A. Nucleation theory of the gene drive population waves

Here we identify different parameter regimes of various types of gene drive waves by establishing an analogy between zero temperature nucleation theory and the reaction-diffusion equation of the prescribed mutagenic chain reaction,

\[ \frac{\partial q}{\partial t} = D \frac{\partial^2 q}{\partial x^2} + \frac{1}{\tau_g} \frac{sq(1-q)(q-q^*)}{1-sq(2-q)}, \quad (S1) \]

using the methods reviewed in [1]. First, we introduce a potential energy function \( U(q) \)

\[ U(q) = -\frac{1}{\tau_g} \int_0^q \frac{sq'(1-q')(q'-q^*)}{1-sq'(2-q')} dq', \quad q^* = \frac{2s-1}{s}, \quad (S2) \]

and rewrite Eq. S1 as

\[ \frac{\partial q}{\partial t} = D \frac{\partial^2 q}{\partial x^2} - \frac{dU(q)}{dq}. \quad (S3) \]

It is useful to recast the reaction-diffusion dynamics in terms of a functional derivative

\[ \frac{\partial q(x,t)}{\partial t} = -\frac{\delta \mathcal{F}[q(y,t)]}{\delta q(x,t)}, \quad (S4) \]

where the functional \( \mathcal{F}[q(y,t)] \) is given by

\[ \mathcal{F}[q(y,t)] = \int_{-\infty}^{\infty} \left\{ \frac{1}{2} D \left( \frac{\partial q(y,t)}{\partial y} \right)^2 + U[q(y,t)] \right\} dy, \quad (S5) \]
and we have

\[
- \frac{\delta F[q(y, t)]}{\delta q(x, t)} = - \lim_{\epsilon \to 0} \frac{F[q(y, t) + \epsilon \delta(y - x)] - F[q(y, t)]}{\epsilon} \\
= - \int_{-\infty}^{\infty} \left\{ D \frac{\partial q(y, t)}{\partial y} \frac{\partial \delta(y - x)}{\partial y} + \frac{dU[q(y, t)]}{dq} \delta(y - x) \right\} dy = D \frac{\partial^2 q(x, t)}{\partial x^2} - \frac{dU[q(x, t)]}{dq}.
\]  

(S6)

Since \( F(t) \) always decreases in time,

\[
\frac{dF(t)}{dt} = \int_{-\infty}^{\infty} \frac{\partial q(x, t)}{\partial t} \frac{\delta F[q(y, t)]}{\delta q(x, t)} dx = - \int_{-\infty}^{\infty} \left( \frac{\partial q(x, t)}{\partial t} \right)^2 dx \leq 0,
\]  

(S7)

\( F[q(y, t)] \) plays the role of the free energy in a thermodynamic system.

The potential energy function \( U(q) \) with various selective disadvantages \( s \) is plotted in Fig. 3. \( U(1) \) becomes the absolute minimum when \( 0.5 < s \) and population waves behave as pushed waves, because both \( U(0) \) and \( U(1) \) are locally stable [1–3]. The pushed gene drive wave stalls out when the two stable points have the same potential energy (blue curve in Fig. 3). The maximum value of the selective disadvantage \( s_{\text{max}} \) supporting the pushed wave of the gene drive allele can be derived by equating \( U(0) = U(1) \), which leads to

\[
0 = \int_{0}^{1} sq(1 - q)(q - q^*) dq = \frac{-2 + s_{\text{max}} + 2\sqrt{-1 + \frac{1}{s_{\text{max}}}} \arcsin(\sqrt{s_{\text{max}}})}{2s_{\text{max}}}.
\]  

(S8)

The excitable gene drive wave of primary interest to us thus arises when the selective disadvantage satisfies

\[
0.5 < s < 0.697.
\]  

(S9)

