4: Mean difference between probability of extinction with and without CSD over the range of parameter combinations for which CSD may drive parasitoid populations to extinction, as function of the number of foundresses (F_0) and the initial allelic diversity (w), for parasitoid populations (A) and for independent populations (B). Below the black line: irrelevant initial conditions ($w > 2F_0$). Other parameter value or range: A: see fig. 1.A; B: $1 < b_1 < 3.5$; $50 < K_1 < 150$; $b_1 = 1.5$.

CSD did not affect the influence of the initial number of foundresses (F_0) on the extinction of parasitoid populations and affected the influence of the initial allelic diversity (w) only when it was very low (Figure 4.4.a). By contrast, it strongly increased the influence of both initial features on the extinction of independent populations (Figure 4.4.b). Independent populations, but not parasitoid populations, show the presence of an extinction vortex induced by CSD.

4. Discussion

Inbreeding depression is considered as one of the most important causes of extinction in small populations (Frankel and Soulé, 1981; Caughley and Gunn, 1996; Saccheri et al., 1998) and have been extensively studied in the context of conservation efforts (Caughley and Gunn, 1996; Palomares et al., 2012; Kampichler et al., 2010). The extinction of small populations resulting from the combined action of 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 dynamics. In more complex situations where, for instance, populations of several species interact with one another, the outcome of interplays between genetic 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 the single locus *Complementary Sex Determination* (CSD) that prevails in insects of the order Hymenoptera. CSD was shown to induced an extinction vortex in non-

interacting populations (Zayed and Packer, 2005; Hedrick et al., 2006). Here, we show that when CSD is applied to parasitoid wasps that interact with their hosts, CSD can promote population persistence, and that the demographic consequences of CSD does not only concern small, threatened populations. Surprisingly, the fate of parasitoid populations with CSD is not determined by population size and genetic diversity, but rather by parasitoid searching efficiency and host dynamics.

By artificially manipulating the degree to which parasitoids depended on their hosts, we demonstrated that the host-parasitoid interaction is the central feature yielding a positive effect of inbreeding depression on population persistence. This counterintuitive result can be explained as follows: by reducing the production of female parasitoids, CSD yields an increase in host abundance and in turn, an increase in parasitoid population growth rate. In other words, CSD limits parasitoid efficiency and thus, promotes a refuge for hosts; in the long run, this refuge serves the persistence of parasitoids. Therefore, the effects produced by CSD resemble that of other processes such as interference between parasitoids and aggregation of parasitoids attacks. Such processes have been largely explored in the last decades, and are deemed responsible for the relative stability of natural parasitoid populations (May, 1978; Hassell, 2000).

4. 1 Damping the cycles

Complementary sex determination promotes the persistence of parasitoid populations when parasitoids have a cyclic dynamic, *i.e.*, when the mortality they induce on their hosts is so strong that is not compensated by an alleviation of host competition. CSD damps the parasitoid cycles through two sequential mechanisms that conversely affect the parasitoid growth rate: 1) when host are abundant and parasitoid abundance consequently increases, CSD reduces parasitoid growth rate due to diploid male production, and 2) when host are rare because of parasitism and parasitoid abundance decreases, hosts remains more numerous with CSD because CSD reduced parasitoid growth rate and its pressure on the host in the previous phase, so CSD increases parasitoid growth rate due to higher host availability. By decreasing parasitoid growth rate when parasitoid abundance increases and increasing parasitoid growth rate when parasitoid abundance decreases, CSD damps the parasitoid cycles. The impact of CSD on cyclic dynamics of non-interacting population differs in two points: i) cycles are damped to a lesser extent and ii) minimal density of cycles decreases. This indicates that the demographic consequences of CSD on cyclic populations strongly depend on biological processes sustaining the cycles. Several authors already noticed 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).

Our results suggest that the decreased production of females offspring caused by CSD has analogous population consequences as a decrease in searching efficiency. The decreased production of female offspring depends on allelic diversity at the *csd* locus. When parasitoids have a cyclic dynamics, the decrease in parasitoid searching efficiency fades within a few generations, after which allelic diversity has reached an equilibrium maintained by frequency-dependent selection. Many deterministic models have tackled the consequences of a decrease in parasitoid searching efficiency or female production, for instance through male biased sex ratio, Allee ef-

fects or interference (Briggs and Collier, 2001; Hassell et al., 1983; Bompard et al., 2013). These phenomena are always stabilizing for cyclic parasitoids, even if their overall effect is not systematically to reduce the parasitoid domain of extinction.

4. 2 Extinctions under CSD

As for non-interacting population, CSD can drive parasitoid populations to extinction, particularly at low parasitoid searching efficiency and high host rate of increase. However, major aspects differentiate CSD-induced extinctions in parasitoids from extinctions proneness through the Diploid Male Vortex in non-interacting populations. Indeed the initiation of this vortex in non-interacting populations is considered as critically depending on the initial population abundance and genetic diversity (Zayed and Packer, 2005). Conversely, CSD-induced extinctions in parasitoids are mostly independent of these two factors, suggesting that the extinction process is mainly demographic. Parasitoid populations with CSD would not be more endangered by an external reduction of population size or genetic diversity than parasitoid populations without CSD. However, they would be more endangered by an external disruption of their searching efficiency (or of their host carrying capacity) as the critical level of these two parameters is higher for parasitoid populations with CSD than without. The reduction of parasitoid efficiency and host carrying capacity occurs quite regularly in agro-ecosystems due to crop rotations or pesticide use (van Driesche et al., 2008), and their consequences may have been underestimated.

4. 3 Diploid male sterility and stochasticity in interacting populations

Demographic impact of single-locus *Complementary Sex Determination* is more complex when diploid males are sterile. In this case, the impact of diploid male production on realized fecundity extends to an extra generation. This additional reduction of female production further increases the domain of asymptotic extinction. Its impact on cyclic dynamics is more intricate. When allelic diversity at the *csd* locus is low, the probability of mating a diploid male and thus producing only unfertilized eggs increases, and so the variability in realized fecundity. This increased stochasticity may drive cyclic parasitoid populations to extinction, as classically observed when demographic and/or environmental stochasticity is introduced in host-parasitoid models (Wilson and Hassell, 1997). An additional analysis (not shown in the paper) including variability on the model parameters suggests that the addition of environmental or demographic stochasticity would not affect the general conclusions of our paper.

4. 4 Natural parasitoid populations

Single-locus *Complementary Sex Determination* presence and impact on demography has not been evaluated in a sufficient number of parasitoid species to test our conclusions over a meta-analysis of the literature. However we can use indirect clues to estimate the likeliness of the dynamical features of CSD in natural populations. While there are evidences of inbreeding avoidance in parasitoids with CSD (Metzger et al., 2010) or of preferential mating with males carrying unmatched alleles at *csd* locus (Thiel et al., 2013), some parasitoid species are known to engage in inbreeding while determining sex with CSD (El Agoze et al., 1994; Cowan and Stahlhut,

2004). This has always been considered as a paradox in regard to the presence of a Diploid Male Vortex (Heimpel and de Boer, 2008). However if parasitoid populations may persist with a low allelic diversity - as suggested by our model - engaging in inbreeding or outbreeding would be likely to produce a similar amount of diploid males and the selective pressure upon mechanisms to avoid inbreeding would not be very strong. Sib-mating behavior is known to invade when this selective pressure is reasonably low, because avoiding inbreeding is costly (Getz et al., 1992).

The presence of the diploid male vortex itself has been tested in the parasitoids *Cotesia glomerata* (Elias et al., 2010b) and *Venturia canescens* (personal communication C. Vassyade). The classical Diploid Male Vortex (entered by reduced population size or genetic diversity) was absent in both. These results support our conclusions, which suggest that the risk of a Diploid Male Vortex in parasitoids may have attracted more fears than it should.

4. 5 Conclusion

Our modeling study reveals how, depending upon underlying assumptions, feedbacks between demography and genetics can follow different routes, and consequently, 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. Our finding is also opposite to our a priori hypothesis in a very interesting way. The demographic cycles, which characterize host-parasitoid systems, were intuitively envisaged as catalyzers of the erosion of allelic diversity; host-parasitoid cycles should thus have accelerated the extinction vortex. Rather, we show that the reduction in allelic diversity buffers the cycles and therefore, promote stability. Hence, genetic diversity and host-parasitoid cycles do interact, but in a way that is drastically different from that expected, yielding the counterintuitive prediction that inbreeding depression might sometimes favor population persistence.

5. Appendix

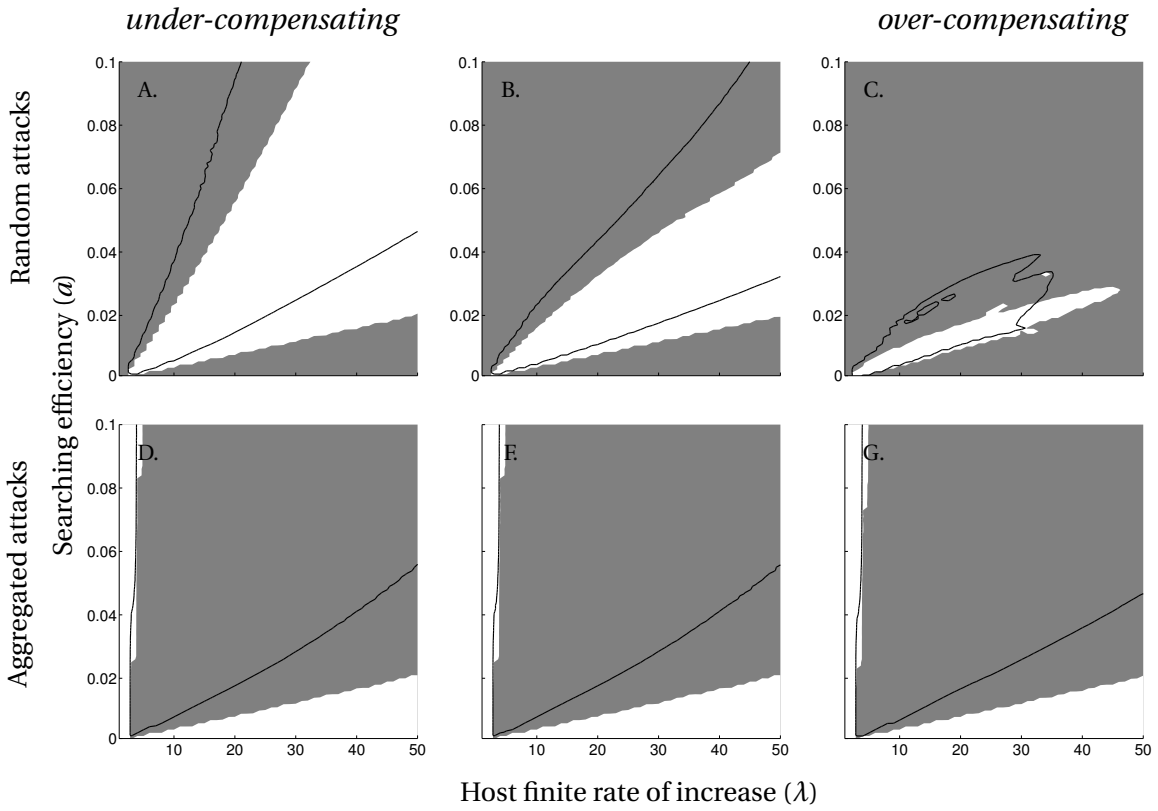

Figure 4.5: Parasitoid extinction area ($P(E) > 0.5$) of various distributions of parasitoid attacks and various severities of host competition, with CSD (bordered by the black line) or without CSD (grey) as function of parasitoid searching efficiency (a) and host finite rate of increase (λ). Other parameter values: $K = 5000$, $w = 25$, $F_0 = 500$.

CHAPTER

5 What alters CSD impact on parasitoid populations?

Article 5

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Résumé

La survie des petites populations de parasitoïdes introduites pour la lutte biologique est un important sujet d'études à la fois théoriques et empiriques. L'interaction entre l'hôte et le parasitoïde peut affecter les facteurs écologiques et génétiques qui peuvent conduire des petites populations de parasitoïdes à l'extinction. Le *Complementary sex determination* (CSD) est un système de détermination du sexe propre aux hyménoptères qui impose un important fardeau génétique à travers la production de mâles diploïdes non viables ou stériles lorsque la diversité génétique est faible dans la population. Le CSD peut induire un « Diploid male vortex» dans les populations non - parasitoïdes, mais a un impact complètement différent dans les populations de parasitoïdes : il stabilise les populations de parasitoïdes efficaces, prévenant leur extinction, et induit des extinction déterministes chez les parasitoïdes peu efficaces. Dans cette étude , nous adoptons une approche théorique plus large pour analyser les facteurs ou traits d'histoire de vie qui peuvent altérer les conséquences du CSD chez les parasitoïdes. Nous utilisons un modèle éco-génétique décrivant un système hôte - parasite où le parasitoïde porte le CSD, et étudions les effets i) des mutations sur le gène *csd*, ii) de la stochasticité environnementale et iii) démographique, iv) des variations dans le nombre de parasitoïdes produits par hôte, v) d'un sex-ratio biaisé en faveur des mâles ou des femelles et vi) de la productions mâles par des femelles fécondées par un mâle diploïde. Nous montrons qu'un nombre trop faible de femelles produites par hôte et des mutations sur le gène *csd* peuvent altérer l'effet stabilisant du CSD sur les parasitoïdes efficaces. Cet effet stabilisant serait au contraire favorisé par la grégarité et les accouplements consanguins. Contrairement aux les vortex d'extinction classique dans les populations non parasitoïdes, les extinctions induites par le CSD ne sont pas plus rapides ou plus nombreuses avec une

forte stochasticité démographique ou environnementale. Enfin, nous montrons que globalement, les impacts démographiques du CSD restent qualitativement similaires quels que soient les traits d'histoire de vie considérés pour les parasitoïdes, et qu'ils sont donc sur ce point très robustes. Ces résultats soulignent l'importance de considérer l'interaction interspécifique dans la prédiction du risque d'extinction d'une population, et démontrent que les impacts démographiques du CSD chez les parasitoïdes ne sont pas limités à des traits d'histoire de vie ou des conditions environnementales et démographiques particulières.

Abstract

The survival of small, introduced parasitoid populations in biological control attracts many theoretical and empirical studies. The interaction between host and parasitoid may affect the features of parasitoid extinctions in small populations, and the ecological and genetic factors that may drive these extinctions. Complementary sex determination (CSD) in Hymenoptera is a sex determination system that imposes substantial genetic load through the production of unviable or sterile diploid males when genetic diversity is low in the population. In parasitoids, CSD stabilizes high efficiency parasitoids and induces deterministic extinctions in low ones. These impacts do not emerge in non-parasitoid populations. In this study, we take a broader theoretical approach to analyze the factors or life history traits that may tamper with the particular consequences of CSD in parasitoids. We investigate the impacts of i) mutations on the *csd* gene, ii) environmental and iii) demographic stochasticity, iv) variations in the number of parasitoids produced per host, v) male-biased or female-biased sex ratio, and vi) male productions by females mated by a diploid male. CSD impact is robust to various life history traits hypothesis, including gregarity and sib-mating. Mate-finding difficulties, developmental mortality in parasitoids and mutations on the *csd* gene may tamper with the stabilizing impact of CSD. These findings underline the importance of considering interspecific interaction in the prediction of population extinction risk, and demonstrate that the demographic impacts of CSD are rather robust to the variety parasitoid life history traits.

KEYWORDS: Hymenoptera; *Complementary sex determination*; host-parasitoid interaction; genetic load; extinction; Diploid Male Vortex; parasitoid; environmental stochasticity; demographic stochasticity; mutations; biased sex-ratios; pseudo-virginity; gregarity; sib-mating.

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1. Introduction

Parasitoid insects have awakened the interest of both empirical and theoretical biologists for many years (Godfray, 1994; Hassell, 2000). The tightness and specificity of their interaction with the host make them valuable for the biological control of insect pest (van Driesche et al., 2008) and shape ecological networks (Ings et al., 2009). Most parasitoids (78%) belong to the Hymenoptera order (Eggleton and Belshaw, 1992) which is entirely composed by insects with haplodiploid sex determination systems (Heimpel and de Boer, 2008).

Haplodiploid organisms are generally less affected by inbreeding depression than diploid organisms, because deleterious mutations are expressed by haploid males and purged by natural selection (Henter, 2003). Various mating strategies in parasitoid wasps (from full to partial sib-mating) have evolved under the condition of a low level of mutational load in the population (Smith, 2000; Werren, 1989; Getz et al., 1992). However, the single-locus *Complementary Sex Determination* (CSD) complicates this picture by introducing a particular form of inbreeding depression (Whiting and April, 1943; Heimpel and de Boer, 2008; Zayed and Packer, 2005). CSD is a sex determination system ancestral to the Hymenoptera, which has been described in four of the five largest Hymenopteran families. Sexual development in wasps with CSD depends on the complementarity of alleles at the *csd* locus: heterozygous develop in females, and hemizygous (haploid) develop in males. If homozygous, eggs develop into diploid males, generally unviable or sterile (Cook and Crozier, 1995; van Wilgenburg et al., 2006; Heimpel and de Boer, 2008). In small, non-parasitoid populations, theoretical studies showed that CSD may provoke the apparition of an extinction vortex that can be of stronger intensity than inbreeding depression in diploid populations (Zayed and Packer, 2005).

In parasitoid wasps, CSD may have the adverse effect: the presence of this genetic load damps the cycles produced by a strong parasitoid searching efficiency and promotes the persistence of parasitoid populations (article 4). When the parasitoid searching efficiency is low, CSD may drive the population to extinction, following a process that differs from the classical Diploid male vortex in its limited dependency to parasitoid genetic diversity and initial abundance. In article 4, the balance between CSD-induced persistence and CSD-induced extinction is in favor of persistence: on a large range or parameter values designed to encompass the diversity of host-parasitoid dynamics, the global probability of extinction of parasitoid populations is decreased by the presence of CSD. Nevertheless, the extent to which CSD beneficial and deleterious effects on population persistence are quantitatively or qualitatively affected by the hypotheses that underlay parasitoid biology in the theoretical model remains unknown.

The diversity of parasitoid biology is impossible to represent in a single theoretical model. In article 4 we considered relatively simple assumptions for parasitoid biology, and the aim of the present work is to explore a wider variety of parasitoid life histories. Among them, we will consider factors that are known to affect population extinction risk when combined with a classical extinction vortex (that is mutations, demographic and environmental stochasticity)(Gilpin and Soule, 1976; Frankham, 2005) and factors that may reflect the diversity of parasitoid biology (gregarity, fertilization rate, forms of diploid male sterility) (Godfray, 1994; Heimpel and de Boer,

2008). Our central question is how much CSD impact on parasitoid demography relays on the hypothesis taken for its biology, and whether its impact on parasitoid dynamics remains similar considering the variety of biological hypotheses previously cited.

Gene flow, under the form of migration between subpopulations or apparition of new viable alleles, renew the genetic diversity and is an important component of population viability (Frankham, 2005; Reed et al., 2003). Hein et al. (2009) demonstrated that in theory, mutations on the *csd* locus could counter the Diploid Male Vortex in non-parasitoid populations. However, the relation between genetic diversity at the *csd* locus and probability of extinction is more complex in parasitoid populations and not necessary positive, so the result of mutations at the *csd* locus on parasitoid demography is not easy to foresee.

Genetic and environmental stochasticity are key components of classical extinction vortices (Gilpin and Soule, 1976) and usually interact with genetic factors to drive populations to extinction (Frankham, 2005; Tanaka, 2000; Higgins and Lynch, 2001). In contrast with classical extinction vortices, strong density-dependent processes within the host parasitoid interaction drive CSD impact on the demography of parasitoid populations article 4. Moreover, Wilson and Hassell (1997) investigated the impact of demographic stochasticity and noise within a deterministic host parasitoid model, and concluded that these impacts depend on parasitoid attack rate a parameter which is also crucial to determine the qualitative impact of CSD on parasitoid dynamics.

The variety of parasitoid uses of their host is rarely considered in classical host-parasitoid models. In solitary species parasitoids lay a single egg in each host, but in gregarious species several eggs are laid and develop in the same host (Godfray, 1994). Hosts may also be killed without any parasitoid emerging because of developmental defects or because the host was consumed rather than used to lay eggs (Godfray, 1994). In classical deterministic models, the average number of adult parasitoid emerging from each host parasitized (further referred as the parasitoid conversion rate) has only a scaling effect on the parasitoid dynamics and is often overlooked (Hassell, 2000). However, CSD decreases parasitoid conversion rate by inducing the production of unviable individuals, and this decrease depends on the population's allelic diversity at the *csd* locus. Moreover, in gregarious parasitoids mating often occur locally, sometimes strictly between sibs (Godfray, 1994; Elias et al., 2010a) but not necessarily (Antolin and Strand, 1992; Gu and Dorn, 2003). In this situation, sex ratio evolves toward a female bias for two reasons: brothers may compete for the same female (Local Mate Competition) and, when inbreeding is the norm, mothers are more related to their daughter than to their sons (Hamilton, 1967; Taylor and Bulmer, 1980; Heimpel and de Boer, 2008). In haplodiploids, this evolution translates by an increase of the fertilization rate. Conversely, a number of constraints, such as mate-finding difficulties and sperm shortage (Godfray, 1990; Boivin, 2013) may prevent parasitoid females to realize the ideal fertilization rate. Because CSD only affects the fertilized offspring, parasitoid fertilization rate could tamper with its demographical consequences. Gregarious parasitoid species may combine the emergence of multiple parasitoids per host, an elevated fertilization rate and an extremely low genetic diversity due to recurrent sib-mating. The demographic consequences of CSD in such parasitoids species may vary from the classical representation of a solitary and outbreeding parasitoid.

When CSD is present and diploid males are sterile, they may either transfer their gene and lead to the production of unviable or sterile triploid offspring, as in *Cotesia vestalis*, or not transfer their genes and lead to pseudo-virgin females that produce haploid males broods (such as in *Habrobacon sp.* and *Venturia canescens*) (Heimpel and de Boer, 2008). Whether in parasitoid or in non-parasitoid populations, the presence of diploid males imposes a huge weight on the population and increases its extinction risk. Heimpel and de Boer (2008) considered sterility with or without gene transfer as equivalent, but Chuine (2014) presented the ability to produce a larger number of haploid males when mated with a diploid as an evolution to reduce CSD fitness cost and impact on population extinction risk. Because the risk of mating with a diploid male is function of the ratio between haploid and diploid males, the impact of sterile diploid males ability to transfer gene may depend on the average fertilization rate in the population.

All these points will be addressed through several eco-genetic models derived from article 4 as an investigation to assess whether the theoretical impact of CSD may hold when the variety of parasitoid biology is considered. We conclude that the qualitative impacts of CSD on parasitoid dynamics - the damping effect of CSD on parasitoid cycles and its increase of low searching efficiency parasitoid extinction risk - are robust to a wide variety of biologically meaningful hypotheses. Nevertheless, frequent mutations and the association of strong demographic stochasticity and high genetic diversity may ultimately neutralize the damping effect of CSD on cycles. Frequent mutations may also neutralize the increase of low searching efficiency parasitoids extinction risk another impact of CSD.

2. Methods

2. 1 Original model

We used the eco-genetic model presented in article 4 that describes the demography of parasitoid populations with CSD (with either unviable or sterile diploid males). This model is composed of two phases: one deterministic which describes the abundance of hosts, fertilized and unfertilized parasitoid offspring according to abundances at the previous generation; and one stochastic which describes the random attribution of mates and offspring, the offspring genotype and discriminate diploid males and unviable triploids from fertilized offspring (see article 4 for more details).

We modified various aspects of this model to investigate the impact of mutations, demographic and environmental stochasticity, male production by females mated to a sterile diploid male. The effect of the fertilization rate and of the number of offspring per parasitized host have been studied by using the original model, assuming a proportion of unfertilized offspring (s_0) that varies between 0.1 and 0.9, and a parasitoid conversion rate that may vary between 0.2 and 10.

2. 2 Mutations

The "mutation" modification allows new alleles to appear at the *csd* locus: the frequency of their apparition depends on the probability of mutation, e.
When parasitoid offspring inherit their genotype from their parents, each allele has a probability e to mutate and to appear as a new viable allele. Thus, females have twice as many chances as males to carry a mutation, and may carry two mutations. A maximum allelic diversity is set to 100 alleles, above which the population ability to mutate is neutralized: at this genetic diversity, diploid male production is already a marginal phenomenon.

