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Optimal barrier zones for stopping the invasion of *Aedes aegypti* mosquitoes via transgenic or sterile insect techniques

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Abstract Biological invasions have dramatically altered the natural world by threatening native species and their communities. Moreover, when the invading species is a vector for human disease, there are further substantive public health and economic impacts. The development of transgenic technologies is being explored in relation to new approaches for the biological control of insect pests. We investigate the use of two control strategies, classical sterile insect techniques and transgenic late-acting bisex lethality (Release of Insects carrying a Dominant Lethal), for controlling invasion of the mosquito *Aedes aegypti* using a spatial stage-structured mathematical model. In particular, we explore the use of a barrier zone of sterile/transgenic insects to prevent or impede the invasion of mosquitoes. We show that the level of control required is not only highly sensitive to the rate at which the sterile/transgenic males are released in the barrier zone but also to the spatial range of release. Our models characterise how the distribution of sterile/transgenic mosquitoes in the barrier zone can be controlled so as to minimise the number of mass-produced insects required for the arrest of species invasion. We predict that, given unknown rates of mosquito dispersal, management strategies should concentrate on larger release areas rather than more intense release rates for optimal control.

Keywords Barrier zone · Biological control · Dengue fever · RIDL · SIT · Transgenic insects

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Introduction

Ecological invasions are increasingly common, primarily due to human-mediated influences (Lodge 1993; MacKenzie et al. 2004). Whilst some invasions are benign and may go unnoticed, others can have devastating effects on ecosystems, inducing enormous costs each year in terms of the inflicted damage and control strategies (Pimentel 2011). This is particularly true of emerging and resurging infectious diseases due to the spread of insect vectors, such as mosquitoes (Mackenzie et al. 2004; Tatem et al. 2006) and midges (Purse et al. 2005), leading to extensive efforts exploring cost-effective invasion control. These counter measures typically have been chemical-based; however, alternatives such as ecological interventions have been sought due to (1) the ever-present risk of pest resistance, (2) an increasing pressure for countries to adopt a low-carbon economy in the future and (3) the desire for environmentally benign pest control (Dyck et al. 2005).

One environmentally friendly alternative is the sterile insect technique (SIT) (Knipling 1955). This species-specific method of insect control relies on the mass rearing, sterilisation and release of large numbers of sterile insects (ideally males) (Dyck et al. 2005) which, it is hoped, mate with wild-type insects, thereby reducing their reproductive output and, potentially, the pest population abundance (see Black et al. (2011) and Wilke et al. (2012) for recent reviews). In general, mixed-sex sterile releases are not preferable: they are often less efficient as released males and females may mate with each other instead of the target wild-type insects. Furthermore, for all mosquito species, it is only the females that bite and therefore releasing sterile female mosquitoes could potentially aid vector-borne disease transmission in the short term (see Alphey et al. (2010) for a recent review).

The SIT has had a number of successes, particularly with the eradication of New World screw worm from the Southern USA, Mexico and Central America (Dyck et al. 2005), using successive area-wide release strategies. New World screw worm adult flies lay their eggs in the wounds of livestock, often with lethal or economically important consequences, and they are therefore a serious problem in cattle farming. Currently, the Panama and US governments have established a permanent barrier zone in the Darien Gap in Panama, where approximately 50 million sterile New World screw worm flies are released per week (Dyck et al. 2005). This biological barrier zone stops species reinvasion and protects economically important industries: it has been estimated that the annual benefit from the campaign to the livestock industry is valued at USD 896 million per annum (Vreysen et al. 2007).

Despite this success, the SIT has several constraints that make the control strategy less than ideal. This is in part due to the damaging effect of sterilising doses of radiation on the mosquitoes, which makes irradiated males less able to compete for mates (Helinski et al. 2009; White et al. 2010). However, radiation dose optimisation has led to greater effectiveness of the technique (Parker and Mehta 2007), with some studies showing little competitive reduction from radiation (Mastrangelo et al. 2012; Oliva et al. 2012; Sow et al. 2012). Nonetheless, transgenic technologies have recently been developed to improve SIT control (Benedict and Robinson 2003; Wimmer 2003; Alphey et al. 2010); these include genetic sexing (Robinson et al. 1999), genetic marking (Peloquin et al. 2000) and genetic female-specific lethality (Seawright et al. 1978).

One such transgenic strategy is Release of Insects carrying a Dominant Lethal (RIDL) (Thomas et al. 2000; Phuc et al. 2007; Harris et al. 2011). Initially, this technology was developed to control *Aedes aegypti* mosquitoes, which can spread yellow fever, dengue fever and chikungunya disease, and are the focus of our study. The released RIDL transgenic male mosquitoes are homozygous for a dominant lethal gene that is expressed in both male and female (bisex) progeny resulting from mating with wild-type insects. Female-specific RIDL strategies have also been developed (Fu et al. 2010), but we focus on bisex RIDL control strategies here. Hereafter, we use the terms 'SIT' and 'sterile' to refer to early acting lethality of the progeny of released insects, for example classical SIT using radiation-induced sterility, and the terms 'RIDL' and 'transgenic' to refer to late-acting lethality in both sexes.

The use of genetically modified (GM) insects as a control strategy is relatively novel, yet for RIDL, extensive caged open and closed field trials (de Valdez et al. 2011; Lee et al. 2012) and full-scale field trials have successfully taken place (Harris et al. 2011). In the first field trial conducted in the Cayman Islands (Harris et al. 2011), males (strain OX513A) were released in a 10-ha area at an average release rate of 465 males/ha/week for 4 weeks, resulting in a significant reduction in wild-type mosquitoes (approximately 80 %), thus demonstrating the feasibility of the technology. Whilst the SIT strategy carries certain fitness costs for the sterilised mosquitoes, there seems to be little costs carried by the transgenic mosquitoes (see White et al. (2010) and references therein). Thus, there is a potential for RIDL to be an effective control for *A. aegypti*; however, the obstacle remains accepting the use of the RIDL GM technology, but such self-limiting strategies are widely viewed as the lowest risk category (Alphey and Beech 2012).

In contrast to SIT, the developmental stage at which the dominant lethal gene is expressed in RIDL, for instance the embryonic or the larval stages, can have a substantial effect on the control strategy. In particular, late-acting genes, which induce death after the density-dependent larval stage, have a significant advantage over SIT strategies because of an additional reduction in pest abundance as a result of larval competition (Atkinson et al. 2007; Phuc et al. 2007; White et al. 2010).

There has been a long history of temporal mathematical models used in SIT (Knipling 1955; Berryman 1967; Barclay and Mackauer 1980). Typically, the models define a pest population in a single equation as either a discrete-time difference equation (see Knipling (1955), for example) or as a continuous-time differential equation (see Barclay and Mackauer (1980), for example), within which sterile insects are released at a constant rate to reduce the pest population. The critical release rate (the minimum rate of sterile release required to eradicate the pest population) is then calculated. However, these models fail to take into account the stage-structured life history of the insects, which can have significant effects on their dynamics and hence the accuracy of the model predictions. This is now being addressed in the literature using delayed differential equations (Phuc et al. 2007; White et al. 2010).

The dispersal dynamics of *A. aegypti* is not well understood (Reiter et al. 1995; Harrington et al. 2005). Harrington et al. (2005) demonstrated empirically, using mark–release–recapture techniques, that both sexes of adult *A. aegypti* mosquitoes disperse similarly and over relatively short distances. However, modern anthropogenic activities have led to greater ranges for this disease vector. For example, global human movement and goods trafficking have led to long-distance dispersal events for *A. aegypti* (Enserink 2010). Furthermore, it is likely that climate change will have a major impact on species distributions, leading to mosquito range expansion, probably by a combination of direct climatic influences (e.g. temperature, wind, rainfall) and indirect changes via human activities (e.g. creation of water storage to mitigate against drought or population increases) (Beebe et al. 2009; Jansena and Beebe 2010). Therefore, understanding the impacts of *A. aegypti* dispersal is critical for effective pest control.

