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Pulsed interaction signals as a route to biological pattern formation

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We identify a mechanism for biological spatial pattern formation arising when the signals that mediate interactions between individuals in a population have pulsed character. Our general population-signal framework shows that while for a slow signal-dynamics limit no pattern formation is observed for any values of the model parameters, for a fast limit, on the contrary, pattern formation can occur. Furthermore, at these limits, our framework reduces, respectively, to reaction-diffusion and spatially nonlocal models, thus bridging these approaches.

Introduction.— One of the striking manifestations of self-organization in complex systems is the emergence of regular spatial patterns at scales much larger than the ones associated to the individual components [1]. In biological populations this phenomenon has been observed in many contexts including semiarid vegetation [2–4], bird swarms [5, 6] or bacteria colonies [7, 8]. Besides being fascinating, pattern formation has been shown to critically affect the stability and resilience of ecosystems [9, 10].

Behind the mechanisms responsible for selforganization there is often an agent or substance working as a signal that mediates the interactions. Signals have distinct emission protocols, propagation dynamics and occur in a wide range of temporal and spatial scales [11]. For example, species might use acoustic [12], visual [13] or chemical [14] signals to attract, repel, harm or support targeted individuals. It is this exchange of signals and the details of its dynamics that ultimately drive self-organization process [15, 16] and, consequently, control other key macroscopic outcomes [9, 17].

Despite the numerous studies analyzing how interactions control pattern formation, the focus has been mostly on continuous and smooth signal dynamics. This overlooks interactions that are mediated by flashing pulsed signals. Therefore, how this fine-scale dynamics scales-up affecting pattern formation is poorly understood. Here we show that a timescale transition from slow (smooth) to fast (pulsed) signal dynamics creates a route to pattern formation alternative to the most studied ones arising from Turing-like mechanisms [18].

This finding is obtained by studying a general activator-inhibitor (population-signal) model, where a population interacts through the release of harmful signals. Our study extends standard activator-inhibitor structure [1, 20], by explicitly describing the fine-scale dynamics associated with the release and spreading of signals. This framework recovers two distinct structures at the regimes of slow and fast signal dynamics that

can lead to qualitative changes in spatial stability. For slow signal (with timescales similar to those of the population), we recover a standard reaction-diffusion system which, for a broad set of population and signal dynamics, does not exhibit Turing instability for any values of model parameters. For the same system dynamics, but with sufficiently fast signals, the system can be described by a single integrodifferential equation, where the toxic effects are captured by a competitive nonlocal spatial interaction. In this limit, spatial instability can occur leading to pattern formation.

Since we explicitly derive the underlying interference competition mechanism behind the nonlocal effective description, these results address a long-standing shortcoming: that paradigmatic nonlocal models of competitive type leading to spatial patterns have been usually proposed phenomenologically with no systematic derivation [4, 21, 22]. In the cases in which such derivation has been provided, the resulting equation did not have the characteristics needed for pattern-forming instabilities (see, for example Ref. [23])

Model.— Our aim is to model an ensemble of simple organisms in a one-dimensional spatial domain (we do not expect this dimensional restriction to be essential for our results). They move, reproduce and release harmful signals in the form of pulses. These pulses can have biochemical origins, such as a toxic substance, but can also be physical, in the form of electricity, heat, sound and light, which can compromise the targets' survival, and lead to a competing dynamics among the individuals [13, 24, 25]. We describe this scenario at the population-level by the following general density-field description,

$$\tau_{\rho}\partial_{t}\rho = \mathcal{L}(\rho, \partial_{x}\rho) - \epsilon\rho\phi, \qquad (1)$$

$$\tau_{\phi}\partial_t \phi = L(\phi, \partial_x \phi) + \mathcal{R}_{\rho}(x, t) , \qquad (2)$$

where ρ and ϕ are the population density and signal intensity, respectively. \mathcal{L} and L give the population and signal dynamics when uncoupled, including diffusion or other transport processes; τ_{ρ} and τ_{ϕ} explicitly set the timescales for the population and signal dynamics, respectively; and ϵ is an exposition factor related to the population sensitivity to the toxin, which is released according to \mathcal{R}_{ρ} .

We consider that signal releases occur in pulses that are controlled by the population density in the following manner: Their starting time-space locations $\{t_i, x_i\}$ are independent Poisson random events with a probability of occurring within small intervals dx and dt given by $\alpha\rho(x,t)dxdt$. The pulses have duration $\overline{\delta}$, negligible spatial extent, and equal intensities I_0 :

$$\mathcal{R}_{\rho}(x,t) = \sum_{i} I_0 \Pi_{\bar{\delta}}(t-t_i) \delta(x-x_i) , \qquad (3)$$

where $\Pi_{\bar{\delta}}(t)$ is the indicator function of the time interval $[0, \bar{\delta}]$. The expected inter-event time, $\langle t_{i+1} - t_i \rangle$, is given by $\tau_R = 1/(N(t)\alpha)$, where $N(t) = \int_{-\infty}^{+\infty} \rho(x) dx$ is the total population size. Eqs. (1-3) together establish the model studied in this work, being constituted by a continuous population model but with a pulsed spatiotemporal dynamics for the signal [26].