2. 3 Environmental and demographic stochasticity

Environmental stochasticity is modeled as a stochastic variation on the parameters a (parasitoid searching efficiency) and λ (the host finite rate of increase). These two parameters are initially set with an initial value λ_0 and a_0 respectively. At each generation, the λ and a parameter values used for the deterministic part of the model are drawn on Gaussian distributions (truncated on 0) of mean λ_0 (resp. a_0) and of standard deviation $\lambda_0\epsilon_s$ (resp $a_0 epsilon_s$). Thus, ϵ_s is the parameter that controls the intensity of the environmental stochasticity by affecting the deviation allowed around the initial value for both parameters.

The demographic stochasticity is modeled as a stochastic variation of the number of offspring produced by each parasitoid female. The deterministic part of the model sets the total number of offspring at generation n from the number of hosts, male and female parasitoids at generation $n-1$, which gives the average number of haploid and diploid offspring per parasitoid females ($\mu_h = \frac{\text{haploid parasitoids at n}}{\text{parasitoid females at n-1}}$ and ($\mu_d = \frac{\text{diploid parasitoids at n}}{\text{parasitoid females at n-1}}$). When $s_0 = 0.5$, $\mu_h = \mu_d$.In the original model one mother was randomly attributed to each offspring, but in the new model each female draw a number of male and female offspring on a Gaussian distribution (truncated on 0) of mean μ_h and of standard deviation ϵ_d. The parameter ϵ_d thus sets the intensity of the demographic stochasticity. The total number of diploid and haplodiploid offspring is updated as the sum of each female's offspring, and host abundance is updated accordingly. This method underestimates the interference between parasitoids that search for a host, because females that realize a high number of offspring do not make hosts unavailable for their conspecifics.

2. 4 Representations

In order to encompass the impact of CSD on parasitoid persistence in a variety of dynamical contexts, we represented the average probability of extinction ($P(E)$) for 5000 parameter sets, resulting from the combination of 50 values for λ (1-50) and 100 values for a (0.001-0.1). Each parameter set is simulated for 3000 generations. The probability of extinction associated to each parameter set is the ratio of the simulations that lead to parasitoid extinction (before 3000 generations) on the total number of simulations per parameter sets (5000). A mean equilibrium allelic diversity (w_e) was assessed for each parameter set from simulations that went without extinction. The average probability of extinction was represented as a function of the focus parameter (e, ϵ_s,ϵ_d, d or s).

3. Results

3. 1 Mutations

As seen in figure 5.1.a, the probability of extinction of a parasitoid population with CSD increases with the probability of viable mutations on the *csd* gene. This result is unusual: most theoretical work on genetic load, even on CSD in non-interacting populations, concludes on a positive impact of gene flow and viable mutations on population persistence.

In parasitoid populations, parasitoid efficiency is roughly the main parameter that controls the impact of CSD: high efficiency populations that undergo large amplitude cycles are stabilized by CSD which reduces their probability of extinction; conversely, low efficiency populations are driven to extinction by the production of diploid males. Mutations on the *csd* locus increase the probability of extinction of high efficiency populations, and decrease the probability of extinction of low efficiency populations (figure 5.1.b). This effect could have been neutral. However, the probability of extinction of high efficiency populations increases faster with e than the probability of extinction of low efficiency populations decreases (figure 5.1.b). Behind this result lay the parasitoid abundances of both populations: high efficiency populations are periodically very numerous due to high amplitude cycles, so mutations are more likely to appear than in low efficiency populations that have a low and rapidly decreasing abundance.

Mutations also increase the equilibrium allelic diversity of persistent parasitoid populations, even when the probability of mutation is low (figure 5.1.c, 5.1.d). Subsequently, while the *csd* equilibrium diversity was of 2 to 10 alleles without mutations, it is of 2 to 18 alleles for $e = 10^{-5}$, which is closer to the genetic diversity observed in natural parasitoid populations.

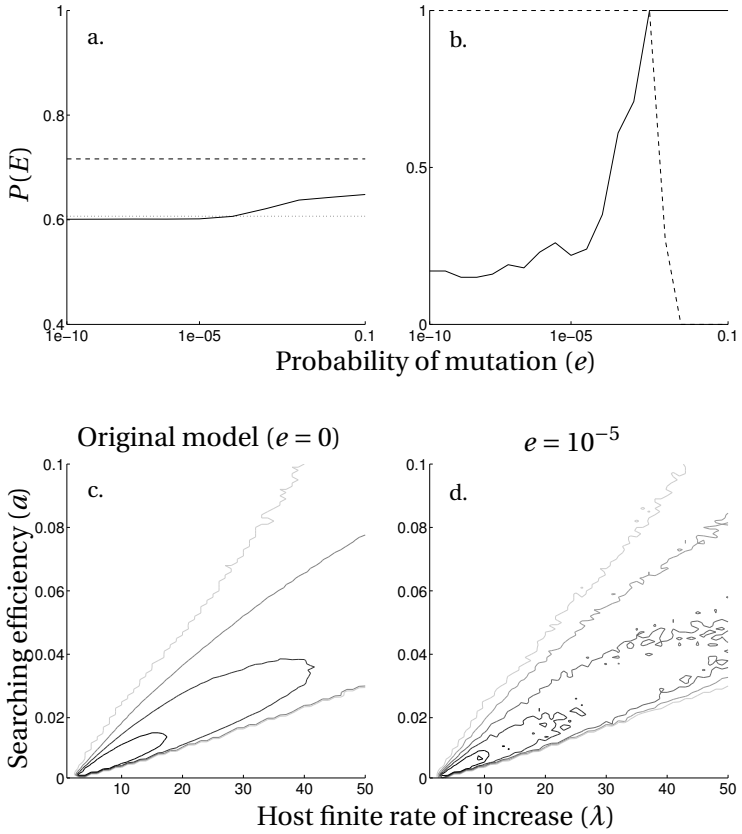

Figure 5.1: Effect of mutations on the *csd* locus on the persistence and allelic diversity of parasitoid populations. a) Plain black line: mean probability of extinction for 5000 a-λ combinations($0.01 \leq a \leq 0.1$, $0.1 \leq \lambda \leq 50$) depending on the probability of mutation (e); dotted (resp. dashed) lines represent the mean probabilities of extinction of populations without mutation, with (resp. without) CSD. b) Probability of extinction of two parasitoid populations, one with high efficiency (plain line) and the other with low efficiency (dashed line). Fig b and c: Contour plot of iso-allelic diversity on the *csd* locus (after 3000 generations) for persistent parasitoid populations without mutations (b) or with relatively rares mutations (c, $e = 10^{-5}$). The four grey levels represent (from lighter to darker): $w_e > 2$; $w_e > 4$; $w_e > 6$; $w_e > 8$; the fifth darker grey level on fig.c represent $w_e > 15$. On all figures, the initial allelic diversity is $w = 10$.

3. 2 Environmental and demographic stochasticity

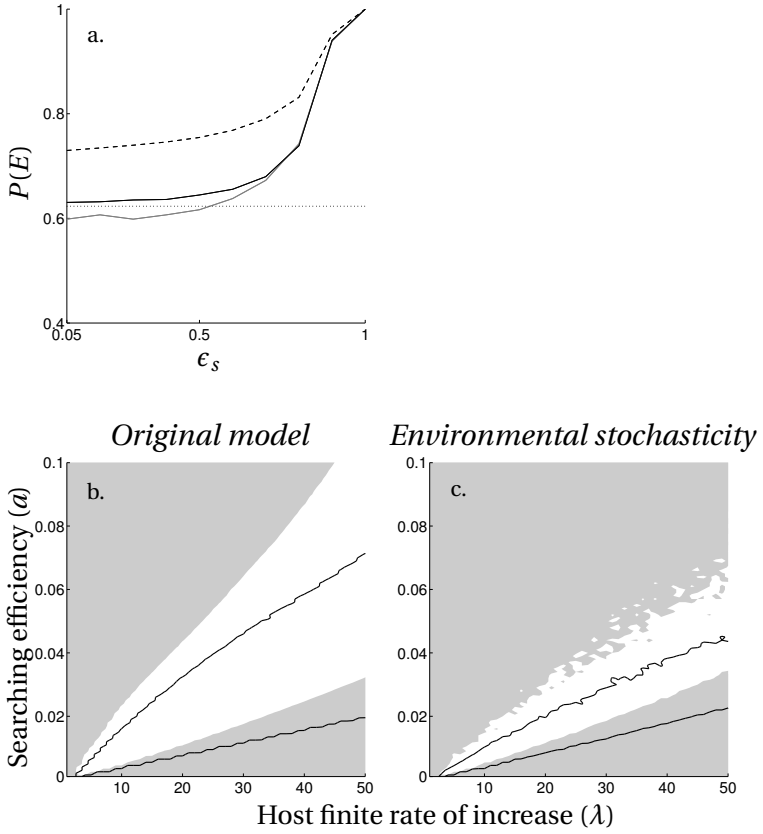

Figure 5.2: Effect of the environmental stochasticity on the probability of extinction of parasitoid populations. a) Mean probability of extinction for 5000 a-λ combinations ($0.01 \leq a \leq 0.1$, $0.1 \leq \lambda \leq 50$) depending on the intensity of the environmental stochasticity ϵ_s, for populations with CSD (plain black line for $w = 20$ and plain grey line for $w = 5$) and without CSD (dashed line); the dotted line represent the mean probability of extinction of populations with CSD but no environmental stochasticity. b) and c) Extinction area ($P(E) > 0.5$) for parasitoid populations without CSD (bordered by the black line) and with CSD (grey filling) without environmental stochasticity (b) or with a moderate level of environmental stochasticity (c, $\epsilon_s = 0.2$) For both b) and c), the initial diversity is $w = 20$

High levels of environmental stochasticity increase the probability of extinction with and without CSD, and reduce the deviation between the probabilities of extinction of populations with and without CSD (figure 5.2.a). A low initial genetic diversity promotes the persistence of

parasitoid populations with CSD only when the environmental stochasticity is moderate (figure 5.2.a). Environmental stochasticity dramatically endangers high efficiency parasitoids with and without CSD, but the impact of CSD on the persistence of parasitoid remains qualitatively similar: high efficiency parasitoids are more persistent and low efficiency parasitoids less persistent.

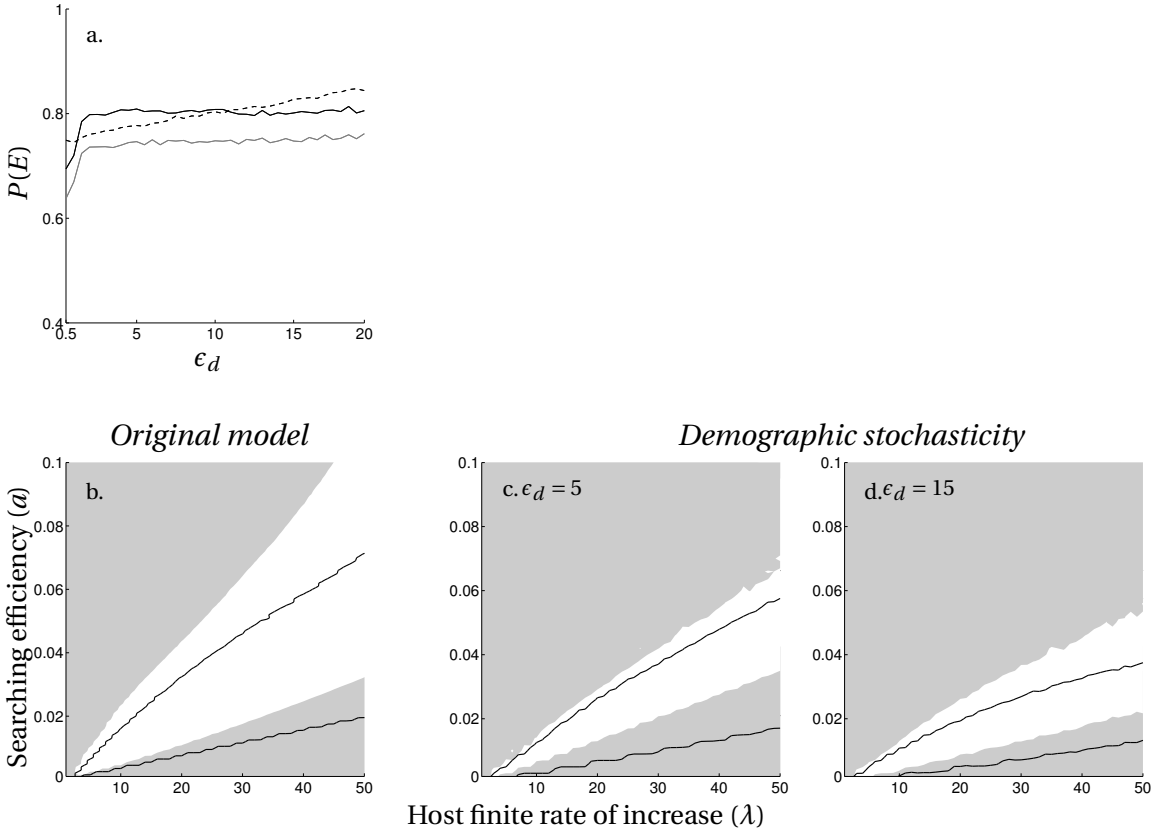

Figure 5.3: Effect of the demographic stochasticity on the probability of extinction of parasitoid populations. a) Mean probability of extinction for 5000 a-λ parameter set ($0.01 \geq a \leq 0.1$, $0.1 \geq \lambda \leq 50$) depending on the intensity of the demographic stochasticity ϵ_d, for populations with CSD (plain black line for $w = 20$ and plain grey line for $w = 5$) and without CSD (dashed line). b), c) and d) Extinction area ($P(E) > 0.5$) for parasitoid populations without CSD (bordered by the black line) and with CSD (grey filling) without demographic stochasticity (b), with a moderate demographic stochasticity (c, $\epsilon_d = 5$) and with a strong demographic stochasticity (d, $\epsilon_d = 15$) For b),c) and d), the initial diversity is $w = 20$.

Conversely, the impact of demographic stochasticity on parasitoid populations depends on the presence or absence of CSD. In parasitoid populations without CSD, demographic stochasticity progressively increases the average extinction risk (figure 5.3.a) by driving cyclic parasitoids to extinction (figure5.3.b). Parasitoid populations with CSD undergo a brutal increase of their extinction risk when the demographic stochasticity increases from low intensities (figure 5.3.a, as ϵ_d increases from 0.5 to 1), which reflects that demographic stochasticity drives

most cyclic parasitoid populations to extinction. Further increase of the demographic stochasticity has no more impact on parasitoid populations with CSD, while it continues to drive cyclic parasitoids without CSD to extinction. Low initial allelic diversity remains equally beneficial to parasitoid persistence independently of the level of demographic stochasticity. Low efficiency parasitoid populations appear to be more persistent at strong demographic stochasticity, with or without CSD, but this could be an artefact of the model construction: assuming a female's realized fecundity is independent of the realized fecundity of its conspecifics underestimates the impact of interference between females, and thus over-estimate their efficiency. The brutal increase in parasitoid extinction rate reflects the massive extinction of cyclic parasitoid populations with CSD. These extinctions occur before the damping effect of CSD may impact, at the bottom of the first cycle. Stability in parasitoid extinction risk at further demographic stochasticity can be attributed to CSD damping effect, and particularly to its increase of the minimum value of parasitoid cycle.

3. 3 Gregarity/host consumption and fertilization rate

The parasitoid conversion rate (d) and proportion of unfertilized offspring (s_0) are key parameters to the demographic effect of CSD in parasitoids: depending on their value, CSD may increase or decrease the average extinction risk of parasitoid populations (figures 5.4.a, 5.4.b). For $d > 0.6$, CSD always decreases the probability of extinction of parasitoid populations (figures 5.4.a). High values of d ($d > 1$) increase the probability of extinction of parasitoid populations for parasitoids with and without CSD. Conversely, for low conversion rates ($d \leq 0.6$), CSD increases the probability of extinction of parasitoid populations up to +22.19% (for $d = 0.2$, on 5000 populations). This increase go through a reduction of the damping effect of CSD on cyclic high efficiency parasitoid, which reduces the extent to which CSD decreases the high efficiency area of extinction (figure 5.4.d). A too small proportion of host killed that effectively produce parasitoids sever the link between unparasitized host and increased parasitoid growth rate, which is a key feature of the beneficial impact of CSD on the persistence of cyclic parasitoid populations.

When the proportion of unfertilized offspring falls below 0.5 (a mother produces more diploid than haploid offspring) CSD always decreases the average risk of extinction of a parasitoid population, particularly when the allelic diversity is low (figure 5.4.b). The tendency is reversed for high proportions of unfertilized offspring (strongly male biased sex ratios, $s_0 > 0.7$) (figure 5.4.b). When a high proportion of haploid males is produced, the production of viable females after mating with a male that share a common allele is particularly low. When the population is at low abundance (as it happens recurrently when the population cycles), a low production of diploids increases the risk to be unable to produce any viable female. As for a low parasitoid conversion rate, a strong proportion of unfertilized offspring reduces the damping effect of CSD on high efficiency parasitoids (figure 5.4.d). A low conversion rate and a strong proportion of unfertilized offspring have very close consequences on parasitoid dynamics: both reduce the efficacy with which the hosts killed are transformed into viable parasitoid females.

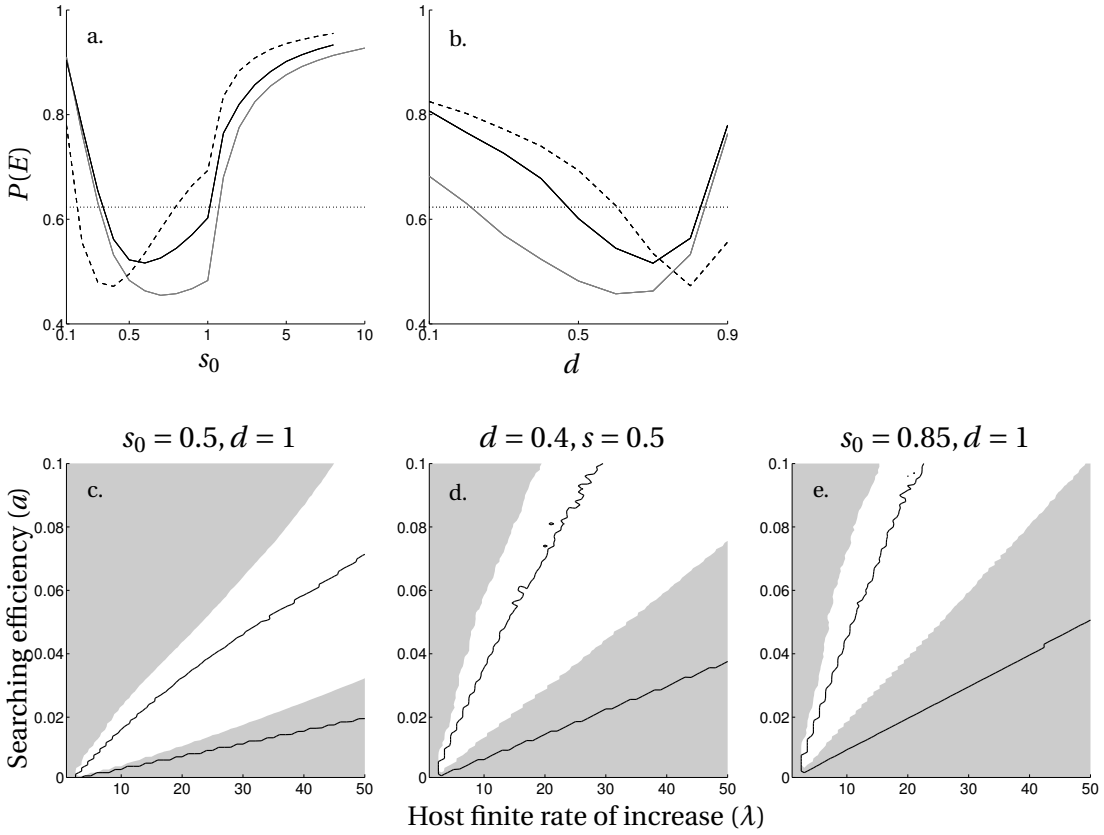

Figure 5.4: Effect of the proportion of unfertilized offspring and the parasitoid conversion rate on the probability of extinction of parasitoid populations. a) and b)Mean probability of extinction for 5000 a-λ combinations ($0.01 \leq a \leq 0.1$, $0.1 \leq \lambda \leq 50$) depending on a) the number of parasitoid produced per host killed (*i.e.* the parasitoid conversion rate, d) and b) the proportion of offspring left unfertilized s_0, for populations with CSD (plain black line for $w = 20$ and plain grey line for $w = 5$) and without CSD (dashed line); the dotted line represent the mean probabilities of extinction of populations with CSD $s_0 = 0.5$, $d = 1$, $w = 20$. b) , c) and d) Extinction area ($P(E) > 0.5$) for parasitoid populations without CSD (bordered by the black line) and with CSD (grey filling) with equilibrated sex ratio and a 1:1 conversion rate (b), with a low conversion rate (d, $d = 0.4$) and with a high proportion of unfertilized offspring (e, $s_0 = 0.85$). For b), c) and d), the initial diversity is $w = 20$

3. 4 Pseudo virginity and sex ratio when diploid males are sterile

Diploid male sterility with gene transfer (that produces unviable triploid offspring) or without gene transfer (that leads to pseudo-virgin females) makes no dramatic difference on parasitoid demography when the proportions of fertilized and unfertilized offspring are equal. Sterility without gene transfer only slightly reduces the population extinction risk (< 1%), because sterile diploid males are diluted among the haploid males produced by pseudo-virgin females (figure 5.5.a). When the proportion of unfertilized offspring decreases (a female produce more

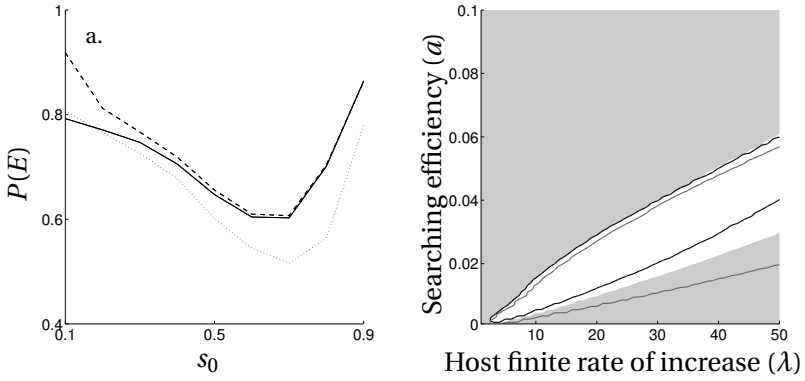

Figure 5.5: Effect of diploid male ability to transfer genes depending on the proportion of unfertilized offspring on the probability of extinction of parasitoid populations. a) Mean probability of extinction for 5000 a-λ combinations ($0.01 \leq a \leq 0.1$, $0.1 \leq \lambda \leq 50$) depending on the proportion of offspring left unfertilized s_0, for populations with CSD and sterile diploid males that sire unviable triploid offspring (gene transfer) (plain black line), with CSD and sterile diploid males that induce pseudo virginity (no gene transfer) (dashed line), and with CSD and unviable diploid males (grey line). b) Extinction area ($P(E) > 0.5$) for parasitoid populations with CSD and unviable males (bordered by the grey line), with CSD and sterile diploid males that transfer genes (bordered by the black line), with CSD and sterile diploid males that do not transfer genes (grey filling) for low proportions of unfertilized offspring $s_0 = 0.2$. For all subfigures, $w = 20$.

diploid than haploid offspring), the dilution effect is on the rise. Pseudo-virginity reduces the population probability of extinction up to -13.2% compared to sterility with gene transfer (figure 5.5.a). Because the proportion of diploid offspring increases, diploid males become more abundant and the risk of mating with one of them increases. This risk is rapidly compensated by the haploid male production of females coupled with a diploid male if diploid males do not transfer their gene, but not if diploid males sire unviable offspring. Subsequently, the extinction risk of low efficiency parasitoid populations is lower with sterile diploid males that do not transfer their genes than with sterile diploid males that do (figure 5.5.a). However, the extinction risk of high efficiency parasitoids is not affected by the biology of diploid males (viability, sterility, etc.) when the proportion of unfertilized offspring is low (figure 5.5.b). When sterile diploid males do not transfer their genes, mating with a diploid male may be considered as a form of constraint that can be freed by increasing the fertilization rate.