Dispersal dynamics have previously been highlighted as an important feature in mathematical models of *A. aegypti* invasion (Lewis and Driessche 1993; Takahashi et al. 2004). In addition, Yakob et al. (2008) and Yakob and Bonsall (2009) studied a spatial model focused on the global effects of controlling an established *A. aegypti* population on a local scale, demonstrating that the migratory behaviour of the female mosquitoes greatly influences the prospects of control success via SIT or RIDL control strategies. These studies of *A. aegypti* within a spatial framework focussed mainly on objectives aimed at minimising pest population levels via the use of transgenic control strategies, including any collateral influences outside of the control region and the question of optimisation. In contrast, here, we are focused on the extent to which sterile or transgenic control strategies can be used as a barrier to prevent or impede invasion, rather than simply a minimisation of an established pest. This firstly raises the prospect of subtle differences in control behaviour as very low pest densities within the control region may nonetheless support a low insect density invasive wave leading to control failure. Thus, a priori, one cannot rely on simple modelling predictions aimed at reducing pest population levels.

Our objective is to consider models of biological barrier controls, targeting *A. aegypti* mosquito populations, whilst highlighting generic features required for preventing or impeding invasions. In addition, we aim to compare and contrast SIT and RIDL strategies within this framework, as well as considering how associated costs (in terms of numbers of sterile/transgenic mosquitoes required for control) may be optimised and the prospects for sensitivity to parameters, especially any that are difficult to estimate, to aid with cost-effective pest control management.

Methods

Mathematical models

In contrast to previous studies, Yakob et al. (2008) for example, we model insect densities in continuous time and space rather than using a discrete approach. This removes difficulties of prospective lattice dependencies within the modelling predictions, though the relationship between continuum and discrete modelling frameworks is very much an open area of research within mathematical biology and is severely underdeveloped for models with both stage and spatial structures. Consequently, comparing and contrasting the results of predictions emerging from both frameworks is certainly a subject for further work in the context of insect pest control. Here, we simply focus on the continuum approach, albeit highlighting similarities with previous results from discrete models where possible.

Temporal model

The temporal model of the wild-type mosquito population we consider here is derived from the framework of Dye (1984) which has been utilised in numerous previous studies (Barclay 2001; Phuc et al. 2007; Yakob et al. 2008; White et al. 2010). We define the density of wild-type female mosquitoes to be $N(t)$ and that of sterile/transgenic male mosquitoes to be $S(t)$, at time t. We assume that: (1) mosquito population growth proceeds via a stage-structured process and density-dependent mortality acts on a pre-adult developmental stage; (2) the sex ratio of the wild-type mosquitoes is 1:1; (3) wild-type male mosquitoes mate in proportion to their relative abundance (Knipling 1955; Phuc et al. 2007), at a rate given by $N(t)/(N(t)+cS(t))$ where $0 < c \leq 1$ represents the reduced mating competitive ability of sterile male or transgenic male mosquitoes so that $c = 1$ implies that the control competes equally with the wild-type female mosquito, whereas small c, in contrast, entails that the biological control is extensively outcompeted in reproducing with females; and (4) the female wild-type and male sterile/transgenic mosquitoes have a per capita death rate μ. Whilst the death rate of the controls could be reduced to reflect their lower fitness, we find the models to be insensitive to this particular detail and so we use the same decay rate for both populations; (5) male sterile/transgenic mosquitoes are released at a constant rate $\kappa = \theta N^*$, where N^* is the control-free equilibrium density of wild-type mosquitoes and θ is the release rate ratio.

These assumptions lead to the SIT/RIDL model:

$$\frac{\mathrm{d}N(t)}{\mathrm{d}t} = rN(t-T)\left(\frac{N(t-T)}{N(t-T)+cS(t-T)}\right)\Phi(t) - \mu N(t), \quad (1a)$$

$$\frac{\mathrm{d}S(t)}{\mathrm{d}t} = \kappa - \mu S(t), \quad (1b)$$

where $\Phi(t)$ is the density-dependent function induced by competition in the larval stage. The parameter r is the per capita daily egg production rate per adult female corrected for survival from the egg to the adult stage and T is the mosquito developmental time.

For SIT, which induces no offspring, the density-dependent function is given by (Phuc et al. 2007; White et al. 2010)

$$\Phi(t) = \exp\left[-\alpha\left(N(t-T)\left(\frac{N(t-T)}{N(t-T)+cS(t-T)}\right)E\right)^{\beta}\right]. \tag{2}$$

Here, $1/\alpha$ is the size at which the wild-type female mosquito population reproduces at maximum rate, E is the egg production rate of adult mosquitoes without correction for density-independent survival between the egg stage and adulthood and β is a parameter derived from fitting empirical data, as detailed in Dye (1984). It describes the qualitative characteristics of density dependence at the larval stage: higher values mean that overcompensatory regulation occurs at lower densities (for a given α) and acts more sharply (a slight increase in initial density causes a large decrease in the number of survivors). In contrast, the late-acting lethal induced by RIDL leads to (Phuc et al. 2007; White et al. 2010)

$$\Phi(t) = \exp\left[-\alpha(N(t-T)E)^{\beta}\right]. \tag{3}$$

In particular, note that for RIDL strategies, the stage-structured larval-dependent competition term has an exponent which scales with the number of reproductions at time $t-T$, following Gurney et al. (1980). For SIT strategies, this is, in contrast, rescaled to reflect the number of futile, sterile matings which therefore implicitly differentiate between how the control interventions affect larval competition. When c is close to unity, RIDL larvae are as competitive as the wild-type and contribute equally to density-dependent mortality. In contrast, when c is close to zero, RIDL larvae contribute nothing to density-dependent mortality and this scenario is equivalent to an early acting conventional SIT system. Thus, we choose c to be close to unity, reflecting a small fitness cost (Lee et al. 2013)—further examination of this parameter can be found in White et al. (2010).

It should be noted that we take the density-dependent parameters from Dye (1984), following many previous studies. However, Legros et al. (2009) have called these values into question by using an alternative technique and finding different parameter estimates. The qualitative results that follow do not change for these alternative values and we detail this further in "Discussion".

Spatial model

We now extend the temporal model (1a–3) to a spatial barrier zone model (see Fig. 1a for a schematic), assuming that the environment for mosquitoes is homogeneous. In particular, we consider a diffusively spreading invasion of wild-type females, which leave a wake of wild-type female mosquitoes at carrying capacity (N^*) behind the invasive front. The barrier zone is constructed by locally releasing sterile/transgenic male mosquitoes in front of the wild-type mosquito travelling wave. Since the larvae mature in a fixed location where the eggs are laid, the adult mosquito distribution depends on the adult density locally, at the current location, without a dispersive kernel. Hence, the spatial model with constant diffusion is given by

$$\begin{aligned}\frac{\partial N(x,t)}{\partial t} &= D_N\frac{\partial^2 N(x,t)}{\partial x^2} + rN(x,t-T)\\ &\quad\times\left(\frac{N(x,t-T)}{N(x,t-T)+cS(x,t-T)}\right)\Phi(x,t)-\mu N(x,t),\end{aligned} \tag{4a}$$

$$\begin{aligned}\frac{\partial S(x,t)}{\partial t} &= D_S\frac{\partial^2 S(x,t)}{\partial x^2}\\ &\quad+\kappa(x)-\mu S(x,t), x\in\Omega, t\in(0,+\infty],\end{aligned} \tag{4b}$$

where D_N and D_S are the diffusion rates of the wild-type female and sterile/transgenic males, respectively, and Ω is a sufficiently wide region of several hundred kilometres. Thus, we implicitly assume that any external motility influences, such as wind patterns and favourable environments, are randomly isotropic and do not bias mosquito motility. Furthermore, we assume that mosquitoes do not emigrate from or immigrate into the region and thus $\partial N/\partial x$ and $\partial S/\partial x$ are zero on the boundary of Ω. The density-dependent function, $\Phi(x,t)$, is given by Eqs. 2 and 3 by simply exchanging $N(t)$, $S(t)$ for $N(x,t)$, $S(x,t)$.