The characteristic timescales are, besides τ_{ρ} and τ_{ϕ} , the duration of the pulses, $\bar{\delta}$, and the mean pulse interevent time, τ_R . We will focus on cases in which pulse duration is much shorter than release inter-event time, which is itself much shorter than population dispersal and other demographic processes, $\bar{\delta} \ll \tau_R \ll \tau_{\rho}$. This means that there is a timescale separation between interaction events and their consequences to population dynamics.

In the following, we investigate how the system spatial stability changes as a function of the signal timescale, τ_{ϕ} . We obtain effective descriptions for the population-toxin dynamics and the respective pattern forming conditions for a) the *slow signal-dynamics limit*, in which the toxin-field relaxation is slow, being comparable to population dynamics timescales $\tau_{\phi}/\tau_{\rho} \sim 1$, and thus $\bar{\delta}, \tau_R \ll \tau_{\phi}$; and b) fast signal-dynamics limit, when signal response is the faster of all the timescales, $\tau_{\phi} \ll \bar{\delta}, \tau_R, \tau_{\rho}$ (see Fig. 1).

Slow signal-dynamics limit.— When $\tau_{\phi}/\tau_{\rho} \sim 1$ the inter-event release time is much shorter than population and signal timescales, $\tau_R \ll \tau_{\phi}, \tau_{\rho}$. Then, the toxin field ϕ in Eq. (2) feels the average of the toxin release pulses, which are many and occur too fast for ϕ to follow them. Consequently, we can replace \mathcal{R}_{ρ} by its average over small time windows $\Delta t \ll \tau_{\phi}$ and small vicinities Δx :

$$\langle \mathcal{R}_{\rho}(x,t) \rangle \equiv \frac{1}{\Delta t \Delta x} \int_{-\Delta t}^{0} \int_{-\frac{\Delta x}{2}}^{\frac{\Delta x}{2}} \mathcal{R}_{\rho}(x+x',t+t') dx' dt'.$$
(4)

Using Eq. (3), $\langle \mathcal{R}_{\rho}(x,t) \rangle = (\Delta x \Delta t)^{-1} \sum_{i=1}^{n_R} I_0 \bar{\delta} = I_0 \bar{\delta} n_R / (\Delta x \Delta t)$, where n_R is the number of pulses that have occurred during the considered space-time window. Noting that pulses are independent events, n_R for each cell follows a Poisson distribution with mean

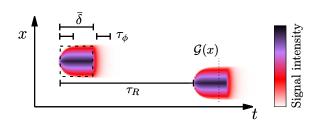


Figure 1: Schematic representation of the timescales of the signal $\phi(x,t)$ in the fast signal-dynamics limit ($\tau_{\phi} \rightarrow 0$). Signal response to two signal release pulses is shown for moderately fast signals. ϕ remains mainly localized in time within the pulse duration $\overline{\delta}$ (dashed rectangle). The rising and decaying parts of the signal are indicated by the two short segments close to the label τ_{ϕ} . The vertical dotted line indicates an intermediate time at which signal intensity attains the steady \mathcal{G} profile. In the plot, the lapse between pulses is set to τ_R (the mean inter-event time) and the field ϕ spreads according to Eq. (7) (with $\nu = 4$ and $\mu = 1$ as in Fig. 3b).

 $\alpha\rho(x,t)\Delta x\Delta t$. If Δt is chosen sufficiently large (but still much smaller than τ_{ϕ}) n_R becomes large and its coefficient of variation (ratio of standard deviation to mean) vanishes so that fluctuations can be neglected. Thus $n_R \approx \alpha\rho(x,t)\Delta x\Delta t$. As a consequence, in the slow signal limit, $\langle \mathcal{R}_{\rho}(x,t) \rangle \approx I_0 \bar{\delta} \alpha \rho(x,t)$. The other terms in Eqs. (1-2) can also be coarse-grained but, due to their slow response times, they remain constant and unaffected by the procedure: $\langle \rho \rangle \simeq \rho, \langle \phi \rangle \simeq \phi, \langle \phi \rho \rangle \simeq \phi \rho$.

