A cascade of destabilizations: combining *Wolbachia* and Allee effects to eradicate insect pests

Julie C. Blackwood[a], Roger Vargas Jr.[a] and Xavier Fauvergue*[b]

[a]Department of Mathematics and Statistics, Williams College, Williamstown, MA 01267, USA

[b]ISA, UMR INRA CNRS, Universite Nicé Côte d'Azur, 06903 Sophia-Antipolis, France

*Corresponding author: xavier.fauvergue@sophia.inra.fr

Summary

1. The management of insect pests has long been dominated by the use chemical insecticides, with the aim of instantaneously killing enough individuals to limit their damage. To minimize unwanted consequences to the environment,

*xavier.fauvergue@sophia.inra.fr

some more recent novel approaches propose biological controls that take advantage of intrinsic demographic processes to eliminate pest populations.

2. We address the feasibility of a novel pest management strategy based on the integration of *Wolbachia*-induced cytoplasmic incompatibility and the enhancement of a pre-existing Allee effect via mating disruption.

3. A stochastic population model is developed that accounts for *Wolbachia*-induced cytoplasmic incompatibilities in addition to an Allee effect that arises from mating failures at low population density. Simulations were run with two objectives: quantifying how cytoplasmic incompatibility and the Allee effect interact to drive insect pest populations toward extinction, and delineating a strategy based on the introduction of *Wolbachia*-infected insects into a population where a mate-finding Allee effect is enhanced by mating disruption.

4. Our modeling results demonstrate that *Wolbachia*-induced cytoplasmic incompatibility and the Allee effect act independently from one an other; the Allee effect does not modify the *Wolbachia*-invasion threshold, and cytoplasmic incompatibility has only a marginal effect on the Allee threshold. Nonetheless, when induced in concert, the two processes can drive even large populations to extinction. Importantly, simulations with successive introductions of two incompatible *Wolbachia* strains into a population where an Allee effect is enhanced by a realistic application of mating disruption suggest that even large pest populations could be driven to extinction.

5. Our study provides novel and tangible perspectives for the use of cytoplasmic

incompatibility and the Allee effect to eradicate insect pests. More generally, it points on the importance of transient dynamics, and the relevance of manipulating a cascade of destabilizatons for pest management. ...

Keywords

Allee effect; biological control; cytoplasmic incompatibility; eradication; extinction; mating disruption; transient dynamics; *Wolbachia*.

Introduction

With one million species and thirty billion tons of accumulated biomass, insects are far more diverse and abundant than any other form of animal life on Earth (Price *et al.* (2011)). Although most species provide crucial ecosystem services (Losey & Vaughan (2006)), a minority of taxa that we consider pests (1%) have an overwhelming influence on the development of population management in theory and in practice. Chemical insecticides have been used extensively since the 1950s, but growing concerns about their negative side effects combined with the widespread emergence of insect resistance urges scientists to search for sustainable alternatives.

Among the various environmentally friendly approaches that have been envisaged to control insect pests, a novel research avenue proposes the exploitation of Allee effects, i.e., the decrease in survival and reproduction at small population sizes and the consequent reduction in population growth (Liebhold & Bascompte (2003); Liebhold & Tobin (2008)). The central ideas surrounding these meth-

ods are twofold: management tactics could be combined in order to (1) reduce a population size down below the Allee threshold – the population size at which the *per capita* growth rate decreases (a "weak" Allee effect) or becomes negative (a "strong" Allee effect) – which, in turn, increases the probability of stochastic extinction, and/or (2) amplify the mechanisms underpinning a pre-existing Allee effect to increase the Allee threshold itself (Liebhold *et al.* (2016); Tobin, Berec & Liebhold (2011); Suckling *et al.* (2012)). Capitalizing on Allee effects to manage undesirable species is especially advantageous because it drives populations into extinction vortexes without needing to kill every individual; the Allee effect could thus be a cornerstone of eradication programs (Liebhold *et al.* (2016)).

The seminal idea of using Allee effects to eradicate insect pests and the subsequent development of theoretical models originate from basic research on, and population of management of, the Gypsy moth *Lymantria dispar*. The damage caused by this invasive forest pest in North America triggered a major containment program to slow its spread toward the western states (Sharov *et al.* (2002); Liebhold, Halverson & Elmes (1992)). *L. dispar* is one of the few insect species for which both a component (mate-finding) and a demographic Allee effect have been explicitly identified (Tobin, Onufrieva & Thorpe (2013); Tobin *et al.* (2007); Johnson *et al.* (2006)). Mating disruption is thus one of the main tactics deployed to control newly established populations along the invasion front, with more efficiency than classic treatments with the pesticide *Bacillus thurengiensis* (Sharov *et al.* (2002)). Therefore, there is a need to identify other invasive pest species that may have pre-existing Allee effects and determine when environmentally friendly forms of control may be effective.

The use of Allee effects to eradicate pests has been the focus of several recent

theoretical developments (e.g. Boukal & Berec (2009); Liebhold & Bascompte (2003); Blackwood *et al.* (2012); Yamanaka & Liebhold (2009)). Models have been developed to integrate one or more component Allee effects and include population management tactics such as culling, release of sterile males, and mating disruption (Fauvergue (2013)) provides a comprehensive review). For example, the model of Blackwood *et al.* 2012 assumes a mate-finding Allee effect and predicts that applying moderate levels of insecticides in conjunction with mating disruption increases the demographic Allee threshold below which the *per capita* rate of increase becomes negative. The two tactics act in synergy, decreasing the time to eradication below the time for either tactic alone.

There is evidence population management strategies centered on taking advantage of Allee effects may be plausible in many insect species. In a meta-analysis focused on natural animal populations (Kramer *et al.* (2009)), terrestrial arthropods were found associated with the largest number of studies (22) and the highest proportion (77%) showing an Allee effect. Mating failure at low density appeared as the most frequent mechanism. Additionally, Fauvergue (2013) found evidence supporting the presence of mate-finding Allee effects in 19 out of 32 published studies. Indirectly, the central role of Allee effects in insect population dynamics is supported by the efficiency of eradication programs based on the disruption of reproduction. Pest management based on the reduction of mating success via mass trapping, mating disruption with sex pheromones, or the release of sterile males has indeed proved successful in several instances (Suckling *et al.* (2014, 2012); Krafsur (1998)).