The sterile/transgenic males are released locally in space and continuously in time with constant rate per unit length, θN^*, which serves to define θ. This is described in detail via the release function

$$\kappa(x)=\theta N^*\chi(x),\ \chi(x)=\begin{cases}1 & x\in A=[\bar{x}-\gamma_s/2,\bar{x}+\gamma_s/2]\\ 0 & x\in\Omega\backslash A\end{cases}, \tag{5}$$

where $\gamma_s \ll |\Omega|$ is the release range and $\bar{x}$ is the centre of the release region (Fig. 1a). The initial conditions are depicted in Fig. 1a, with a travelling wave for wild-type female, $N(x,t)$, approaching the barrier zone from the right.

Parameter values

The life history parameter values for *A. aegypti* were estimated by Dye (1984) and we adopt them here following

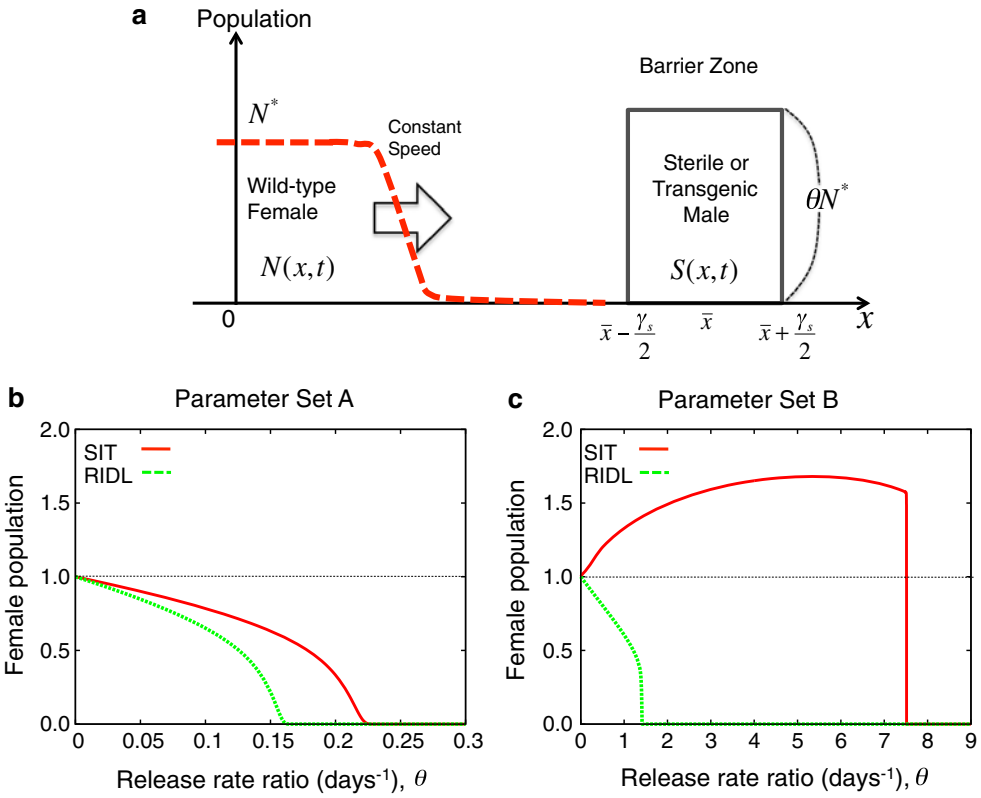

Fig. 1 **a** An illustration of the barrier zone strategy in the presence of spatial heterogeneity. Sterile/transgenic male mosquitoes are released (and disperse) in front of the invading travelling wave of wild-type mosquitoes. Control insects are released over an area γ_s and are released at rate θN^* per unit length, where N^* is the female wild-type equilibrium density and θ is the release rate ratio. After release, control insects disperse via diffusion. **b**, **c** An illustration of the quantitative dynamics associated with wild-type female mosquitoes for SIT/RIDL strategies in the spatially homogenous setting. The figures show the wild-type female population dynamics for each control strategy in the purely temporal models, Eqs. 1a–3, given the parameter sets A and B. The plots give the normalised equilibrium female population in terms of θ, the control release rate ratio

Phuc et al. (2007), Yakob et al. (2008) and White et al. (2010). However, Dye estimated a range of values for the intrinsic birth rate, r, and the density-dependent coefficient, β, which yields contrasting values of the equilibrium density, N^*. We focus on two sets of parameters representing the extremes of r and β: these are relatively small for parameter set A and give rise to a stable equilibrium which is approached monotonically in the absence of control strategies. In contrast, parameter set B has substantially larger values of r and β and exhibits damped oscillatory dynamics with a rapidly growing population.

Whilst these parameters give qualitatively similar mosquito dynamics, the two parameter sets result in very different predictions concerning the control of *A. aegypti* mosquitoes (Phuc et al. 2007; White et al. 2010), as illustrated in Fig. 1b, c. Whilst both SIT and RIDL are similarly influenced by control strategies in the case of parameter set A as shown in Fig. 1b, a moderate release rate of sterile mosquitoes for parameter set B may undesirably increase the wild-type mosquito population as there is a reduction in competition that offsets the reduced birth rate. Thus, these two parameter sets can be thought of as best and worst case scenarios (Phuc et al. 2007; Yakob et al. 2008; White et al. 2010), and given the uncertainty for these parameter estimates (see also Legros et al. (2009)), we consider both parameter sets when exploring barrier zone control strategies aimed at preventing pest invasion.

Empirical estimates for the diffusion rates of *A. aegypti* mosquitoes are generally lacking (Reiter et al. 1995; Harrington et al. 2005). Thus, we assume several scales of diffusion rates from hundreds of square metres per day to several square kilometres per day; for simplicity, we also assume that the sterile/transgenic mosquitoes have the same diffusion rates as the wild type.

The parameter values are listed in Table 1. Note that although we present numerical results with representative diffusion rates and the parameter sets in Table 1, simple parameter rescaling such as non-dimensionalisation leads to the same results for a large range of parameter choices so that our results are not restricted to the parameters listed therein.

Results

Due to its complexity, an analytical exploration of the model (4a–5) is not possible. Therefore, we use extensive numerical simulations (see Appendix 1 for details).

Mosquito wave speed and barrier zone dynamics

Before exploring the influence of the two parameter sets on the barrier zone strategy, we first consider the wave speed of the invasion of wild-type female mosquitoes in

Table 1 Parameter values used in simulations. With the exception of the diffusion coefficient and control parameters, we have followed numerous studies in the literature, e.g. Phuc et al. (2007) and White et al. (2010), and based our parameter choices on Dye (1984). Further details of the parameter estimations can be found in this study which used extensive empirical data to estimate the two extremes of mosquito population dynamics in the absence of control insects, yielding parameter sets A and B below.

Parameter/variable	Definition	Value	Ref.
N	Density/number of female wild-type mosquitoes		
S	Density/number of male sterile or transgenic mosquitoes		
Ω	Whole spatial region size	500 or 250 km	
γ_s	Width of release region	[125 m, 50 km]	
D_N	Diffusion coefficient for female wild-type mosquitoes	[0.01, 1.0] (km^2/day)	
D_S	Diffusion coefficient for male control strategy mosquitoes	[0.1, 1.0] (km^2/day)	
T	Mosquito development time	18.84 days	Dye (1984)
c	Coefficient of reduced mating competitive ability of sterile/transgenic male mosquitoes	0.95	Phuc et al. (2007)
α	Density-dependent coefficient ($1/\alpha$ is the size for which the wild-type female mosquitoes reproduce most rapidly)	0.01[a]	White et al. (2010)
E	Average egg production rate of adult females	1.0	White et al. (2010)
μ	Death rate of wild-type adult females	0.12 days^{-1}	Dye (1984)
κ	Release rate of control strategy males	θN^* days^{-1}[a]	
θ	Release rate ratio of control strategy males	[0, 20](days^{-1})	
Parameter set A			
r	Birth rate of adults corrected for egg to adult survival	0.367 days^{-1}	Dye (1984)
β	Density-dependent coefficient	0.302	Dye (1984)
N^*	Control-free female mosquito equilibrium $([(1/\alpha)\ln(r/\mu)]^{1/\beta}/E)$	6.064×10^6[a]	
Parameter set B			
r	Birth rate of adults corrected for egg to adult survival	1.31 days^{-1}	Dye (1984)
β	Density-dependent coefficient	1.0	Dye (1984)
N^*	Control-free female mosquito equilibrium	239.0[a]	

[a]For the spatial model, this value is given per unit length. Note that since we scale the wild-type population relative to the pre-control equilibrium in our simulations, the relative measure of adult population numbers is independent of the magnitude of $\alpha > 0$ and $E > 0$ (Phuc et al. 2007).

the absence of control strategies. In Fig. 2a, a linear dependence can be observed between the invasive wave speed and the square root of the wild-type mosquito diffusion coefficient (see Appendix 2 for a proof). Nonetheless, the differences between parameter sets A and B are sufficient to induce a change in the constant of proportionality within this relationship; at higher diffusion coefficients, the differences in invasive wave speeds will be magnified between the two parameter sets, suggesting that there will be a greater sensitivity to barrier control strategies.