Fast signal-dynamics limit.— In this fast limit, $\tau_{\phi}/\tau_{\rho} \rightarrow 0$, the signal dynamics is much faster than any other process. Then, we can expect the signal field to be always in constant equilibrium with the release events: it immediately reaches a fixed stationary profile, $\mathcal{G}(x)$ during the pulse duration, $0 < t - t_i < \overline{\delta}$, and dissipates immediately when release ceases (we assume a L dynamics that leads to signal dissipation in the absence of releases). In Fig. 1, we present a schematic representation of the fast signal propagation, highlighting with a dashed rectangle the area in which signals would be confined taking $\tau_{\phi} \to 0$. The profile \mathcal{G} at intermediate times (such as the vertical dotted line in Fig. 1) can be obtained by solving Eq. (2) under the limit $\tau_{\phi} \to 0$. For a single pulse in (3) at x = 0, $L(\mathcal{G}, \partial_x \mathcal{G}) + I_0 \delta(x) = 0$. The conditions $\tau_{\phi} \ll \bar{\delta} \ll \tau_R$ guarantee that pulses are nonoverlapping, so that the solution of Eq. (2) can be built just adding up the successive responses to the different pulses: $\phi(x,t) \approx \sum_{i} \mathcal{G}(x-x_i) \prod_{\bar{\delta}} (t-t_i)$. We now perform, as in Eq. (4), an average of Eq. (1) over small intervals Δx and $\Delta t \ll \tau_{\rho}$. Because of timescale separation, all terms remain unaltered except the last one containing ϕ , which becomes $\langle \rho \phi \rangle \approx \rho \langle \phi \rangle$. Calculation of this last average is performed in detail in the Supplemental Material [27], with the final result $\langle \phi \rangle \approx \bar{\delta} \alpha [\mathcal{G} * \rho]$, where $\mathcal{G} * \rho \equiv \int \mathcal{G}(x - x')\rho(x', t)dx'.$

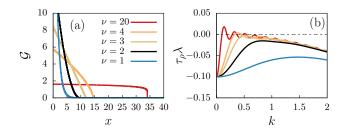


Figure 2: (a) Profile $\mathcal{G}(x)$ from Eq. (8) and several ν . (b) Corresponding growth rates $\lambda(k)$ of perturbations to the homogeneous solution, as a function of perturbation wavenumber k. Parameters are $D_{\rho} = 0.01$, $\mu = 1$, $\bar{\epsilon} = r = 1$, $D_{\phi} = \gamma = 1$ and $I_0 = 10^2$.

In summary, from model (1-3), the slow signaldynamics limit $(\bar{\delta} \ll \tau_R \ll \tau_{\phi}, \tau_{\rho})$ leads to

$$\tau_{\rho}\partial_{t}\rho = \mathcal{L}(\rho,\partial_{x}\rho) - \epsilon\rho\phi,$$

$$\tau_{\phi}\partial_{t}\phi = L(\phi,\partial_{x}\phi) + \bar{R}\rho, \text{ with } \bar{R} \equiv \alpha\bar{\delta}I_{0}.$$
 (5)

Fast signal dynamics $(\tau_{\phi} \ll \bar{\delta} \ll \tau_R \ll \tau_{\rho})$ gives

$$\tau_{\rho}\partial_{t}\rho = \mathcal{L}(\rho,\partial_{x}\rho) - \bar{\epsilon}\rho \left[\mathcal{G}*\rho\right], \quad \text{with} \quad \bar{\epsilon} \equiv \epsilon\bar{\delta}\alpha \;. \tag{6}$$

Regardless of the choice of $\mathcal{L}(\rho, \partial_x \rho)$ and $L(\phi, \partial_x \phi)$, the fact that the two regimes lead to different coarse-grained models suggests that their spatial stability also differs. In fact, it can be shown that pattern formation does not occur in the slow signal limit (Eq. (5)) for a large class of operators (see [27] for precise conditions on \mathcal{L} and L). However, for this same class in the fast limit, it is well known that Eq. (6) can lead to spatial patterns when the signal profile \mathcal{G} is sufficiently platykurtic [28, 29].

A particular example. — We illustrate the above developments with the following dynamics,

$$\mathcal{L}(\rho, \partial_x \rho) = (D_\rho \partial_{xx} + r)\rho, \qquad (7)$$
$$L(\phi, \partial_x \phi) = D_\phi \partial_x (\phi^{\nu-1} \partial_x \phi) - [\gamma \phi^{\mu-1}]\phi,$$

which models populations of organisms moving Brownianly with diffusion coefficient D_{ρ} and reproducing with growth rate r. This choice is a fundamental building block for more complex population dynamics models [30]. For the signal dynamics, Eq. (7) gives a generalized nonlinear diffusion-decay process characterized by exponents $\nu, \mu > 0$. It allows to consider the case where diffusion and decay are sensitive to signal intensity in a negative ($\nu, \mu < 1$) or positive ($\nu, \mu > 1$) manner, unraveling important channels through which environment structure (e.g. propagation in porous media, leading to $\nu > 1$, see Ref. [31]) and mediator inter-specific biochemical interactions [24, 30, 32–36] can affect signal propagation dynamics.