4. Discussion

In parasitoid population, CSD is a genetic load that can promote population persistence, a result in contradiction with its effect on non-interacting populations (Zayed and Packer, 2005), and with the classical effects of genetic load in non-interacting populations (Frankham, 2005; Keller and Waller, 2002; Gilpin and Soule, 1976). In article 4 we presented the mechanisms behind this effect: a stabilization of parasitoid cycles for high efficiency parasitoid populations. But CSD is not fully beneficial for population persistence: low efficiency parasitoid populations can be driven to extinction by CSD, following a mostly demographic process in which CSD reduces their relative host attack rate. With this work, we show that these phenomena are robust to many variations on the hypotheses that underlay parasitoid biology or environment: parasitoids may be able to mutate at the *csd* locus, they may suffer strong environmental or demographic stochasticity, produce unequal proportion of haploid and diploid offspring and more (or less) than one parasitoid per host, females may be able to produce supplementary haploid offspring or not when they mate with a diploid male: the qualitative effects of CSD on the population remain similar. In addition to these factors, we tested a variety of others (not presented in this paper) such as interference between parasitoids, asynchronous host and parasitoid generations and position of the host density dependence after (rather than before) parasitism, with the same conclusions (unpublished results). We confirm that rather than issuing from a particular set of conditions in host-parasitoid populations, the theoretical impact of CSD is a result that emerges from the interaction itself.

However, CSD is not necessarily always as efficient at damping parasitoid cycles. Particularly, the intensity of the damping effect relays on: 1) the level of disturbance in parasitoid growth during the growing phase of the cycles (as witnessed by the impact of mutations), and 2) the relation between unparasitized host and later increase in female parasitoid production (as witnessed by the impact of parasitoid conversion rate and fertilization rate). The extent of CSD-induced extinction on low-efficiency parasitoids relays on the biology of diploid males (unviable, sterile with gene transfer or without gene transfer), particularly when females fertilize a large proportion of their offspring.

4. 1 Mutations, migrations and the two fold aspects of genetic diversity on the *csd* locus.

The role of genetic diversity at the *csd* locus on the persistence of parasitoid populations is quite complex. In low efficiency parasitoid populations, maintaining the allelic diversity above a threshold is critical for population persistence (article 4). Mutations may prevent the extinction of low efficiency populations only if frequent enough ($e > 10^{-3}$) to counter the decay of genetic diversity by drift. Conversely, high efficiency parasitoid populations suffer from an initially elevated genetic diversity because parasitoid go extinct before the damping effect of CSD may impact (article 4). Mutations increase the extinction risk of high efficiency populations with CSD because they introduce a disturbance in parasitoid cycles that translates into an increase of parasitoid growth rate. Because mutations are more likely to appear in abundant parasitoid populations, this disturbance occurs near the apex of parasitoid cycles: the boost of parasitoid

growth at this point is particularly likely to drive the host, and therefore the parasitoid, to extinction.

The rate of apparition of favorable mutations at a locus depends on the rate of DNA replication errors (Kimura, 1968), which may vary between species and locii but typically ranges between 10^{-6} and 10^{-8} errors per base (Kunkel, 2004). Not all mutations lead to the apparition of a new viable sex allele at *csd* locus, so the rate of mutations in natural populations is likely to not alter the extinction risk of parasitoid populations. It would be tempting to expand the interpretation of our results to the impact of migration from genetically different populations, a phenomenon that could be more frequent. On this assimilation, one must take caution: the impact of mutations in high efficiency populations lays on the relation between parasitoid abundance and likeliness of apparition of a new gene. Thus, migrations could be assimilated to mutations only if they are more likely to happen in abundant population, for instance as a result of pheromones of aggregation (French and Travis, 2001). Relatively to mutations, random migrations may induce less disturbances in parasitoid cycles because they could appear in less traumatic instants of parasitoid cycle.

4. 2 Demographic and environmental stochasticity: CSD and extinction in more realistic parasitoid populations

In parasitoid populations without CSD, demographic and environmental stochasticity drive high efficiency parasitoids to extinction, but do not affect low efficiency parasitoids. This result contrasts with the impact of stochasticity in non-interacting populations, in which populations with low growth rate and abundance would typically be driven to extinction. However, this result has been previously highlighted in host parasitoid systems by Wilson and Hassell (1997). They suggested that in low efficiency parasitoids, the density dependent processes related to the interaction quickly offset the effect of stochasticity.

In non-interacting populations, demographic stochasticity combines with genetic load to reduce genetic diversity and increase the extinction risk (Gilpin and Soule, 1976). In contrast, parasitoid populations that could be driven to extinction by CSD are little affected by demographic stochasticity. This point highlights the differences between CSD-induced extinctions, mostly piloted by deterministic processes, and classical extinction vortices, where stochastic processes play an important part (article 4). Host selection by Asobara tabida Nees (Braconidae, Alysiinae), a larval parasitoid of fruit inhabiting Drosophila species.

4. 3 Mating difficulties and developmental defects with CSD

In high efficiency populations, CSD damps the parasitoid cycles by decreasing parasitoid growth rate when high and increasing parasitoid growth rate when low. The increase of parasitoid growth rate when low lays on the increased host availability due to prior decrease in parasitoid population size (article 4). Figuratively speaking, parasitoids save up hosts to be used when they will be critically rare. When the relationship between saved up hosts and later gain in parasitoid females is severed, the damping effect of CSD on cyclic parasitoid populations is limited. This rupture may come either from low fertilization rate or from low number of parasitoid

produced per host.

Natural parasitoid populations often have high fertilization rates as the result of kin-competition (Hamilton, 1967; Taylor and Bulmer, 1980; Godfray, 1994). Low fertilization rates may however occur when mate-finding difficulties rise in the population and constraint a proportion of females to remain virgin (Godfray, 1990). In parasitoids, mate-finding difficulties alone are deleterious, because they induce a strong demographic Allee effect in low efficiency populations and may drive high efficiency parasitoids to extinction depending on the severity of the host intraspecific competition (article 2). Further, the combination of CSD and mate-finding Allee effect could be particularly dramatic because parasitoids are less able to withstand a low fertilization rate with CSD than without.

A low parasitoid conversion rate may rise when parasitoid females feed on unparasitized hosts, or when parasitoids encounter mortality at a late stage of their development (*e.g.* due to low host quality) (Godfray, 1994). Parasitoid females select hosts as function of their quality to maximize their offspring survival (van Alphen and Janssen, 1981) and when they feed, do so on low quality host (Godfray, 1994). Janssen (1989) studied the survival rate to parasitism of two parasitoids (*Leptopilina heterotoma* and *Asobara tabida*) and various host species. The proportion of viable parasitoid per host killed is usually high (60-90%) when the parasitoid is on its preferred host, but may drop to very low values (4-10%) when it is forced to develop on an alternative host (Janssen, 1989; Godfray, 1994). Thus, a highly efficient parasitoid transferred on a less suitable host may encounter both an increased developmental mortality, a change in the condition allowing its coexistence with the host, and a loss of CSD stabilizing impact. Such transfer are sometime inevitable in biological control, and may yield an important decrease of parasitoid abilities to control the host population (van Driesche et al., 2008).

4. 4 Gregarious parasitoids with CSD

Among natural parasitoid populations that have CSD, some are gregarious, reproduce through full sib-mating and have strongly female biased sex ratios (El Agoze et al., 1994; Cowan and Stahlhut, 2004; Elias et al., 2010b). The maintenance of sib-mating has been considered puzzling in species with CSD, because it reduces the genetic diversity and would drive the population into a Diploid Male Vortex (Heimpel and de Boer, 2008). In a previous study, we demonstrated that a low genetic diversity at the *csd* locus does not necessarily drive parasitoid populations into an extinction vortex (article 4). With this work, we show that assuming the parasitoid to be gregarious and to have a high fertilization rate maintain the damping effect of CSD on parasitoid cycles. In this frame, a low genetic diversity largely reduces population risk of extinction. In conclusion, their is no particular reason to dread the presence of a Diploid Male Vortex in gregarious parasitoids that mate between sibs, which explains why natural populations that are in this case easily persist.

However, the evolution of sib-mating under CSD remains surprising, because it is difficult to assess how it could be advantageous in terms of fitness. Partner choice with CSD is rather supposed to evolve toward the avoidance of kin and males that share a *csd* allele, as some parasitoid species already display these characteristics (Herzner et al., 2006; Metzger et al., 2010;

Chuine, 2014; Thiel et al., 2013). Further research will include the evolution of mate-choice and its demographic impact on parasitoid populations.

Through this work, we advance in the understanding of how the demo-genetic feedback articulates in parasitoids. Many results are counterintuitive: for instance, that the renewal of genetic diversity can endanger the survival of a population, whereas the reverse is always presented in populations considered without interactions. There is still a lot of work before understanding how and to which extent genetic load impacts the demography and evolution of species. Such knowledge is a key asset for the implementation of conservation programs for declining species, for the fight against invasive species, and for biological control.

CHAPTER

6 Demographic and evolutionary consequences of mate-choice under *Complementary Sex Determination* constraints

Article 6

AUTEURS : A. Bompard, T. Spataro

Abstract

Chez les individus sexués, le choix de partenaire vise à maximiser la fitness du parent qui choisit. Refuser des partenaires potentiels permet d'améliorer les chances de survie de sa descendance, mais peut également augmenter le risque de rester non accouplée. Chez les hyménoptères le système de reproduction est haplodiploïde, c.à.d que les gamètes non fécondés se développent en embryons mâles, tandis que les gamètes fécondés se développent en embryons femelles. Le coût lié au refus de partenaire se traduit principalement par une diminution de la capacité de fertilisation et une augmentation de la proportion de mâles dans la descendance. Le *Complementary sex determination* (CSD) est un mécanisme de détermination du sexe basé sur la complémentarité des allèles, qui induit la production d'individus non viables lorsque le père et la mère ont un allèle commun au gène *csd*. Le choix d'un partenaire en fonction de sa compatibilité allélique au *csd* peut donc procurer un avantage en terme de fitness, puisqu'il permet d'éviter qu'une fraction de la descendance fertilisée soit non viable. A l'échelle d'une population d'hyménoptères parasitoïdes, le CSD peut soit stabiliser les dynamiques hôte-parasitoïde et permettre à la population de persister, soit induire des extinctions, selon différentes caractéristiques associées à l'interaction hôte-parasitoïde. A l'aide de modèles mathématiques et éco-génétiques, nous étudions i) dans quelles conditions le choix de partenaire pour éviter les individus appariés au gène du *csd* peut évoluer dans une population d'hyménoptères, en considérant que le coût associé à ce choix est une diminution des capacités de fertilisation et donc une augmentation de la proportion de mâles dans la descendance et ii) quelles en sont les conséquences sur le risque d'extinction d'une population de parasitoïdes hyménoptères. Nous montrons que la diversité génétique au locus du *csd* joue un rôle majeur sur l'évolution du choix de partenaire chez les hyménoptères. Deux types de populations se distinguent : 1) les populations de parasitoïdes de faible efficacité, qui peuvent maintenir une forte diversité et évoluer

vers l'évitement des mâles appariés au locus du CSD, dans lesquelles les conséquences démographiques du CSD sont habituellement délétères pour la population mais peuvent être prévenues par le choix de partenaire, et 2) les populations de parasitoïdes de forte efficacité, qui ne peuvent maintenir qu'une très faible diversité génétique au locus du *csd* et ne peuvent évoluer vers un choix de partenaire efficace, dans lequel le CSD est stabilisant et permet à la population de persister. Nous discutons également de l'évolution de systèmes de reproduction consanguins ou thélytoques malgré la présence du CSD chez certaines populations naturelles de parasitoïdes. Nos résultats permettent de mieux comprendre la diversité des systèmes d'accouplement des hyménoptères parasitoïdes, ainsi que les facteurs d'extinction associé au CSD chez ceux-ci.

Abstract

In sexual individuals, the choice of a mate aims to maximize the fitness of the choosy parent, but may face costs, among which a greater difficulty to find the ideal partner. Hymenoptera have a haplodiploid reproductive system, *i.e.* the unmated gametes become males, while fertilized gametes become females. The cost of mate choice is thus mainly traduced by an increase in the proportion of males in the offspring. The Complementary sex determination (CSD) is a mechanism for sex determination based on the complementarity of alleles, which induces the production of non-viable individuals when father and mother share a common allele at *csd* gene. On the demography of parasitoid Hymenoptera, the CSD can either promote or induce extinctions. Using mathematical models and eco-genetic , we investigate 1) the conditions under which mate-choice to avoid assorted males can evolve in Hymenoptera and 2) what are the demographic consequences of this choice in parasitoid wasps. We conclude that two types of populations can be distinguished: 1) high genetic diversity populations, in which mate choice may evolve and prevent CSD induced extinctions, and 2) low genetic diversity populations in which mate-choice cannot evolve but CSD is usually stabilizing. We discuss the evolution of sib-mating and thelytoky despite the presence of CSD in some natural populations of parasitoids. Our results provide insight into the diversity of Hymenoptera parasitoids mating systems, but also into the extinction factors associated with CSD in these highly valuable populations.

KEYWORDS: mate-choice; parasitoid; *Complementary sex determination*; genetic load; extinction risk; fitness; selective value; evolution; demography.

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1. Introduction

The loss of genetic diversity is an important component in the extinction of small populations (Lynch et al., 1995; Lande, 1995; Reed et al., 2003). As population size becomes smaller, genetic diversity is lost by drift and the relatedness between individuals increases. Reproduction between related individuals (*i.e.* inbreeding) increases homozygoty. This may have a number of deleterious consequences on individual fitness - named inbreeding depression - , among which the increased expression of recessive deleterious mutations and the production of unviable individuals due to homozygoty at self-incompatible loci (Lynch et al., 1995; Frankham, 2005).

Behavioral processes such as mate selection may defuse the threat of inbreeding depression (Frankham, 2005; Pusey and Wolf, 1996; Manning and Thompson, 1984). The variety of mating systems goes from the strict avoidance sibs, assorted or mutated individuals as mating partners, where sub-optimal phenotypes are rarely expressed (Pusey and Wolf, 1996) to full sib-mating, where deleterious mutations are quickly expressed and purged by natural selection (Knapp and Rice, 1998; Crow and Kimura, 1970; Hedrick and Parker, 1997). Mate choice allows increases the probability of survival and/or of mating of the offspring (Kondrashov and Shpak, 1998) and, in naturally outbreeding species, maintain the genetic diversity and reduces the mutational load in the population (Kondrashov and Shpak, 1998; Manning and Thompson, 1984). Mutational load is directly linked to the strength of inbreeding depression, and inbreeding depression may compromise population persistence, so a careful partner selection benefits both the individual and the population.

In Hymenopteran species, the reproductive system is haplodiploid (females are diploid, and males are haploid), which induces the expression and purge of most of the deleterious mutations through haploid males (Goldstein, 1994; Henter, 2003; Heimpel and de Boer, 2008). However, the presence of *Complementary Sex Determination* (CSD) in numerous species may induce a specific form of genetic load (Zayed and Packer, 2005; Antolin et al., 2003; Heimpel and de Boer, 2008) The allelic set at the *csd* locus affects sexual development: heterozygous individuals develop as females, while hemizygous (resp. homozygous) individuals develop as haploid (resp. diploid) males (Cook, 1993). Haploid males are normal, but diploid males are generally unviable or sterile (Heimpel and de Boer, 2008; van Wilgenburg et al., 2006). Thus, when mating occurs between individuals whom share one allele at the *csd* locus ("assorted" individuals), half of the diploid offspring is unable to contribute to its parent's fitness because it is either sterile or unviable.

At the population level, CSD induces an extinction vortex in non-interacting Hymenoptera, and may drive low-density populations to extinction at a faster rate than classical mutational load in diploid species (Zayed and Packer, 2005). However, the complex density-dependent pattern in host-parasitoids interactions may lead to adverse results when CSD is in a parasitoid wasp interacting with its host (article 4). For parasitoids that have a strong impact on their host demography ("efficient"), CSD reduces the population's extinction risk by largely stabilizing the cyclic dynamics induced by the parasitoid. Conversely, for parasitoids that have a weak impact on their host, CSD increases the population extinction risk by reducing the population growth

rate. Because diploid male production may promote parasitoid population persistence, mate choice to avoid this production may conflict with population persistence. In natural populations, such conflicts between fitness benefits and population persistence generally arise on sexually selected traits (ornaments), and are known to have driven multiple species to extinction. This phenomenon is named evolutionary suicide (Zahavi, 1975; Hamilton and Zuk, 1982; Kokko et al., 2003; Jennions and Petrie, 1997).

Mate-choice has costs: rejecting potential mates increases the risk of remaining unmated, the time and energy employed to mate, and sometime the quality of available mating partners (Jennions and Petrie, 1997). The evolution of mate-choice lays on a balance between costs and benefits. In haplodiploid species, unmated females may nonetheless produce their male offspring, and parasitoid females may even begin oviposition before any attempt at mating (Godfray, 1994). When mated, sperm quantity and quality may limit the female's ability to fertilize their offspring. Delaying the mating attempts may results in mating with a male that already mated before and have a lower sperm quantity(Boivin, 2013; Damiens, 2005). Thus, mate choice in parasitoids wasps may constraint females to fertilize less eggs and thus to produce more haploid males than the optimal proportion (Godfray, 1990).

In a parasitoid wasp population with CSD and unviable diploid males, we investigate through mathematical modeling (1) the evolution of mate-choice strategy to avoid assorted males at the *csd* locus and (2) the impact of mate choice on population dynamics and extinction risks. In order to address the conditions for the evolution of mate choice strategies, we will consider the selective value of each strategy depending on the dominant strategy in the population. Then, to consider the impact of mate choice on population dynamics, we will proceed in two steps: first, we investigate the average proportion of female produced at each generation (as a proxy of the direct impact of mate selection on host-parasitoid dynamics). Second, we use a stochastic model to simulate the dynamics and extinction risk of parasitoid populations with CSD for various parasitoid efficiency and mate-choice strategies. We focus on the potential conflicts between fitness benefits and populations survival, and discuss the results in terms of evolution of mate-choice in parasitoid populations.

2. Methods

We consider a theoretical population of Hymenopteran parasitoids with CSD and unviable diploid males. Females mate only once but can make several attempts. During an attempt at mating, the female may reject a partner if it shares one of her alleles at the *csd* locus (an "assorted" male), but never reject a male that do not share one of her alleles (an "unassorted" male). Mate choice is assumed to only occurs on this criteria. The rejection of a potential partner has a cost that is expressed as a loss of the female's fertilization abilities, *i.e* an increase in the proportion of males she produces. Thus, mate choice strategies may take two directions: an increase of the discriminating abilities of the females (*i.e* of the probability of rejecting an assorted male) or a decrease of the cost of rejecting a mate. A female with its full fertilization abilities (that mates on its first attempt) will fertilize a proportion $s_1 = 0.5$ of their eggs, which leads to a balanced sex ratio in the population when no diploid males are produced.

2. 1 Selective value of mutations on traits associated with mate-choice

We define the selective value of a mate choice strategy as the advantage in terms of fitness it confers on a mutant using this strategy when arriving in a population in which residents use a different strategy. We assume that the total number of eggs available to a female does not depend on its mate choice strategy. In order to compute the selective value of a mate-choice strategy, we first compute the average fitness of the population depending on the presence of CSD and of its current mate-choice strategy.

2. 1. a Avergage individual fitness in an homogeneous population without mate-choice

Fitness is calculated as a female's average number of viable grandchildren in a population of fixed size N. This population is composed of viable females (F), viable males (M) and unviable diploid males (DM) in proportions that may vary depending on mate-choice strategies and CSD. The sex ratio of the population, defined as the proportion of females among viable individuals, is $s_r = \frac{F}{F+M}$ Each mother has b offspring, separated in viable females (b_f), viable males (b_m) or unviable diploid males (b_{dm}).
Each viable daughter will in turn mother a fraction of the offspring of the next generation, that is given by the ratio of the population size and the number of mothers $\frac{N}{Ns_r}$. Each son fathers $\frac{N}{N(1-s_r)}$ Thus, the number of grandchildren (*i.e.* the fitness) of a female is :

$$W = \frac{b_f}{s_r} + \frac{b_m}{1-s_r} \tag{6.1}$$

In optimal conditions, a female fertilizes $s_1 b$ eggs and leaves $(1-s_1)b$ eggs unfertilized. In a population without CSD, unfertilized eggs become viable males and all fertilized eggs become viable females, so the sex ratio is equal to s_1 and the average fitness in such population is:

$$W_{max} = \frac{s_1 b}{s_1} + \frac{(1-s_1)b}{1-s_1} = 2b \tag{6.2}$$

When CSD is present in the population, not all fertilized offspring will become viable females. If mating occurs completely at random in a population in which the allelic diversity at the *csd* gene is w, a female has a probability $P = \frac{2}{w}$ to encounter an assorted male. The frequency of each allele is assumed to be equal, since *csd* alleles are submitted to frequency-dependent selection. A female mated with an assorted male produce only half of her fertilized offspring as viable individuals, and the other half is homozygous and develops as unviable diploid males. On average, a mother produces $b_f = \left(1-\frac{P}{2}\right)s_1 b$ viable female offspring. The sex ratio in such a population is:

$$s_r = \frac{(1-\frac{P}{2})}{(1-\frac{P}{2})+1} \tag{6.3}$$

and according to eq. 1.1, the average fitness becomes:

$$W_{csd} = (1-\frac{P}{2})\frac{s_1 b}{s_r} + \frac{(1-s_1)b}{1-s_r} = b(2-\frac{P}{2}) \tag{6.4}$$

With a large allelic diversity, P is close to 0 and $W_{csd} = W_{max}$. Otherwise, we always have $W_{csd} < W_{max}$

2. 1. b Average individual fitness in a population with mate-choice

Two parameters are associated with mate choice:
- δ, the probability of rejecting mating when facing an assorted male, *i.e.*. the discriminating ability
- γ, the loss in fertilization capacity associated with rejecting a mate, *i.e.* the rejection cost

Mate-choice affects the proportion of male and female among the offspring, because a female that rejects a mate looses a fraction of its ability to fertilize offspring. As females multiply the attempts at mating, the cost of rejection adds up and the proportion of fertilized offspring becomes $s_t = s_1(1-\gamma t)$ (for t+1 attempts at mating). The female stop discriminating between mates when it ran out of fertilization capacity, *i.e* when $\gamma t \geq 1$.

Lets take f_x as the proportion of female offspring among the $s_1 b$ progeny and m_x as the proportion of haploid males among the $s_1 b$ progeny, so that:

$$\begin{cases} b_f = f_x s_1 b \\ b_m = (1-s_1)b + m_x s_1 b b_{dm} = s_1 b(1-f_x-m_x) \end{cases} \tag{6.5}$$

The proportions f_x and m_x depend on the discriminating abilities (δ) and rejection cost (γ) of the mother. Their equations will be presented in the next section.

In an homogeneous population, each mother has the same discriminating abilities and rejection cost, so each female produces, on average, the same proportion of males and females. The population sex ratio becomes :

$$s_r = \frac{b_f}{b_m + b_f} = \frac{f_x}{1 + m_x + f_x} \tag{6.6}$$

Thus, the average fitness become:

$$W_{chx} = \frac{f_x s_1 b}{s_r} + \frac{m_x s_1 b + (1-s_1)b}{1-s_r} = b(1 + m_x + f_x) \tag{6.7}$$

When mate-choice rarely occurs, m_x approach 0 and f_x approach $1 - \frac{P}{2}$, so $W_{chx} = W_{csd}$. Otherwise, we always have $W_{chx} > W_{csd}$

2. 1. c Individual fitness of a mutants and selective value of mate-choice strategy

We now consider that a mutant for the mate choice strategy appears in an otherwise homogeneous population. The mutation is assumed to be a small increase of the female's discriminating abilities relatively to the discriminating abilities of the populations ($\delta + 0.01$) or a small

reduction of the rejection cost relatively to the rejection cost of the population ($\gamma - 0.01$).
We assume that the modification in the population sex ratio due to the mutant or its progeny can be neglected, so we still have s_r is given by eq. 1.6 . However, the mutant produces proportions of males and females that may differ from the average individual in the population. $f\prime_x$ and $m\prime_x$ are respectively the proportions of viable males and viable females in the mutant $s_1 b$ progeny.
The fitness of a mutant is :

$$\begin{cases} W_{mut} = \frac{f\prime_x s_1 b}{s_r} + \frac{m\prime_x s_1 b + (1-s_1)b}{1-s_r} \end{cases} \tag{6.8}$$

We evaluate the selective value of the mutation as the difference $W_{mut} - W_{chx}$. If this selective value is positive, we consider that the mutation may settle in the population. However, we do not evaluate wether or not the mutation may become dominant, and how it may affect the average fitness or sex ratio in the population.
We will represent the selective value of a mutation on the discriminating ability, depending on the discriminating abilities of the population and for various allelic diversities and rejection cost. We also represent the selective value of a mutation on the rejection cost, depending on the rejection cost of the population and for various allelic diversities and discriminating abilities.