The behaviour of wild-type female mosquito invasive waves governed by parameter set B, and encroaching a biological barrier, is depicted in Fig. 2b. In Fig. 2c–e, the behaviour of these wild-type female mosquitoes on penetrating the biological barrier, consisting either of SIT or RIDL control insects, is highlighted for different barrier zone release areas. In Fig. 2c, when the release area is large, we see that both SIT and RIDL control strategies prevent propagation of the wild-type mosquito wave. However, the resulting wave of wild-type mosquitoes for the SIT strategy exhibits an increase in abundance within the barrier zone, demonstrating the well-known phenomenon that sterile insect biological controls can locally and undesirably increase wild-type pest densities due to larval competition (Phuc et al. 2007; Yakob et al. 2008; White et al. 2010).

In contrast, in Fig. 2d, where the release area is reduced, the SIT strategy is unsuccessful and the barrier is penetrated; however, the RIDL strategy maintains its barrier function, illustrating that late-acting transgenic RIDL strategies are superior, in general, to SIT releases and that the release region size is crucial in the design of a control strategy. Finally, in Fig. 2e, where the release area is small,

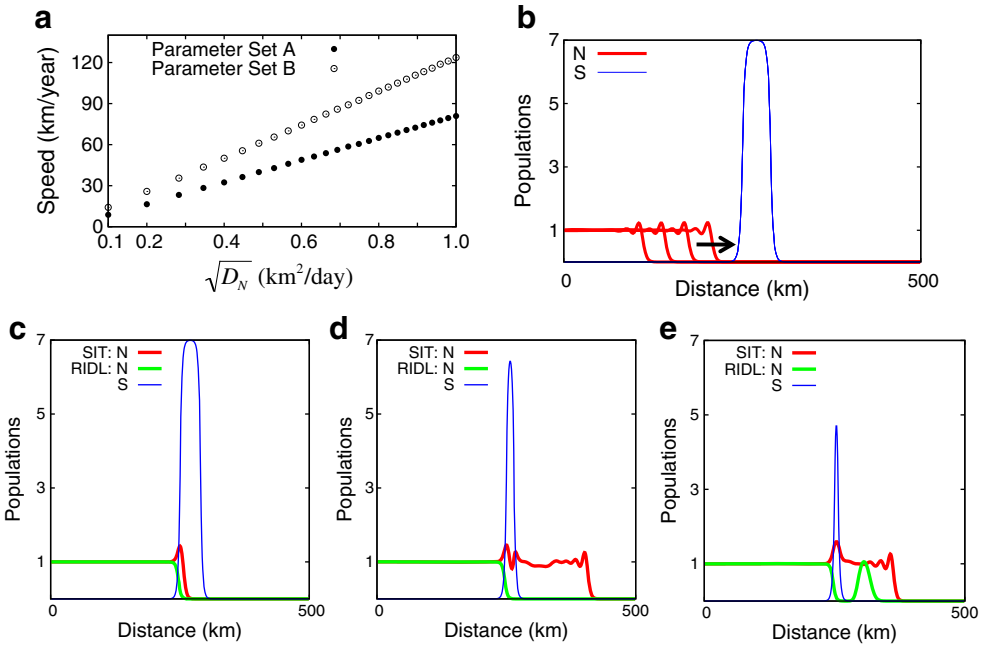

Fig. 2 Example results for barrier intervention strategies. The wild-type population is governed by the worst case scenario of parameter set B with the diffusion rates $D_N = D_S = 1$ km^2/day within plots (**b**–**e**). **a** The dependence of the wild-type female mosquito invasion speed in the absence of control as the diffusion coefficient is varied. **b** An invasive wave of female mosquitoes approaching a barrier zone. The wild-type profile has settled to a constant wave speed propagating at approximately 130 km/year. **c**–**e** show three cases of control for SIT/RIDL strategies with variations in the release area for a fixed release rate ratio $\theta = \kappa/N^*(= 7.0)$, with γ_s=40 km in (**c**), γ_s=15 km in (**d**) and γ_s=7 km in (**e**)

both the SIT and RIDL strategies fail to arrest the invasive front of wild-type mosquitoes. Note that, in the case of RIDL, very low wild-type densities are responsible for barrier penetration. This enables small numbers of mosquitoes to breach the barrier zone, thereby initiating a new wave that subsequently propagates away from the barrier.

Barrier size, the release effort and optimality

In the following, we explore the influence of different release rates and release region sizes associated with the control strategy via variation of the control parameters θ (the release rate ratio) and γ_s (the size of the release region), in particular highlighting the threshold for successful control in Fig. 3. Here, the region above the plotted thresholds corresponds to successful control, in contrast to the lower regions where wild-type mosquitoes penetrate the prospective biological barrier.

Unsurprisingly, it is difficult to extract insight concerning effective barrier sizes from purely temporal models, in that one cannot predict the threshold curves given in Fig. 3 by inspection of Fig. 1b, c. However, one particular aspect is clearly inherited from the temporal modelling predictions: for parameter set A, SIT and RIDL perform similarly, but the latter is clearly substantially superior for parameter set B. In particular, here the reduction in larval competition induced by SIT strategies is especially pronounced and undesirable. Under such circumstances, Fig. 3 indicates that SIT strategies require substantially higher release rates rather than substantially larger barrier regions for the prevention of mosquito invasion.

We also observe in Fig. 3 that, at sufficiently small release rates, denoted by θ_R^* and θ_S^* for RIDL and SIT, respectively, barrier failure occurs regardless of the release region. Thus, there exist minimal release rates for population control; failure to exceed them will result in the barrier zone failing, regardless of the size of the release region. In contrast, no such minimal positive value exists for the release region, γ_s, and therefore any release region may be employed for barrier control so long as a sufficiently high release rate is used. These results also suggest that, whilst theoretical invasion control strategies may be used, they need not be particularly cost-effective or optimal.

Hence, from a management perspective, we are particularly interested not only in the release rate required to stop an invading wave but also in reducing the numbers of control insect required. To this end, we define the release effort, [EF], as the 'size' of the released barrier zone. Mathematically, this is defined as

$$[\mathrm{EF}] = \gamma_s \times \theta N^*, \tag{6}$$

where γ_s is the release region and θN^* is the release rate. This is denoted by the shaded area in the barrier zone schematic in Fig. 1a and provides a measure of the effort required in order to achieve population control.