Linear stability analysis and pattern formation. — The pattern-forming stability conditions of model (1-3) with the choice (7) can be obtained in the above studied

timescale limits. For slow signal dynamics (Eq. (5)) the non-trivial homogeneous steady state is $\rho_0 = \gamma(r/\epsilon)^{\mu}/\bar{R}$, $\phi_0 = r/\epsilon$. Standard linear perturbation around this state identifies that all perturbation growth rates are negative for any value of parameters, implying the stability of the homogeneous state. Hence, no pattern-forming instability can arise.

For fast signal dynamics the model reduces to a single nonlocal equation, Eq. (6), with integral kernel \mathcal{G} . This is the solution of $L(\mathcal{G}, \partial_x \mathcal{G}) + I_0 \delta(x) = 0$, an equation that can be solved exactly [5] for the particular choice (7) discussed here (additional details are in [27]) giving:

$$\mathcal{G}(x) = A \left[1 - (1 - q) \left| sx \right| \right]^{\frac{1}{1 - q}}, \ A = \left[\frac{I_0}{2D_{\phi}} \sqrt{\frac{\mu + \nu}{2\kappa}} \right]^{\frac{2}{\mu + \nu}}$$
(8)

with $q = 1 + (\mu - \nu)/2$, $s^2 = 2\kappa A^{\mu-\nu}/(\mu + \nu)$, and $\kappa = \gamma/D_{\phi}$. If q < 1 the support of this solution is restricted to $|x| \leq 1/(1-q)$.

Fig. 2a presents the different shapes of $\mathcal{G}(x)$ as ν increases, while assuming linear decay ($\mu = 1$). The homogeneous steady solution is $\rho_0 = r/(\bar{\epsilon}\mathcal{G}(0))$, where $\mathcal{G}(k)$ is the Fourier transform of \mathcal{G} . Growth rates of periodic perturbations of wavenumber k to the homogeneous state are given by $\tau_{\rho}\lambda(k) = -D_{\rho}k^2 - D_{\rho}k^2$ $r\tilde{\mathcal{G}}(k)/\tilde{\mathcal{G}}(0)$ and are shown in Fig. 2b. Pattern formation requires that, for some k, $\tilde{\mathcal{G}}$ assumes a sufficiently negative value, yielding $\lambda(k) > 0$ [28]. For the present case, this occurs if toxin diffusion has a stronger sensitivity to concentration when compared to the decay process, $\nu > \mu + 2$. The marginal case, $\nu = \mu + 2$ ($\nu = 3$ with $\mu = 1$ in Fig. 2), corresponds to the triangular kernel and the limit case $\nu \to \infty$ to the (most used) top hat kernel, which is well-known to lead to pattern formation [21, 38–40]. Thus, in contrast to the slow signal-dynamics limit, pattern formation can occur under fast signal dynamics, showing the importance of pulsed dynamics on the macroscopic behavior of the system.

To support these analytical findings, we show in Fig. 3 direct numerical simulations (see Supplemental Material for the numerical integration scheme [27]) of Eqs. (1-3)and (7) (with $\mu = 1, \nu = 4$) for a slow (a) and a fast (b) signal-dynamics regimes. This is done by keeping the population timescale at $\tau_{\rho} = 1/r = 1$ for both plots, and selecting the signal timescale corresponding to $\tau_{\phi} = 1 \sim \tau_{\rho}$, and $\tau_{\phi}/\tau_{\rho} = 1/500 \ll 1$, respectively. In agreement with the analytical results, for the slow signal dynamics pattern formation does not occur for any of the parameter values we have checked (Fig. 3a). On the contrary, in the fast limit patterns develop, since $\nu > \mu + 2$. Spatial population periodicity is seen to emerge at long times in Fig. 3b, and the spatial pattern remains stable afterwards (see [27]). The wavelength of the final pattern can be analytically estimated as $2\pi/k^{\star} \simeq 16.5$, where k^{\star} is the fastest growing mode in

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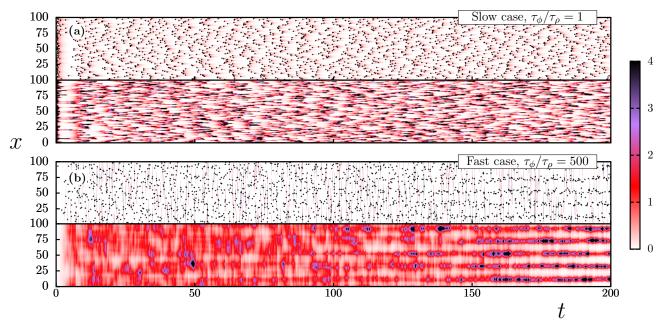