In this article, we investigate *Wolbachia*-induced cytoplasmic incompatibility (CI) as a novel method for triggering reproductive failures and thereby increasing

pushing populations below pre-existing mate-finding Allee effects. *Wolbachia* are endosymbiotic bacteria that infect at least 20% of all insect species and up to two thirds in some estimations (Hilgenboecker *et al.* (2008)). It has various effects on its insect hosts, the most widespread and prominent being cytoplasmic incompatibly (Stouthamer, Breeuwer & Hurst (1999)). Under CI, matings involving an infected male result in offspring mortality during embryonic development if the female is either uninfected or infected with a different incompatible strain. Fitness advantages of infected females and maternal inheritance provide key features underlying the spread of *Wolbachia* within a population: above a threshold frequency, a given *Wolbachia* strain is expected to invade until near-fixation (Barton & Turelli (2011); Hancock, Sinkins & Godfray (2011); Caspari & Watson (1959); Hoffmann & Turelli (1997); Turelli & Hoffmann (1991)). The introduction of cytoplasmic incompatibility within a population therefore promotes the release of individuals infected with *Wolbachia* as a candidate biological control agent against Arthropod pests (Bourtzis (2008)).

There are two different implementations of these techniques that can be considered in practice. First, similar to the use of "Sterile Insect Technique" (SIT), males bearing a *Wolbachia* strain incompatible with that of the target population can be released in large numbers, leading to CI and a consequent decrease in population growth rate, possibly driving populations to local extinction (Laven (1967); Zabalou *et al.* (2004); Atyame *et al.* (2015)). Incompatible males can be obtained via transfection, even between completely different species of host insects (e.g. Braig *et al.* (1994)). At the population level, the underpinnings for mass-releases of incompatible males do not depart from that of SIT, for which interactions with the Allee effect have already been thoroughly analyzed (Boukal & Berec (2009);

Yamanaka & Liebhold (2009); Fauvergue (2013)).

The second management tactic using CI relies on the inoculation of a relatively small number of insects of both sex infected with a *Wolbachia* strain incompatible with that of the target population. This method is investigated in the theoretical model introduced in Dobson *et al.* (2002), which combines *Wolbachia* invasions with insect population dynamics and predicts a transient reduction in insect population size. This decline results from the temporary increase in the fraction of incompatible matings, which peak in the midst of the invasion process. Hence, fine-tuned sequential introductions of different Wolbachia strains could be applied to sustain artificially an unstable coexistence of multiple incompatible infections within an insect population, allowing the population size to be reduced and maintained at low level (Dobson, Fox & Jiggins (2002)).

Our aim is to determine the potential for *Wolbachia*-induced CI to drive a population to extinction in the presence of the Allee effect. Specifically, we derive a theoretical model built upon Dobson *et al.*'s (2002) approach of CI management that additionally accounts for Allee effects and environmental and demographic stochasticity. We also consider mating disruption in our model as a potential complementary tactic. We use this model to address three primary questions: (1) What is the influence of Allee effects present within a host population on *Wolbachia* invasion dynamics? (2) What is the influence of cytoplasmic incompatibility on the demographic Allee effect? (3) What is the influence of a combination of *Wolbachia*-induced CI, mating disruption, Allee effects, and stochasticity on the probability of host extinction?

Methods

Population model

In this section, we first introduce a model that considers the population dynamics in the absence of individuals infected with *Wolbachia*. Our model extends the modeling framework introduced by Dobson, Fox & Jiggins (2002) by additionally accounting for pre-existing Allee effects, the release of pheromone sources as a method of mating disruption, as well as both demographic and environmental stochasticity.

Similar to Dobson, Fox & Jiggins (2002), we considered populations such that the dynamics can be modeled in discrete time with non-overlapping generations. The model explicitly tracks the total population size at each time t, given by N_t, and also tracks the distribution of infected and uninfected individuals within the population. We assume that each time step can be broken into two stages: the first (at time $t + 0.5$) captures reproduction, and the second (at time $t + 1$) captures density dependent survivorship of offspring to adults. The total number of offspring is given by

$$N_{t+0.5} = mN_t f(N_t) g(N_t), \tag{1}$$

where m is the maximum *per capita* fecundity. $f(N_t)$ captures a component Allee effect that results from the failure to find mates at low densities such that

$$f(N_t) = \frac{N_t}{N_t + \theta}. \tag{2}$$

Here, θ measures the strength of the Allee effect; a convenient interpretation of this term is that θ represents the population size at which half of the females successfully mate. Further, $g(N_t)$ in Equation 1 captures the decline in fecundity resulting from techniques to control populations via mating disruption. We assume that P pheromone sources are maintained within the population; given that there are F_t females in the population, only a fraction $F_t/(F_t + S)$ males successfully find a mate (Fauvergue (2013)). We assume a 50:50 sex ratio (i.e. $F = N_t/2$) so that

$$g(N_t) = \frac{N_t}{N_t + 2P} \tag{3}$$

Finally, we assume that survivorship of offspring to adults is density dependent so that

$$N_{t+1} = N_{t+0.5} S_N \tag{4}$$

where

$$S_N = \frac{S_0}{1 + (\alpha N_{t+0.5})^{\gamma}} \tag{5}$$

where the constant α is related to the carrying capacity, γ is related to intraspecific competition, and S_0 is survivorship in the absence of intraspecific competition (Slatkin & Smith (1979)).

This modeling framework allows us to analytically determine the equilibrium values of carrying capacity K as well as the Allee threshold. Equilibrium values in discrete time models occur when $N_{t+1} = N_t$, and the equilibria N^* of our model are given by the roots of the polynomial

$$\lambda\alpha(N^*)^2 + (1 - \lambda S_0)\, N^* + \theta = 0. \tag{6}$$

This expression is used to analytically determine the carrying capacity as used in Figures 1, 3, 4, and 5.

We build in environmental and demographic stochasticity based on this deterministic framework. Using methods similar to Schoener *et al.* (2003) and Melbourne & Hastings (2008), we first account for environmental stochasticity by rewriting Equation 1 as

$$N_{t+0.5} = m_e N_t f(N_t) g(N_t), \tag{7}$$

where the fecundity m_e is drawn at each generation from a normal distribution with mean m and a standard deviation σ. We also imposed the restriction that $m_e \geq 0$. Although we imposed this restriction, however, we note that the probability that of choosing a negative value is less than 10^{-6} as a result of the relatively

high fecundity. Therefore, the normal distribution is a reasonable choice to model environmental stochasticity.