It can be shown (see Appendix 3) that the release effort satisfies

$$[\mathrm{EF}] = \mu \lim_{t\to\infty} \int_\Omega S \, \mathrm{d}x. \tag{7}$$

Hence, this effort function provides a pragmatic measure for the total number of control insects in the environment due

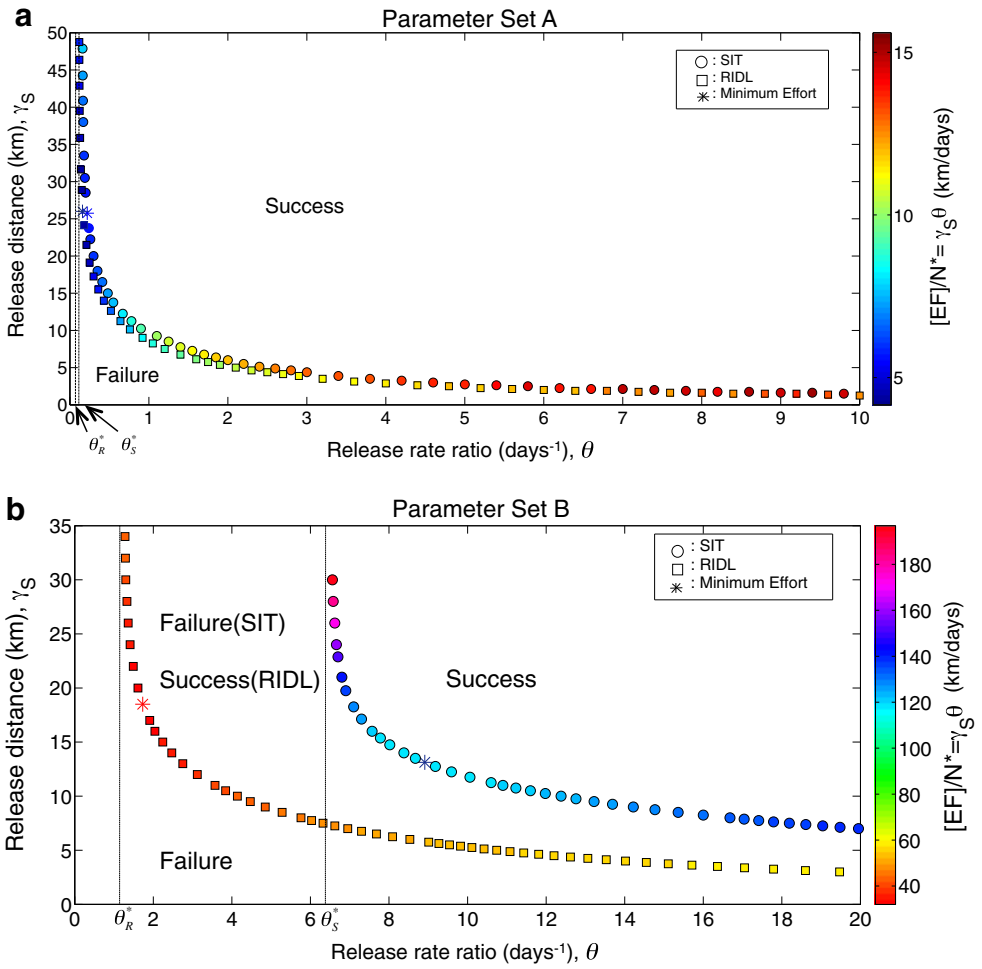

Fig. 3 Threshold curves for successful barrier zone control strategies given variations in the size of the release region, γ_s, and the release rate ratio, θ. The *colours* denote levels of normalised release effort, $[EF]/N^*$, on these curves with *asterisks* indicating the minimum release effort required for successful barrier zone strategies. The parameters are as given in Table 1 with $\Omega = 250$ km and $D_N = D_S = 1$ km^2/day

to the control strategy, given a fixed death rate μ which, in turn, is a convenient, if crude, surrogate for the economic costs associated with the control intervention.

In Fig. 3, we plot the normalised release effort ($[EF]/N^*$) for each control strategy and each parameter set, along the success/failure boundary curves, denoted by the colours in the plots. Here, the effort required in order to maintain a successful barrier zone varies depending on the release rate and the release region. Furthermore, one can observe that a distinct optimum, minimising the effort function, given the non-trivial constraint of a successful control, occurs at intermediate values of θ and γ_s, thus demonstrating a prediction that optimisation does not occur at parameter extremes. Hence, we conclude, from a management perspective, that one may minimise the effort required to maintain a successful barrier zone by releasing at a predetermined release rate and over a predetermined release region.

Comparing and contrasting SIT and RIDL release strategies for parameter set B, we observe that the SIT release rate under optimal conditions is over five times larger than that of RIDL and this, in particular, is responsible for the substantial difference in the effort function between these two control strategies. In addition, we note that the effort function associated with the RIDL strategy on the threshold curve for parameter set B is remarkably and encouragingly insensitive, in contrast to the moderate sensitivity exhibited by the SIT curve. For the scenario of parameter set A, the release efforts on the threshold curve are more sensitive for both control strategies, though once again, the use of transgenic insects is preferable in a direct comparison of the two strategies.

Given the difficulties in estimating the diffusion coefficient, it is useful to consider how the results presented thus far vary with this parameter. Using rescaling analysis, it is in fact straightforward to generate how the threshold curves and release effort minima are altered with the diffusion coefficient, as detailed in Appendix 2. It can be shown that for a diffusion coefficient D, the minimum release effort, $[EF]_{min}$, satisfies

$$[EF]_{min}(D) = \bar{\gamma}_s \bar{\theta} N^* \sqrt{\frac{D}{D_0}}, \tag{8}$$

where $\bar{\gamma}_s$ and $\bar{\theta}$ are, respectively, the minimising values of the release region and release rate ratio for a given diffusion

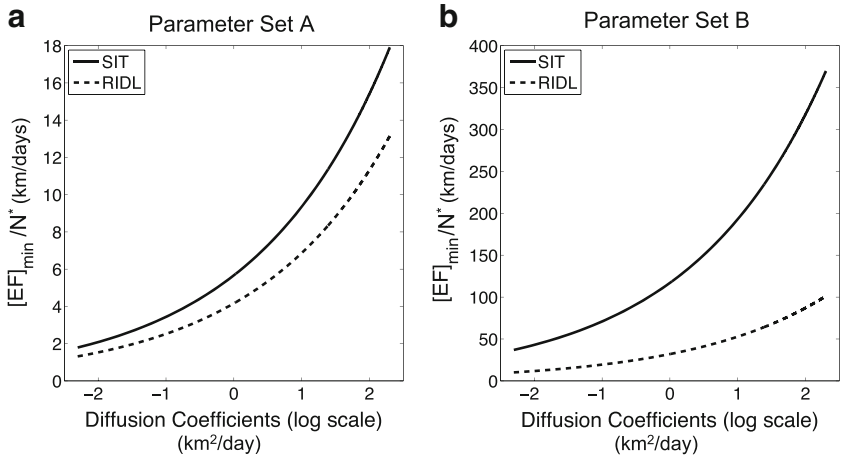

Fig. 4 Dependence of the minimal release effort on the mosquito diffusion rate. The variation in the normalised minimum release effort required to achieve barrier zone control is plotted as the mosquito diffusion rate is varied for SIT/RIDL strategies and parameter sets A and B. The parameters are as given in Table 1 with the diffusion rates varying from 0.1 to 8 km^2/day. See Appendix 2 for further details

coefficient of D_0. Therefore, the release effort and its minimum scale with the square root of the diffusion coefficient, as depicted explicitly in Fig. 4 where $D_0 = 1\text{km}^2/\text{day}$. It is worth noting the divergence in minimal release effort for each of the strategies as the diffusion coefficient increases, demonstrating that RIDL's superiority is especially pronounced for high levels of mosquito diffusion. As expected, this is exaggerated for SIT control strategies when larval competition is high, as in parameter set B. In addition, note the significant increases in release effort as the diffusion coefficient increases, indicating that the number of control insects will also vary substantially with insect motility.

From Eq. 8 and the observation that the minimising value of the release rate ratio is independent of the diffusion rate in the rescaling analysis (Appendix 2), we see that, in fact, the minimising value of the release region is proportional to the square root of D. Thus, the minimising point, $(\bar{\gamma}_s, \bar{\theta})$, in Fig. 3 will move up as the diffusion rate is increased. This implies that we always have the same optimal release rate ratio for any release effort value when diffusion rates are varied, and the minimal barrier zone is more sensitive to the release region size than the release rate ratio. Hence, when the diffusion rate is unknown or changeable, it is more efficient to release the sterile/transgenic mosquitoes over a wider area rather than at higher density.

Mosquito wave speed through a failed barrier zone

We now consider the dynamics within the barrier zone should the release strategy fail, that is, when the release rate ratio, θ, is sufficiently small. In particular, the wave speed of the invasive front within the barrier is plotted in Fig. 5a, b for both SIT and RIDL control strategies given both parameter sets A and B, with the spatiotemporal details of barrier penetration for specific examples presented in Fig. 5c, d. Firstly, note the extreme and desirable sensitivity of the wave speed as the size of the release rate ratio is increased. Thus, even when the control strategy is woefully incapable of stopping the mosquito invasion, it is nonetheless predicted to substantially slow down the wave speed within the barrier zone. Furthermore, in this specific scenario, there is no substantial difference in the effects of SIT or RIDL control strategies for sufficiently weak release rates.