2. 2 Proportion of diploid females and haploid males among the theoretically fertilized offspring.

In a population with CSD and mate-choice, females may make multiple attempts at mating, until they find an unassorted male or until they lost all of their fertilization capacity. The fraction f_x of the $s_1 b$ offspring that develops as viable females and the fraction m_x of the $s_1 b$ offspring that develop as viable males depend on the probability of mating with an assorted male at each mating attempts, and the proportion of males and females corresponding to each mating attempt.

Let $Z = \frac{1}{\gamma}$ be the number of attempts at re-mating (after the first refusal) before the female stop choosing and leaves all her eggs unfertilized. For $t < Z$, $P_{a,t}$ is the probability of mating with an assorted male after exactly t+1 attempts at mating, and $P_{u,t}$ is the probability of mating with an unassorted male after exactly t+1 attempts. P_{at} and $P_{u,t}$ depend on δ, P (the probability of mating with an assorted male) and t:

$$\forall\, t < Z \begin{cases} P_{a,t} = P(1-\delta) \times (P\delta)^t \\ P_{u,t} = (1-P) \times (P\delta)^t \end{cases} \tag{6.9}$$

For $t < Z$, $f_{a,t}$ is the proportion of viable females among the $s_1 b$ offspring when mating with an assorted male after exactly t+1 attempts at mating; $f_{u,t}$ is the proportion of viable females among the $s_1 b$ offspring when mating with an unassorted male after exactly t+1 attempts. $f_{a,t}$ and $f_{u,t}$ depend on γ and t:

$$\forall\, t < Z \begin{cases} f_{a,t} = \frac{1-\gamma t}{2} \\ f_{u,t} = 1 - \gamma t \end{cases} \tag{6.10}$$

The average proportion of viable females among the $s_1 b$ offspring (f_x) is expressed as:

$$f_x = \sum_{t=0}^{Z} (P_{a,t} \times f_{a,t} + P_{u,t} \times f_{u,t}) \tag{6.11}$$

Wether the females mate with an assorted or with an unassorted male, she always produce γt haploid males among its $s_1 b$ offspring.Thus, as derived from eq. 1.10, The average proportion of haploid males among the $s_1 b$ offspring (m_x) is:

$$\begin{cases} m_x = \sum_{t=0}^{Z}((P_{a,t} + P_{u,t})\gamma t) + (P\delta)^{Z+1} \\ m_x = \gamma(1 - (P\delta)^{N+1}(2Z+1)) \end{cases} \tag{6.12}$$

The average proportion of viable females will be represented as function of the discriminating abilities in the population, depending on the allelic diversity and rejection cost, to underpin the direct impact of mate-choice on population growth. To assess the global impact of mate choice on parasitoid population demography, we will present simulations of parasitoid populations with mate-choice and CSD including the host parasitoid interaction and gene flow on the *csd* gene, as described in the following paragraph.

2. 3 Simulation of parasitoid population dynamics.

To describe the dynamics of parasitoid populations with mate choice, we started from the model described in article 4 for parasitoids with CSD and unviable diploid males. This eco-genetic model is composed of a deterministic model, which describes the abundance host and parasitoid, and of a stochastic model, which describes the random attribution of mates and offspring, the offspring genotype and the development of diploid males (see article 4 for more details).

To investigate the impact of mate choice, we modified the stochastic part of the model so that females may reject an assorted male. For each female, mating occurs after the abundance of her offspring has been attributed.
1) A male is randomly drawn in the population
2) If assorted, the females has a probability δ to reject it.
3) If the male is rejected, γ is added to the cost of selection. If $\gamma \times t > 1$ the females stops trying to choose between mates (step 2 is canceled); if not, the females draws an other male (back to step one).
4)The loop closes when the females mates or when it has run out of fertilization capacity.

After the mating sequence, a proportion $\gamma \times t$ of the offspring attributed to this female as would-be fertilized (the $s_1 b$ offspring) develop as haploid males instead.

We compute the probability of extinction $P(E)$ as the average proportion of simulations ending because the parasitoid went extinct on 3000 iterations, for simulations lasting 1000 generations. We represented $P(E)$ of to representative parasitoid populations, one of high impact and one of low impact as function of the discriminating abilities of the population and for various rejection costs.

3. Results

3. 1 Fitness and the selective value of mate choice

The evolution of a physiological trait in a population depends on the selective advantage it provides, *i.e.* the difference in fitness between an individual that possesses this trait and the rest of the population. We consider the selective value associated with two traits as determinants of the evolution of mate-choice strategies: the discriminating ability the cost of rejection. The selective values of these two traits mainly depend on their average value in the population and on the population's allelic diversity.

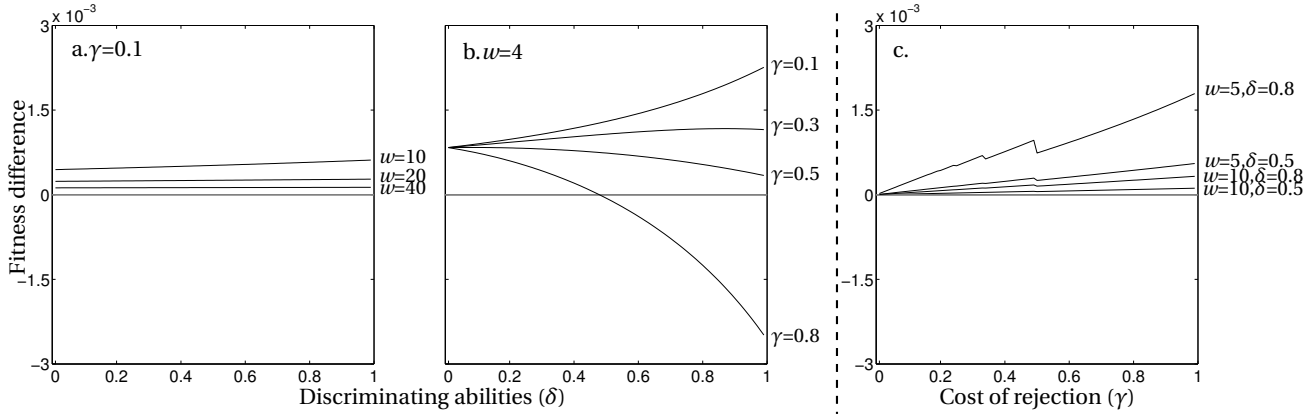

Figure 6.1: Fitness difference between the fitness of an individual with better discriminating abilities (δ+0.01, subfigures a, b) and with lower rejection cost (γ-0.01, subfigures c) for populations with unviable diploid males that select against assorted males depending on the mean parameter value in the population, for various genetic diversity (w), average discriminating abilities (δ) and average rejection cost (γ). The grey line is set on 0 (no difference)

A better ability to discriminate than the rest of the population is a selective advantage when the allelic diversity is high (Figure 6.1) . For $w > 10$, this selective advantages depends on the allelic diversity (w) but not on the cost of rejection(γ), or on the discriminating abilities of the population (Figure 6.1.a). Conversely, when the allelic diversity is low ($w = 4$, Figure 6.1.b), good discriminating abilities can be aselective advantage when the cost of rejection is low ($\gamma \leq 5$), but a disadvantage when this cost and the discriminating abilities of the population are high (Figure 6.1.b). This particular results rises because the probability of drawing an unassorted male is low (low genetic diversity), each event of rejection produces a high proportion of haploid males (high cost of rejection), and haploid males participate little to their mother's fitness because population sex ratio is heavily skewed (high discriminating abilities in the population). Rejection is too costly, for too little chances of drawing a better male at a second attempt at coupling.

A reduction of the cost of rejection is always a selective advantage, regardless of the allelic diversity, ability to discriminate and maximum cost (Figure 6.1.c). This advantage is particularly

strong when the discriminating abilities and cost of rejection of the population are important and the genetic diversity is low. In this situation, the population produces an important proportion of haploid males, so the sex ratio is skewed and haploid males participate little in their mother's fitness. Consequently, the supplementary females that can be produced by having a low rejection cost yield a particularly high selective advantage.

3. 2 Proportion of females

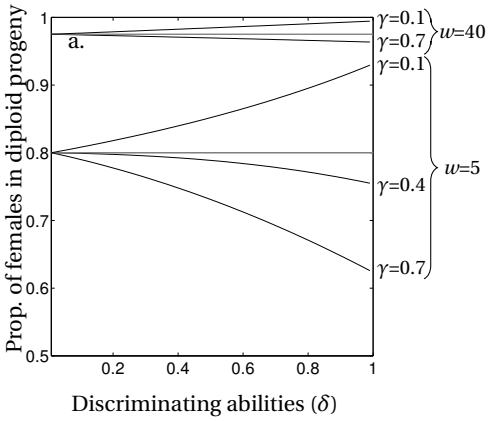

Figure 6.2: Proportion of females among diploid progeny for populations with CSD that select against assorted males, depending on the discriminating abilities of the population. The black line represent the proportion of females in populations with mate choice and the plain grey line represent the proportion of females in populations without mate choice, for various initial allelic diversity (w) and cost of rejection (γ).

The average proportion of females among the diploid progeny describes the direct impact of mate-choice on the dynamics of parasitoids, which are impacted only by females (that attack the host to lay eggs). The proportion of females in the population increases with the discriminating abilities as long as the cost of rejection remains low (Figure 6.2, $\gamma = 0.01$). In contrast, a high cost of rejection (> 0.5) can reduce the proportion of females among the diploid offspring, especially since the discriminating abilities are high in the population and the allelic diversity is low (Figure 6.2). Indeed, with a rejection cost higher than 0.5, mating without discrimination maximize the production of females compared to rejecting a male, even assorted. When the genetic diversity of the population is low, this effect rises for lower rejection costs because many more attempts at mating will be necessary to find an unassorted male and the costs of successive rejections will sum up.

The impact of assorted males on the proportion of females in the progeny can be completely neutralized by mate choice, but only if the allelic diversity remains high (see figure 6.2, line $\gamma = 0.1$, $w = 40$) . In this case only, the evolution of traits associated with mate-choice (toward high discriminating abilities and low cost of rejection) may compensate the impact of CSD on the dynamics of the parasitoid population.

3. 3 Persistence of the population

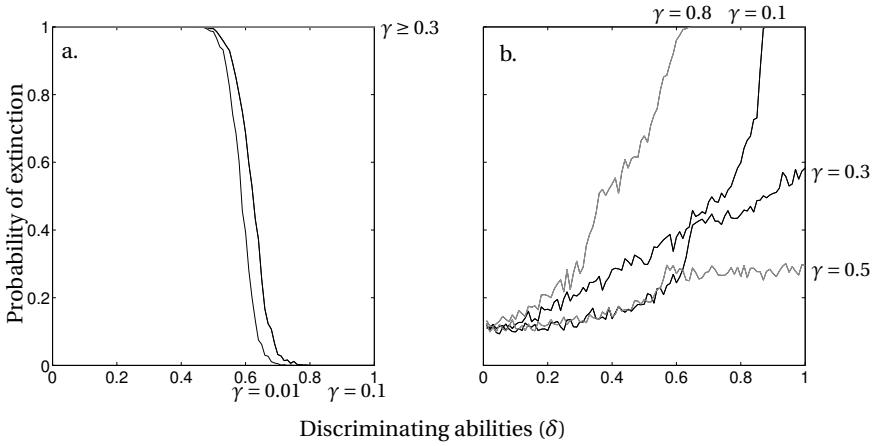

Figure 6.3: Probability of extinction for low-efficiency (a) and high-efficiency (b) parasitoid populations with CSD and selection to avoid assorted males, depending on the discriminating abilities of the populations, for various rejection costs.

Population a) (Figure 6.3.a) is a parasitoid population with a low searching efficiency and a high host rate of increase, *i.e* a low efficiency parasitoid population. This population persists close to the area of asymptotic extinction without CSD and goes extinct with CSD (without mate-choice), because it registers a decrease in female production that reduces its ability to parasitize the host (article 4). In such population, survival is correlated with the proportion of females in the progeny. When females select their mate to avoid assorted males, population a) may persist for sufficient discriminating abilities (0.7 to 0.8) and and sufficiently low mating cost ($\gamma < 0.3$) (Figure 6.3.a). For higher rejection costs, betted discriminating abilities are required to achieve the same probability of survival. The initial allelic diversity is without impact, because most of it is lost and the population, when it persists, settles to relatively low equilibrium diversity.

Population b) (Figure 6.3.b) is a parasitoid population with a high searching efficiency and a low host rate of increase, *i.e* a high efficiency parasitoid population. Without CSD, the population is unable to persist because its efficiency induces high amplitude cycles that drive the parasitoid to too low abundances. CSD (without mate choice) damp these cycles and increase their minimum value, allowing the population to persist (article 4). When females avoid assorted males, population b probability of extinction increases with the discriminating abilities (Figure 6.3.b). A too low cost of rejection drives the population to extinction because the effect

of diploid male production on cycles is neutralized. A high rejection cost also drives the population to extinction, because parasitoid females produces extremely few female offspring and thus are more vulnerable to extinction due to demographic stochasticity.A moderate rejection cost is the least deleterious to population persistence, because the production of diploid males is replaced by the production of haploid males.

4. Discussion

In Hymenopteran populations, the presence of single-locus *Complementary Sex Determination* (CSD) is assimilated to a genetic load (Zayed and Packer, 2005; Hein et al., 2009). In parasitoid wasps, CSD is not always deleterious to population persistence (article 4) but remains deleterious to fitness because half of the fertilized offspring is replaced by individuals that will not contribute to their mother's fitness (unviable or sterile diploid males). Thus, the avoidance of such mating is critical to preserve one's fitness. However, as diploid male production may decrease the extinction risk of parasitoid populations, mate selection on CSD-related criteria could conflict with population persistence.

Our investigations showed that in theory, mate-choice against assorted males could invade in a population under many circumstances. In low diversity populations, the discriminating abilities of females to exclude assorted males may reach equilibrium at intermediary values, particularly if mate rejection induces an important loss of fertilization capacity. In populations with higher genetic diversity, the females discriminating abilities may increase until they are perfectly able to discriminate between assorted and unassorted males. Unfortunately, it is also only in populations with high genetic diversity that mate-choice may restore the female production to its level without CSD (half of the eggs if the fertilization is balanced). Moreover, the chances of mating with an assorted male when mating without selection are clearly higher in low -diversity than in high diversity populations. Two patterns emerge: high genetic diversity populations, where females may become perfectly able to select against assorted males and counter the effects of CSD on population dynamics; and low genetic diversity populations, where females may gain only mild discriminating capacities that are of moderate impact against the strong effect of CSD on their dynamics.

The evolution of mate-choice against assorted males may compromise population persistence in various situations. The maximization of fitness drives the evolution of mate choice toward low rejection costs and high discriminating abilities: in high searching efficiency parasitoid populations, these two features increase the risk of extinction. Thus, high searching efficiency populations could be subjected to a form of evolutionary suicide underpinned by CSD. However, high efficiency parasitoids usually settle to an extremely low genetic diversity on the *csd* locus, because the recurrent cycles induce bottlenecks during which most of the genetic diversity is lost (articles 4,5). Moreover, these populations do not appear to be able to suffer a renewal of their genetic diversity, as this renewal drives them to extinction (article 5). Subsequently, high efficiency parasitoids may never reach the genetic diversity necessary for high discriminating abilities to be of selective value. If the cost of partner rejection is initially high, discriminating abilities may never emerge and high efficiency parasitoids may remains unable

to select unassorted males.

In contrast with high efficiency parasitoids, low efficiency parasitoids may particularly benefit from mate choice. While they persist, these populations generally maintain a higher genetic diversity than high efficiency populations, particularly if mutations or migrations maintain the genetic diversity (articles 4, 5). Thus, the population may become perfectly able to discriminate between assorted and unassorted males, effectively blocking the extinction risk induced by CSD even with a mild rejection cost.

Biologically, discrimination may be difficult to achieve on the basis of *csd* genotype only. The genotype at *csd* locus, if complementary, translates into a protein that activates a gene cascade responsible for female development. Other than this protein, the genotype for *csd* is not assumed to be otherwise expressed on its bearer's phenotype, which makes selection on this basis rather difficult (Heimpel and de Boer, 2008). However, we know at least one natural parasitoid species that actively avoid mating with males assorted at the *csd* locus, independently of familial links (*Bracon brevicornis*, Thiel et al. (2013)). In a population where the genetic diversity is high, avoiding assorted males is equivalent to avoiding kins, a selection much more easily realized in insect populations (Godfray, 1994). Some parasitoid species with CSD indeed avoid mating with their kin (Metzger et al., 2010; Herzner et al., 2006; Chuine, 2014) but some other do not (Ruf et al., 2013; Elias et al., 2010b). If discrimination against assorted males is achieved through kin avoidance, this discrimination becomes less efficient when the population looses its genetic diversity, because a larger proportion of males is assorted but not a close kin. This particular feature may extend the gap between high diversity populations that can neutralize the impact of CSD, and low diversity populations that suffer a high diploid male production without possibilities of escape.

The evolution of low rejection costs could take a variety of paths, some of which have been observed in natural parasitoid populations. In parasitoid females, signaling for males or gathering in swarms could reduce the risk of remaining virgin, thus allowing the females to be choosy (Godfray, 1994). A longer reproductive time and an oviposition signal associated with mating may allow the female to save her eggs for fertilization, reducing the risk of running out of eggs before mating (Fauvergue et al., 2008). In parasitoid males, an increase of sperm production allows successive mating without sperm depletion, which reduces the cost associated with mating with a male who already mated (Damiens, 2005). All theses adaptations are balanced by costs: 1) signaling for a mate or gathering in swarms may increase the predation and parasitism risk 2) if the female wait for too long before beginning oviposition, she risk dying before producing any eggs and 3) a large sperm production is costly in energy (Godfray, 1994; Boivin, 2013). If the costs of these adaptations are too high to allow their appearance, the cost of rejecting a mate cannot evolve toward lower values, thus limiting the selective value of discriminating abilities and mate-choice.

Diploid male production may yield a supplementary cost to female's fitness when diploid males are sterile rather than unviable, because females mated with a sterile diploid male are unable to produce any viable fertilized egg. Selection on the criteria of male ploidy could in theory be achieved relatively easily, since ploidy affects male phenotype on various traits asso-

ciated with mating, impacting the time to mate and mating success (Chuine, 2014). Even while mating with a diploid male is highly costly, active avoidance of diploid males by parasitoid females have not been previously demonstrated. Diploid males however tend to have a lower mating success when competing with haploid males (Chuine, 2014) and avoidance of diploid males through killing of diploid males larvae have been observed in social Hymenoptera (Zayed and Packer, 2001). Future directions of research include the study of the evolutionary and demographic consequences associated with mate-choice to avoid diploid males.

CSD is not the prerogative of arrhenotokous Hymenopteran species, but may also be found in thelytokous species, among which some are parasitoids (Beukeboom, 2001; van Wilgenburg et al., 2006). Thelytoky is scattered toward all Hymenopteran superfamilies, and probably evolved after the apparition of CSD (Heimpel and de Boer, 2008; Godfray, 1994; van Wilgenburg et al., 2006). van Wilgenburg et al. (2006) noticed that the presence of this reproductive system was puzzling, because most forms of thelytoky increase the level of homozygoty and are thus incompatible with CSD. However, this lays on the assumption that thelytoky would yield a diploid male production so large compared to the average production, that it would overbalance the other benefits it may provide. In contrast, populations with a low genetic diversity at the *csd* locus (such as high efficiency parasitoids) produce a high average proportion of diploid males, and females maximize their fitness by not being too discriminative against assorted males. In this picture, thelytoky could be of higher selective value than classical haplodiploidy (arrhenotoky) because the reduction of mating costs and the increase of the female's genetic relatedness to its offspring could compensate for diploid male production.

As first suggested by Hein et al. (2009), circumstances may render the production of diploid males less costly than its avoidance. We show that for Hymenopteran species with CSD, the loss of genetic diversity at the *csd* locus may not only decrease individual fitness and increase the population's extinction risk(Zayed and Packer, 2005) but also reduce their chances to avoid diploid male production by mate-choice. For Hymenopteran parasitoids specifically, this feature may prevent a conflict between the fitness benefits of mate-choice and population persistence. Low efficiency parasitoids that risk being driven to extinction by CSD usually maintain a relatively high genetic diversity, which may allow the evolution of mate choice and prevent diploid male production. In contrast, high efficiency parasitoids that relay upon CSD to persist maintain a low genetic diversity, which prevents the evolution of mate-choice against assorted males and maintain CSD effects on their dynamics.

Discussion Générale

Les mécanismes s'exprimant à faible densité, tels les effets Allee et les fardeaux génétiques, ont une réputation négative: ils sont moteur d'extinction (Gilpin and Soule, 1976; Frankham, 2005; Courchamp et al., 2008). Nous montrons qu'il est au contraire possible pour de tels mécanismes d'avoir un rôle stabilisatant sur les dynamiques des parasitoïdes, ce qui peut parfois prévenir leur extinction (article 2,4). L'effet Allee et le fardeau génétique ne sont pas pour autant "bénéfiques" pour tous les parasitoïdes: certains sont amenés à s'éteindre à cause de ces phénomènes.

1. L'effet des mécanismes s'exprimant à faible densité : une question de dynamiques.

Les effets bénéfiques ou délétères du fardeau génétique et de l'effet Allee chez les parasitoïdes dépendent de leurs dynamiques. Ces dynamiques sont principalement déterminées par qui, de l'hôte ou du parasitoïde, contrôle la dynamique de la population hôte. Lorsque l'efficacité de recherche du parasitoïde et la capacité de charge de l'hôte sont faibles par rapport au taux de croissance de l'hôte, c'est l'hôte (et plus particulièrement sa compétition intraspécifique) qui détermine sa dynamique: le parasitoïde est dit "peu efficace" (Figure 0.4). Dans cette situation les dynamiques du parasitoïde sont généralement asymptotiques qu'elles s'orientent vers un équilibre à faible abondance (Figure 0.4, zone 2) ou vers l'extinction (Figure 0.4, zone 1). Au contraire, lorsque l'efficacité de recherche du parasitoïde et la capacité de charge de l'hôte sont fortes par rapport au taux de croissance de l'hôte, c'est le parasitoïde (à travers la mortalité liée au parasitisme) qui détermine la dynamique de l'hôte : le parasitoïde est dit "très efficace". Les dynamiques du parasitoïde sont généralement cycliques/ chaotiques et peuvent entraîner l'extinction du parasitoïde (Figure 0.4, zone 5), lui permettre de se maintenir malgré des cycles d'amplitude importante (Figure 0.4, zone 4), ou bien être amorties jusqu'à un équilibre à forte abondance (Figure 0.4, zone 3).

Les parasitoïdes peu efficaces et très efficace répondent de manière diamétralement opposée aux mécanismes s'exprimant à faible densité.