Next, we included demographic stochasticity in two ways: in fecundity and in density dependent survivorship. Note that the total number of individuals that successfully reproduce is given by

$$B = N_t f(N_t) g(N_t). \tag{8}$$

We assume that each of these individuals at a given time t reproduces with fecundity m_e (as described above), and the total number of eggs produced is a Poisson random variable. Since the sum of independent Poisson random variables is also a Poisson random variable, the total offspring of all adults at is

$$N_{t+0.5} \sim \text{Poisson}\left(m_e B\right) \tag{9}$$

Finally, we include demographic stochasticity in density dependent survivorship. Given that S_N (as defined in Equation 5) is the probability that offspring survive to adults, we assume that survivorship is a binomially distributed so that

$$N_{t+1} \sim \text{Binomial}\left(N_{t+0.5}, S_N\right). \tag{10}$$

Infection dynamics

We consider the infection dynamics of up to two different cytotypes of *Wolbachia* (referred to as cytotypes X and Y) and denote the number of uninfected individuals as W. Note that all variables and parameters with subscripts X (or Y) are related to cytotype X (or Y). Note that the infection dynamics have the same assumptions as Dobson, Fox & Jiggins (2002).

In the presence of a single cytotype of *Wolbachia*, there are only unidirectional cytoplasmic incompatibilities (CI); in contrast, in the presence of multiple cytotypes there may be bi-directional CI. Therefore, we first introduce the case of a single cytotype and then modify the model to include two cytotypes.

One cytotype

To capture the *Wolbachia* dynamics, we first determine the proportions of infected and uninfected individuals in our population at time t. For example, if there are W_t uninfected individuals and X_t infected with cytotype X then, under the assumption that there is a 50:50 sex ratio, the fraction of females infected with cytotype X at $t + 0.5$ is given by

$$a_t = \frac{X_t}{W_t + X_t},$$

Similarly, we find the fraction i_t of all males are infected (where $i_t = a_t$), the fraction q_t of all males are uninfected, and the fraction c_t of all females are uninfected (again note that $q_t = c_t$).

Based on the proportions of uninfected and infected populations, we can now

192 determine the fraction of offspring that are infected. Crosses between pairs with
193 at least one infected individual suffer a fecundity loss due to infection $(1 - F_X)$.
194 Vertical transmission of *Wolbachia* occurs maternally and we assume that trans-
195 mission is successful with probability μ_X. The proportion of viable offspring that
196 are infected with cytotype X after reproduction (i.e. at time $t + 0.5$) is therefore
197 given by

$$X_{t+0.5} = a_t \left(1 - \mu_X\right) F_X \left(i_t + q_t\right) \tag{11}$$

198 Second, we can identify the proportion of viable offspring that are uninfected
199 $(W_{t+0.5})$. Uninfected individuals can arise from crosses both uninfected females
200 and males. Further, matings between both infected females and males can have
201 viable uninfected offspring. This results from failure to transmit *Wolbachia* to
202 their offspring (i.e. with probability μ_X). When one type of *Wolbachia* is present
203 within a population, then only unidirectional cytoplasmic incompatibility (CI) is
204 possible. This type of CI occurs through matings between infected males and
205 uninfected females. Therefore, we assume that pairings between infected males
206 and uninfected females undergo CI and a fraction H_X survives. The proportion
207 of viable offspring that are not infected with *Wolbachia* following reproduction is
208 given by

$$W_{t+0.5} = \left(\mu_X F_X a_t + c_t\right) \times \left(i_t H_X + q_t\right) \tag{12}$$

Notice that due to cytoplasmic incompatibilities and the fecundity cost due to infection with *Wolbachia*, the fraction of the total population that successfully reproduces ($X_{t+0.5} + W_{t+0.5}$) is less than one. Therefore, the total number of offspring as governed by Equation 1 can be rewritten as

$$N_{t+0.5} = m\left(X_{t+0.5} + W_{t+0.5}\right) N_t g(N_t) f(N_t). \tag{13}$$

In other words, the product $g(N_t)f(N_t)$ captures the total fraction of adults at time t who successfully find a mate, and the sum $X_{t+0.5} + W_{t+0.5}$ is the fraction of all offspring that are viable. Finally, as described in the previous section, density dependent mortality limits the total number of adults at time t (Equation 5).

Two cytotypes

When two cytotypes of *Wolbachia* are present within a population, bidirectional CI occurs when a male with one cytotype mates with a female infected with an incompatible *Wolbachia* cytotype. Similar to the previous section, we assume that a fraction H_X (or H_Y, depending on the infection type of the male and female) survives.

Therefore, in the presence of two strains we rewrite Equation 11 as

$$X_{t+0.5} = a_t\left(1 - \mu_X\right) F_X\left(i_t + j_t H_Y + q_t\right) \tag{14}$$

where j_t is the fraction of males infected with cytotype Y and b_t is the fraction

of females infected with cytotype Y. Similarly, the proportion of viable offspring infected with cytotype Y following reproduction is given by

$$Y_{t+0.5} = b_t \left(1 - \mu_Y\right) F_Y \left(i_t H_X + j_t + q_t\right). \tag{15}$$

The proportion of viable uninfected offspring is now given by

$$W_{t+0.5} = \left(\mu_X F_X a_t + \mu_Y F_Y b_t + c_t\right) \times \left(i_t H_X + j_t H_Y + q_t\right) \tag{16}$$

For simplicity, we assume that fecundity loss, transmission failure, and survival of CI are equal between cytotypes.

Results

In the following sections we first test our model against well-established results related to *Wolbachia* invasion as a method of model validation, establish the relationship between *Wolbachia* and the location of the Allee threshold, and finally evaluate the potential for the release of infected insects to control a population.

Model validation

We first determine whether our model captures the same features of the important earlier work (Hoffmann, Turelli & Harshman (1990); Turelli & Hoffmann (1991); Hancock, Sinkins & Godfray (2011)). Hoffmann *et al.* (1990) derived an analytic

expression for the expected equilibrium infection frequencies. After adjusting their notation to match ours and simplifying, the equilibrium infection frequency for a single cytotype of *Wolbachia* X should be the roots of

$$(1 - H_X) * (1 - \mu_X F_X) p^2 + (F_X + H_X - 2) p + 1 - F_X (1 - \mu_X) = 0.$$

Their work predicts that there is an unstable equilibrium, below which invasion is unsuccessful and above which invasion is successful. This equilibrium is therefore referred to as the invasion threshold. Initial infection frequencies above this value will increase until reaching the higher stable equilibrium that indicates a successful invasion.