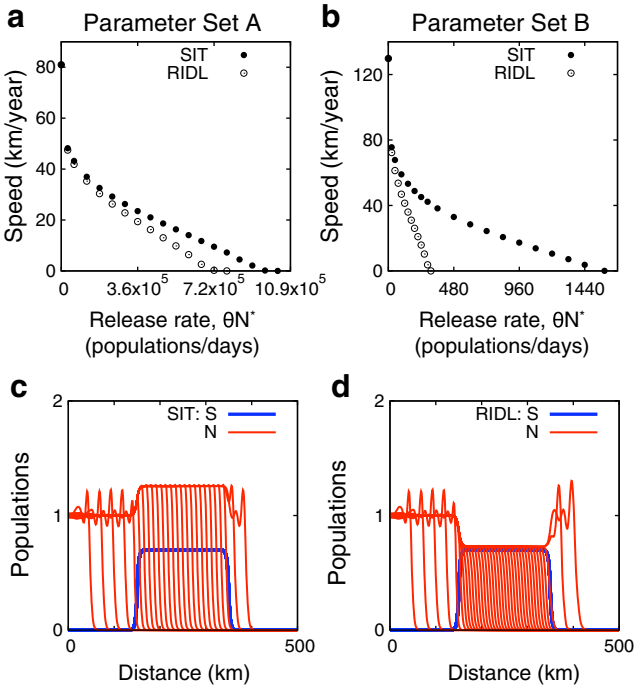

Fig. 5 Wave speeds in the barrier zone. **a** and **b** show the wave speed of the wild-type mosquitoes in the failed barrier zone for varying release rates, $\kappa = \theta N^*$. Diffusion coefficients are 1 km^2/day and the release region is $\gamma_s = 300$ km. All other parameters are as given in Table 1. In (**c**) SIT and (**d**) RIDL, snapshots of the travelling wave through the barrier zone are plotted for parameter set **B**, with $\theta = 0.7$. All other parameters are as in **B**. The speed of the wild-type females in the barrier wall is approximately 50 km/year in (**c**) SIT and 31 km/year in (**d**) RIDL. The speed in the control-free case is approximately 130 km/year in both (**c**) and (**d**)

In Fig. 5c, d, the spatiotemporal dynamics of barrier penetration are depicted for both SIT and RIDL control strategies given parameter set B and a relatively small release ratio of $\theta = 0.7$, which is insufficient for mosquito control even in the temporal model (see Fig. 1b, c). Note that, during penetration of an SIT barrier zone, there is an increase in wild-type female mosquito density throughout the release region behind the invading wave, as expected given the detrimental influence of SIT on larval competition within the modelling framework. Nonetheless, this local increase in pest population does not prevent the SIT substantially reducing the invasion speed. Intuitively, one might expect that an increased population size may lead to a faster wave speed, but since the reproductive potential of the wild-type mosquitoes is reduced at the front of the travelling wave, the wave speed slows. In contrast, RIDL control strategies not only slow the wave speed of the wild-type mosquitoes but may also reduce their abundance.

Discussion

The prevention of insect pest invasion using barrier zones of biological controls has already proven to be successful, though the indefinite release of sterile or transgenic insects is a heavy economic burden in practice (Vreysen et al. 2007), reducing its applicability and the cost-to-benefit ratio. The costs involved are substantial, for example classical sterile insect technologies require irradiation facilities and, in addition, obtaining and interpreting reliable field data to implement and maintain control strategies is notoriously expensive. This combination motivates and encourages detailed modelling studies to inform understanding of when barrier zone control strategies may be successful, in particular which aspects of both the pest and control are likely to be important and how may control insect numbers be reduced without sabotaging barrier function. Consequently, we have constructed a model for biological barrier control focusing on preventing or impeding the invasion of *A. aegypti* mosquitoes, contrasting SIT and late-acting RIDL control strategies.

A fundamental observation from our results is the inability to assess potential barrier success or failure from purely temporal models. Control insect release rates that are sufficient for population suppression in the temporal model need not prevent a biological invasion, which is capable of proceeding via very small densities of pest insects within the release region, especially for barrier control strategies of relatively small spatial extent. Such observations highlight the importance of the barrier zone release region in designing control strategies and further illustrate that any field studies monitoring the effectiveness of barrier control need to be especially fine-tuned. More generally, these results emphasise that predictions from spatial models concerning the minimisation of pest populations need not translate into strategies for invasion impediment or prevention and, consequently, that the management of barrier regions should not a priori be based upon observations of other aspects of biological control.

An immediate difficulty in modelling studies is determining the levels of insect dispersal, with very limited empirical data and, potentially, a very wide range of estimates (Reiter et al. 1995; Harrington et al. 2005). However, a simple rescaling analysis demonstrates that the influence of motility can be readily accounted for. Thus, the speed of invading wild-type insects and the minimal total number of insects that need to be released whilst ensuring barrier success, which can be considered as an approximate surrogate for the associated economic burden, are proportional to the insect diffusion rate, at least given the assumption that both the control and wild-type insects have the same motility. Consequently, even though insect dispersal rates are very difficult to estimate, the qualitative features emerging from this model are robust to variations in dispersal rates. Our analysis also predicts that for increasing dispersal rates, the optimal release strategy should be changed by increasing the release area and not the local release rate. However, adopting this strategy will incur additional distribution costs, and therefore a detailed economic analysis is required to accommodate this trade-off. These observations, in combination with detailed predictions for a specific value of the diffusion coefficient, also indicate that extensive insect mobility leads to a substantial difference between the effectiveness of RIDL control strategies compared to SIT strategies, especially if larval competition is particularly intensive, as with the scenario of parameter set B. Consequently, substantial gains in barrier effectiveness can be achieved by adopting transgenic RIDL technologies over the conventional SIT control strategy.

Observations of the improved outcomes associated with RIDL strategies are inherited from the temporal model dynamics. In particular, once the suppression of larval competition by SIT interventions induces dynamically significant effects, as in the scenario of parameter set B, RIDL strategies are substantially more effective in almost all aspects of control. Consequently, the typical conclusions that RIDL interventions are superior as a result of previous modelling (Phuc et al. 2007; White et al. 2010) do transfer in the context of barrier control strategies. Similarly, local increases in pest population are often associated with SIT barrier interventions, as observed in other contexts (Yakob et al. 2008; Yakob and Bonsall 2009). These conclusions hinge on the fact that sterile insect technologies reduce larval populations, enhancing the survival of insects resulting from wild-type matings and thus offsetting the reductions in proliferation. The one exception to this trend is

that substantially ineffective barriers nonetheless slow down an invading wave to almost the same extent, regardless of whether the intervention is via sterile insect or RIDL technologies, even though there is a local population increase associated with the release of sterile insects. As such, if a temporary impediment is all that is required, for example due to seasonality, then the additional advantages of RIDL need not be apparent.

We have also observed that barrier failure will occur at the extreme of too small a control insect release rate, but not at too small a release region with sufficiently high release rate, as migratory effects have the potential to compensate for the latter. Regardless, optimal strategies, as measured by the release effort, occur far from the extremes of control strategy parameters, but instead at intermediate values of release region size and insect release rate per unit length. This is perhaps not unexpected: for an extremely high release rate coupled with a small spatial extent, wild-type insects must be eradicated before the invasive wave can cross the release region. This demands not only population control but also sufficiently rapid population control of the wild-type mosquitoes, which is all the more demanding in terms of insect release strategies. In contrast, for an extensive barrier (large release region) with a low control insect release rate, the control perturbatively reduces the effective wild-type population over a large region. In the absence of additional competition, or some other feature sensitising the wild-type dynamics, this simply reduces the equilibrium wild-type density within the barrier zone, which is insufficient to prevent invasion.