1. Chez les parasitoïdes peu efficaces, la croissance de la population est principalement limitée par l'efficacité de recherche et l'impact sur l'hôte est faible. La dynamique d'une telle population est proche de celle d'une population « libre », c.à.d. considérée sans interactions inter-spécifiques. Les impacts de l'effet Allee et du fardeau génétique chez les parasitoïdes peu efficaces sont proches de ceux habituellement décrits chez les populations libres : ces deux phénomènes augmentent le risque d'extinction (articles 2,4, Courchamp et al. (2008); Zayed and Packer (2005)). Cependant, les facteurs qui peuvent avoir un impact synergique avec l'effet Allee ou le fardeau génétique ne sont pas nécessairement les mêmes chez les populations libres et chez les parasitoïdes peu efficaces. Par exemple, une faible diversité génétique et une forte stochasticité démographique sont des facteurs qui augmentent le risque d'extinction dû au fardeau génétique dans les populations libres (Gilpin and Soule, 1976; Frankham, 2005; Zayed and Packer, 2005), mais qui n'ont que très peu d'impact sur le risque d'extinction des parasitoïdes peu efficaces (article 4). En revanche, les facteurs affectant la population hôte (comme par exemple une réduction de

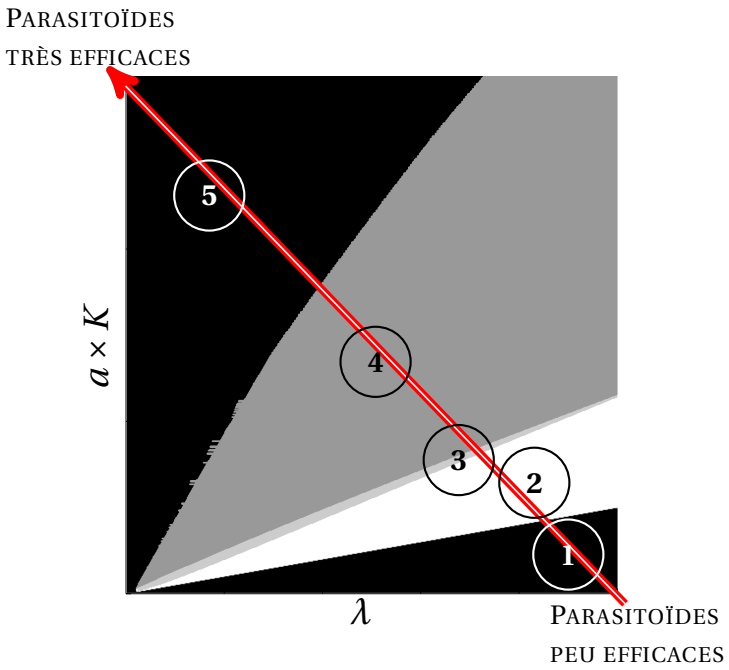

Figure 0.4: Dynamiques et efficacité des parasitoïdes

sa capacité de charge) induisent des risques d'extinction spécifiques aux parasitoïdes peu efficaces (article 2).

2. La démographie des parasitoïdes très efficaces est très différente de celle d'une population libre. Lorsque les hôtes sont abondants, la croissance du parasitoïde est limitée par son abondance et son efficacité de recherche, et son impact sur l'hôte est tellement fort que celui-ci devient rare. Lorsque l'hôte est rare, la croissance du parasitoïde est principalement limitée par l'abondance des hôtes et le parasitoïde devient rare: c'est à ce point de leurs dynamiques que les parasitoïdes efficaces sont les plus susceptibles de s'éteindre. Les conséquences des mécanismes s'exprimant à faible densité chez les parasitoïdes efficaces sont très différentes de celles décrites dans les populations libres: le CSD amorti les cycles du parasitoïde, réduisant son risque d'extinction (article 4) et l'effet Allee peut soit amortir soit amplifier les cycles, selon la sévérité de la compétition intra-spécifique chez l'hôte (article 2). Les facteurs qui affectent le risque d'extinction des populations libres peuvent avoir un impact complètement opposé sur les populations de parasitoïdes efficaces. Par exemple, la diversité génétique au locus du *csd* ou son maintien par l'apparition de nouveaux allèles permet de réduire le risque d'extinction dû au Diploid Male Vortex chez les populations libres (Zayed and Packer, 2005; Hein et al., 2009). Chez les parasitoïdes, ces facteurs ont au contraire tendance à augmenter le risque d'extinction (articles 4,5).

Au sein d'une interaction antagoniste il n'est pas rare qu'un phénomène apparemment délétère ait un impact stabilisant sur le système. C'est le cas de l'effet Allee chez le prédateur (Verdy, 2010; Zhou et al., 2005), de l'augmentation de la mortalité du prédateur (l'effet *Hydra,* (Abrams and Matsuda, 2005), la diminution des ressources en proies disponibles (le paradoxe de l'enrichissement

de Rosenzweig and MacArthur (1963)), du cannibalisme et de l'infanticide (Volker, 2007; Claessen et al., 2004), et du superparasitisme ou de l'autoparasitisme chez les parasitoïdes (Briggs and Collier, 2001). Le CSD et l'effet Allee peuvent être perçus comme deux autres phénomènes qui en réduisant la croissance du parasitoïde sont capables de stabiliser les dynamiques hôte-parasitoïde et de permettre le maintien du système.

2. Quels facteurs d'extinctions pour les petites populations de parasitoïdes?

Les facteurs qui entraînent l'extinction des parasitoïdes peuvent être très différents de ceux qui créent des vortex d'extinction chez les populations libres. Or, l'apparition et l'impact de ces facteurs d'extinction sont au coeur des problématiques de succès d'installation en lutte biologique classique (Hopper, 2003; Fauvergue et al., 2012), de conservation et de risque d'invasion biologique (King et al., 2010) Le tableau suivant (table 0.1) rassemble les différents facteurs d'extinction dont nous avons pu étudier l'impact:

Extinction Libre	Extinction Para.	Facteur	Interprétation
+	+ si LE - si HE	Présence du CSD	Le CSD amorti l'amplitude et augmente le minimum des cycles des parasitoïdes efficaces, réduisant leur risque d'extinction. Il réduit également le taux de parasitisme des parasitoïdes peu efficaces, entraînant leur extinction (article 4)
+	+	Présence de mâles diploïdes stériles	La présence de mâles diploides stériles augmente le risque d'extinction des parasitoïdes efficaces (en augmentant le risque qu'aucun descendant femelle ne soit produit à faible densité) et des parasitoïdes peu efficaces (en réduisant leur taux de parasitisme) (article 4).
+	= si LE - si HE	CSD+ faible diversité génétique initiale/faible abondance initiale	L'extinction des parasitoïdes de faible efficacité est principalement gouvernée par des processus déterministes (réduction du taux de parasitisme) et intéragit peu avec l'abondance initiale ou la diversité génétique initiale si elles sont au dessus d'un seuil faible (article 4. Une forte diversité génétique initiale et/ou une forte abondance initiale entraînent l'extinction des parasitoïdes très efficaces avant que les cycles ne puissent être amortis.

Extinction Libre	Extinction Para.	Facteur	Interprétation
-	- si LE + si HE	Maintien de la diversité génétique du *csd* par des mutations	L'apparition de nouveaux allèles déstabilise les cycles des parasitoïdes très efficaces et entraîne leur extinction. Si elle est très fréquente, l'apparition de nouveaux allèles permet le maintien des populations de faible efficacité en maintenant la production de mâles diploïdes à un niveau faible (article 5).
-	- si LE + /= si HE	Evitement des mâles appariés au gène *csd*	Le choix de partenaire permet d'éviter la production de mâles diploïdes et l'extinction dans les populations de faible efficacité (et de forte diversité génétique). Chez les parasitoïdes très efficaces (et de faible diversité génétique), le choix de partenaire a peu de risques d'apparaître, mais peu augmenter le risque d'extinction de la population si il apparaît.(article 6).
+	=	CSD + stochasticité démographique CSD + stochasticité environnementale	La combinaison de la stochasticité démographique ou environnementale et du CSD n'affecte pas le risque d'extinction des parasitoïdes peu efficaces (les processus déterministes gouvernent l'impact du CSD) (article 5)
+	+ si HE	CSD + faible taux de fertilisation et de conversion	L'impact démographique du CSD sur les parasitoïdes efficaces est lié à la conservation d'hôtes qui seront plus bénéfiques a un point ultérieur des dynamiques. Le fait de réduire le nombre de parasitoïdes femelles/hôte réduit le bénéfice associé au fait de "conserver" des hôtes et donc les avantages du CSD (article 5).
+	+ si LE +/- si HE	Difficultés à trouver un partenaire sexuel (EA)	Lorsque les parasitoïdes font face à un effet Allee, les parasitoïdes peu efficaces s'éteignent, tandis que les parasitoïdes efficaces soit ont des dynamiques plus stables, soit s'éteignent, selon la dynamique de la population hôte (article 2)
+	+/= si HD + si DD	EA + faible densité initiale EA + mortalité EA + compétition	Lorsque les parasitoïdes font face à un effet Allee, l'haplodiploïdie permet au parasitoïde de conserver ses capacités d'invasion sur un hôte seul ou sur un système hôte-parasitoïde établi, ainsi que ses capacités de survie à des causes extérieures de mortalité ou à l'arrivée d'un nouveau parasitoïde (article 3).

Table 0.1: Synthèse des résultats du projet de thèse. HE = Parasitoïdes de forte efficacité; LE= Parasitoïdes de faible efficacité; HD = haplodiploïdes; DD = diploïdes

2. 1 Implications pour la lutte biologique

Parmi les mécanismes j'ai pu mettre en évidence, un certain nombre ont de possibles implications pour la lutte biologique classique (table 0.1). Celles-ci concernent le choix de l'espèce et son élevage (1), le nombre d'individus à relâcher et les chances de maintien de la population (2) et le risque d'affecter des espèces non-cibles (3). Nous n'avons pas examiné dans quelle mesure l'effet Allee et le CSD pouvaient affecter la propension du parasitoïde à réduire la population du ravageur ciblé.

2. 2 Choix de l'espèce

L'utilisation de parasitoïdes dont le sexe est déterminé à l'aide du sl-CSD en lutte biologique classique n'est pas particulièrement contre-indiquée. Dans la mesure où les parasitoïdes sélectionnés pour la lutte biologique sont généralement très efficaces - l'efficacité du parasitoïde est corrélée avec son taux de parasitisme, un des principaux critères de choix - un maintien particulièrement attentionné de la diversité génétique au locus du *csd* n'est pas nécessaire en théorie.

Cependant, attention : dans les populations naturelles de parasitoïdes où les mâles diploïdes sont rarement observés, le CSD peut ne se manifester qu'une fois que la diversité génétique de la population est réduite. Par exemple, le CSD peut être porté par plusieurs locus, et n'induire aucune production de mâles diploïdes: lorsque la diversité génétique se réduit, les locii se fixent par dérive et le ml-CSD devient sl-CSD (de Boer et al., 2012). De même, dans une population où la diversité génétique est forte, un choix de partenaire efficace permettant d'éviter les individus apparenté peut être apparu en réponse à la pression évolutive du CSD (article 6), et prévenir la production de mâles diploïdes. Lorsque la diversité génétique se réduit, éviter les individus apparentés ne suffit plus à prévenir la production de mâles diploïdes, puisque des individus peu apparentés peuvent partager le même génotype au locus du *csd*. Ces deux phénomènes peuvent induire une apparition brutale de la production de mâles diploïdes. Chez les populations de parasitoïdes peu efficaces, une telle apparition peut entraîner des extinctions brutales et difficiles à prévenir. L'efficacité du parasitoïde est donc la clé: un parasitoïde très efficace, en plus d'être généralement un meilleur choix pour le contrôle de la population hôte, est relativement immunisé contre ce type de phénomène.

2. 3 Introduction du parasitoïde

Lorsqu'il est introduit dans un nouvel environnement, la densité du parasitoïde peu fortement décroître, selon ses capacités de dispersion et la structure spatiale de la population de parasitoïdes. Bien que je n'ai pas eu l'occasion d'explorer ces points au cours de ma thèse, de nombreux auteurs l'ont fait à la fois par la modélisation et par les expériences de terrain (Cronin, 2004; Hassell, 2000; Bueno et al., 2012; Godfray, 1994). Il n'est pas rare d'observer des densités de parasitoïdes très faibles après une introduction (Hopper, 1993; Stiling, 1993; van Driesche et al., 2008).

La faible densité du parasitoïde introduit peut entraîner des difficultés à trouver un partenaire et donc un effet Allee comportemental (Dennis, 1989; Hopper, 1993). Dans l'article 3), je montre qu'en présence d'un effet Allee comportemental et à densité égale, les diploïdes ont plus de chances de s'éteindre parce qu'ils ont été introduits en trop petites populations ou parce qu'une variation environnementale a induit de la mortalité que les haplodiploïdes. A l'échelle de l'ordre, cette prédiction est assez bien vérifiée, puisque les Diptères ont un succès d'introduction moindre que les hyménoptères (Hopper, 1993). Cependant, il serait malvenu d'utiliser ce critère à l'échelle de l'espèce, puisque les systèmes d'accouplements et les capacités de dispersion propres à chaque espèce peuvent avoir un impact très fort sur le risque d'apparition d'un effet Allee.

L'impact de l'effet Allee comportemental sur la population de parasitoïdes introduite semble dépendre fortement de la dynamique de l'hôte cible (article 2). L'évaluation expérimentale de cette théorie pose des problèmes techniques importants, car la dynamique naturelle de la population hôte est rarement reproduite de manière satisfaisante en laboratoire. En conséquence, les caractéristiques démographiques de la population hôte sont peut être largement sous-estimées dans la planification des actions de lutte biologique et dans la recherche des facteurs ayant pu entraîner l'échec d'une introduction(Stiling, 1993).

2. 4 Impact sur les espèces non cibles

Au delà du succès d'introduction et du contrôle de la population cible, limiter les impacts des introductions de parasitoïdes sur les populations non-cibles est un des objectifs majeurs de la lutte biologique du XXIe siècle (Rossi and Fowler, 2004). Parmi ces effets non-cible, on cite parfois l'extinction des populations natives de parasitoïdes (Simberloff and Stiling, 1996). Les critères entraînant ces extinctions sont variés et encore mal connus. Comme démontré dans l'article 3, mes travaux suggèrent que les parasitoïdes diploïdes pourraient être particulièrement mis en danger par l'arrivée d'un nouveau parasitoïde, surtout si celui-ci est haplodiploïde, en présence d'un Allee comportemental.

2. 5 Implications des mécanismes de détermination du sexe

Dans quelle mesure les facteurs d'extinctions que nous avons identifiés chez les parasitoïdes dépendent-il de leur mécanisme de détermination du sexe? Cette question a plus de poids qu'il n'y parait: l'idée que l'haplodiploidie rend les hyménoptères (et donc les parasitoïdes) immunes aux effets Allee est répandue chez les biologistes, tout comme l'idée récente que le CSD induit une dépression de consanguinité particulièrement catastrophique (Heimpel and de Boer, 2008).

L'haplodiploïdie permet effectivement de maintenir les capacités d'invasion et la robustesse des populations de parasitoïdes malgré la présence d'un effet Allee comportemental (article 3). Dans cette situation, elle permet aussi l'évolution de taux de fertilisation élevés, ou ajustés à la disponibilité des mâles, qui permettent de maximiser la fitness individuelle mais n'ont que très peu d'impact sur la démographie de la population (article 3). Cependant, l'impact de l'haplodiploïdie sur la réponse démographique des parasitoïdes à un effet Allee est très limité

comparé à l'impact de la dynamique de l'hôte ou de l'efficacité du parasitoïde.

L'haplodiploïdie permet également de limiter le fardeau de mutations, laissant le CSD comme principal fardeau génétique(Henter, 2003). Déterminer si le CSD est l'équivalent d'un fardeau génétique classique ou si il a un impact spécifique sur les facteurs d'extinctions des parasitoïdes n'est pas trivial. Plusieurs points distinguent le CSD d'un fardeau génétique classique :

1. Les allèles du *csd* sont sous sélection fréquence-dépendante. Les descendants homozygotes étant non viables, tout allèle rare dans la population porte une fitness supérieure à la moyenne. La sélection fréquence dépendante est à l'origine de la « diversité génétique à l'équilibre » constatée au locus du *csd* (articles 4, 5), alors qu'un gène sans sélection fréquence dépendante voit sa diversité génétique progressivement décroître par dérive génétique(Wright, 1922). Certaines populations de parasitoïdes peu efficaces ayant un seuil minimal de diversité génétique nécessaire à leur persistance (article 4), un fardeau génétique sur un gène qui ne serait pas sous sélection fréquence dépendante pourrait impliquer un risque d'extinction plus fort.
2. L'impact du CSD sur la fitness a une limite lorsque les mâles diploïdes sont non viables. Une femelle accouplée avec un mâle partageant un de ces allèles au locus du *csd* perd au maximum 50% de ses descendants fertilisés, le reste étant hétérozygote. Ce chiffre en apparence très fort est à contraster avec l'expression des mutations délétères, qui peuvent s'accumuler et affecter la fitness de manière beaucoup plus importante quand elles sont en grand nombre dans la population(Frankham, 2005).

La simulation d'un fardeau de mutation classique chez des parasitoïdes est une piste de recherche qui pourrait nous permettre d'isoler les résultats propres aux caractéristiques du CSD. Quoiqu'il en soit à l'issue de ces travaux il me semble beaucoup plus problématique lorsqu'on veux prédire le risque d'extinction d'un parasitoïde d'ignorer son intéraction avec l'hôte - dont dépend la plupart des facteurs d'extinctions que j'ai mis en évidence - que d'ignorer son caractère haplodiploïde.

3. Pourquoi l'effet Allee et le CSD n'ont pas le même impact sur les populations de parasitoïdes cycliques? Effet Allee génétiques et effets Allee comportementaux

L'effet Allee comportemental et le CSD réduisent tous deux le taux de croissance des populations qu'ils affectent, particulièrement lorsqu'elles sont à faible densité. L'un comme l'autre, ils augmentent le risque d'extinction des populations libres et des populations de parasitoïdes peu efficaces. A cause de cette similitude de conséquences démographiques, les fardeau génétiques (tels le CSD) sont considéré comme des effets Allee élémentaires au même titre que la difficulté à trouver un partenaire sexuel (Vayssade et al., 2014).

Pourtant, effet Allee comportemental et CSD ont des conséquences drastiquement différentes lorsqu'ils sont présents chez des parasitoïdes très efficaces. Alors que le CSD a toujours un impact stabilisant sur les populations de parasitoïdes cycliques, l'effet Allee comportemental est

très souvent déstabilisant, sauf si l'hôte subit une sévère compétition intra-spécifique. La différence entre fardeau génétique et effet Allee « classique » tient en une caractéristique: l'expression de l'effet Allee comportemental dépend à tout instant de la densité de la population, tandis que l'expression du fardeau génétique ne dépend que de l'existence d'un épisode à faible densité dans le passé. La figure 0.5 décrit schématiquement les impacts opposés du CSD et de l'effet Allee sur les cycles des parasitoïdes:

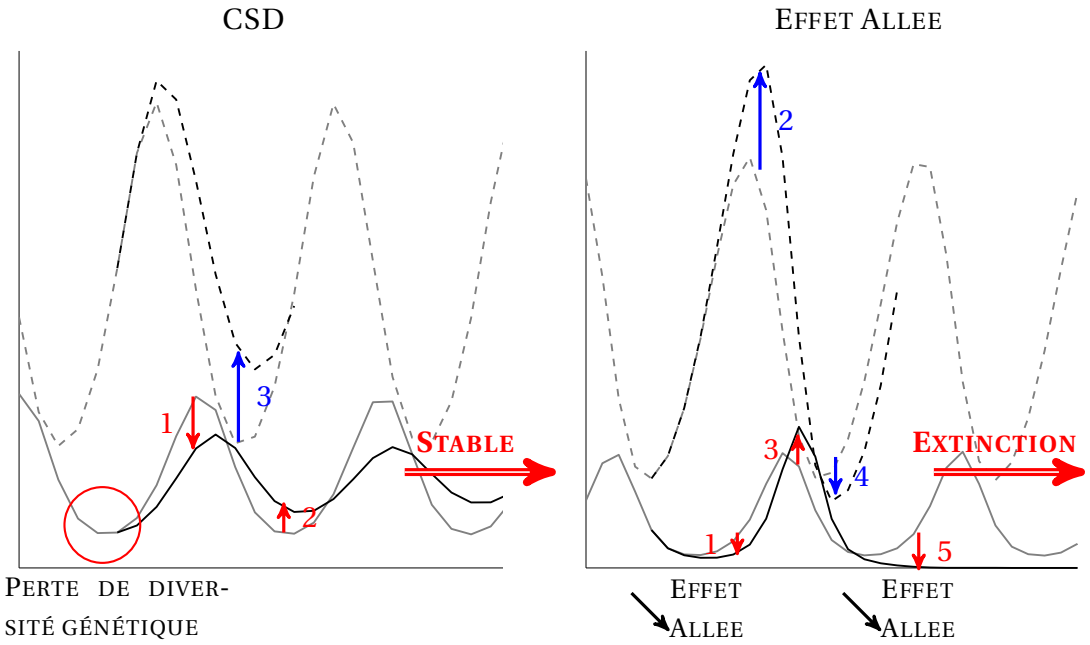

Figure 0.5: Impact du CSD et de l'effet Allee comportemental sur les parasitoïdes cycliques

Figure 0.5.a Lorsque le CSD est présent chez les parasitoïdes efficaces, le premier cycle entraîne un goulot d'étranglement et la réduction de la diversité génétique du CSD. Son impact s'exprime ensuite en trois temps : 1) des mâles diploïdes sont produits dans la phase de croissance du parasitoïde, réduisant son abondance ; 2) moins d'hôtes sont parasités durant la phase de décroissance des hôtes, car il y a moins de parasitoïdes ; 3) davantage de parasitoïdes persistent dans la phase de décroissance du parasitoïde, parce qu'il y a plus d'hôtes disponibles. Dans le cas du CSD, la sélection fréquence dépendante tend à maintenir la diversité alléliques après le premier goulot d'étranglement (si la diversité génétique était perdue au cours des cycles successifs, ceux-ci seraient plus rapidement amortis).

Figure 0.5. b. Lorsque l'effet Allee est présent chez les parasitoïdes efficaces, son impact est plus complexe, et distribué en cinq temps: 1) à faible densité, l'effet Allee s'exprime et le taux de croissance du parasitoïde est réduit ; 2) une proportion d'hôte est libérée du parasitisme durant la phase de croissance de l'hôte, aboutissant à des densités d'hôte élevées ; 3) lorsque l'abondance du parasitoïde est forte, il est libéré de l'effet Allee et davantage d'hôtes sont disponibles, ce qui augmente sa croissance 4) comme le parasitoïde est plus abondant, son impact sur l'hôte est plus fort, réduisant l'abondance de la population hôte déjà en déclin ; 5) à faible densité, l'abondance du parasitoïde est réduite à la fois parce que l'effet Allee s'exprime

et parce que l'hôte est moins abondant. Dans ce schéma, la compétition intra-spécifique de l'hôte joue un rôle à l'étape 2: si cette compétition est très surcompensée, la libération des hôtes entraîne une diminution (et non une augmentation) de l'abondance des hôtes au sommet de leur croissance, ce qui limite la croissance du parasitoïde et peut amener l'effet Allee à jouer un rôle stabilisant sur les cycles hôte-parasitoïde.

La diversité génétique, une fois perdue, ne peut pas être retrouvée rapidement par un accroissement de la population: ce point critique a été soulevé par Vayssade et al. (2014). Fardeau génétique et effet Allee "classique" peuvent donc être assimilés dans le cas des petites populations en déclin, où le taux de croissance est progressivement réduit par le fardeau génétique, mais pas dans le cas des populations cycliques où une phase de croissance succède à la phase de déclin.

L'origine écologique des cycles a également une importance majeure pour déterminer l'impact du CSD et de l'effet Allee comportemental. Une population libre soumise à une sévère compétition intra-spécifique présente des cycles qui peuvent être en apparence similaires à ceux des parasitoïdes, mais reposent sur une ressource fixe. Le CSD a un impact stabilisant moindre sur une telle population que sur les parasitoïdes et entraîne la population vers des minimas plus bas, alors qu'il augmente ceux des parasitoïdes (article 4). A l'inverse, l'effet Allee a un effet strictement stabilisant sur les populations libres dans lesquelles compétition intra-spécifique est sévère (Courchamp et al., 2008), alors qu'il est le plus souvent déstabilisant chez les parasitoïdes.

4. Facteurs génétique chez les parasitoïdes et adaptation à l'hôte

En s'appuyant sur les conséquences démographiques du CSD, on constate que la perte de diversité génétique apparaît moins dramatique chez les parasitoïdes que chez les populations libres. Le signal déterministe lié à l'interaction hôte-parasitoïde a un tel poids sur la dynamique du parasitoïde qu'il compense rapidement les variations liées aux facteurs génétiques (articles 4, 5).