Figure 3: Temporal evolution of signal and population fields for the slow (a) and fast (b) signal regimes. The system is a line of length 100 with periodic boundary conditions. Colors indicate field intensity $\phi(x,t)$ (upper panels), and population density relative to the (fast-signal limit) homogeneous state, $\rho(x,t)/\rho_0$ (bottom panels). In the upper panels crosses indicate release instants and positions (not all releases are captured by the finite resolution of the heatmap). Data from numerical integration of Eqs.(1-3), using an Euler scheme with $\delta t = 10^{-6}$ and $\delta x = 1.0$. The dynamics is given by Eq. (7) with $D_{\rho} = 10^{-2}$, $r = \tau_{\rho} = 1$, $\alpha = 10^2$, $\overline{\delta} = 10^{-2}$ and $\epsilon = 10$. Signal dynamics is set by $\mu = 1$ and $\nu = 4$, scaled by τ_{ϕ} , in such way that $D_{\phi}/4 = \gamma = 1/\tau_{\phi}$ and $I_0 = 10^2/\tau_{\phi}$. The slow and fast regimes were obtained setting $\tau_{\phi} = 1$ and $\tau_{\phi} = 1/500$, respectively. Mean interpulse interval is in both cases $\tau_R = 1/(N(t)\alpha) \simeq 10^{-1}$ (for a schematic close-up of the signal field in the fast-signal regime see Fig. 1).

Fig. 2b. This is roughly close to the periodicity seen in Fig. 3b (see also Fig. S2 in Supplemental Material [27]).

Final remarks and discussion.— Our framework allowed us to see how different fine-scale signal dynamics impact at a coarser scale. It recovers standard reactiondiffusion [1, 20] schemes in the slow-signal limit and integrodifferential schemes [28, 41] in the fast-signal limit, working as a bridge between the two mostly used formalisms to describe interacting populations.

We crucially note that these two descriptions can lead to different macroscopic outcomes. In this work, we focused on showing that, for the same population and mediator dynamics, a transition from slow to fast pulsed signals can effectively lead to spatiallyextended interference competition in such way that pattern formation occurs [21, 23, 28].

Our findings are of relevance in situations, from chemistry to ecology, in which interactions between the entities are mediated by pulses that are short and fast compared to reaction processes. More broadly, they stress crucial channels through which environment and individual-level behavior can control system spatial organization [42]. For example, our approach can be extended to cases in which signals regulate individual mobility [43, 44], a mediation that has already shown to be relevant for population survival and spatial patterns [15, 45]. Developmental programs can also explore these channels to engineer specific morphologies [26, 46]. Further extension aiming at concrete problems should include realistic features such as: state-dependent signal emissions [47, 48] accounting for individuals response to attacks; memory [16]; persistence [15]; and multi-signal mediation where signals establish a set of distinct biochemical interactions [11].

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Supplemental material: "Pulsed interaction-signals as a route to pattern formation"

Eduardo H. Colombo, Cristóbal López and Emilio Hernández-García

COARSE-GRAINING OF THE TOXIN FIELD IN THE FAST TOXIN-DYNAMICS LIMIT

We demonstrated in the main text that the signal field in the fast toxin-dynamics limit $\tau_{\phi}/\tau_{\rho} \rightarrow 0$ takes the approximate form

$$\phi(x,t) = \sum_{i} \mathcal{G}(x-x_i) \Pi_{\bar{\delta}}(t-t_i) , \qquad (S1)$$

where $\mathcal{G}(x)$ is the steady profile attained under a single persistent pulse at x = 0 and $\{t_i, x_i\}$ are the starting times of the different toxin release events and their locations. Here, we calculate the average or coarse-graining of this field, $\langle \phi(x,t) \rangle$, over small temporal and spatial intervals:

$$\langle \phi(x,t) \rangle \equiv \frac{1}{\Delta t \Delta x} \int_{-\Delta t}^{0} \int_{-\frac{\Delta x}{2}}^{\frac{\Delta x}{2}} \sum_{i} \mathcal{G}(x+x'-x_i) \Pi_{\bar{\delta}}(t+t'-t_i) dt' dx' .$$
(S2)

The temporal interval Δt should satisfy $\Delta t \ll \tau_{\rho}$ but we will also assume that it is larger than other microscopic time scales: $\bar{\delta}, \tau_R \ll \Delta t$. At difference with the slow toxin case, here the spatial coarse-graining is not really needed, so that we eliminate it from the expression by taking the limit $\Delta x \to 0$. The remaining temporal integral acts on the indicator function $\Pi_{\bar{\delta}}$, selecting at any time t only the m pulses that have occurred anywhere in the system during the interval $[t - \Delta t, t]$. Thus we have:

$$\langle \phi(x,t) \rangle \approx \bar{\delta}(\Delta t)^{-1} \sum_{i}^{m_R} \mathcal{G}(x-x_i).$$
 (S3)

 m_R is the total number of releases for the population which follows a Poisson distribution with mean $\alpha N(t)\Delta t$, where $N(t) = \int \rho(x, t) dx$ is the total population size. We have neglected the values of t for which a pulse is only partially contained in Δt . Because of the condition $\bar{\delta} \ll \Delta t$ such time intervals are very small and negligible at the population scale τ_{ρ} .