As expected (Phuc et al. 2007; Yakob et al. 2008; White et al. 2010), RIDL control strategies are superior in terms of requiring smaller release efforts. A further predicted advantage of RIDL strategies, especially for parameter set B, is a reduced sensitivity for the balance between barrier zone release region and release rate at optimal release efforts. Hence, RIDL strategies do not need as extensive a fine-tuning to approach the balance between barrier size and insect release rate required to minimise the total number of insects released, and thus reduce the associated economic burdens. In addition, we see that the difference in efficiency between SIT and RIDL strategies emerges due to the prediction that the former requires substantially higher release rates rather than substantially larger release regions. However, such observations are not intuitively explained by the features of larval competition, though this is the only difference between the two control strategies, as modelled here.

In this manuscript, we have made the simplifying assumption that releases of sterile and transgenic males will be constant, whereas, in practise, releases will be periodic or punctuated. Studies by White et al. (2010) and Kean et al. (2011), where purely temporal models have been analysed, the authors have shown that smaller, more frequent releases may be more effective than larger, less frequent releases because the former minimises the amount of time during which the overflooding ratio may become very low. There is no reason to suggest that the same will not be true for the spatial model considered here. However, this investigation is beyond the scope of this manuscript and we will report on the results elsewhere.

In summary, we have explored barrier interventions for pest insects, motivated by the need for environmentally friendly controls of emerging and resurging disease spread, as illustrated by the fact that insect disease vectors for malaria and dengue fever have recently been found in Europe (Enserink 2010). Our exemplar study has considered the mosquito *A. aegypti* and explored how traditional sterile insect controls as well as more recent transgenic technologies, such as RIDL, can be used to effect biological barriers. Whilst both interventions can be successful, RIDL is robustly predicted to be highly preferable, especially when extensive larval competition is prevalent.

Our study concerning improvements in control strategies only considers surrogates for the total control insect release and thus does not consider detailed costing information, such as the operation of a radiation source or distributed insect release, which are also important in decision-making (Alphey et al. 2011). Nonetheless, our results illustrate the general characteristics of optimal strategies, for instance that there is a balance between release rates and release areas, that the influence of dispersion rates can be accounted for and that effective and optimal barrier design cannot be inferred from modelling explorations considering pest insect minimisation or eradication. Furthermore, we illustrate that numerous observations and predictions emerge for barrier dynamics and management that would be difficult to deduce from intuition alone.

In practical terms, estimating the optimal release distance and release rate ratio for the minimal release effort may not always be straightforward. This issue is compounded by our limited ability to estimate diffusion coefficients: our study suggests that for mosquito diffusion rates on the order of hundreds of square metres per day, the difference in the minimal release effort required for SIT and RIDL is significantly smaller for parameter set A as compared to parameter set B (Fig. 4). However, a successful barrier zone can be secured with the use of a small release distance as long as a sufficiently large release rate ratio is chosen, regardless of the choice of parameter set and control method (Fig. 3). These results imply that a high release rate ratio in combination with a small release distance is a more sensible strategy as it reduces the sensitivity of the control to parameter choice and control method when the mosquito diffusion rate is small enough.

As a word of caution, we note that the results presented in this manuscript depend heavily on the functional form of the density-dependent function and the model parameters. As Legros et al. (2009) discuss, the functional forms and parameters chosen may be interpreted differently, depending on the fitting method. Following Phuc et al. (2007), Yakob et al. (2008) and White et al. (2010), we have chosen two parameter sets (A and B) which reflect the best and worst cases so that we can make predictions over the full range of scenarios, with the aim of enlightening pest managers to the potential range of outcomes, based upon the current data available. It should be noted that there are large differences in the wild-type mosquito population levels over the estimated values of r and β (Dye 1984). This has an effect on the SIT and RIDL release rates required for successful population control. In particular, it can be shown (see Appendix 4) that lower values of density dependence, β, and birth rate, r, lead to lower required release rates, as one might expect. Furthermore, the transition through parameter space gives rise to no counterintuitive behaviour. Therefore, given the uncertainty in dispersal rates, birth rates and density-dependent coefficients of *A. aegypti* mosquitoes, our predicted release values for the barrier zone should be viewed with extreme caution. In particular, in the absence of detailed information, barrier overengineering with worst case life history and motility parameters should govern barrier management decision-making, due to the relative sensitivity of the modelling to life history parameters in particular. Nonetheless, this also suggests that less extreme barriers may be successful but such implementations will require careful parameter estimation studies and/or monitoring, especially given the modelling prediction that low pest densities can nonetheless induce barrier penetration.

Throughout this manuscript, we have used fecundity and density-dependent parameter values based upon Dye (1984), concentrating on the extreme best and worst case scenarios, following previous approaches (Phuc et al. 2007; Yakob et al. 2008; White et al. 2010). It can be shown that intermediate parameter values lead to qualitatively similar results (see Appendix 4). However, there are significant quantitative differences which have implications for the release rates required for population control. These parameters are derived from field data to which a simple regression is used to obtain the values. Legros et al. (2009) questioned this method and used a two-stage fitting method. They concluded that for their two-stage method: (a) when density-independent processes are taken into consideration, they account for a large part of the mortality of immature stages and density dependence is much weaker than the Dye approach; (b) the functional responses of the two approaches are significantly different for the range of densities in the study; and (c) whilst both methods give reasonable accounts of the characteristics of density dependence, they deviate where low densities are concerned, primarily due to the lack of data. Hence, it is critical that full life-table analyses are conducted in order to ensure that suitable estimates of these and other life history parameters are calculated (e.g. development periods, dispersal distances, differential density-dependent coefficients throughout the larval stages). The types of potential studies required are discussed at length in Legros et al. (2009), which we also encourage. In particular, it should be noted that from a practical management perspective, the life history parameters are likely to be highly dependent on the local environmental biotic and abiotic factors. Thus, local parameter estimation should be advocated to avoid under- or overrelease rates and hence provide effective pest management.

Finally, we remark that seasonality and fitness costs may influence the system dynamics (White et al. 2010). For example, it has recently been shown that the dispersal ability of two lines of RIDL *A. aegypti* mosquitoes may be reduced compared to their wild-type counterparts (Bargielowski et al. 2012). This is likely to have an impact of the effectiveness of barrier zone techniques for population control. However, the difference in diffusion rates of the transgenic and wild-type mosquitoes is likely to add greater model complexity (Billingham and King 2001). Thus, combining the above modelling framework with a temporal variation in the release rate and detrimental control insect fitness, so as again to explore how one can reduce the number of control insects that needs to be released, and hence the associated economic burden, constitutes a topic of current exploration.

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Appendix 1: Numerical methods

The delayed PDE equation (4a) was solved by constructing a finite difference method. The kinetics are considered explicitly with a fully implicit treatment of diffusive transport (Morton and Mayers 1994). This numerical algorithm has been validated against the simulations of Seirin-Lee et al. (2010), and it has also been confirmed that refinements in the time steps and grid size do not influence the results presented. In simulations, we used 10^{-3} or 10^{-4} for the time step and 10^{-3} for the spatial step.

Appendix 2: The influence of mosquito diffusion on optimal release ratio, optimal release distance, release effort minima and wave speeds

The region Ω is chosen sufficiently large to ensure that the boundary conditions do not influence the prospects for wild-type invasion through the barrier zone. Thus, there are two parameters in the model which are changed by a rescaling of length: the diffusion coefficient and the barrier zone size. For example, we can obtain a rescaled diffusion rate, $D_{\text{esti}} = D_0/L_0^2$, for some given diffusion rate, D_0, and length scale, L_0. For a different diffusion rate, $D = kD_0$, with k constant, we can rescale the spatial length from L_0 to $L = \sqrt{k}L_0$ which leads to the same rescaled diffusion rate, D_{esti}, such that $D_{\text{esti}} = D_0/L_0^2 = D/L^2$.