Chez les hyménoptères, l'haplodiploïdie réduit l'intensité de la dépression de consanguinité (hors CSD), et facilite donc la transition vers des systèmes de reproduction qui ne maintiennent pas la diversité génétique (comme la parthénogénèse thélytoque ou les accouplements fortement consanguins) (Werren, 1989). Ces systèmes sont notamment assez fréquents chez les parasitoïdes(Godfray, 1994). Il est possible que l'évolution vers ce type de système soit également facilitée par l'interaction hôte-parasitoïde parce qu'elle compense les variations liées aux facteurs génétiques, et donc minimise l'impact démographique direct d'une perte de diversité génétique. Ce faisant, la relation hôte- parasitoïde permettrait à des populations ayant une très faible diversité génétique de se maintenir suffisamment longtemps pour voir apparaître des systèmes de reproduction consanguins ou thélytoques.

4. 1 Spécificité et risque d'extinction

Cependant, l'interaction hôte-parasitoïde n'a pas que des avantages : même si elle prévient une partie des conséquences démographiques d'une perte de diversité génétique, elle rend aussi la persistance des parasitoïdes sensible à la démographie de l'hôte. Un effet Allee chez l'hôte entraîne une augmentation dramatique du risque d'extinction du parasitoïde (Jang, 2006), et on peut supposer qu'une dépression de consanguinité chez l'hôte aurait le même effet.

Cette sensibilité est un problème commun aux espèces spécialistes (Simpson, 1944; Futuyma and Moreno, 1988; Stireman, 2005), et induit des fréquences d'extinction plus élevées chez les spécialistes que chez les généralistes en conditions naturelles (Jablonski, 1986; Smith and Jeffery, 1998; Purvis et al., 2000). Chez les parasitoïdes, certaines espèces sont généralistes, mais d'autres sont extrêmement spécialisées, un caractère qui a intrigué de nombreux scientifiques (Godfray, 1994). La principale hypothèse expliquant cette spécialisation est que la performance des parasitoïdes résulte d'un trade-off entre leurs performances sur les différents hôtes (Rausher, 1984; Jaenike, 1990; Stireman, 2005). Le parasitoïde ne pourrait donc améliorer son efficacité sur un hôte - par exemple à travers des adaptations physiologiques à son système immunitaire, ou des méthodes de recherche plus appropriées - sans abandonner une partie de son efficacité sur ses hôtes alternatifs (Janz and Nylin, 1997), suggérant que la spécialisation limite les capacités de diversification. L'hypothèse de l'existence d'un tel trade-off est n'est pas toujours bien supportée par les expériences empiriques (Jaenike, 1990; Carrière and Roitberg, 1994; Desneux et al., 2012; Fry, 1996).

4. 2 Capacités d'adaptation et spécialisation

Les capacités d'adaptation des parasitoïdes pourraient être un second obstacle à la diversification. Nous avons suggéré que la relation hôte-parasitoïde pouvait minimiser les impacts démographiques directs d'une perte de diversité génétique; mais elle n'affecte pas ses impacts indirects, dont la perte des capacités d'adaptation de la population. Or, l'hôte évolue constamment pour éviter le parasitisme, et le parasitoïde doit s'adapter pour survivre. En autorisant l'apparition de traits d'histoire de vie qui ne maintiennent pas la diversité génétique, l'haplodiploïdie et la relation hôte-parasitoïde ne permettent peut être pas au parasitoïde de conserver suffisamment de capacité d'adaptation pour se maintenir efficacement sur plusieurs hôtes.

Dans les populations naturelles de parasitoïdes, les proportions relatives de généralistes et de spécialistes sont techniquement difficiles à évaluer (Godfray, 1994; Smith and Jeffery, 1998). Cependant, on remarque que chez les hyménoptères parasitoïdes, la spécialisation semble évoluer à partir de lignées généralistes (Gauld, 2000), tandis que chez les parasitoïdes tachinaires (diploïdes) la reconstruction des arbres phylogénétiques montre que la généralisation a très souvent évolué à partir de lignées spécialistes (Stireman, 2005). Cette différence montre que l'évolution de la spécialisation n'est peut être pas simplement une question de trade-offs, mais pourrait être affectée par les traits d'histoire de vie particuliers des hyménoptères.

5. Présence et persistance du CSD dans les populations d'hyménoptères parasitoïdes

Quel que soit l'effet du CSD sur la démographie des parasitoïdes, son effet délétère sur la fitness individuelle est loin d'être négligeable (Heimpel and de Boer, 2008) Certains groupes de parasitoïdes présentent des mécanismes de détermination du sexe alternatifs, généralement mal connus mais suggérant que l'évolution vers un autre mécanisme est possible (Asplen et al., 2009; Beukeboom and van de Zande, 2010). La persistance du CSD chez les hyménoptères apparaît donc comme assez surprenante(van Wilgenburg et al., 2006; Heimpel and de Boer, 2008).

5. 1 Echapper aux conséquences du CSD

Il n'est peut être pas nécessaire de changer de mode de détermination du sexe pour atténuer l'impact du CSD. van Wilgenburg et al. (2006) a rassemblé les différents traits d'histoires de vie qui permettent de limiter la production de mâles diploïdes et donc les effets du CSD. Certains sont présents ou pourraient apparaître chez les parasitoïdes:

1. L'évitement des accouplements entre apparentés, notamment à travers le choix de partenaire, la dispersion post-natale, et la ségrégation temporelle entre la production et/ou l'émergence des mâles et des femelles. Ces comportements existent chez les parasitoïdes (Hardy, 1994; Ode and Hardy, 1995; Godfray, 1994), mais ne sont efficaces que lorsque la diversité génétique est relativement importante dans la population (article 6). De plus, ces caractéristiques ne sont pas systématiques puisque certaines espèces de parasitoïdes s'accouplent principalement entre apparentés malgré la présence du CSD (Elias et al., 2010b; Heimpel and de Boer, 2008).
2. La fertilité des mâles diploïdes. Quelques espèces de parasitoïdes produisent des mâles diploïdes fertiles, tels que *Euodynerus foraminatus* (Cowan and Stahlhut, 2004). La fertilité des mâles diploïdes rend le CSD en pratique comparable à une simple contrainte sur l'allocation du sexe, qui peut être surmontée par une augmentation du taux de fertilisation (Cowan and Stahlhut, 2004; Elias et al., 2010b; Godfray, 1990). Pourtant, si le CSD est bien le mode ancestral de détermination du sexe des hyménoptères, et antérieur à l'apparition de l'haplodiploïdie (Asplen et al., 2009), la fertilité des mâles diploïdes à été perdue chez la plupart des espèces et les hyménoptères évoluent vers une moindre viabilité des mâles diploïdes. Cette hypothèse est bien supportée par les comportements de cannibalisme des mâles diploïdes stériles observés chez certaines espèces d'abeille, rendant les mâles diploïdes stériles en pratique non viable (Zayed and Packer, 2001). Le retour ou la conservation de la fertilité des mâles diploïdes n'a pour le moment été observé que chez très peu d'espèces (Heimpel and de Boer, 2008; Elias et al., 2010b).
3. La parthénogénèse thélytoque mitotique (apomixie), la perte du génome paternel (PGL) et l'ovicide sélectif. L'apomixie est une forme de parthénogénèse thélytoque où les descendants diploïdes sont des clones de leur mère, ce qui maintient l'hétérozygotie et prévient donc l'apparition de mâles diploïdes. Cependant, elle n'est connue que chez quatre espèces d'hyménoptères, dont aucune n'est parasitoïde (van Wilgenburg et al., 2006). Chez quelques hyménoptères, le génome paternel d'un oeuf fertilisé peut être inactivé (PGL)

ou l'oeuf peut être détruit afin de réemployer ses ressources ou l'hôte dans lequel il était pondu (Godfray, 1994). Il n'y a cependant aucune indication qu'une femelle hyménoptère soit capable d'inactiver le génome paternel ou de détruire spécifiquement ses oeufs homozygotes au locus du *csd* (van Wilgenburg et al., 2006).

4. La multiplicité des locus du *csd* (ml-CSD). Le ml-CSD permet une plus faible production de mâles diploïdes que le sl-CSD, et plusieurs auteurs ont suggéré qu'il s'agissait d'une adaptation évolutive aux effets du CSD (de Boer et al., 2008). La duplication du gene *csd* est le mécanisme le plus fréquemment suggéré comme permettant le passage du sl-CSD au ml-CSD. Pourtant, le ml-CSD réalisé par ce biais ne permet pas de réduire la production de mâles diploïdes puisque les gènes issus de duplication sont liés (Heimpel and de Boer, 2008; Asplen et al., 2009). Même dans le cas de locii indépendants, le ml-CSD pose deux problèmes :

   – Si la dépression de consanguinité est très forte et que tous les locii sauf un sont fixés, le ml-CSD devient sl-CSD, un point que nous avons déjà évoqué. Son avantage dépend donc d'une dépression de consanguinité modérée (Heimpel and de Boer, 2008)
   – Les mâles haploïdes pouvant posséder plusieurs allèles différents du gène *csd*, le ml-CSD pourrait entraîner leur féminisation (Crozier, 1971).

La phylogénie des hyménoptères ne permet pas de trancher sur l'ancestralité du ml-CSD ou du sl-CSD (Asplen et al., 2009). L'évolution du gène *csd* chez les abeilles suggère que ml-CSD pourrait avoir été antérieur au sl-CSD, la multiplicité des locus ayant ensuite été perdue par dérive (Asplen et al., 2009).

5. 2 Le CSD: un rôle évolutif?

Une possible hypothèse serait donc que la production de mâles diploïdes chez les hyménoptères parasitoïdes est une "erreur" évolutive en passe d'être corrigée. Très peu d'auteurs se sont intéressés aux possibles avantages sélectifs du CSD, et ce il me semble pour deux raisons: 1) au premier abord, rien ne semble venir compenser la mort certaine de la moitié des femelles viables, et 2) l'impact du CSD sur le risque d'extinction des populations libres dresse clairement le portrait d'un fardeau génétique.

Pourtant, le CSD est fonctionnellement très similaire aux gènes d'auto-incompatibilité largement présents chez les plantes, et dont on sait qu'il sont apparus à plusieurs reprises (Charlesworth et al., 2005; Allen and S.J., 2008). Chez les plantes, l'auto-incompatibilité permet d'éviter l'autofécondation et les croisements entre apparentés par un système de reconnaissance des allèles. Lorsque deux gamètes incompatibles se rencontrent, la fécondation n'est pas réalisée et l'auto-incompatibilité n'entraîne donc pas la production d'individus non viables, contrairement au CSD. Cependant, lorsque les partenaires sexuels sont rares, l'auto-incompatibilité peut empêcher la fertilisation des gamètes (ce qui revient à les perdre) et donc avoir un coût similaire voir supérieur au CSD. La réduction de la taille de la population, en induisant une diminution du nombre de partenaires sexuels et de la diversité génétique, augmente donc le coût de l'auto-incompatibilité chez les plantes comme le coût du CSD (Busch et al., 2010).

Comme le CSD, l'auto-incompatibilité est ancestrale et s'est maintenue dans un grand nombre de populations. Les conditions de son apparition sont mal connues mais différentes hypothèses ont été proposées, parmi lesquelles la limitation de la quantité de pollen disponible et la présence d'un fardeau génétique au locus auto-incompatible. Cette dernière hypothèse est particulièrement intéressante à mettre en relation avec le CSD. Chez les hyménoptères, l'expression des mutations délétères chez les mâles accélère leur élimination, mais les mutations sur les caractères uniquement femelle s'accumulent normalement (Antolin, 1999). Serait-il possible que le CSD soit situé à proximité de certains gènes clé du développement des caractères reproducteurs femelle? Dans ce cas, l'auto-incompatibilité au gène du *csd* préviendrait la collision de deux génomes proches génétiquement, évitant ainsi la fixation progressive de mutations délétères récessives lorsque la consanguinité augmente. Génétiquement, l'hypothèse n'est pas absurde: chez les abeilles au moins, l'environnement du gène *csd* contient le gène *feminizer*, qui active une cascade de gènes pilotant la différenciation des traits femelles (Hasselmann et al., 2008). Ces cascades de gènes sont souvent spatialement proches dans le génome; on pourrait donc supposer qu'un certain nombres gènes impliqués dans le développement femelle pourraient être proche du locus du *csd*, une hypothèse qui reste à vérifier.

Sur le plan évolutif, cette hypothèse pourrait avoir des ramifications intéressantes. Les mécanismes de reproduction thélytoques ou strictement consanguins (frère-soeurs) chez les parasitoïdes haplodiploïdes sont fonctionnellement proche des systèmes d'accouplement par autofécondation chez les plantes. Dans ces trois cas, les coûts liés à la recherche de partenaire sont réduits (Taylor and Bulmer, 1980) et les descendants sont des clones de leur mère. Chez les parasitoïdes, l'existence d'espèces ayant à la fois le CSD et des accouplements consanguins (Ruf et al., 2011; Elias et al., 2010b) ou une reproduction thélytoque (van Wilgenburg et al., 2006) est apparue comme un mystère évolutif, puisque ces modes de reproduction induisent une production maximale de mâles diploïdes (50% des descendants diploïdes). Lande and Schemske (1985) ont étudié l'évolution des systèmes d'accouplement chez les plantes et prédit deux points évolutifs stables selon l'intensité de la dépression de consanguinité: l'allo-fécondation, et la promotion d'accouplement non consanguins lorsque la dépression de consanguinité est forte ($\delta = 1 - \frac{\text{fitness des descendants consanguins}}{\text{fitness des descendants non consanguins}} > 0.5$) et l'autofécondation quand la dépression de consanguinité est faible ($\delta < 0.5$). La dépression de consanguinité associée au CSD lorsque tous les descendants sont fertilisés est au maximum $\delta = 0.5$, donc si le CSD permet de limiter drastiquement la dépression de consanguinité en dehors de celle qu'il induit il peut promouvoir, et non contredire, l'apparition de systèmes d'accouplement fortement consanguins ou thélytoques. Imaginons une population d'hyménoptères parasitoïdes, ayant au départ un système d'accouplement non consanguin, dans laquelle la recherche de partenaire devient difficile : le taux de fertilisation des oeufs (et donc δ) diminue, éviter un partenaire apparié au gène du *csd* devient plus difficile et refuser des accouplements pour choisir son partenaire est de moins en moins avantageux (article 6), alors qu'être capable de produire des femelles est de plus en plus avantageux puisque les femelles non accouplées produisent des mâles. Si le CSD restreint la dépression de consanguinité à celle qu'il induit, les systèmes de reproduction consanguins ou thélytoques sont très avantageux et peuvent donc se fixer dans la population si ils apparaissent.

Seule une meilleure connaissance de la génétique des hyménoptères et de l'environnement du gène *csd* pourrait permettre d'évaluer plus clairement cette hypothèse. Associée aux impacts

théoriques du CSD sur la persistance des populations de parasitoïdes que j'ai pu mettre en évidence durant cette thèse, cette hypothèse dresse un tableau très nouveau de la présence du CSD chez les hyménoptères.

6. Alien *vs.* Predator (le fardeau génétique arbitre)

Au cours de cette étude, nous avons mis en évidence un certain nombre de résultats complexes, parfois contre-intuitifs, sur l'impact des mécanismes s'exprimant à faible densité sur les populations de parasitoïdes. Peut-on les généraliser aux relations antagonistes? Serions nous susceptibles de les retrouver chez un prédateur? La question n'est pas innocente: parmi les espèces au coeur des programmes de conservation, un grand nombre sont des prédateurs, à la fois parce qu'ils ont une forte valeur culturelle, parce qu'ils ont un impact structurel fort sur l'écosystème qui les soutient, et parce que le risque d'extinction tend à augmenter avec le niveau trophique des espèces, les grands prédateurs étant les plus susceptibles de s'éteindre(Purvis et al., 2000). Comprendre et prévenir les extinctions chez les prédateurs est donc un challenge de conservation majeur.

L'impact de l'effet Allee a été étudié à plusieurs reprises chez les prédateurs, à la fois sur le plan théorique et expérimental: sur le plan théorique, les résultat obtenus sont assez proches de ceux que j'ai mis en évidence chez les parasitoïdes (voir article 2) et les études mettent surtout en avance l'effet délétère des effets Allee sur la persistance des prédateurs (Jager et al., 2006). Les conséquences possibles d'un fardeau génétique chez un prédateur sont moins faciles à évaluer. En rassemblant des pistes sur conséquences théoriques d'un fardeau génétique chez des populations en interaction (article 1) j'ai conclu que le fardeau génétique pouvait être supporté très différemment chez les parasitoïdes et chez les prédateurs, en particulier parce que le lien entre proie consommées et reproduction est moins étroit chez les prédateurs. Les études postérieures que j'ai pu mener sur l'impact du CSD chez les parasitoïdes semblent confirmer que le relâchement de la relation hôte attaqué-parasitoïde produit minimise l'effet stabilisant du CSD(articles 4, 5). Le taux de conversion du prédateur (nombre de prédateur produits/ proies consommées) est un paramètre fonctionnellement proche mais généralement beaucoup plus faible que le nombre de parasitoïdes produits par hôte ("taux de conversion des parasitoïdes"). Un faible taux de conversion chez les parasitoïdes minimise l'effet stabilisant du CSD, ce qui suggère que cet effet serait peut être beaucoup moins fort chez un prédateur. Les individus stériles - comme par exemple des mâles diploïdes - ont coût supplémentaire chez un prédateur puisqu'ils consomment des proies. Ce coût ne peut pas être neutralisé simplement en évitant leur accouplement, comme chez les parasitoïdes.

L'ordre des hyménoptères contient des prédateurs, particulièrement au sein de la superfamille des Vespidae (guêpes sociales et solitaires) où le CSD a été mis en évidence chez au moins huit espèces (van Wilgenburg et al., 2006). Il est intéressant de remarquer que l'assassinat des mâles diploïdes juvéniles a évolué chez de telles guêpes (dont *Polybiodes tabidus*) et chez des insectes sociaux (notamment *Apis mellifera*)(Zayed and Packer, 2001; van Wilgenburg et al., 2006) mais pas chez les parasitoïdes, où les femelles à la recherche d'un partenaire pourraient

éventuellement éviter les mâles diploïdes (Hein et al., 2009; Chuine, 2014). Ces différences sont peut-être le témoin de l'impact plus dramatique du CSD et des mâles diploïdes stériles chez les prédateurs que chez les parasitoïdes.

Parce que les parasitoïdes maintiennent un lien plus étroit entre consommation des ressources et reproduction, parce que leur mode de vie est plus conservateur en termes d'énergies, ils supportent probablement mieux la présence d'un fardeau génétique que les prédateurs. Aussi bien parmi les hyménoptères que parmi les Diptères, la prédation semble avoir été une étape de l'évolution du mode de vie parasitoïde(Godfray, 1994; Eggleton and Belshaw, 1992). Peut-on supposer que le fardeau génétique ait joué un rôle dans cette évolution? Cette hypothèse reste inexplorée.

Conclusion

Comme c'est bien souvent le cas quand on s'attaque à un problème, les points sur lesquels nous nous sommes penchés soulèvent plus de questions qu'ils n'en résolvent. Lorsque j'ai débuté ma thèse, nous avions l'à-priori suivant: "chez les parasitoïdes, le CSD va être particulièrement délétère, surtout chez les populations cycliques puisqu'elles perdent leur diversité génétique à chaque cycle". Ce préjugé, qui s'est par la suite avéré être complètement faux, illustre bien la facilité avec laquelle on peut commettre des erreurs en inférant l'impact d'un phénomène dans une population en interaction à partir de son impact sur une population libre.

L'effet du CSD est-il généralisable à celui d'un fardeau génétique? Quel est le rôle du fardeau génétique dans l'évolution des hyménoptères, et en particulier des parasitoïdes? Quel est l'impact d'un tel fardeau chez l'hôte, chez le prédateur, dans d'autres formes d'interactions, sur l'évolution de ces interactions? Autant de questions dont les réponses permettraient de mieux comprendre le monde qui nous entoure et les lois qui le dessinent.

Pour finir, rappelons que cette étude est basée sur des modèles mathématiques théoriques. Il leur manque une multitude de dimensions qui permettraient de mieux décrire la population de parasitoïdes, mais également l'appui d'études empiriques qui permettraient d'évaluer leur valeur. Rien ne peut remplacer l'étude des populations naturelles, et la modélisation n'a pas cette ambition. Par ce travail, j'espère proposer des hypothèses plausibles sur les mécanismes qui participent à l'extinction, à l'invasion ou au maintien des populations de parasitoïdes, et sur les facteurs qui jouent un rôle dans ces évènements.

Bibliography

Abrams, P. a. (2001). The effect of density-independent mortality on the coexistence of exploitative competitors for renewing resources. The American naturalist, 158(5):459–70.

Abrams, P. a. (2003). Effects of altered resource consumption rates by one consumer species on a competitor. Ecology Letters, 6(6):550–555.

Abrams, P. a. and Kawecki, T. J. (1999). Adaptive host preference and the dynamics of host-parasitoid interactions. Theoretical population biology, 56(3):307–24.

Abrams, P. A. and Matsuda, H. (2005). The effect of adaptive change in the prey on the dynamics of an exploited predator population. Canadian Journal of Fisheries and Aquatic Sciences, 62:758–766.

Abrams, P. a. and Quince, C. (2005). The impact of mortality on predator population size and stability in systems with stage-structured prey. Theoretical population biology, 68(4):253–66.

Agrawal, a. a. (2001). Phenotypic plasticity in the interactions and evolution of species. Science (New York, N.Y.), 294(5541):321–6.

Agrawal, A. F. and Whitlock, M. C. (2012). Mutation Load: The Fitness of Individuals in Populations Where Deleterious Alleles Are Abundant. Annual Review of Ecology, Evolution, and Systematics, 43(1):115–135.

Allen, A. M. and S.J., H. (2008). Evolution and Phylogeny of Self-Incompatibility Systems in Angiosperms. In Franklin-Tong, V. E., editor, Self-Incompatibility in Flowering Plants: Evolution, Diversity, and Mechanisms.

Antolin, M. F. (1999). A genetic perspective on mating systems and sex ratios of parasitoid wasps. Researches on Population Ecology, 41(1):29–37.

Antolin, M. F., Ode, P. J., Heimpel, G. E., O'Hara, R. B., and Strand, M. R. (2003). Population structure, mating system, and sex-determining allele diversity of the parasitoid wasp Habrobracon hebetor. Heredity, 91(4):373–81.

Antolin, M. F. and Strand, M. R. (1992). Mating system of Bracon hebetor (Hymenoptera: Braconidae). Ecological Entomology, 17(1):1–7.

Asplen, M. K., Whitfield, J. B., DE Boer, J. G., and Heimpel, G. E. (2009). Ancestral state reconstruction analysis of hymenopteran sex determination mechanisms. Journal of evolutionary biology, 22(8):1762–9.

Bellows, T. (2001). Restoring Population Balance through Natural Enemy Introductions. Biological Control, 21(3):199–205.

Belovsky, G. E., Mellison, C., Larson, C., and Van Zandt, P. A. (1999). Experimental Studies of Extinction Dynamics. Science, 286(5442):1175–1177.

Bernstein, C. (1996). Density dependence and the stability of host-parasitoid systems. Oikos, 47:176–180.

Bernstein, C. (2000). Host-parasitoid models: the story of a successful failure. In Hochberg, M. E. and Ives, A. R., editors, Parasitoid Population Biology, pages 41–57. New Jersey, U.S.A., princeton edition.

Beukeboom, L. W. (2001). Single locus complementary sex determination in the ichneumonid Venturia canescens (Gravenhorst)(Hymenoptera). Nether, 51(1):1–15.

Beukeboom, L. W. and van de Zande, L. (2010). Genetics of sex determination in the haplodiploid wasp Nasonia vitripennis (Hymenoptera: Chalcidoidea). Journal of genetics, 89(3):333–9.

Beye, M., Hasselmann, M., Fondrk, M. K., Page, R. E., and Omholt, S. W. (2003). The gene csd is the primary signal for sexual development in the honeybee and encodes an SR-type protein. Cell, 114(4):419–29.

Boivin, G. (2013). Sperm as a limiting factor in mating success in Hymenoptera parasitoids. Entomologia Experimentalis et Applicata, 146(1):149–155.