As in the calculation for the slow signal case, the condition $\tau_R \ll \Delta t$ implies a large value of m_R so that, by the law of large numbers, its fluctuations can be neglected. Also in this case we can use $m_R^{-1} \sum_i \mathcal{G}(x-x_i) \approx \int dx' \mathcal{G}(x-x') \text{pdf}_t(x')$, where $\text{pdf}_t(x) = \rho(x,t)/N(t)$ is the probability density of the locations x_i . Combining these results we arrive at

$$\langle \phi \rangle \approx \bar{\delta} \alpha \int \mathcal{G}(x - x') \rho(x', t) dx' \equiv \bar{\delta} \alpha [\mathcal{G} * \rho].$$
 (S4)

CONDITIONS FOR THE ABSENCE OF PATTERN FORMATION IN THE SLOW SIGNAL-DYNAMICS LIMIT

In this Section we establish conditions on the dynamics which are sufficient to guarantee that pattern formation is absent in the slow signal-dynamics limit. In this limit, Eqs. (5) of the main text were found:

$$\partial_t \rho = \mathcal{L}(\rho, \partial_x \rho) - \epsilon \rho \phi \tag{S5}$$

$$\partial_t \phi = L(\phi, \partial_x \phi) + R\rho . \tag{S6}$$

By inspecting this structure we note that it is likely to not produce patterns, since it has a linear coupling between the equations [1]. To obtain the exact class of operators for which we can guarantee that the homogeneous solution of Eqs. (S5-S6) is stable, we perform the following calculations.

To begin with, the steady and homogeneous solution ($\rho_0 > 0, \phi_0 > 0$) of (S5)-(S6) is

$$\rho_0 = -\frac{L_0(\phi_0)}{\bar{R}} \quad , \quad \phi_0 = -\frac{\bar{R}}{\epsilon} \frac{\mathcal{L}_0(\rho_0)}{L_0(\phi_0)} \; , \tag{S7}$$

where we have defined $\mathcal{L}_0(\rho_0) \equiv \mathcal{L}(\rho_0, 0)$ and $L_0(\phi_0) \equiv L(\phi_0, 0)$. Note that positivity of ρ_0 requires $L_0(\phi_0) < 0$. To check the stability properties, we linearize: $\rho(x,t) = \rho_0 + \delta\rho(x,t)$, $\phi(x,t) = \phi_0 + \delta\phi(x,t)$, $\mathcal{L} = \mathcal{L}_0(\rho_0) + \mathcal{L}_1\delta\rho(x,t) + \mathcal{O}(\delta\rho)^2$, $L = L_0(\phi_0) + L_1\delta\phi(x,t) + \mathcal{O}(\delta\phi)^2$. Under Fourier transformation, $\delta\rho(x,t) \to \delta\tilde{\rho}(k,t)$, $\delta\phi(x,t) \to \delta\tilde{\phi}(k,t)$, $\mathcal{L}_1 \to \tilde{\mathcal{L}}_k$ and $L_1 \to \tilde{\mathcal{L}}_k$, the linear stability of (ρ_0, ϕ_0) is guaranteed if the following linear growth rates $\lambda_{\pm}(k)$ have negative real parts $\forall k$:

$$\lambda_{\pm}(k) = \frac{1}{2} \left[\tilde{\mathcal{L}}_k + \tilde{L}_k - \epsilon \phi_0 \pm \sqrt{(\tilde{\mathcal{L}}_k + \tilde{L}_k - \epsilon \phi_0)^2 - 4(\tilde{\mathcal{L}}_k - \epsilon \phi_0)\tilde{L}_k - 4\bar{R}\rho_0} \right] .$$
(S8)

We now impose additional restrictions on \mathcal{L} and L that would be sufficient to guarantee stability of (ρ_0, ϕ_0) . First, we assume that $L_k < 0$. This is the case if the dynamics of the toxin in the absence of release is some diffusiondecay process. For the population dynamics implemented in \mathcal{L} we assume that the maximum of $\tilde{\mathcal{L}}_k$ is achieved at k = 0, i.e. $\tilde{\mathcal{L}}_k \leq \tilde{\mathcal{L}}_{k=0} = \mathcal{L}_0(\rho_0)'$. This is the typical case in which gradient terms in the population dynamics are diffusion-like, but excludes models in which higher order derivatives induce instabilities [2–4]. Finally, we restrict to $\mathcal{L}_0(\rho_0)' \leq \mathcal{L}(\rho_0)/\rho_0 \ (= \epsilon \phi_0)$. This is for example the case if $\mathcal{L}_0(\rho_0)$ is linear, or the well known logistic model $\mathcal{L}_0(\rho_0) = a\rho_0 - b\rho_0^2$ with $b \geq 0$. But excludes the case b < 0, which could lead to additional homogeneous instabilities, as in [3, 4]. The stated conditions are sufficient to guarantee that $\hat{\mathcal{L}}_k + \hat{\mathcal{L}}_k - \epsilon \phi_0 < 0$, and then $\text{Re}\lambda_{\pm}(k) < 0 \ \forall k$, and (ρ_0, ϕ_0) is stable so that no pattern-forming instability occurs. In the main text we focus on showing that, keeping the same dynamics for \mathcal{L} and L, in the fast-signal limit the result for the linear stability changes qualitatively and allows pattern formation to occur for some classes of \mathcal{L} and L.