**B. The range of the pushed wave regime with an arbitrary conversion rate**

In the main text, we assumed perfect conversion efficiency \((c = 1)\) of the mutagenic chain reaction. However, in reality, some fraction of the reactions can be unsuccessful and the conversion rate \( c \) will be \( 0 < c < 1 \). As a result there will be heterozygous individuals with fitness \( 1 - hs \), where \( h \) controls dominance of the gene drive allele. When \( h = 1 \), the gene drive allele is dominant and the fitness of the heterozygous genotype is \( 1 - s \). The choices \( h = 0, 0.5 \) correspond to the recessive and additive cases respectively. As derived by Unckless et al. [4], the reaction term in Eq. 5 is now given by

\[
q(t + \tau_g) - q(t) = \bar{f}(q) = \frac{q^2(1 - s) + q(1 - q)[(1 - c)(1 - hs) + 2c(1 - s)]}{q^2(1 - s) + 2q(1 - q)(1 - c)(1 - hs) + 2q(1 - q)c(1 - s) + (1 - q)^2} - q.
\]  

(S10)
FIG. S1. $s_{\text{min}}$ and $s_{\text{max}}$ as a function of the conversion rate $c$ when the fitness of heterozygotes individuals is (A) recessive ($h=0$), (B) additive ($h=0.5$) and (C) dominant ($h=1.0$) of gene drives, where the fitness of heterozygotes is $1-hs$. The socially responsible pushed wave regime ($s_{\text{min}} < s < s_{\text{max}}$) is always widest when $c=1$, i.e., for 100% conversion efficiency. Note that the results become independent of $h$ when $c=1$. The gene drive wave reverses direction and dies out in the white regions of this diagram.

There are again three fixed points $q = 0, 1, q^*$ where the third fixed point is

$$q^* = \frac{c + cs(h-2) - hs}{s(1-2c-2h+2ch)},$$  \hspace{1cm} \text{(S11)}

Following [4], we find that $q^*$ first becomes positive for $s > s_{\text{min}}$, where

$$s_{\text{min}} = \frac{c}{2c-(c-1)h}.$$  \hspace{1cm} \text{(S12)}

For $0 \leq s \leq s_{\text{min}}$, $q^* < 0$ and the spatial dynamics is again controlled by pulled waves. We can also calculate $s_{\text{max}}$ by recalculating the potential function analogy discussed in SI, Sec A and in the main text,

$$\bar{U}(q) = -\frac{1}{\tau_g} \int_0^q \bar{f}(q')dq',$$  \hspace{1cm} \text{(S13)}

and numerically solving for $\bar{U}(q=0,c,h,s_{\text{max}}) = \bar{U}(q=1,c,h,s_{\text{max}})$ to obtain $s_{\text{max}}(c)$ given $h$, with the results shown in Fig. S1. The gene drive spreads spatially as a pushed excitable wave for $s_{\text{min}} < s < s_{\text{max}}$. Note that the relevant range of $s$ when $c < 1$ shrinks compared to $c = 1$.

C. Spatial evolutionary games in one dimension

In this SI section, we show that genetic waves mathematically quite similar to the pushed gene drive waves studied here arise in spatial evolutionary games of two interacting asexual species that are colored red (“R”) and green (“G”) using the analogy with nucleation theory
FIG. S2. A schematic phase diagram of the spatial evolutionary games in one dimension ignoring genetic drift. The parameters $\alpha$ and $\beta$ describe interactions between red and green genetic variants, with growth rates written as $w_R(x,t) = g + \alpha(1 - f(x,t))$ and $w_G(x,t) = g + \beta f(x,t)$ respectively. (The parameter $g > 0$ is a background growth rate.) Inserted graphs show schematically the potential energy function $U(f)$, where each of the green and red dot corresponds to $f = 0$ and $f = 1$ respectively ($0 \leq f \leq 1$). By searching for barriers in $U(f)$ as a function of $\alpha$ and $\beta$, we identify the bistable regimes that require a critical nucleus and pushed excitable waves to reach a stable dynamical state and the pulled Fisher wave regimes which do not require the nucleation process. The two regimes are separated by two solid black lines $\alpha = 0, \beta < 0$, and $\alpha < 0, \beta = 0$, which correspond limits of metastability. The solid line along $\alpha = \beta < 0$ between the two bistable states is analogous to a first-order phase transition line (equal depth minima in $U(q)$), along which the excitable genetic wave separating red and green stalls out.