Using similar methods to Charlat *et al.* (2005), Figure 1A displays the infection frequency at generation $t + 1$ as a function of the frequency at generation t (blue curve). Specifically, we compute the infection frequency between two subsequent generations with generation t on the horizontal axis and generation $t + 1$ on the vertical axis. Therefore, if the blue curve lies above the line $y = x$, then the infection frequency is decreasing. Similarly, if the dashed curve lies below the line $y = x$, then the infection frequency is increasing. The intersections of the black line and the blue curve indicate the equilibria, and the horizontal dashed lines display the analytically derived results of Hoffmann, Turelli & Harshman (1990). Unless otherwise stated, all parameters used are listed in Table 1. As evidenced by the figure, our simulation results is consistent with the analytically derived equilibrium infection frequencies.

Since our aim is to determine the interaction between Allee effects and *Wol-*

bachia invasion, we also determine whether the invasion threshold varies with the strength of the Allee effect θ. Using the same methods as used in finding the invasion threshold in Figure 1A, we identify the threshold as θ varies. As displayed in Figure 1B, the invasion threshold does not change. Additionally, the invasion threshold is not effected by the parameter S_0 (results not shown).

The effect of *Wolbachia* on the Allee threshold

To determine the dynamical effects of the presence of *Wolbachia* infection within a population, we find the Allee threshold both in the presence and absence of infection. In this section, we ignore stochasticity as well as the release of pheromones ($S = 0$). The Allee threshold at a given population size is found by calculating the reproductive rate between two consecutive generations (i.e. N_{t+1}/N_t). Because our model is in discrete time, reproductive rates above one result in positive growth, whereas population growth rates below one correspond to a declining population.

Figure 2A displays the probability of mating as determined by the strength of the Allee effect. We considered both a weak Allee effect – the population reproductive rate declines at low densities – (displayed in black) and a strong Allee effect – the population growth rate drops below one at low densities (displayed in red). In Figure 2B, the solid lines display the reproductive rates in the absence of *Wolbachia*-infected individuals. The intersection of these curves with the horizontal black line indicate equilibria, or

$$N_{t+1} = N_t. \tag{17}$$

As stated above, the black curve represents a weak Allee effect such that the only equilibrium is the carrying capacity, whereas the red curve has two equilibria: the first indicating the Allee threshold and the second is the carrying capacity.

In Figure 2B, the dashed and dash-dotted curves represent the reproductive rates of the population when the population size is comprised of 10% and 50%, respectively, infected individuals. These values are above the invasion threshold; therefore, this figure captures the dynamics during the replacement process when the population contains the specified distribution of infected and uninfected individuals. As a consequence of cytoplasmic incompatibilities, the maximum reproductive rate for both values of θ drops. However, although there is an increase in the Allee threshold in the case of strong Allee effects, the increase is relatively small. Interestingly, when the population is comprised of 50% *Wolbachia*-infected individuals, the invasion causes a large enough decrease in reproductive rate that it creates a strong, rather than weak, Allee effect. Although the Allee threshold is very small, it is an interesting result to consider especially in the contest of invasion management.

Implications for population management

Deterministic predictions

In this section, we directly characterize implications for population management through the release of infected individuals. As observed by Dobson, Fox & Jiggins (2002), there is a transient decline in the population size during the replacement of uninfected hosts by *Wolbachia*-infected individuals. Therefore, we first find the magnitude of this decline in the presence of Allee effects to deter-

mine the conditions under which the replacement process brings the population size below the Allee threshold, thereby forcing extinction.

To ensure that the introduction size is above the invasion threshold, in all simulations we assume that the introduction is large enough so that the infection frequency is 10%. This value lies just above the actual threshold of $\sim 9\%$. Therefore, the number of infected individuals introduced directly depends on the current host population size. When a single release is considered, we always assume that the release occurs in the first generation. When a second cytotype is released, we assume it occurs five generations after the initial release.

Initially, we consider the dynamics in the absence of stochasticity (Figure 3). Figure 3A-B displays the minimum population size relative to K during the replacement process following one and two releases, respectively. Notice that values of zero for the minimal population size indicate that the transient reduction in population size brought the population below the Allee threshold, therefore leading to deterministic extinction. To determine the success of the releases under varying reproductive rates, we replicated the results for four different values of S_0 . We observe that for all reproductive rates, releases of individuals infected with different cytotypes of *Wolbachia* leads to greater success of these methods. Importantly, we also find that the effectiveness of control via releases of infected individuals critically depends on the reproductive rate of a population: for low reproductive rates, even a single release is often capable of driving a population to extinction. In contrast, however, populations with relatively high reproductive rates are only driven to extinction with unrealistically strong Allee effects. For example, if $S_0 = 0.15$, a value of θ that is close to half of the carrying capacity is required for extinction to occur (Figure 3B).

While pest management through *Wolbachia* may be independently effective for species with relatively low reproductive rates, complementary tactics may be required when reproductive rates are high. Specifically, a tactic that either decreases the population size or further increases the Allee threshold may amplify the effects of *Wolbachia* introductions. Therefore, we consider the use of mating disruption through the release of pheromones (P) as a supplemental management tactic (see Equations 3 and 1). Because there is greater uncertainty surrounding the effectiveness of *Wolbachia* introductions in bringing pest populations to extinction, hereafter we use a relatively high reproductive rate ($S_0 = 0.15$). In Figure 3C, we fix the strength of the Allee effect relative to K at a value that is likely biologically reasonable ($\theta/K = 0.1$), and determine the minimum population size as the number of pheromone sources is varied. The solid curve displays the results in the absence of a release of *Wolbachia*-infected individuals, whereas the dotted and dashed-dotted curves display the results when one and two, respectively, cytotypes are released. Note that in the absence of pheromones, releases of infected individuals does not bring the population size below the Allee threshold (Figure 3AB). In contrast, combining both methods is significantly more effective than either tactic alone.

Stochastic predictions

The analysis in the previous section was centered on the deterministic outcome of population management strategies in the presence of Allee effects. Additionally, Figure 3 evaluates the success of releases of *Wolbachia*-infected individuals as well as techniques centered directly on mating disruption when a population begins at its carrying capacity. However, we did not consider the interaction of

Allee effects and stochasticity which can be especially important at low population sizes. Therefore, in this section we determine the ability of *Wolbachia* and mating disruption to drive population sizes with variable initial population sizes to extinction in the presence of Allee effects and stochasticity. As in Figure 3C, we implement a reproductive rate that is relatively high and releases of *Wolbachia*-infected individuals does not guarantee population extinction in the deterministic setting.