STATIONARY SIGNAL DENSITY FIELD FOR A SINGLE RELEASE IN THE FAST SIGNAL-DYNAMICS LIMIT

Under the dynamics

$$\tau_{\phi}\partial_t \phi = L(\phi, \partial_x \phi) + \mathcal{R}_{\rho}(x, t) , \qquad (S9)$$

in the case of a single persistent and localized release, say at x = 0: $\mathcal{R}_{\rho}(x,t) = I_0\delta(x)$ if $t \in [t_i, t_t + \overline{\delta}]$, and in the fast signal-dynamics limit $\tau_{\phi} \to 0$, ϕ immediately achieves a stationary profile $\mathcal{G}(x)$ which lasts while the pulse is present $(t \in [t_i, t_t + \overline{\delta}])$. Assuming that the signal dynamics is ruled by

$$L(\phi, \partial_x \phi) = D_\phi \partial_x (\phi^{\nu-1} \partial_x \phi) - [\gamma \phi^{\mu-1}] \phi, \qquad (S10)$$

the stationary profile $\mathcal{G}(x)$ satisfies

$$D_{\phi}\partial_x(\mathcal{G}^{\nu-1}\partial_x\mathcal{G}) - \gamma\mathcal{G}^{\mu} = -I_0\delta(x).$$
(S11)

This stationary solution can be found rewriting the above equation as

$$\partial_{xx}Z - \nu\kappa Z^{\frac{\mu}{\nu}} = \frac{-\nu I_0}{D_\phi}\delta(x) \tag{S12}$$

where $Z = \mathcal{G}^{\nu}$ and $\kappa = \frac{\gamma}{D_{\phi}}$. Outside the release point we need that $\partial_{xx}Z = \nu\kappa Z^{\frac{\mu}{\nu}}$. The solution can be found [5] using as ansatz a generalization of the exponential function, namely

$$e_q(x) \equiv [1 - (1 - q)|x|]^{\frac{1}{1 - q}},$$
 (S13)

which is valid for $|x| \in [0, +\infty)$ for $q \ge 1$ and $|x| \in [0, 1/(1-q)]$ if q < 1. This function recovers the exponential function in the $q \to 1$ limit. Its derivative is given by $\frac{de_q(sx)}{dx} = -se_q^q$, then, consequently $\frac{d^2e_q(sx)}{dx^2} = s^2qe_q^{2q-1}$. Hence, substituting $Z = A'e_{q'}(s'x)$ in Eq. (S12) we find $s'^2 = (A')^{\mu/\nu-1}\kappa\nu/q'$, $q' = \frac{\mu+\nu}{2\nu}$ and $A' = A^{\nu}$. Using that $\mathcal{G} = Z^{1/\nu}$, we find that

$$\mathcal{G}(x) = Ae_q(sx),$$

$$q = 1 + \frac{\mu - \nu}{2}, s^2 = \frac{2\kappa A^{\mu - \nu}}{(\mu + \nu)}, A = \left[\frac{I_0}{2D_\phi}\sqrt{\frac{\mu + \nu}{2\kappa}}\right]^{\frac{2}{\mu + \nu}},$$
(S14)

where the value of the amplitude A is found by considering the flux constrain introduced by the point release, $\partial_x Z|_{x=0} = -\nu I_0/(2D_{\phi})$. For q < 2, the area under the stationary profile is finite and it is given by $\tilde{\mathcal{G}}(0) \equiv \int dx \mathcal{G}(x) = \frac{2A}{(2-q)s}$.

NUMERICAL INTEGRATION SCHEME

In order to numerically integrate Eqs. (1-3) of the main text, we follow a standard forward Euler scheme complemented with the generation of the stochastic state-dependent toxic release \mathcal{R}_{ρ} . We discretize space in small cells of size δx and time in small intervals of duration δt and define $\rho_{j,n} \equiv \rho(x = j\delta x, t = n\delta t)$, and analogously with $\phi_{j,n}$. For the particular dynamics given by Eq. (7) of the main text, the evolution after one time-step δt of the population and signal fields is obtained as follows:

$$\rho_{j,n+1} = \rho_{j,n} + [D_{\rho}(\rho_{j+1,n} + \rho_{j-1,n} - 2\rho_{j,n})/(\delta x^2) + r\rho_{j,n} - \epsilon \rho_{j,n}\phi_{j,n}]\delta t/\tau_{\rho}, \qquad (S15)$$

$$\phi_{j,n+1} = \phi_{j,n} + [D_{\phi}(\phi_{j+1,n}^{\nu} + \phi_{j-1,n}^{\nu} - 2\phi_{j,n}^{\nu})/(\nu \delta x^2) - \gamma \phi_{j,n}^{\mu} + \mathcal{R}_{j,n}]\delta t/\tau_{\phi}.$$