Now, we redefine $[\text{EF}]_{\min}$ directly for an arbitrary diffusion rate. From $L = \sqrt{k}L_0$ and $D = kD_0$, we have

$$L(\equiv L(D)) = \sqrt{k}L_0 = \sqrt{\frac{D}{D_0}}L_0. \tag{9}$$

This shows that an optimal release region size can be described by a minimising value of the release region size, $\bar{\gamma}$, at the minimum release effort for the diffusion rate, D_0, such that

$$\gamma_{\text{s}}^{\text{opt}} = \sqrt{\frac{D}{D_0}}\bar{\gamma}. \tag{10}$$

Then, the release effort function for the arbitrary diffusion coefficient D is given by

$$[\text{EF}]_{\min}(D) = \gamma_{\text{s}}^{\text{opt}}\theta^{\text{opt}}N^* = \sqrt{\frac{D}{D_0}}\bar{\gamma}\bar{\theta}N^*, \tag{11}$$

where $\bar{\theta}$ is the minimising value of the release rate ratio at the minimum release effort, $[\text{EF}]_{\min}(D_0)$, and is not affected by the spatial length scaling so that $\theta^{\text{opt}} = \bar{\theta}$. From the numerical results of Fig. 3, we know the values of $D_0 = 1\ \text{km}^2/\text{day}$ and the detailed values of $(\bar{\gamma}, \bar{\theta})$, so that we obtain Fig. 4 directly from noting

$$[\text{EF}]_{\min}(D) = \bar{\gamma}_{\text{s}}\bar{\theta}N^*\sqrt{D}, \tag{12}$$

with D measured in units of square kilometre per day.

Invasion speed and the diffusion rate

Analogous observations apply for the invasive wave speed of pest insects in the absence of control. In particular, noting independence with respect to the overall domain size, the only length scale in the current context within the model occurs in the diffusion coefficient. This is of degree two with respect to powers of the length dimension whereas the speed of invasion is of degree one; thus, the invasion speed must scale linearly with the square root of the diffusion coefficient. Any other relation will conflict with the need for dimensional consistency.

Appendix 3: The release effort function

The sterile/transgenic males are released locally in space and continuously in time with constant rate, κ; this is described by the release function

$$\kappa(x) = \theta N^* \chi(x),$$
$$\chi(x) = \begin{cases} 1 & x \in A = [\bar{x} - \gamma_{\text{s}}/2, \bar{x} + \gamma_{\text{s}}/2] \\ 0 & x \in \Omega \backslash A \end{cases}, \tag{13}$$

where $\gamma_{\text{s}} \ll |\Omega|$ is the release region size and $\bar{x}$ is the release region centre.

Here, we show that the *release effort function*, defined by $[\text{EF}] = \gamma_{\text{s}}\theta N^*$, is also proportional to the number of control males present in the environment after the initial transient dynamics, in particular,

$$[\text{EF}] = \mu \lim_{t\to\infty} \int_\Omega S\,\mathrm{d}x. \tag{14}$$

Integrating Eq. 4b,

$$\frac{\partial S(x,t)}{\partial t} = D_S \frac{\partial^2 S(x,t)}{\partial x^2} + \kappa(x) - \mu S(x,t), x \in \Omega, t \in (0, +\infty], \tag{15}$$

over the spatial variable, with use of Eq. 13, we have

$$\int_\Omega \frac{\partial S}{\partial t}\,\mathrm{d}x = \int_\Omega D_S \frac{\partial^2 S}{\partial x^2}\,\mathrm{d}x + \int_\Omega [\kappa(x) - \mu S]\,\mathrm{d}x, \tag{16}$$

and thus

$$\frac{\mathrm{d}}{\mathrm{d}t}\int_\Omega S\,\mathrm{d}x = \theta N^*\gamma_{\text{s}} - \mu \int_\Omega S\,\mathrm{d}x. \tag{17}$$

This is a linear ordinary differential equation in $\int_\Omega S\,\mathrm{d}x$, which can be readily be solved and, for general initial conditions, this yields

$$\lim_{t\to\infty} \int_\Omega S\,\mathrm{d}x = \frac{\theta N^*\gamma_{\text{s}}}{\mu} = \frac{1}{\mu}[\text{EF}].$$

Hence, we obtain the required result, Eq. 14.

Appendix 4: Equilibrium relativity on varying r and β

In Fig. 6, we vary the birth rate and density-dependent coefficient for four different release rates (rows) and for

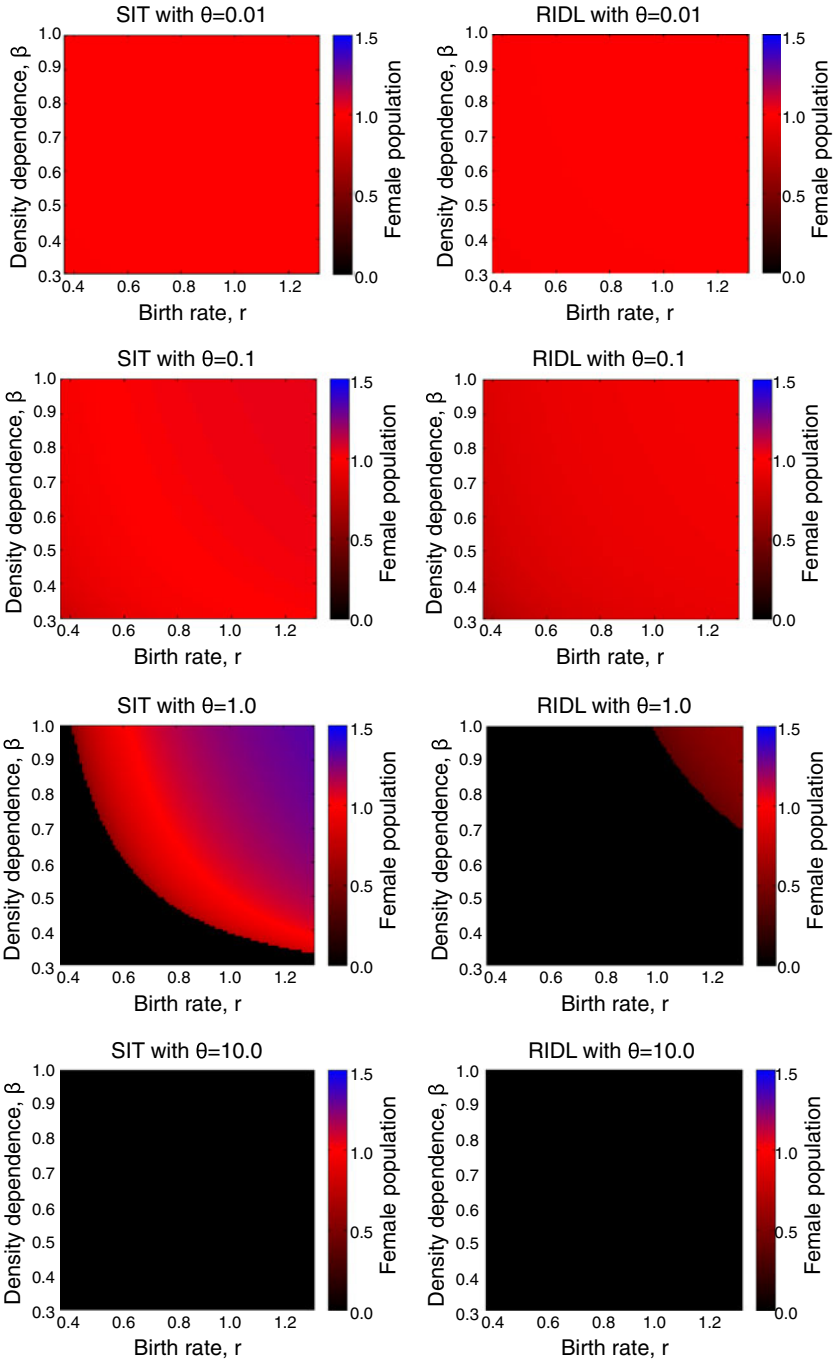

Fig. 6 The equilibrium relative to the no-strategy equilibrium in the non-spatial model when varying the birth rate, r, and density-dependent coefficient, β, for each of four different release rates (*rows*) and two control strategies, SIT vs. RIDL (*columns*)

the two control strategies (columns) and calculate the equilibrium relative to the no-strategy equilibrium in the non-spatial model. As one might expect, lower values of density dependence and birth rate lead to a lower required release rate for population control. Also, as one can see, the transition across parameter space is relatively smooth, with no counterintuitive behaviour for intermediate values. Similar qualitative results may be obtained for the spatial model, but we omit these for the sake of brevity.

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