We find the probability of extinction based on 500 realizations of the stochastic model (i.e. Equations 7-10) for each relevant parameter combination of initial population size and strength of the Allee effect. To determine the relative roles of environmental and demographic stochasticity, we simulate the model while including both types of stochasticity (bottom row of Figure 4) as well as demographic stochasticity alone (top row of Figure 4). Further, we find the extinction probability under three scenarios: no introduction of *Wolbachia*-infected individuals, one introduction, and two introductions (each implemented as in Figure 3).

Similar to the deterministic results, we find that introducing two cytotypes of *Wolbachia* is more effective than one (Figure 4). However, as also noted in the deterministic results, the strength of the Allee effect (θ) required to force extinction is very high. Therefore, combining *Wolbachia* introductions with other methods that increase the Allee threshold (e.g. mating disruption) are critical for management success. Interestingly, local extinction is possible for large initial population sizes – including those at carrying capacity – for very strong Allee effects.

While there is little difference in whether both demographic and environmental stochasticity are simultaneously included in the model, environmental stochastic-

ity may increase the uncertainty of local extinction near the boundary between extinction and population survival. Moreover, when the strength of the Allee effect is high, demographic stochasticity does not increase extinction probability significantly as compared to the deterministic setting (e.g. Figure 3). This result follows from the relatively large initial population sizes near the Allee threshold when θ is large.

Our finding that the value of θ (strength of the Allee effect) required for population extinction in the deterministic setting is echoed in the stochastic simulations. This again highlights that the release of individuals infected with *Wolbachia* might be the most successful when combined with another tactic. Therefore, as in Figure 3C, we fix θ relative to K at a more realistic value of 0.1 and consider the dynamics with mating disruption only as well as mating disruption with the addition with one and two strains of *Wolbachia*. As shown in Figure 5, utilizing both mating disruption and CI is much more effective than using mating disruption alone. Additionally, it is important to note that when θ/K is fixed at 0.1, releases of infected individuals in the absence of mating disruption has little effect on the extinction probability. Therefore, these two methods can serve as complementary tactics for pest management.

Discussion

We investigated a population management strategy that considers *Wolbachia*-induced cytoplasmic incompatibility in the presence of Allee effects. In particular, we developed a stochastic population model, building upon the seminal approach of Dobson *et al.* (Dobson2002) and emerging ideas on the use of Allee effects for

the eradication of pest species (Liebhold & Bascompte (2003); Tobin, Berec & Liebhold (2011); Liebhold *et al.* (2016)). Our model demonstrates that the introduction of a small number of incompatible individuals into a pest population that has a strong pre-existing Allee effect can drive the pest population to extinction with no further intervention. We also demonstrate that extinction is possible for surprisingly large pest populations, and that combinations of more than one strain of *Wolbachia* and mating disruption via sex pheromones greatly increases the population's extinction risk. Our study thus unveils promising research avenues on a novel environmentally-friendly and elegant pest management strategy based on the sole manipulation of intrinsic population processes.

An important component of our modeling work was to uncover the basic interactions between Allee effects and cytoplasmic incompatibility (CI). We show that the interactions between Allee effects and CI are weak or non-existent: the invasion threshold does not depend on the strength of the Allee effect, and the Allee threshold has a marginal decrease in the presence of CI in the host population. Therefore, invasion of a particular *Wolbachia* strain into a population only depends on the threshold population size (or infection frequency) above which invasion begins in a deterministic setting (Barton & Turelli (2011)). This invasion threshold corresponds to a proportion of infected hosts above which infection spreads up to almost fixation, and is determined by parameters such as the reduction in egg hatch-rate caused by CI, the fitness costs of Wolbachia carriage, and the fraction of offspring that inherit the bacteria from an infected mother (Turelli (1994)). The invasion threshold found with our simulation model is consistent with that derived analytically (Turelli & Hoffmann (1991)), and unaffected by the intensity of a mate-finding Allee effect (Fig. 1B). In addition to adding validation to our model,

this also result holds interest because most theoretical approaches of *Wolbachia* invasion dynamics are purely genetic and consider changes in invasion frequency without considering host population dynamics. Our result follows that of Hancock *et al* (2011) in suggesting that *Wolbachia* invasion thresholds predicted analytically hold for closed populations, even when, as assumed here, host reproductive rate is affected by two opposite forms of density dependence.

In the context of strong Allee effects, the Allee threshold is the unstable equilibrium corresponding to the population size below which the population growth rate becomes negative (Wang & Kot (2001); BOUKAL & BEREC (2002); DeReDeC & COuRCHAmp (2007)). A population below the Allee threshold is therefore doomed to deterministic extinction, making the Allee effect a central paradigm for conservation (DeReDeC & COuRCHAmp (2007); Stephens & Sutherland (1999)), invasions (Taylor & Hastings (2005)), biological control introduction (Fauvergue *et al.* (2007, 2012)), and as hypothesized in the present work, eradication (Tobin, Berec & Liebhold (2011)). Whether an Allee effect is weak or strong, and in the latter case, the value of the Allee threshold depends on the strength of the underlying component Allee effect(s) relative to other density-dependent processes. Our simulations of various levels of cytoplasmic incompatibilities in a population with a mate finding Allee effect suggest that the Allee threshold itself is much less sensitive to extreme variations in CI (0-50% infected individuals), than it is to variations the mate-finding Allee effect (Fig. 2B). *Wolbachia*-induced cytoplasmic incompatibility does decrease population growth rate, as expected, but it has a minimal effect on the extinction threshold. Hence, *Wolbachia*-induced CI may be considered as a culling population management tactic, whereby population size is temporarily decreased as a result of cytoplasmic incompatibilities (Dobson, Fox &

Jiggins (2002)).