We used that the nonlinear diffusion term $\partial_x(\phi^{\nu-1}\partial_x\phi)$ can be written as $\nu^{-1}\partial_{xx}\phi^{\nu}$ to help with numerical instabilities. The stochastic variable $\mathcal{R}_{j,n}$ is the discretized version the pulse release function $\mathcal{R}_{\rho}(x,t)$ given by Eq. (3) of the main text. It is implemented as follows: Initially all $\mathcal{R}_{j,n}$ are set to zero. At each time step we check if a pulse will occur somewhere in the system, with probability $\alpha N(t)\delta t$, where $N(t) = \sum_k \rho_{k,n}\delta x$ is the total population. If so, the needed pulse location j is sampled from its probability $\rho_{j,n}/\sum_k \rho_{k,n}$. Then, the value $\mathcal{R}_{j,n}$ is set to $I_0/\delta x$ during $\bar{\delta}/\delta t$ time steps, being reset to zero afterwards. The denominator δx arises from the discretization of the spatial delta function. To ensure that fast pulses are resolved by the numerical integration we consider $\delta x = 1.0$, smaller than signals' reach (~ 10), and $\delta t = 10^{-6}$, much smaller than the signal duration time $\bar{\delta} = 0.01$.

NUMERICAL STEADY SOLUTIONS FOR FAST SIGNAL DYNAMICS

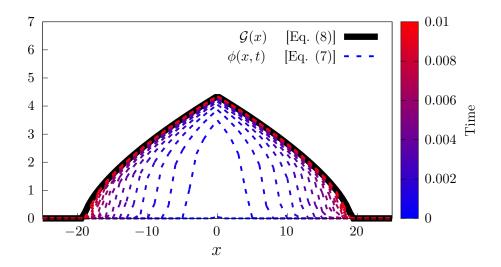


Figure S1: Signal dynamics (dashed lines) from integration of Eq. (2) with (7) of the main text starting from vanishing toxic signal, but with signal release fixed to a delta function at the origin ($\mathcal{R}_{\rho} = I_0 \delta(x)$). Time is indicated by color as shown in the colorbar. Parameters as in Fig. 3b of the main text. After a short transient the profile correctly approaches the stationary profile calculated in the fast signal-dynamics limit from the integrodifferential description of Eq. (8) of the main text (solid black line).

Both as a check of the numerical algorithm of the previous Section, and to show the validity of the integrodifferential approximation in Eq. (6) of the main text, obtained in the fast signal-dynamics limit, we show in this Section two simulations of the system (1-3) with (7) in a fast signal regime.

First, Fig. S1 displays an integration of Eq. (2) with (7) using the second equation of the numerical algorithm (S15) in a situation of fast signal dynamics (parameters as in Fig. 3b of the main text) but with the release fixed as a delta function at the origin $\mathcal{R}_{\rho}(x,t) = I_0 \delta(x)$ ($\mathcal{R}_{j,n} = I_0 / \delta x$ in the discretized version). We see that the signal intensity profile correctly achieves in a short time the analytical steady form given by Eq. (8) of the main text, which is adequate for this parameter regime.

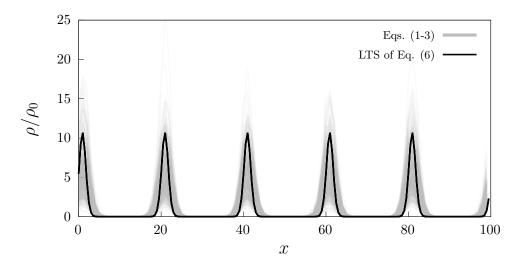


Figure S2: Snapshots of the density profiles at the long-time regime are shown as gray (95% transparent) solid lines. Snapshots are from the same simulation as in Fig. 3b of the main text, but for 200 < t < 1000, being displayed at every unit time interval (800 samples). The solid black line is the long-time state (LTS) of the integrodifferential Eq. (6), appropriate for this fast signal-dynamics situation. This last solution was spatially shifted so that the peaks of the two solutions match.

Second, in Fig. S2, we show that the pattern produced by the numerical integration of the stochastic system (1-3) with (7) of the main text, in the fast-signal regime (same simulation as in Fig. 3b of main text) matches the stationary profile predicted by the integrodifferential description Eq. (6) of the main text, obtained by numerical simulation at long times, using random fluctuations around the homogeneous solution as initial condition.

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