introduced in the previous SI section. We start from the continuum description of the one dimensional stepping stone model (following [5, 6]),

$$\frac{\partial f(x,t)}{\partial t} = D \frac{\partial^2 f(x,t)}{\partial x^2} + s[f]f(1-f) + \sqrt{D_g f(1-f) \Gamma(x,t)},$$  \hspace{1cm} \text{(S14)}$$

where $f(x,t)$ is the frequency of red species and $D$ is the spatial diffusion constant representing migration. The last term, where $\Gamma(x,t)$ is an Ito correlated Gaussian white noise source and $D_g$, proportional to an inverse effective population size, represents genetic drift. We henceforth neglect genetic drift and set this term to zero. The function $s[f]$ represents
the difference in relative reproduction rates between the two species, and is given by [5]

\[ s[f] = w_{\text{eff}} = \frac{w_R - w_G}{\frac{1}{2}(w_R + w_G)}, \]  

(S15)

where \( w_R \) and \( w_G \) are fitnesses of alleles \( R \) and \( G \). If \( g \) is a background reproduction rate, we have

\[
\begin{align*}
    w_R(x,t) &= g + \alpha(1 - f(x,t)), \\
    w_G(x,t) &= g + \beta f(x,t),
\end{align*}
\]  

(S16)

where the interactions between the two competing variants are characterized by constants \( \alpha \) and \( \beta \). With the definitions above, we have

\[
    s[f] = -\frac{(\alpha + \beta)(f - \frac{\alpha}{\alpha + \beta})}{g + \frac{1}{2}\alpha(1 - f) + \frac{1}{2}\beta f},
\]  

(S17)

which leads to a reaction term similar to that in Eq. 5 and introduces an additional fixed point into the dynamics of Eq. S14 at \( f^* = \frac{\alpha}{\alpha + \beta} \) in addition to \( f = 0, 1 \). A diagram summarizing the dynamics of this model is shown in Fig. 2. This “phase diagram” was worked out including genetic drift in Eq. S14 which affects the shape and location of the phase transition lines in the first quadrant of Fig. 1. [6]. If the genetic drift term in Eq. S14 is neglected, the lines labelled “DP” in Fig. 2 would coincide with the positive \( \alpha \) and \( \beta \) axes and would merge at the origin. Upon setting \( D_g = 0 \) in Eq. S14, we employ the argument presented above and define a potential energy function,

\[
    U_b(f) = -\int_0^f s[f']f'(1 - f')df'.
\]  

(S18)

The schematic picture of \( U_b(f) \) in different parameter regimes is drawn in Fig. S2. The mutualistic regime (\( \alpha > 0, \beta > 0 \)) has already been studied in detail, including effects of genetic drift [6]. By studying shapes of the potential energy function \( U[f] \) we identify two important parameter regimes. In the bistable regime (dark green), there is a finite energy barrier between the two locally stable states and a nucleation process is required to establish an excitable wave.

However, in the Fisher wave regimes (light green and light red), there is no energy barrier to reach the unique stable configuration and thus nucleation is not required. The two regimes are separated by the two black solid lines \( \alpha = 0, \beta < 0 \) or \( \alpha < 0, \beta = 0 \), which are limits of metastability. We also draw a solid black line between the two bistable states along \( \alpha = \beta < 0 \), where the pushed waves stall out. This line is analogous to a line of first-order transitions. When \( \alpha \neq \beta \), the integral in Eq. S18 for the effective thermodynamic potential is given by
\[ U[f] = \frac{1}{3(\alpha - \beta)^4} \left( (\alpha - \beta) f \left\{ \alpha^3 f (2f - 3) + \alpha^2 f (9\beta - 2\beta f + 6g) + \alpha \left( \beta^2 (12 - f (3 + 2f)) + 36\beta g + 24g^2 \right) + \beta \left( \beta^2 f (-3 + 2f) - 6\beta (-2 + f) g + 24g^2 \right) \right\} + 12(\alpha + 2g)(\beta + 2g) (\alpha \beta + (\alpha + \beta) g) \log \left[ 1 - \frac{\alpha - \beta}{\alpha + 2g f} \right] \right). \]  

(S19)

When \( \alpha = \beta \), we can simplify \( s[f] \)

\[ s[f] = -\frac{2\alpha (f - \frac{1}{2})}{g + \frac{1}{2} \alpha}, \]  

and the integral gives

\[ U[f] = \frac{2\alpha}{g + \frac{1}{2} \alpha} \int_0^f f' \left( 1 - f' \right) \left( f' - \frac{1}{2} \right) df' = -\frac{\alpha}{2g + \alpha} f^2 (f - 1)^2. \]  

(S21)

When \( \alpha = -\beta \), \( \alpha \ll g \) and \( 1 \ll \left| \frac{g}{\alpha} + \frac{1}{2} \right| \), we have

\[ s[f] = \frac{\alpha}{g + \frac{1}{2} \alpha (1 - 2f)} \]  

(S22)

and

\[ U[f] = -\int_0^f \frac{f'^2 - f'^{\prime}}{f' - \left( \frac{g}{\alpha} + \frac{1}{2} \right)} df' = -\frac{1}{2} f \left( f + \frac{2g}{\alpha} - 1 \right) - \left( \frac{g}{\alpha} + \frac{1}{2} \right) \left( \frac{g}{\alpha} - \frac{1}{2} \right) \log \left[ 1 - \frac{2\alpha f}{\alpha + 2g} \right] \]  

(S23)

The last term diverges at \( f = \frac{g}{\alpha} + \frac{1}{2} \), but we focus on the weak interaction limit \( 1 \ll \left| \frac{g}{\alpha} + \frac{1}{2} \right| \), where the biologically relevant regime \( 0 \leq f \leq 1 \) will not be affected. If we substitute \( \alpha = -\beta \) into Eq. S19, we recover Eq. S23, as expected.

**D. Calculation of the critical propogules in one dimension**

In this SI section, we describe details of the calculation of the critical propogules shown in Fig. 5. Reaction-diffusion equations in one dimension with a general reaction term \( R[q(x, t)] \) can be written as

\[ \tau_g \frac{\partial q(x, t)}{\partial t} = \tau_g D \frac{\partial^2 q(x, t)}{\partial x^2} + R[q(x, t)]. \]  

(S24)

The critical propogule profile \( q_c(x) \) can be defined as a stationary solution of Eq. S24, i.e.,

\[ 0 = \tau_g D \frac{\partial^2 q_c}{\partial x^2} + R[q_c]. \]  

(S25)
Upon multiplying both sides by $\frac{dq_c}{dx}$ and integrating we obtain,

$$\tau_g D \left( \frac{dq_c}{dx} \right)^2 = 2 \int_q^0 R[\tilde{q}] d\tilde{q}. \quad (S26)$$

If we assume a symmetric critical propagule about $x = 0$, so that $\frac{dq_c}{dx} = 0$ at $x = 0$, we can obtain $q_m \equiv q_c(0)$ from

$$\int_{q_m}^0 R[\tilde{q}] d\tilde{q} = 0. \quad (S27)$$

Since the slope $\frac{dx_c(q)}{dq}$ is given by

$$\frac{dx_c(q)}{dq} = \frac{\sqrt{\frac{\tau_g D}{2}}}{\sqrt{\int_q^0 R[\tilde{q}] d\tilde{q}}}, \quad (S28)$$

we obtain the critical propagule profile $x_c(q)$ by integrating both sides from $q_m$ to $q$. The calculations described above can be carried out analytically for the cubic reaction term Eq. 7 and critical propagules for $s = 0.66, 0.58, 0.51$ are plotted in Fig. 5 with dashed lines. For the full MCR equation, the corresponding numerical results are plotted with solid lines.

**E. Critical radius and allele concentration in two dimensions**

In practice, it is important to model the distribution of MCR alleles to be released locally to initiate its traveling genetic wave in a two-dimensional space. Upon assuming circular symmetry of the traveling wave solution, the reaction-diffusion equation governing the radial

![FIG. S3. In two dimensions the gene drive allele is introduced uniformly over a disk-shaped region with radius $r_0$ with uniform frequency $q_0$ inside as illustrated in the inset image. We numerically determined the critical frequency $q_0$ and radius $r_0$ just sufficient to initiate an excitable wave in two dimensions.](image-url)
frequency profile of the MCR allele $q(r, t)$ reads in radial coordinates,

$$
\tau_g \frac{\partial q}{\partial t} = \tau_g D \left( \frac{\partial^2 q}{\partial r^2} + \frac{1}{r} \frac{\partial q}{\partial r} \right) + \frac{sq(1 - q)(q - q^*)}{1 - sq(2 - q)}, \tag{S29}
$$

The only correction to the one dimensional case is the derivative term $\frac{1}{r} \frac{\partial q}{\partial r}$, which can be neglected relative to $\frac{\partial^2 q}{\partial r^2}$ in the limit of $r \to \infty$. However, we keep this term in the calculation of the critical nucleus as this term is not negligible where $r$ is comparable to or smaller than the width of the excitable wave being launched. In our numerical calculations, instead of a Gaussian initial condition, it is convenient to introduce the gene drive allele with a uniform frequency $q_0$ over a circular region with radius $r_0$. Indeed, in an actual release of a gene drive organism, it is plausible that the release would be implemented by creating a gene drive concentration $q_0$ in a circular region of radius $r_0$ with a sharp boundary. To model the radial frequency profiles, we used a circularly symmetric steep logistic function as an initial condition,

$$
q(r, t = 0) = \frac{q_0}{1 + e^{10(r - r_0)/\sqrt{\tau_g D}}}, \tag{S30}
$$

instead of a step function to insure numerical stability. Fig. S3 shows the parameter regimes where a pushed wave is excited for various selective disadvantages $s$. The pushed waves successfully launched for initial conditions whose parameters are above the curves $q_0(r_0)$, shown for a variety of selective disadvantages $s$ in the pushed wave regime.

**F. Line tension, energy difference and analogy with nucleation theory in two dimensions**

The scenario studied in the previous section (sharp boundary, adjustable initial drive concentration $q_0$ and inoculation radius $r_0$) seems appropriate for many engineered releases of gene drives, at least in situations with large effective population sizes $N_{\text{eff}}$, so that genetic drift can be neglected. (See the discussion of genetic drift in SI Sec. J.)

However, when genetic drift is important, stochastic contributions like the term $\sqrt{D_g f(1 - f)} \eta(x, t)$ in, e.g., Eq. S14, can act on spatial gradients at the interfaces of pushed and pulled waves [7, 8] in a manner somewhat reminiscent of thermal fluctuations near a first-order phase transition. Provided strong genetic drift is able to produce something analogous to local thermal equilibrium after a gene drive release, it is interesting to explore an analogy with classical nucleation theory. Nucleation leads to a pushed wave when $s_{\text{min}} < s < s_{\text{max}}$. One might then expect the two-dimensional analog of the total energy function discussed in SI Sec. A for an equilibrated circular droplet with $q_0 = 1$ and radius $r_0$ in two dimensions to
take the form

$$
\mathcal{F}[q(r)] = \int dr \left\{ \frac{1}{2} D (\nabla q(r))^2 + U[q(r)] \right\} \\
= 2\pi \int_0^\infty dr \frac{r D}{2} \left( \frac{dq}{dr} \right)^2 + 2\pi \int_0^\infty dr U[q(r)] \\
\approx 2\pi r_0 \int_0^\infty dr \frac{D}{2} \left( \frac{dq}{dr} \right)^2 + \pi r_0^2 (U(1) - U(0)) \\
\equiv 2\pi r_0 \gamma - \pi r_0^2 |\Delta U| 
$$

(S31)

where we have assumed a sharp interface between saturated gene drive and wild-type states.

Here, $\Delta U$, the “energy” difference between the gene drive and wild type, causes the droplet to expand, and the role of an energy barrier to nucleation is played by the line tension term $\gamma$ [9]. This is indeed the case. For simplicity, we illustrate the nucleation approach with the cubic reaction term given by Eq. 7 in the main text.

First, we assume the logistic form of the spatial profile derived in the 1d limit by Barton and Turelli [1]

$$
q(r) = \frac{1}{1 + e^{s/2\tau_g D(r-r_0)}},
$$

(S32)

and the line tension term is

$$
\gamma = \int_0^\infty dr \frac{D}{2} \left( \frac{dq}{dr} \right)^2 = \frac{\sqrt{s D}/2\tau_g (e^{3r_0 \sqrt{s/2\tau_g D}} + 3e^{2r_0 \sqrt{s/2\tau_g D}})}{12 (e^{r_0 \sqrt{s/2\tau_g D}} + 1)^3} \approx \frac{\sqrt{s D}/2\tau_g}{12},
$$

(S33)

in the limit of $1 \ll r_0 \sqrt{s/2\tau_g D}$. The energy difference is given by

$$
\Delta U = U(1) - U(0) = \frac{3s - 2}{12\tau_g},
$$

(S34)

and the critical radius of the nucleus $r_c$ which corresponds to the saddle point barrier of the free energy landscape is

$$
r_c = \frac{\sqrt{s\tau_g D/2}}{2 - 3s}
$$

(S35)

as plotted in Fig. S4. This result shows the divergence of $r_c$ in the limit of $s \to s_{\text{max}} (= 2/3)$ and the above approximation ($r_0 \sqrt{s/2\tau_g D} \gg 1$) becomes exact in this limit. The diverging $r_c(s)$ shown in Fig. S4 is qualitatively consistent with the behavior found for the simplified gene drive initial condition in two dimensions shown in Fig. S3 in the limit $q_0 \to 1$
G. Wave velocities of the excitable waves

The reaction-diffusion equation admits traveling wave solutions with a continuous family of velocities. It selects the slowest speed asymptotically in the large time limit [10]. The pink circular dots in Fig. S5 are numerically calculated asymptotic wave velocities for the MCR model in the pushed wave regime. We also plot the known wave velocity for the cubic approximation \( v(s) = (2 - 3s)\sqrt{D/2\tau_g}s \) [1–3] for comparison. Due to the larger reaction term \( f_{\text{MCR}}(q) > f_{\text{cubic}}(q) \) (see discussion in Fig. 5), the wave velocity for the MCR model is always faster than the cubic approximation given the same selective disadvantage \( s \). In both cases, a larger selective disadvantage \( s \) decreases the wave velocity, which eventually becomes zero at \( s_{\text{max}} = 0.697 \) for the MCR model and the slightly smaller value \( s_{\text{max}} = 2/3 \).
within the cubic approximation.

**H. Calculation of the speed of the excitable waves**

In this section, we review the numerical method for calculating the speed of the excitable waves, following [1]. First, we assume a traveling waveform of the solution

\[ q(x,t) = Q(x - vt) = Q(z), \quad z \equiv x - vt, \tag{S36} \]

with boundary conditions

\[ Q(z) \rightarrow 1 \quad (z \rightarrow -\infty), \quad Q(z) \rightarrow 0 \quad (z \rightarrow +\infty), \]
\[ \frac{dQ}{dz} \rightarrow 0 \quad (z \rightarrow \pm \infty). \tag{S37} \]

By substituting \( Q(z) \) into

\[ \tau_g \frac{\partial q}{\partial t} = \tau_g D \frac{\partial^2 q}{\partial x^2} + R[q], \tag{S38} \]

we obtain

\[ 0 = \tau_g D \frac{d^2 Q}{dz^2} + v \tau_g \frac{dQ}{dz} + R[Q]. \tag{S39} \]

If we define the gradient \( G \) as a function of \( Q \), \( G[Q] \equiv \frac{dQ}{dz} \) we arrive an ordinary differential equation

\[ 0 = \tau_g DG \frac{dG}{dQ} + v \tau_g G + R[Q], \tag{S40} \]

with boundary conditions

\[ G[0] = G[1] = 0. \tag{S41} \]

It is known that there exists a unique velocity of the excitable wave \( v \) that has solution \( G[Q] \) of the above differential equation with the boundary condition [11]. We used a shooting method to determine such \( v \) and plotted the results in Fig. S5.

**I. Critical barrier strength**

Fig. S6 shows how the excitable wave can be slowed down and finally stopped by increasing the strength of a selective disadvantage barrier \( s_b > s \). As a reference, we first show dynamics of the excitable wave without a barrier \((s_b = 0.625 \text{ matches the selective disadvantage } s = 0.625 \text{ outside})\) in Fig. S6A. When a small barrier is erected \((s_b = 0.688 < 0.697)\), the excitable wave significantly slows down within the barrier as expected from the results shown in Fig. S5. However, the wave recovers and propagates through the barrier as in Fig. S6B. When the barrier strength exceeds a critical value \((\text{in Fig. S6C we plot the case } s_b = 0.708)\) the excitable wave is stopped.
In Fig. S7, we plot the critical width $L$ and selective disadvantage within the one dimensional barrier region $s_b$ just sufficient to stop the excitable population wave of the gene drive species. The values are numerically obtained by placing the barrier in a region $25\sqrt{\tau g D} < x < (25 + L)\sqrt{\tau g D}$. For example, when the selective disadvantage outside the barrier region is set to be $s \approx 0.65$, the excitable gene drive wave can be stopped by increasing $s$ by $\sim 20\%$ within the barrier region of thickness $\sim \sqrt{\tau g D/s}$.

**FIG. S6.** Stopping power of a selective advantage barrier in one dimension. Numerical solutions of Eq. 5 are shown with time increment $\Delta t = 10.0\tau_g$. The early time response is shown in red with later times in blue. The selective disadvantage of the barrier is $s_b$ within the purple bar of width $L = 5$ occupying the spatial region $25\sqrt{\tau g D} < x < 30\sqrt{\tau g D}$ (shaded in blue) and $s = 0.625$ otherwise. (A) The excitable wave propagates with constant speed when the barrier vanishes for $s_b = 0.625$. (B) With $s_b = 0.688 > s = 0.625$, the wave significantly slows down at the barrier, but recovers and propagates onwards. (C) The excitable wave is stopped when $s_b = 0.708$. 
FIG. S7. Critical width $L$ and the selective disadvantage $s_b$ of a barrier that is just sufficient to stop a pushed gene drive wave in one dimension. The values are numerically obtained by placing the barrier in a region $25\sqrt{\tau_g D} < x < (25 + L)\sqrt{\tau_g D}$. Results are plotted for a variety of selective disadvantages $s$ outside the barrier region. Given $s$, the excitable population wave can be stopped by a barrier whose parameters $(s_b, L/(\sqrt{\tau_g D}))$ lie above the curves.

J. Fluctuations due to finite population size

In this section, we estimate effects of fluctuations due to a finite population size using mosquitoes as an example. First, we define the effective spatial population size $N_{\text{eff}}$ to be the number of mosquitoes with which an individual might conceivably mate during its generation time $\tau_g$ [12]. Given a diffusion constant $D$, the two dimensional area an individual can explore during its life time $\tau_g$ is $\pi(\sqrt{4D\tau_g})^2$ and the effective population size in two dimensions is

$$N_{\text{eff}} \equiv 4\pi D\tau_g n,$$  \hfill (S42)

where $n$ is the area density of organisms. Here, we estimate $N_{\text{eff}}$ using parameters appropriate to mosquitoes: $\tau_g \sim 10$[days] [13], $D \sim 0.1$[km$^2$/day] and $n \sim 1$[m$^{-2}$] = 10$^6$[km$^{-2}$] to get $N_{\text{eff}} \sim 10^5 - 10^6$. With such a large effective population size, we believe that the dynamics can be well described by the deterministic limit explored here. Fluctuations can play a role for systems with smaller populations and such effects have been thoroughly investigated in the physics literatures [10, 14–17]. Pulled waves are more sensitive to fluctuations, with a Fisher wave velocity that changes according to

$$v = v_F[1 - O(1/\ln^2 N_{\text{eff}})],$$  \hfill (S43)

where $v_F$ is the velocity of the pulled wave in the deterministic limit [14].