Despite their apparent independence, cytoplasmic incompatibility and the Allee effect yield very interesting emerging properties when acting in concert within transient dynamics that are triggered by the invasion of a new *Wolbachia* strain into an uninfected host population. Our first analysis that considered the combined occurrence of Allee effects and CI in a deterministic context reveals that the transient decrease in population size (resulting from the invasion of an incompatible *Wolbachia* strain into an uninfected insect population) is large enough to trigger extinction when the reproductive rate of the host species is relatively low. However, extinction is only observed in populations with higher reproductive rates for very strong Allee effects (Fig. 3A). However, the strength of the Allee effect required for extinction lowers for the introduction of an additional incompatible *Wolbachia* strain. The resulting insect extinction probability, estimated from simulations assuming stochasticity, confirmed the interaction between the two processes. Without CI, extinction probability is determined by population size and the strength of the component Allee effect (Fig 4A and 4D). Introducing infected individuals results in the extinction of populations that would have persisted otherwise (i.e., populations above the Allee threshold), and further increasing incompatibility via the release of an additional Wolbachia strain increases the extinction domain by reducing the severity of Allee effect necessary to trigger extinction (Fig 4).

Nonetheless, with the exception of species with low reproductive rates, our model predicts that although Allee effects and CI combine to drive populations to extinction, even in surprisingly large populations, these extinctions occur for unrealistically severe Allee effects. For instance, after the introduction of two incompatible *Wolbachia* strains, extinction is expected for $\theta/K \geq 0.4$, that is, if

half of the females fail to mate in a population that is at 40% of the carrying capacity. Unfortunately, field estimations of mate-finding Allee effects in insects are rare, but it is probable that mating failures only occur at very low densities. For instance, in the Gypsy moth *Lymantria dispar*, mating failures occurred below a density (estimated via the rate of male captures on sex-pheromone traps) of about 4 whereas the carrying capacity was estimated around 800 Tobin *et al.* (2007); Tobin, Onufrieva & Thorpe (2013), so that estimation of θ/K in this species could be one or two orders of magnitude lower than that yielding extinction in our model.

Importantly, we show here that cytoplasmic incompatibility management may independently be an effective strategy for populations with low reproductive rates, and effective more generally when applied in conjunction with mating disruption. Regardless of reproductive rate, our simulations demonstrate that eradication can be obtained via the combined introduction of *Wolbachia*-infected individuals and sex pheromone sources into an insect population subject to pre-existing Allee effect (Fig. 5). Two important conclusions can be drawn from this part of the study. First, this result is obtained with a set of realistic assumptions including relatively low intensity of Allee effect ($\theta/K = 0.1$), the introduction of only two different *Wolbachia* strains, and conceivable application of mating disruption (density of pheromone sources at 10% of K). Second, eradication is not restricted to small populations, but also applies to populations that have reached carrying capacity. Hence, via a specific focus on cytoplasmic incompatibility, our model supports conclusions from previous studies concerning the benefit of combining several population management tactics (Blackwood *et al.* (2012); Suckling *et al.* (2012)). Interestingly, *Wolbachia*-induced CI and mating disruption are fully compatible, and other methods for population control such as parasitism or predation

by native natural enemies may also be complimentary .

There is a long and prolific body of research in population dynamics that focuses on understanding the mechanisms stabilizing species near their carrying capacities (e.g. Hassell & May (1973); Robert M. May (1978); Bernstein (2000)). More recently, global climate change and the biodiversity crisis, including population declines, extinctions, or biological invasions, points towards the increasing relevance of nonequilibrium ecology (Rohde (2006)) and the biology of small populations (Fauvergue *et al.* (2012)). Transient dynamics are increasingly emphasized (Hastings (2004)) and sometimes considered in the specific context of population management (Ezard *et al.* (2010); Kidd & Amarasekare (2012)). The theoretical model developed in this study contributes to this perspective, and suggests that pest populations can be eradicated via the implementation of a cascade of intrinsic destabilizing processes. The introduction of individuals infected with *Wolbachia* strains incompatible to that of the resident pest population – few individuals, but numerous enough to exceed the *Wolbachia* invasion threshold – is akin to a butterfly effect, that is, a small perturbation with dramatic consequences. As first highlighted by Dobson *et al.* (2002), cytotype replacement which occurs in the course a successful *Wolbachia* invasion yields a transient coexistence of incompatible infections within a host population, and as a consequence, a transient decrease in reproductive rate. Here, the transients only last a few generations and this perturbation of the population's microbiome the first step in a destabilizing cascade. We show here that the population can then be pushed toward a second step of destabilization, triggered by a mate-finding Allee effect that can be reinforced by the application of mating disruption, which potentially drives the population to extinction.

Acknowledgements

We thank Sylvain Charlat for helpful comments in earlier stages of this work.

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658 bin, Patrick C. Onufrieva, Ksenia S. Thorpe, Kevin W. gypsy moth agency We
659 thank the Gypsy Moth Mating Disruption Working Group for thoughtful discus-
660 sions and support for much of the research conducted that enabled this analysis.
661 We also would like to thank the countless number of field workers, industry rep-
662 resentatives, collaborating scientists, and gypsy moth research funding agencies
663 that also made this analysis possible. Wiley-blackwell Hoboken Si.

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Table 1. Unless otherwise stated, all figures use these parameter values.

Parameter	Description	Value	Source
N_0	initial population size	varies	
m, m_e	*per capita* fecundity	25	
σ	standard deviation of fecundity	5	
S_0	survivorship in absence of competition	varies	
α	related to carrying capacity	0.00002	Dobson, Fox & Jiggins (2002)
γ	related to intraspecific competition	1	Dobson, Fox & Jiggins (2002)
μ_X, μ_Y	maternal transmission failure	0.03	Dobson, Fox & Jiggins (2002)
F_X, F_Y	fecundity effect	0.95	Dobson, Fox & Jiggins (2002)
H_X, H_Y	survivorship of cytoplasmic incompatibilities	0.05	Charlat *et al.* (2005), varies
θ	strength of Allee effect	varies	
P	number of false pheromones	varies	

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Figure captions

Figure 1. (A) Verification that our model accurately predicts the invasion threshold as analytically determined in (Hoffman *et al.* 1990). Here, we ignore Allee effects, stochasticity, and assume $P = 0$. The blue line is the frequency of infected individuals at time $t+1$ given the frequency at t; equilibria occur when the blue and black lines intersect. The red dashed lines indicate the analytically predicted equilibria. The smaller intersection is an unstable equilibrium that defines the invasion threshold: i.e. frequencies of *Wolbachia*-infected individuals above this threshold will successfully invade the population and approach the higher stable equilibrium. (B) demonstrates that Allee effects do not change the invasion threshold (dashed line is our model prediction, solid line is the analytically predicted equilibrium). In this figure, we use more extreme values related to CI to more clearly demonstrate the location of the invasion threshold (specifically, $\mu_X = 0.2$, $H_X = 0.1$).