Bolnick, D. I., Amarasekare, P., Araújo, M. S., Bürger, R., Levine, J. M., Novak, M., Rudolf, V. H. W., Schreiber, S. J., Urban, M. C., and Vasseur, D. a. (2011). Why intraspecific trait variation matters in community ecology. Trends in ecology & evolution, 26(4):183–92.

Bompard, A., Amat, I., Fauvergue, X., and Spataro, T. (2013). Host-parasitoid dynamics and the success of biological control when parasitoids are prone to allee effects. PloS one, 8(10).

Boukal, D. S., Sabelis, M. W., and Berec, L. (2007). How predator functional responses and Allee effects in prey affect the paradox of enrichment and population collapses. Theoretical population biology, 72(1):136–47.

Boyce, M. S. (1992). POPULATION VIABILITY ANALYSIS. Annual Review of Ecology and Systematics, 23(1992):481–506.

Briggs, C. J. and Collier, T. R. (2001). Autoparasitism, interference, and parasitoid-pest population dynamics. Theoretical population biology, 60(1):33–57.

Bueno, R. C., Parra, J. R. P., and Bueno (2012). Trichogramma pretiosum parasitism and dispersal capacity: a basis for developing biological control programs for soybean caterpillars. Bulletin of entomological research, 102(1):1–8.

Bull, J. J. (1981). Coevolution of Haplo-Diploidy and Sex Determination in the Hymenoptera. Evolution, 35(3):568–580.

Busch, J. W., Joly, S., and Schoen, D. J. (2010). Does mate limitation in self-incompatible species promote the evolution of selfing? The case of Leavenworthia alabamica. Evolution; international journal of organic evolution, 64(6):1657–70.

Caro, T. and Laurenson, M. (1994). Ecological and genetic factors in conservation: a cautionary tale. Science, 263(5146):485–486.

Carrière, Y. and Roitberg, B. D. (1994). Trade-offs in responses to host plants within a population of a generalist herbivore, Choristoneura rosaceana. Entomologia Experimentalis et Applicata, 72(2):173–180.

Caughley, G. (1994). Directions in Conservation Biology. Journal of Animal Ecology, 63(2):215.

Caughley, G. and Gunn, A. (1996). Conservation Biology in Theory and Practice. Blackwell Science, Cambridge.

Çelik, C. and Duman, O. (2009). Allee effect in a discrete-time predator,Äìprey system. Chaos, Solitons & Fractals, 40(4):1956–1962.

Chapman, A. P., Brook, B. W., Clutton-Brock, T. H., Grenfell, B. T., and Frankham, R. (2001). Population viability analyses on a cycling population: a cautionary tale. Biological Conservation, 97(1):61–69.

Charlesworth, D. (1987). Inbreeding Depression And Its Evolutionary Consequences. Annual Review of Ecology and Systematics, 18(1):237–268.

Charlesworth, D., Vekemans, X., Castric, V., and Glémin, S. (2005). Plant self-incompatibility systems: a molecular evolutionary perspective. The New phytologist, 168(1):61–9.

Chen, X.-w., Fu, X.-l., and Jing, Z.-j. (2013). Dynamics in a discrete-time predator-prey system with Allee effect. Acta Mathematicae Applicatae Sinica, English Series, 29(1):143–164.

Chen, Y.-Y. and Hsu, S.-B. (2011). Synchronized reproduction promotes species coexistence through reproductive facilitation. Journal of theoretical biology, 274(1):136–44.

Christensen, B. (1996). Predator foraging capabilities and prey antipredator behaviours : pre- versus postcapture constraints on size-dependent predator-prey interactions. Oikos, 76(2):368–380.

Chuine, A. (2014). Des mécanismes aux conséquences adaptatives du choix du partenaire sexuel pour la comp PhD thesis, Lyon 1.

Claessen, D., de Roos, A. M., and Persson, L. (2004). Population dynamic theory of size-dependent cannibalism. Proceedings. Biological sciences / The Royal Society, 271(1537):333–40.

Clausen, C. (1936). Insect parasitism and biological control. Annals Entomological Society of Americal, 29(2):201–223.

Cook, J. M. (1993). Sex determination in the Hymenoptera : a review of models and evidence. Heredity, 71(March):421–435.

Cook, J. M. and Crozier, R. H. (1995). Sex determination and population biology in the hymenoptera. Trends in ecology & evolution, 10(7):281–6.

Courchamp, F., Berec, L., and Gascoigne, J. (2008). Allee Effects in Ecology and Conservation. Oxford University Press.

Cowan, D. P. and Stahlhut, J. K. (2004). Functionally reproductive diploid and haploid males in an inbreeding hymenopteran with complementary sex determination. Proceedings of the National Academy of Sciences of the United States of America, 101(28):10374–9.

Crnokrak, P. and Roff, D. A. (1999). Inbreeding depression in the wild. Heredity, 83(3):260–270.

Cronin, J. T. (2004). Host-parasitoid extinction and colonization in a fragmented prairie landscape. Oecologia, 139(4):503–14.

Crow, J. F. and Kimura, M. (1970). An introduction to population genetics theory.

Crozier, R. H. (1971). Heterozygosity and Sex Determination in Haplo-Diploidy. The American naturalist, 105(945):399–412.

Damiens, D. (2005). Why do sperm-depleted parasitoid males continue to mate? Behavioral Ecology, 17(1):138–143.

Darwin, C. (1876). The Effects of Cross and Self Fertilisation in the Vegetable Kingdom. London.

de Boer, J. G., Kuijper, B., Heimpel, G. E., and Beukeboom, L. W. (2012). Sex determination meltdown upon biological control introduction of the parasitoid Cotesia rubecula? Evolutionary applications, 5(5):444–54.

de Boer, J. G., Ode, P. J., Rendahl, A. K., Vet, L. E. M., Whitfield, J. B., and Heimpel, G. E. (2008). Experimental support for multiple-locus complementary sex determination in the parasitoid Cotesia vestalis. Genetics, 180(3):1525–35.

Dennis, B. (1989). Allee effects : population growth, critical density, and the chance of extinction. Natural resource modeling, 3(4):481–538.

Dennis, B., Munholland, P. L., and Scott, J. M. (1991). Estimation of Growth and Extinction Parameters for Endangered Species. Ecological Monographs, 61(2):115.

Deredec, A. and Courchamp, F. (2006). Combined impacts of Allee effects and parasitism. Oikos, 3(August 2005):667–679.

Desneux, N., Blahnik, R., Delebecque, C. J., and Heimpel, G. E. (2012). Host phylogeny and specialisation in parasitoids. Ecology letters, 15(5):453–60.

Doebeli, M. (1997). Genetic Variation and Persistence of Predator-prey Interactions in the Nicholson,ÄìBailey Model. Journal of Theoretical Biology, 188(1):109–120.

Doebeli, M. and de Jong, G. (1999). Genetic variability in sensitivity to population density affects the dynamics of simple ecological models. Theoretical population biology, 55(1):37–52.

Eggleton, P. and Belshaw, R. (1992). Insect parasitoids : an evolutionary overview. Philosophical transactions of the Royal Society of London. Series B, Biological sciences, 337(1279):1–20.

El Agoze, M., Drezen, J. M., Renault, S., and Periquet, G. (1994). Analysis of the reproductive potential of diploid males in the wasp Diadromus pulchellus (Hymenoptera: Ichneumonidae). Bulletin of Entomological Reseach, 84(02):213–218.

Elias, J., Dorn, S., and Mazzi, D. (2010a). Inbreeding in a natural population of the gregarious parasitoid wasp Cotesia glomerata. Molecular ecology, 19(11):2336–45.

Elias, J., Dorn, S., and Mazzi, D. (2010b). No evidence for increased extinction proneness with decreasing effective population size in a parasitoid with complementary sex determination and fertile diploid males. BMC evolutionary biology, 10(1):366.

Ellstrand, N. C. and Elam, D. R. (1993). Genetic Consequences of Small Population Size: Implications for Plant Conservation. Annual Review of Ecology and Systematics, 24:217–242.

Fagan, W. F. and Holmes, E. E. (2006). Quantifying the extinction vortex. Ecology letters, 9(1):51–60.

Fauvergue, X. and Hopper, K. R. (2009). French wasps in the New World: experimental biological control introductions reveal a demographic Allee effect. Population Ecology, 51(3):385–397.

Fauvergue, X., Hopper, K. R., Antolin, M. F., and Kazmer, D. (1998). Does time until mating affect the progeny sex ratio? A manipulative experiment with the parasitoid wasp Aphelinus asychis. Journal of Evolutionary Biology.

Fauvergue, X., Lo Genco, A., and Lo Pinto, M. (2008). Virgins in the wild: mating status affects the behavior of a parasitoid foraging in the field. Oecologia, 156(4):913–20.

Fauvergue, X., Vercken, E., Malausa, T., and Hufbauer, R. a. (2012). The biology of small, introduced populations, with special reference to biological control. Evolutionary applications, 5(5):424–43.

Fawcett, T. W. and Johnstone, R. A. (2003). Optimal assessment of multiple cues. Proceedings. Biological sciences / The Royal Society, 270(1524):1637–43.

Ferdy, J.-b. and Molofsky, J. (2002). Allee Effect , Spatial Structure and Species Coexistence. Journal of Theoretical Biology, 2017:413–424.

Ferreira, J. D., Salazar, C. A. T., and Tabares, P. C. (2013). Weak Allee effect in a predator,Äìprey model involving memory with a hump. Nonlinear Analysis: Real World Applications, 14(1):536–548.

Fisher, R. (1930). The Genetical Theory of Natural Selection. Clarendon Press, Oxford.

Frankel, O. and Soulé, M. E. (1981). Conservation and Evolution. Cambridge University Press, Cambridge.

Frankham, R. (1995). Conservation genetics. Annual review of genetics, 29:305–27.

Frankham, R. (2002). Population Viability Analysis, volume 419. University of Chicago Press.

Frankham, R. (2005). Genetics and extinction. Biological Conservation, 126(2):131–140.

Frankham, R., Lees, K., Montgomery, M. E., England, P. R., Lowe, E. H., and Briscoe, D. A. (1999). Do population size bottlenecks reduce evolutionary potential? Animal Conservation, 2(4):255–260.

French, D. R. and Travis, J. M. J. (2001). Density-dependent dispersal in host-parasitoid assemblages. Oikos, 95(1):125–135.

Fry, J. D. (1996). The Evolution of Host Specialization: Are Trade-Offs Overrated? The American Naturalist, 148(s1):S84.

Futuyma, D. J. and Moreno, G. (1988). The Evolution of Ecological Specialization. Annual Review of Ecology and Systematics, 19(1):207–233.

Gao, Y. and Li, B. (2013). Dynamics of a ratio-dependent predator-prey system with a strong Allee effect. Discrete and Continuous Dynamical Systems - Series B, 18(9):2283–2313.

Gates, D. J. and Nason, J. D. (2012). Flowering asynchrony and mating system effects on reproductive assurance and mutualism persistence in fragmented fig-fig wasp populations. American journal of botany, 99(4):757–68.

Gauld, I. (2000). The Labeninae (Hymenoptera: Ichneumonidae): a study in phylogenetic reconstruction and evolutionary biology. Zoological Journal of the Linnean Society, 129(3):271–347.

Getz, W. M., Kaitala, V., and Ratnieks, F. L. W. (1992). Invasion of sibmating genes in diploid and haplodiploid populations. Evolutionary Ecology, 6(4):312–330.

Gilpin, M. E. and Soule, M. E. (1976). Minimum viable populations: processes of species extinction.

Godfray, H. C. J. (1990). The causes and consequences of constrained sex allocation in haplodiploid animals. Journal of evolutionary biology, 3:3–17.

Godfray, H. C. J. (1994). Parasitoids: Behavioral and Evolutionary Ecology.

Goldstein, D. B. (1994). Deleterious mutations and the Evolution of Male Haploidy. The American naturalist, 144(1):176–183.

González-Olivares, E., Mena-Lorca, J., Rojas-Palma, A., and Flores, J. D. (2011). Dynamical complexities in the Leslie Gower predator prey model as consequences of the Allee effect on prey. Applied Mathematical Modelling, 35(1):366–381.

Goodman, D. (1987). Viable Populations for Conservation. Cambridge University Press, Cambridge.

Gu, H. and Dorn, S. (2003). Mating system and sex allocation in the gregarious parasitoid Cotesia glomerata. Animal Behaviour, 66(2):259–264.

Hadjiavgousti, D. and Ichtiaroglou, S. (2008). Allee effect in a prey predator system. Chaos, Solitons & Fractals, 36(2):334–342.

Haldane, J. (1957). The Cost of Natural Selection. Genetics, 55:511–524.

Hamilton, W. and Zuk, M. (1982). Heritable true fitness and bright birds: a role for parasites? Science, 218(4570):384–387.

Hamilton, W. D. (1967). Extraordinary Sex Ratios. Science, 156(April):477–488.

Hardy, I. C. W. (1994). Sex ratio and mating structure in the parasitoid Hymenoptera. Oikos, 69:3–20.

Hartt, L. and Haefner, J. W. (1998). How phenotypic variation and life history trait correlation enhance mean fitness in prey populations. Theoretical population biology, 54(1):50–61.

Hassell, M. P. (2000). The Spatial and Temporal Dynamics of Host-Parasitoid Interactions. Oxford ser edition.

Hassell, M. P., Waage, J., and May, R. M. (1983). Variable parasitoid sex ratios and their effect on host-parasitoid dynamics. Journal of Animal Ecology, 52(3):889–904.

Hasselmann, M., Gempe, T., Schiø tt, M., Nunes-Silva, C. G., Otte, M., and Beye, M. (2008). Evidence for the evolutionary nascence of a novel sex determination pathway in honeybees. Nature, 454(7203):519–22.

Hedrick, P. W. (2001). Conservation genetics: where are we now? Trends in Ecology & Evolution, 16(11):629–636.

Hedrick, P. W., Gadau, J., and Page, R. E. (2006). Genetic sex determination and extinction. Trends in ecology & evolution, 21(2):55–7.

Hedrick, P. W. and Parker, J. D. (1997). Evolutionary Genetics and Genetic Variation of Haplodiploids and X-Linked Genes. Annual Review of Ecology and Systematics, 28(1):55–83.

Heimpel, G., Antolin, M., and Strand, M. (1999). Diversity of sex-determining alleles in bracon hebetor. Heredity, 82(June 1998):282–91.

Heimpel, G. E. (1997). Extraordinary sex ratios for extraordinary reasons. Trends in ecology & evolution, 12(3):298–299.

Heimpel, G. E. and de Boer, J. G. (2008). Sex determination in the hymenoptera. Annual review of entomology, 53:209–30.

Hein, S., Poethke, H.-J., and Dorn, S. (2009). What stops the diploid male vortex ? A simulation study for species with single locus complementary sex determination. Ecological Modelling, 220(13-14):1663–1669.

Henter, H. J. (2003). Inbreeding depression and haplodiploidy: experimental measures in a parasitoid and comparisons across diploid and haplodiploid insect taxa. Evolution;, 57(8):1793–803.

Herzner, G., Schmitt, T., Heckel, F., Schreier, P., and Strohm, E. (2006). Brothers smell similar: variation in the sex pheromone of male European Beewolves Philanthus triangulum F. (Hymenoptera: Crabronidae) and its implications for inbreeding avoidance. Biological Journal of the Linnean Society, 89(3):433–442.

Higgins, K. and Lynch, M. (2001). Metapopulation extinction caused by mutation accumulation. Proceedings of the National Academy of Sciences of the United States of America, 98(5):2928–33.

Ho, E. K. H. and Agrawal, a. F. (2012). The effects of competition on the strength and softness of selection. Journal of evolutionary biology, 25(12):2537–46.

Hopper, K. R. (1993). Management of genetics of Biological control introductions. Annual Review of Entomology, 38:27–51.

Hopper, K. R. (2003). Within - generation bet hedging : a seductive explanation ? Oikos, 101(1):219–222.

Hughes, a. R., Inouye, B. D., Johnson, M. T. J., Underwood, N., and Vellend, M. (2008). Ecological consequences of genetic diversity. Ecology letters, 11(6):609–23.

Imura, D., Toquenaga, Y., and Fujii, K. (2003). Genetic variation can promote system persistence in an experimental host-parasitoid system. Population Ecology, 45(3):205–212.

Ings, T. C., Montoya, J. M., Bascompte, J., Blüthgen, N., Brown, L., Dormann, C. F., Edwards, F., Figueroa, D., Jacob, U., Jones, J. I., Lauridsen, R. B., Ledger, M. E., Lewis, H. M., Olesen, J. M., van Veen, F. J. F., Warren, P. H., and Woodward, G. (2009). Ecological networks–beyond food webs. The Journal of animal ecology, 78(1):253–69.

Jablonski, D. (1986). Background and mass extinctions: the alternation of macroevolutionary regimes. Science (New York, N.Y.), 231(4734):129–33.

Jaenike, J. (1990). Host Specialization in Phytophagous Insects. Annual Review of Ecology and Systematics, 21:243–273.

Jager, H. I., Carr, E. A., and Efroymson, R. A. (2006). Simulated effects of habitat loss and fragmentation on a solitary mustelid predator. Ecological Modelling, 191(3-4):416–430.

Jang, S. R.-J. (2006). Allee effects in a discrete-time host-parasitoid model. Journal of Difference Equations and Applications, 12(2):165–181.

Janssen, A. (1989). Optimal Host selection by Drosophilia parasitoids in the Field. Functional Ecology, 3(4):469–479.

Janz, N. and Nylin, S. (1997). The role of female search behaviour in determining host plant range in plant feeding insects: a test of the information processing hypothesis. Proceedings of the Royal Society B: Biological Sciences, 264(1382):701–707.

Jennions, M. D. and Petrie, M. (1997). Variation in mate choice and mating preferences: a review of causes and consequences. Biological reviews of the Cambridge Philosophical Society, 72(2):283–327.

Jensen, A. (1987). Simple models for exploitative and interference competition. Ecological Modelling, 35:113–121.

Jiménez, J. A., Hughes, K. A., Alaks, G., Graham, L., and Lacy, R. C. (1994). An experimental study of inbreeding depression in a natural habitat. Science (New York, N.Y.), 266(5183):271–3.

Kaitala, V. and Getz, W. M. (1992). Sex ratio genetics and the competitiveness of parasitic wasps. Bulletin of mathematical biology, 54(2):295–311.

Kampichler, C., Calmé, S., Weissenberger, H., and Arriaga-Weiss, S. L. (2010). Indication of a species in an extinction vortex: The ocellated turkey on the Yucatan peninsula, Mexico. Acta Oecologica, 36(6):561–568.

Kang, Y. (2013). Scramble competitions can rescue endangered species subject to strong Allee effects. Mathematical biosciences, 241(1):75–87.

Keane, R. M. and Crawley, M. J. (2002). Exotic plant invasions and the enemy release hypothesis. Trends in ecology & evolution, 17(4):164–170.

Keller, L. F. and Waller, D. M. (2002). Inbreeding effects in wild populations. Trends in ecology & evolution, 17(5):19–23.

Kent, A., Patrick Doncaster, C., and Sluckin, T. (2003). Consequences for predators of rescue and Allee effects on prey. Ecological Modelling, 162(3):233–245.

Kim, M.-S., Brunsfeld, S. J., McDonald, G. I., and Klopfenstein, N. B. (2003). Effect of white pine blister rust (Cronartium ribicola) and rust-resistance breeding on genetic variation in western white pine (Pinus monticola). Theoretical and applied genetics., 106(6):1004–10.

Kimura, M. (1968). Evolutionary rate at the molecular level. Nature, 217:624–626.

King, B. (2002). Sex ratio response to conspecifics in a parasitoid wasp: test of a prediction of local mate competition theory and alternative hypotheses. Behavioral Ecology and Sociobiology, 52(1):17–24.

King, C. B. A., Haines, W. P., and Rubinoff, D. (2010). Impacts of invasive parasitoids on declining endemic Hawaiian leafroller moths (Omiodes : Crambidae) vary among sites and species. Journal of Applied Ecology, 47(2):299–308.

Knapp, E. E. and Rice, K. J. (1998). Comparison of Isozymes and Quantitative Traits for Evaluating Patterns of Genetic Variation in Purple Needlegrass (Nassella pulchra). Conservation Biology, 12(5):1031–1041.

Kokko, H., Brooks, R., Jennions, M. D., and Morley, J. (2003). The evolution of mate choice and mating biases. Proceedings. Biological sciences / The Royal Society, 270(1515):653–64.

Kondrashov, A. S. and Shpak, M. (1998). On the origin of species by means of assortative mating. Proceedings. Biological sciences / The Royal Society, 265(1412):2273–8.

Kramer, A. M., Dennis, B., Liebhold, A. M., and Drake, J. M. (2009). The evidence for Allee effects. Population Ecology, 51(3):341–354.

Krueger, C. and May, B. (1991). Ecological and genetic-effects of salmonid introductions in North-America. Canadian Journal of Fisheries and Aquatic Sciences, 48:66–77.

Kunkel, T. a. (2004). DNA replication fidelity. The Journal of biological chemistry, 279(17):16895–8.

Lacy, R. C. (1993). VORTEX: A Computer Simulation Model for Population Viability Analysis. Wildlife Research, 20:45–65.

Lacy, R. C. (1997). Importance of genetic variation to the viability of mammalian populations. Journal of Mammology, 78(2):320–335.

Laikre, L. and Ryman, N. (1991). Inbreeding Depression in a Captive Wolf (Canis lupus) Population. Conservation Biology, 5(1):33–40.

Lande, R. (1988). Genetics and Demography in Biological Conservation. Science, 241(4872):1455–1460.

Lande, R. (1995). Mutation and Conservation. Conservation Biology, 9(4):782–791.

Lande, R. (1998). Anthropogenic , Ecological and Genetic Factors in Extinction and Conservation. Population Ecology, 40(3):259–269.

Lande, R. and Schemske, D. (1985). The Evolution of Self Fertilization and Invreeding depression in Plants. Evolution, 39(1):24–40.

Leimu, R., Mutikainen, P., Koricheva, J., and Fisher, M. (2006). How general are positive relationships between plant size , fitness and genetic variation ? population. Journal of Ecology, 94(5):942–952.

Liao, W. and Reed, D. H. (2009). Inbreeding-environment interactions increase extinction risk. Animal Conservation, 12(1):54–61.

Lichstein, J. W., Dushoff, J., Levin, S. a., and Pacala, S. W. (2007). Intraspecific variation and species coexistence. The American naturalist, 170(6):807–18.

Lynch, M., Conery, J., Burger, R., Url, S., and Conery, I. J. (1995). Mutation Accumulation and the Extinction of Small Populations. The American Naturalist, 146(4):489–518.

Macarthur, R. and Levins, R. (1967). The Limiting Similarity, Convergence , and Divergence of Coexisting Species. The American naturalist, 101(921):377–385.

Madsen, T., Stille, B., and Shine, R. (1996). Inbreeding depression in an isolated population of adders Vipera berus. Biological Conservation, 75(2):113–118.

Manning, J. T. and Thompson, D. J. (1984). Muller's ratchet and the accumulation of favourable mutations. Acta Biotheoretica, 33(4):219–225.

Mather, K. (1969). Selection through Competition. pages 529–540.

May, R. M. (1978). Host-Parasitoid Systems in Patchy Environments: A Phenomenological Model. The Journal of Animal Ecology, 47(3):833.

May, R. M., Hassell, M. P., Anderson, R. M., and Tonkyn, D. W. (1981). Density dependence in host-parasitoid models. Journal of Animal Ecology, 50(3):855–865.

Maynard Smith, J. and Slatkin, M. . (1973). The Stability of Predator-Prey Systems. Ecology, 54(2):384–391.

Memmott, J., Godfray, H. C. J., and Gauld, I. D. (1994). The structure of a tropical host-parasitoid community. Journal of animal ecology, 63(3):521–540.

Merdan, H. and Gümüs, O. A. (2012). Stability analysis of a general discrete-time population model involving delay and Allee effects. Applied Mathematics and Computation, 219(4):1821–1832.

Metzger, M., Bernstein, C., Hoffmeister, T. S., and Desouhant, E. (2010). Does kin recognition and sib-mating avoidance limit the risk of genetic incompatibility in a parasitic wasp? PloS one, 5(10):e13505.

Morozov, A. and Li, B.-L. (2007). On the importance of dimensionality of space in models of space-mediated population persistence. Theoretical population biology, 71(3):278–89.

Mougi, A. and Kishida, O. (2009). Reciprocal phenotypic plasticity can lead to stable predator-prey interaction. The Journal of animal ecology, 78(6):1172–81.

Müller, M. J. I., Neugeboren, B. I., Nelson, D. R., and Murray, A. W. (2014). Genetic drift opposes mutualism during spatial population expansion. Proceedings of the National Academy of Sciences of the United States of America, 111(3):1037–42.

Munther, D. (2013). The ideal free strategy with weak Allee effect. Journal of Differential Equations, 254(4):1728–1740.

Newman, D. and Pilson, D. (1997). Increased Probability of Extinction due to Decreased Genetic Effective Population Size: Experimental Populations of Clarkia pulchella. Evolution, 51(2):354–362.

O Grady, J. J., Brook, B. W., Reed, D. H., Ballou, J. D., Tonkyn, D. W., and Frankham, R. (2006). Realistic levels of inbreeding depression strongly affect extinction risk in wild populations. Biological Conservation, 133(1):42–51.

Obrycki, J. J. and Kring, T. J. (1998). Predaceous Coccinellidae in Biological control. Annual Review of Entomology, 43:295–321.

Ode, P. J., Antolin, M. F., and Strand, M. R. (1997). Constrained oviposition and female-biased sex allocation in a parasitic wasp. Oecologia, 109(4):547–555.

Ode, P. J. and Hardy, I. C. W. (1995). Parasitoid sex ratios and biological control. pages 253–291.

Okuyama, T. (2007). Individual behavioral variation in predator,Äìprey models. Ecological Research, 23(4):665–671.

Packer, L., Zayed, A., Grixti, J. C., Ruz, L., Owen, R. E., Vivallo, F., and Toro, H. (2005). Conservation Genetics of Potentially Endangered Mutualisms: Reduced Levels of Genetic Variation in Specialist versus Generalist Bees. Conservation Biology, 19(1):195–202.

Pal, P. J., Saha, T., Sen, M., and Banerjee, M. (2011). A delayed predator,Äìprey model with strong Allee effect in prey population growth. Nonlinear Dynamics, 68(1-2):23–42.

Palomares, F., Godoy, J. A., López-Bao, J. V., Rodríguez, A., Roques, S., Casas-Marce, M., Revilla, E., and Delibes, M. (2012). Possible extinction vortex for a population of Iberian lynx on the verge of extirpation. Conservation biology : the journal of the Society for Conservation Biology, 26(4):689–97.

Purvis, A., Gittleman, J. L., Cowlishaw, G., and Mace, G. M. (2000). Predicting extinction risk in declining species. Proceedings of the Royal Society B: Biological Sciences, 267(1456):1947–52.

Pusey, A. and Wolf, M. (1996). Inbreeding avoidance in animals. Trends in ecology & evolution, 5347(1993):298–301.

Ralls, K. and Ballou, J. (1983). Extinction: Lessons from zoos. BIOL. CONSERV. SER.

Rausher, M. D. (1984). Tradeoffs in performance on different hosts: evidence from within and between site variation in the beetle Deloyala guttata. Evolution, 38(3):582–595.

Reed, D. H., O'Grady, J. J., Brook, B. W., Ballou, J. D., and Frankham, R. (2003). Estimates of minimum viable population sizes for vertebrates and factors influencing those estimates. Biological Conservation, 113(1):23–34.

Rempel, R. S., Elkie, P. C., Rodgers, A. R., and Gluck, M. J. (1997). Timber-management and natural disturbance effects on moose habitat: landscape evaluation. Journal of wildlife management, 61(1):1997.

Richman, A. D. and Kohn, J. R. (1996). Learning from rejection: the evolutionary biology of single-locus incompatibility. Trends in ecology & evolution, 11(12):497–502.

Robert, A. (2011). Find the weakest link. A comparison between demographic, genetic and demo-genetic metapopulation extinction times. BMC evolutionary biology, 11(1):260.

Rosenzweig, M. and MacArthur, R. (1963). Graphical Representation and Stability Conditions of Predator-Prey Interactions. American naturalist, 97(895):209–223.

Rossi, M. N. and Fowler, H. G. (2004). Spatial and temporal population interactions between the parasitoids Cotesia flavipes and Tachinidae flies: considerations on the adverse effects of biological control practice. Journal of Applied Entomology, 128(2):112–119.

Ruf, D., Dorn, S., and Mazzi, D. (2011). Females leave home for sex: natal dispersal in a parasitoid with complementary sex determination. Animal Behaviour, 81(5):1083–1089.

Ruf, D., Dorn, S., and Mazzi, D. (2013). Unexpectedly low frequencies of diploid males in an inbreeding parasitoid with complementary sex determination. Biological Journal of the Linnean Society, 108(1):79–86.

Rusch, A., Valantin-Morison, M., Sarthou, J.-P., and Roger-Estrade, J. (2011). Multi-scale effects of landscape complexity and crop management on pollen beetle parasitism rate. Landscape Ecology, 26(4):473–486.

Saccheri, I., Kuussaari, M., Kankare, M., Vikman, P., and Hanski, I. (1998). Inbreeding and extinction in a butterfly metapopulation. Nature, 45:1996–1999.

Saloniemi, I. (1993). A coevolutionary predator-prey model with quantitative characters. The American naturalist, 141(6):880–96.

Sato, H., Okabayashi, Y., and Kamijo, K. (2002). Structure and Function of Parasitoid Assemblages Associated with Phyllonorycter Leafminers (Lepidoptera: Gracillariidae) on Deciduous Oaks in Japan. Environmental Entomology, 31(6):1052–1061.

Schmieder, S. (2012). Tracing back the nascence of a new sex-determination pathway to the ancestor of bees and ants. Nature communications, 3:895.

Schoener, T. W., Clobert, J., Legendre, S., and Spiller, D. a. (2003). Life-history models of extinction: a test with island spiders. The American naturalist, 162(5):558–73.

Shaffer, M. L. (1981). Minimum Population Sizes for Species Conservation. BioScience, 31(2):131–134.

Shea, K. and Possingham, H. P. (2000). Optimal release strategies for biological control agents: an application of stochastic dynamic programming to population management. Journal of Applied Ecology, 37(1):77–86.

Simberloff, D. (1988). The Contribution of Population and Community Biology to Conservation Science. Annual Review of Ecology and Systematics, 19:473–511.

Simberloff, D. (2008). We can eliminate invasions or live with them. Successful management projects. Biological Invasions, 11(1):149–157.

Simberloff, D. and Stiling, P. (1996). Risk of species introduced for biological control. Biological Conservation, 3207(96):185–192.

Simpson, G. G. (1944). Tempo and Mode in Evolution.

Smith, A. B. and Jeffery, C. H. (1998). Selectivity of extinction among sea urchins at the end of the Cretaceous period. 392(6671):69–71.

Smith, N. G. C. (2000). The evolution of haplodiploidy under inbreeding. Heredity, 84(2):186.

Soule, M. E. (1980). Thresholds for survival: maintaining fitness and evolutionary potential. In Soule, M. E., editor, Conservation Biology: An evolutionary-ecological perspective, pages 111–124. Sinauer.

Spielman, D., Brook, B. W., and Frankham, R. (2004). Most species are not driven to extinction before genetic factors impact them. Proceedings of the National Academy of Sciences of the United States of America, 101(42):15261–4.

Stiling, P. (1993). Why Do Natural Enemies Fail in Classical Biological Control Programs? pages 31–37.

Stireman, J. O. (2005). The evolution of generalization? Parasitoid flies and the perils of inferring host range evolution from phylogenies. Journal of evolutionary biology, 18(2):325–36.

Tanaka, Y. (1997). Extinction of Populations Due to Inbreeding Depression with Demographic Disturbances. Researches on Population Ecology, 39(1):57–66.

Tanaka, Y. (2000). Extinction of populations by inbreeding depression under stochastic environments. Population Ecology, 42(1):0055.

Taylor, P. and Bulmer, M. (1980). Local mate competition and the sex ratio. Journal of Theoretical Biology, 86(3):409–419.

Theodorou, K., Souan, H., and Couvet, D. (2009). Metapopulation persistence in fragmented landscapes: significant interactions between genetic and demographic processes. Journal of evolutionary biology, 22(1):152–62.

Thiel, A., Weeda, A. C., de Boer, J. G., and Hoffmeister, T. S. (2013). Genetic incompatibility drives mate choice in a parasitic wasp. Frontiers in zoology, 10(1):43.

Traill, L. and Bradshaw, C. (2007). Minimum viable population size: A meta-analysis of 30 years of published estimates. Biological Conservation, 139:159 – 166.

Tscharntke, T. and Brandl, R. (2004). Plant-insect interactions in fragmented landscapes. Annual review of entomology, 49:405–30.

Turner, J. R. G. and Williamson, M. H. (1968). Population Size, Natural Selection and the Genetic load. Nature, 218(May):700.

van Alphen, J. and Janssen, A. (1981). Host Selection By Asobara Tabida Nees (Braconidae; Alysiinae) a Larval Parasitoid of Fruit Inhabiting Drosophila Species. Netherlands Journal of Zoology, 32(2):194–214.

van Driesche, R., Hoddle, M., and Center, T. (2008). Control of Pests and Weeds by Natural Enemies: An Introdu

van Staaden, M. J., Hamilton, M. J., and Chesser, R. K. (1995). Genetic variation of Woodland caribou (Rangifer tarandus) in North America. International Journal of mammalian biology, 60:150–158.

Van Valen, L. (1973). A new evolutionary law. 30:1–30.

van Wilgenburg, E., Driessen, G., and Beukeboom, L. W. (2006). Single locus complementary sex determination in Hymenoptera: an "unintelligent" design? Frontiers in zoology, 3:1.

Vayssade, C., Luque, G., and Courchamp, F. (2014). What is a genetic Allee effect? Trends in ecology & evolution.

Vellend, M. (2006). The Consequences of Genetic Diversity in Competitive Communities. Ecology, 87(2):304–311.

Verdy, A. (2010). Modulation of predator prey interactions by the Allee effect. Ecological Modelling, 221(8):1098–1107.

Volker, R. H. W. (2007). The interaction of cannibalism and omnivory: consequences fo community dynamics. Ecology, 88(11):2697–2705.

Wang, G., Liang, X.-G., and Wang, F.-Z. (1999). The competitive dynamics of populations subject to an Allee effect. Ecological Modelling, 124(2-3):183–192.

Wang, W., Jiao, Y., and Chen, X. (2013). Asymmetrical Impact of Allee Effect on a Discrete-Time Predator-Prey System. Journal of Applied Mathematics, 2013:1–10.

Wearing, H. J., Sait, S. M., Cameron, T. C., and Rohani, P. (2004). Stage-structured competition and the cyclic dynamics of host-parasitoid populations. Journal of Animal Ecology, 73(4):706–722.

Welton, N. J. and Houston, a. I. (2001). A theoretical investigation into the direct and indirect effects of state on the risk of predation. Journal of theoretical biology, 213(2):275–97.

Wernegreen, J. J. and Wheeler, D. E. (2009). Remaining flexible in old alliances: functional plasticity in constrained mutualisms. DNA and cell biology, 28(8):371–82.

Werren, J. H. (1989). The evolution of inbreeding in Haplodiploid organisms.

West, S. a., Shuker, D. M., and Sheldon, B. C. (2005). Sex-ratio adjustment when relatives interact: a test of constraints on adaptation. Evolution; international journal of organic evolution, 59(6):1211–28.

White, J. W., Rassweiler, A., Samhouri, J. F., Stier, A. C., and White, C. (2013). Ecologists should not use statistical significance tests to interpret simulation model results. Oikos, pages no–no.

Whitham, T. G., Difazio, S. P., Schweitzer, J. a., Shuster, S. M., Allan, G. J., Bailey, J. K., and Woolbright, S. a. (2008). Extending genomics to natural communities and ecosystems. Science (New York, N.Y.), 320(5875):492–5.

Whitham, T. G., Young, W. P., Martinsen, G. D., Catherine, A., Schweitzer, J. A., Shuster, S. M., Wimp, G. M., Fischer, D. G., Bailey, J. K., Lindroth, R. L., Woolbright, S., and Kuske, C. R. (2003). Community and Ecosystem Genetics : A Consequence of the Extended Phenotype. Ecology, 84(3):559–573.

Whiting, P. W. and April, R. (1943). Multiple alleles in Complementary Sex Determination of Habrobracon. Genetics, 28(365):365–382.

Whitlock, M. C. (2002). Selection, Load and Inbreeding Depression in a Large Metapopulation. Genetics, 160(3):1191–1202.

Wilson, H. B. and Hassell, M. P. (1997). Host-parasitoid spatial models: the interplay of demographic stochasticity and dynamics. Proceedings of the Royal Society B: Biological Sciences, 264(1385):1189–1195.

Wimp, G. M., Martinsen, G. D., Floate, K. D., Bangert, R. K., and Whitham, T. G. (2005). Plant Genetic Determinants of Arthropod Community Structure and Diversity. Evolution, 59(1):61–69.

Witter, M. S. and Cuthill, I. C. (1993). The ecological costs of avian fat storage. Philosophical transactions of the Royal Society of London. Series B, Biological sciences, 340(1291):73–92.

Wittmann, M. J., Hutzenthaler, M., Gabriel, W., and Metzler, D. (2013). Ecological and genetic effects of introduced species on their native competitors. Theoretical population biology, 84:25–35.

Wittmer, H. U., Sinclair, A. R. E., and McLellan, B. N. (2005). The role of predation in the decline and extirpation of woodland caribou. Oecologia, 144(2):257–67.

Wright, S. (1922). Coefficients of inbreeding and relationship. The American Naturalist, 56(645):330–338.

Wright, S. (1931). Evolution in Mendelian Populations. Genetics, 16(2):97–159.

Zahavi, A. (1975). Mate selection,ÄîA selection for a handicap. Journal of Theoretical Biology, 53(1):205–214.

Zayed, A. (2009). Bee genetics and conservation. Apidologie, 40:237–262.

Zayed, A. and Packer, L. (2001). High levels of diploid male production in a primitively eusocial bee (Hymenoptera: Halictidae). Heredity, 87(6):631–636.

Zayed, A. and Packer, L. (2005). Complementary sex determination substantially increases extinction proneness of haplodiploid populations. Proceedings of the National Academy of Sciences of the United States of America, 102(30):10742–6.

Zayed, A., Roubik, D. W., and Packer, L. (2004). Use of diploid male frequency data as an indicator of pollinator decline. Proceedings of the Royal Society B: Biological Sciences, 271 Suppl(Goudet 1995):S9–12.

Zhou, S.-R., Liu, Y.-F., and Wang, G. (2005). The stability of predator-prey systems subject to the Allee effects. Theoretical population biology, 67(1):23–31.

Zu, J., Mimura, M., and Yuichiro Wakano, J. (2010). The evolution of phenotypic traits in a predator-prey system subject to Allee effect. Journal of theoretical biology, 262(3):528–43.

Annexes

Annexe 1 : Le Projet Sextinction

Le projet ANR Sextinction, piloté par Xavier Fauvergue, a pour but la compréhension du fonctionnement et des conséquences démographiques et adaptatives du *Complementary Sex Determination* chez les hyménoptères parasitoïdes. Dans un cadre plus large, il s'attèle à la compréhension des conséquences des goulots d'étranglement chez les hyménoptères, un point clé pour la conservation et l'introduction de populations de parasitoïdes en lutte biologique. Ce projet interdisciplinaire implique plusieurs laboratoires français et combine des approches variées:

1. *génétique moléculaire* : recherche du gène *csd* et mise en place de marqueurs microsatellites chez *Venturia canescens* (Laboratoire de Biologie des Populations Introduites de l'INRA, Sophia Antipolis)
2. *biologie des populations* : description des populations naturelles de *V. canescens* à travers l'étude de populations expérimentales goulotées (Laboratoire de Biologie des Populations Introduites de l'INRA, Sophia Antipolis)
3. *écologie comportementale* et *écologie chimique* : étude du choix de partenaire de *V. canescens* pour l'évitement des accouplements consanguins et/ou assortis au locus du *csd*(Laboratoire de Biométrie et Biologie Evolutive de l'Université Claude Bernard, Lyon1). Identification des composés chimiques impliqués dans la reconnaissance des partenaires et le choix (Centre d'Ecologie Fonctionnelle et d'Ecologie Evolutive du CNRS, Montpellier et Centre d'Etudes des Substances Naturelles de l'Université Claude Bernard, Lyon1).
4. *modélisation* Modélisation des conséquences démographiques du CSD sur les systèmes hôte-parasitoïdes (Laboratoire d'Ecologie et d'Evolution de l'Université Pierre et Marie Curie).

Figure 0.6: Illustration d'une femelle *Venturia canescens* parasitant une larve d'*Ephestia kuehniella*. (Anna Chuine)

Le modèle biologique utilisé pour les parties expérimentales de ce projet, *V. canescens*, est un hyménoptère parasitoïde solitaire de la famille des Ichneumons. Chez *V. canescens*, le sexe est déterminé à l'aide du sl-CSD et les mâles diploïdes sont viables mais stériles. La recherche de partenaire est effectuée par les mâles, les femelles recherchant principalement des hôtes dans lesquels elles commencent à pondre avant d'être accouplées. Les femellessont monoandres et effectuent l'essentiel du choix de partenaire, évitant les individus apparentés.

Ma thèse s'inscrit dans ce projet ANR à l'étape de modélisation des conséquences démographiques du CSD chez les parasitoïdes. Bien que mes modèles aient été conçus pour représenter des systèmes hôte-parasitoïde en général et non une espèce en particulier, un certain nombres de leurs hypothèses sont basées sur le modèle *V. canescens*.

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EMPLOI

2013-2014 : **ATER** à temps plein à l'Université Pierre et Marie Curie (UFR 927), **doctorante** au laboratoire Ecologie et Evolution (nouvellement au sein de l'Institut d'Ecologie et des Sciences de l'Environnement de Paris). Cours magistraux et TDs en mathématique (L1), systèmes dynamiques (L3) et statistiques (L3) à destination de licences de biologie et d'informatique.

2010-2013 : **Doctorante** de l'Université Denis Diderot (école doctorale Frontières du Vivant). Directeur & co-directeur de thèse : Thierry Spataro & Minus van Baalen. Encadrement d'une stagiaire de M2, organisation de séminaires inter-équipes au laboratoire. Présidente d'un club de réflexion scientifique de l'ED. Enseignement de travaux dirigés de dynamique des populations à AgroParisTech (72 heures).

FORMATION

2010 (6 mois) : **Stage de M2 à l'INRA (Sophia Antipolis)** "Interactions indirectes entre insectes phytophages médiées par un prédateur généraliste". Expérimentations de comportement (en laboratoire) et de dynamiques de population (sous serre) sur des insectes ravageurs des cultures. Modélisation de dynamiques de population.

2007-2010 : **Préparation du diplôme d'ingénieur agronome** à l'Ecole Nationale Supérieure d'Agronomie de Rennes (Agrocampus Ouest). Obtention du diplôme spécialisation "Protection des Plantes et Environnement".

2008-2009 (6 mois) : **Stage de M1 au CIRAD (Dakar)** "Effets des conditions agro-climatiques sur les communautés d'arthropodes des cultures de chou dans la région des Niayes (Sénégal)". Identification d'insectes, expérimentation en champ et interprétation statistique de données d'écologie des communautés.

2005-2007 : **Classe préparatoire BCPST** au lycée Henri IV, Paris. Admission au concours Agro-Véto, spécialisation en informatique.

2005 : **Baccalauréat scientifique**, mention bien.

CAPACITÉS LINGUISITIQUES : Anglais (courant), espagnol (scolaire)

CAPACITÉS INFORMATIQUES : C/C++, Matlab, Mathematica, R, Scilab, Populus, NetLogo, LateX, pack office (Excel, Powerpoint, etc.).

COMMUNICATIONS SCIENTIFIQUES

2013 : **INTECOL** Poster "Demographic consequences of Complementary Sex Determination in Hymenoptera parasitoids", Londres, Royaume-Uni.

2012 : **International Entomophagous Insects Conference.** Présentation orale "Effet Allee comportemental chez les parasitoïdes : impacts démographiques et conséquences pour la lutte biologique", Montpellier, France.

2012 : **Ecology Workshop (Frontiers of Life).** Présentation orale "Mécanismes de détermination du sexe et effet Allee génétique dans les systèmes hôte-parasitoïde", Paris, France.

2011 : **Entomological Society of Canada Annual Meeting**. Présentation orale "Conséquences démographiques des Effets Allee comportementaux dans les systèmes hôte-parasitoïde", Halifax, Canada.

2010 : **Entomological Society of America.** Poster “Sharing a predator: can invasive species affect the biological control of an endemic pest”, San Diego , USA.

PUBLICATIONS

Bompard, A., Jaworski, C., Bearez, P, Desneux, N. (2013): “Sharing a predator : can invasive species affect the predation pressure on a local pest ?” Population Ecology, *vol. 55 (3) pp 433-440.*

Bompard, A., Amat, I., Fauvergue, X. Spataro, T. (2013): “Host-parasitoid dynamics and the success of biological control when parasitoids are prone to Allee effects”. *Plos One, vol. 8 (10).*

Jaworski, C., Bompard, A., Genies, L., Amiens-Desneux, E., Desneux, N. (2013):"Preference and Prey Switching in a Generalist Predator Attacking Local and Invasive Alien Pests". *Plos One, vol.8 (12).*

Autres

Sports pratiqués: Parapente, rock ’n’ roll, karaté, randonnée.

Activités : Aquarelle, chorale, piano.

RÉSUMÉ

Dans une population de faible densité, la diversité génétique est réduite et les partenaires sexuels sont plus difficiles à trouver, ce qui augmente le risque d'extinction si la démographie est simple. Dans ces travaux, j'étudie par la modélisation l'impact de tels phénomènes sur les populations de parasitoïdes, chez qui la démographie est plus complexe en raison de l'interaction avec l'hôte. Les parasitoïdes sont souvent utilisés en lutte biologique. A cette occasion ils sont introduits dans un nouvel environnement en faibles densités, ce qui justifie particulièrement l'étude du devenir des petites populations de parasitoïdes. Ma thèse est sur articles et composée de trois parties : i) une synthèse de la bibliographie ; ii) mes travaux sur les conséquences de l'effet Allee comportemental sur la démographie des parasitoïdes; iii) mes travaux sur les conséquences du *Complementary Sex Determination* (CSD), un mécanisme qui induit un fardeau génétique propre aux hyménoptères, sur la démographie des parasitoïdes et l'évolution du choix de partenaire. La prise en compte des interactions interspécifiques est absolument indispensable pour prédire les conséquences des mécanismes s'exprimant à faible densité. La sensibilité des parasitoïdes aux effets Allee dépend de la dynamique de leur hôte. Contrairement à ce qui a été décrit chez les populations non parasitoïdes, le CSD n'induit pas de vortex d'extinction à proprement parler chez les parasitoïdes, mais au contraire il permet de limiter leur risque d'extinction. Ce résultat très novateur apporte un nouveau regard sur les conséquences démographiques et évolutives des fardeaux génétiques dans les populations en interaction.

ABSTRACT

In low-density populations, genetic diversity is decreased and mate-finding is increasingly difficult, two phenomena that may induce extinctions if the demography is simple. During my thesis, I used mathematical modeling to investigate the impact of such phenomena on parasitoids in which population dynamics are more complex (due to the close interaction between the parasitoid and its host. The study of the fate of small parasitoid populations is particularly justified in the context of biological control, in which parasitoids are widely used and often introduced in low densities. This manuscript is built on scientific papers and organized into three parts: i) a literature synthesis; ii) the theoretical impact of mate-finding Allee effect on the demography of parasitoids and iii) the theoretical impact of *Complementary Sex Determination* (CSD), which induces a genetic load specific to the Hymenoptera, on the demography of parasitoids on the evolution of mate-choice to avoid the deleterious consequences of this burden at the individual level. I conclude that considering the presence of an interspecific interaction, such as the host-parasitoid interaction, is crucial to predict the consequences of Allee effects and genetic load. We show that parasitoid survival can be very sensitive to mate-finding Allee effects, depending on the host dynamics. We also show that contrary to what has been described in non parasitoid populations, CSD does not induce an extinction vortex in parasitoids, but on the contrary limits their extinction risk. This highly innovative result provides a new look at the demographic and evolutionary consequences of genetic load in interacting populations.