Figure 2. (A) displays the probability of successful mating as determined by Allee effects as a function of population size. The black line corresponds to a weak Allee effect and the red corresponds to a strong Allee effect. The corresponding vertical lines indicate the population sizes at which half of the individuals successfully find a mate. (B) displays the reproductive rate between generations as a function of population size. Values above one correspond to population growth, and values below one correspond to decline. The colors have the same representation as in (A). The populations corresponding to the solid lines have no *Wolbachia*-infected individuals, dashed lines are comprised of 10% infected individuals, and dash-dotted lines have 50% of the population infected.

712

713 **Figure 3.** Deterministic results. (A) single introduction; (B) two introductions.
714 Plot displays the minimum population size relative to K over 50 generations as-
715 suming that $N_0 = K$. The solid line has $S_0 = 0.08$ (maximum reproductive rate
716 of 2 in the absence of AE, as in Dobson, Fox & Jiggins (2002)), the dashed line
717 has $S_0 = 0.15$ (maximum reproductive rate of 3.75 in the absence of AE), dash-
718 dotted line has $S_0 = 0.2$ (maximum reproductive rate of 5 in the absence of AE),
719 and dotted line has $S_0 = 0.25$ (maximum reproductive rate of 6.25 in absence of
720 AE). (C) assumes that θ relative to K is fixed 0.1 (as displayed in A and B) with
721 $S_0 = 0.15$, and instead varies the number of pheromone sources P relative to K.
722 The black line assumes there is no *Wolbachia*-infected individuals, dashed assumes
723 a single release, and dash-dotted assumes a release of two cytotypes. In all plots,
724 each release is created so the initial infection frequency of that cytotype is 10%.
725 The first release is at generation one, and the second is at generation six.

726

727 **Figure 4.** Using $S_0 = 0.15$, the colors of each plot represent the extinction
728 probability for a given parameter combination based on 500 realizations of the
729 model. In each plot, the initial population size and the strength of the Allee effect
730 θ relative to K are varied. Top row: demographic stochasticity only. Bottom
731 row: both demographic and environmental stochasticity. First column: no intro-
732 duction. Second column: introduction such that infection frequency is at 10%.
733 Third column: two subsequent introductions. Gray line is the Allee threshold (i.e.
734 initial populations below the gray line go to extinction in the deterministic model).

735

736 **Figure 5.** Using $S_0 = 0.15$ and fixing $\theta/K = 0.1$,, the colors of each plot represent

the extinction probability for a given parameter combination based on 500 realizations of the model. In each plot, the initial population size and the number of pheromone sources (P) relative to K are varied. Top row: demographic stochasticity only. Bottom row: both demographic and environmental stochasticity. First column: no introduction. Second column: introduction such that infection frequency is at 10%. Third column: two subsequent introductions.

Figure 1:

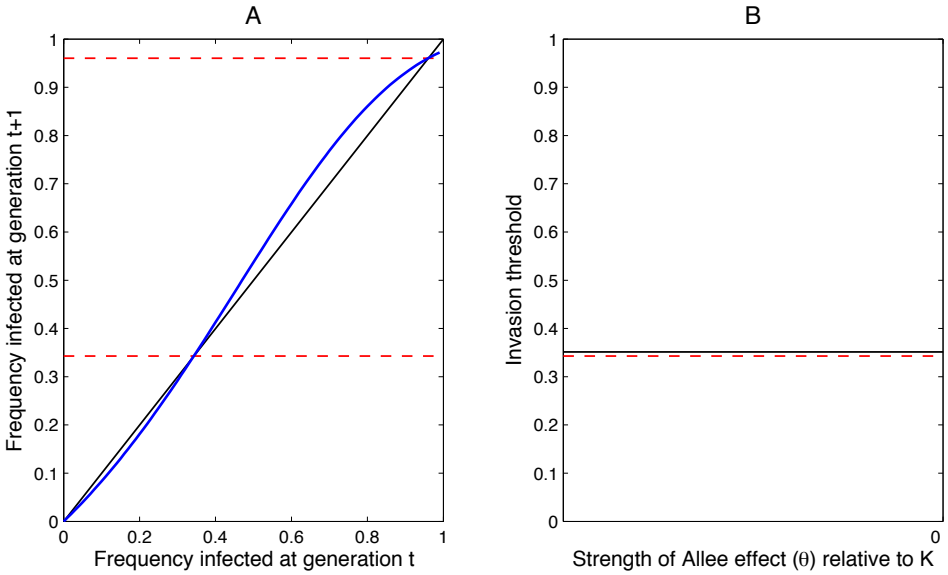

Figure 2:

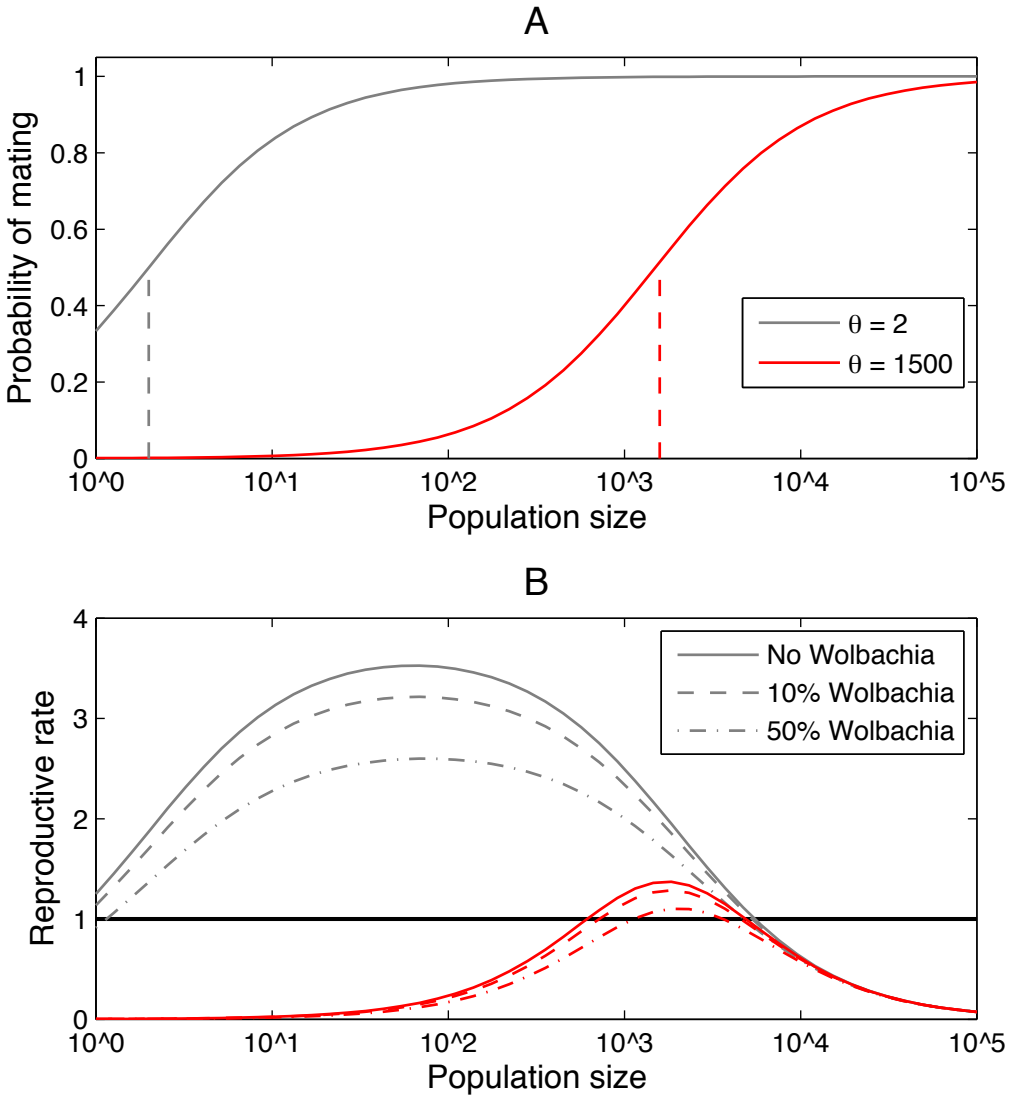

Figure 3:

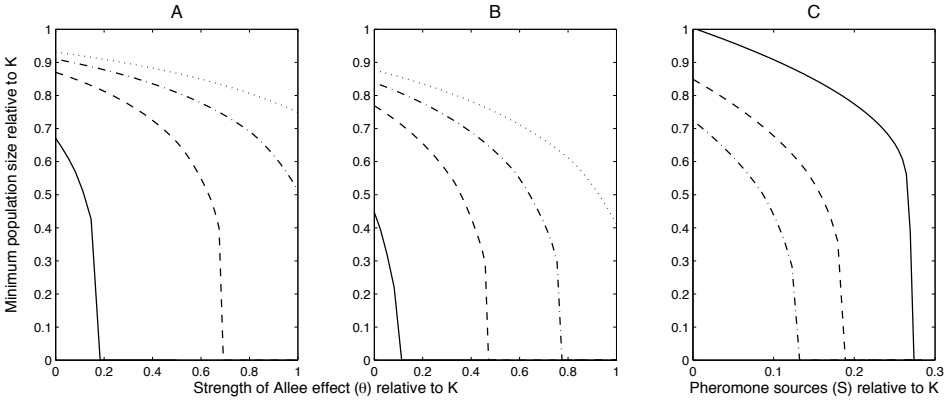

Figure 4:

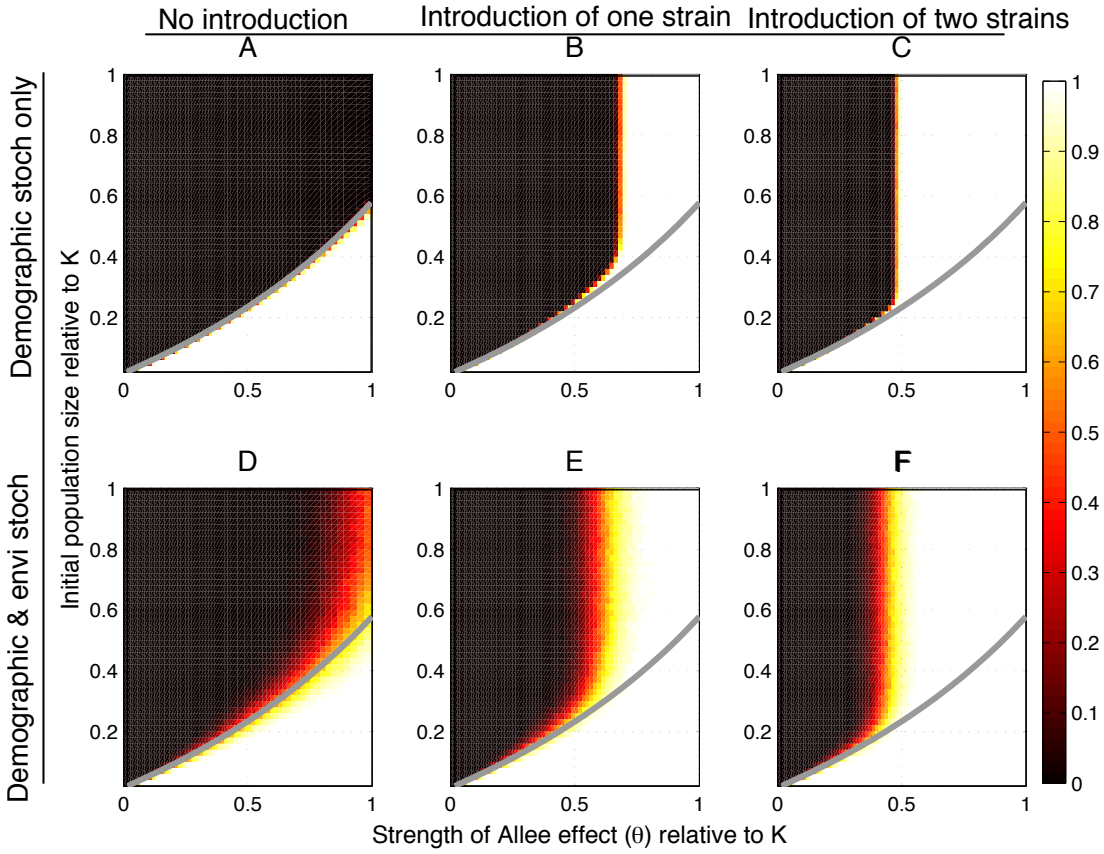

Figure 5: