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ABSTRACT

Most studies of collective phenomena in oscillator networks focus on directly coupled systems, as exemplified by the classical Kuramoto model. However, there are a growing number of examples in which oscillators interact indirectly via a common external medium, including bacterial quorum sensing (QS) networks, pedestrians walking on a bridge, and centrally coupled lasers. In this paper, we analyze the effects of stochastic phase resetting on a Kuramoto model with indirect coupling. All the phases are simultaneously reset to their initial values at a random sequence of times generated from a Poisson process. On the other hand, the external environmental state is not reset. We first derive a continuity equation for the population density in the presence of resetting and show how the resulting density equation is itself subject to stochastic resetting. We then use an Ott–Antonsen (OA) *Ansatz* to reduce the infinite-dimensional system to a four-dimensional piecewise deterministic system with subsystem resetting. The latter is used to explore how synchronization depends on a cell density parameter. (In bacterial QS, this represents the ratio of the population cell volume and the extracellular volume.) At high densities, we recover the OA dynamics of the classical Kuramoto model with global resetting. On the other hand, at low densities, we show how subsystem resetting has a major effect on collective synchronization, ranging from noise-induced transitions to slow/fast dynamics.

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Spontaneous synchronization of coupled oscillators occurs in a wide range of natural and artificial systems. One of the most studied examples of collective synchronization is the Kuramoto model of globally coupled phase oscillators, whose interactions are taken to be a sinusoidal function of their phase differences. However, there are a number of systems in which the coupling between oscillators is indirectly mediated by a common external medium. One major example is quorum sensing in a network of genetically engineered bacterial cells that communicate via the secretion and subsequent uptake of signaling molecules diffusing in the extracellular environment. One finds that the collective behavior of the bacteria is strongly dependent on the cell density. In this paper, we consider the Kuramoto model with indirect coupling and global phase resetting, where all the oscillators are simultaneously reset to their original phases at a random sequence of times generated by a Poisson process. Using a combination of continuum modeling, dimensional reduction, and slow/fast analysis, we explore how collective synchronization in the presence of resetting depends on the cell density.

I. INTRODUCTION

A number of natural and synthetic systems involve populations of oscillators that interact indirectly via a common external medium rather than via direct connections. The best known example is a quorum sensing (QS) network of genetically engineered bacteria.^{1,2} (Other examples include pedestrians walking on a bridge,^{3,4} which was inspired by the Millennium Bridge Problem in London, semiconductor lasers coupled through a central hub,⁵ and social networks under the influence of social media.⁶) Bacterial QS involves the production and extracellular secretion of certain signaling molecules known as autoinducers (see Fig. 1). Each cell also has receptors that can detect the autoinducers via ligand–receptor binding. If the probability of detection is sufficiently high then this can activate gene transcription, including the synthesis of the autoinducers. However, since there is a low likelihood of an individual bacterium detecting its own secreted autoinducers, the cell must encounter signaling molecules secreted by other cells in its environment in order for gene transcription to be activated. When only a few other bacteria of the same kind are in the vicinity

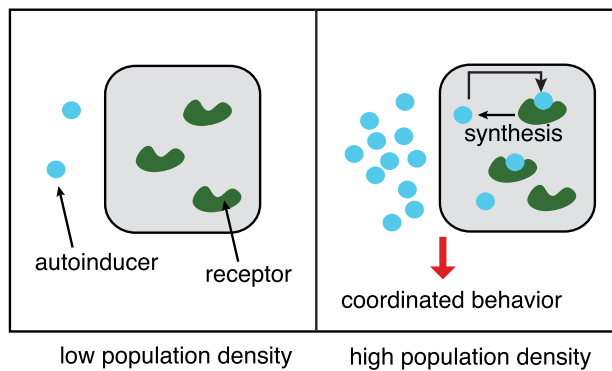


FIG. 1. A schematic illustration of bacterial quorum sensing (QS) at the single-cell level. An increase in cell density leads to an increase in the local extracellular and intracellular autoinducer concentrations. This results in some form of coordinated behavior due to the simultaneous activation of protein synthesis across the cell population. See text for details.

(low bacterial population density), diffusion reduces the concentration of the inducer in the surrounding medium to almost zero, resulting in small amounts of inducer being available to bind to receptors. On the other hand, as the population grows, the concentration of the inducer passes a threshold, causing more inducers to be synthesized. This generates a positive feedback loop such that all of the cells initiate transcription at approximately the same time, resulting in some form of collective behavior.

In wild-type bacterial colonies, QS drives the production and release of various effector molecules that aid in bioluminescence, biofilm formation, virulence, and antibiotic resistance.^{7–10} On the other hand, genetically engineered bacterial colonies can exhibit intrinsic chemical oscillations that are synchronized by diffusive coupling. This population density-dependent form of synchronization is an example of the so-called dynamic QS. Other examples include suspensions of yeast in nutrient solutions¹¹ and starving cellular colonies of the social amoeba *Dictyostelium*.¹²

Most theoretical studies of collective phenomena in oscillator networks focus on directly coupled systems,¹³ although there are some notable exceptions.^{14–18} In particular, Schwab *et al.*¹⁶ considered a Kuramoto model of dynamical QS with indirect coupling via an external medium. As in the original Kuramoto model,^{19–21} each cellular oscillator is described by a phase $\theta_j(t)$ and natural frequency ω_j , $j = 1, \dots, M$. Moreover, the natural frequencies are drawn from a prescribed probability density $h(\omega)$ with mean ω_0 . However, in contrast to the original Kuramoto model with direct coupling, the phase oscillators are diffusively coupled to an external medium whose state is represented by a complex amplitude $Z(t) = R(t)e^{i\Phi(t)}$, see Fig. 2. Following previous studies of direct coupling, Schwab *et al.*¹⁶ investigated the existence of partially coherent states in the continuum limit $M \rightarrow \infty$. The resulting continuity equation for the density of oscillators was solved by projecting the dynamics onto the corresponding Ott–Antonsen (OA) manifold with $h(\omega)$ taken to be Lorentzian. In contrast to the effective one-dimensional OA manifold found for direct coupling,²² the OA

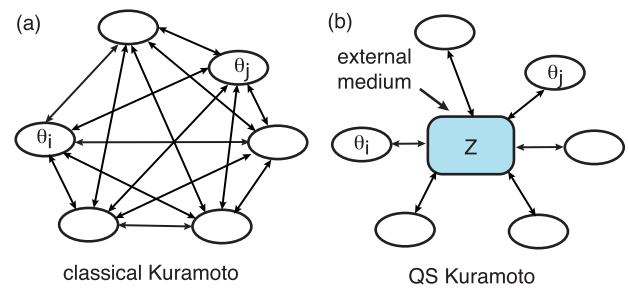


FIG. 2. Schematic illustration of the Kuramoto model with (a) direct pairwise coupling and (b) indirect coupling via an external medium with complex amplitude Z . The latter is an example of a QS network.

manifold for indirect coupling is three-dimensional with coordinates $(r(t), R(t), \psi(t))$, where $\psi(t) = \Phi(t) - \phi(t)$ and $r(t)e^{i\phi(t)}$ is the first circular moment of the phases. Consequently, certain phenomena arise that are not found in the original model,¹⁶ including bistability.

A key parameter in the Kuramoto model with indirect coupling is an effective cell density σ_0 that acts as a multiplier of the coupling constant in the dynamics of $Z(t)$. In applications to bacterial QS, σ_0 represents the ratio of the population cell volume to the extracellular volume and is a dimensionless quantity. (It is denoted by ρ in Ref. 16.) In the high-density limit $\sigma_0 \rightarrow \infty$, one finds that the OA dynamics reduces to the 1D dynamics of the standard Kuramoto model. On the other hand, in the low density regime, the system exhibits bistability between an incoherent state and a highly coherent state or between coherent states with different amplitudes R . Moreover, in the limit $\sigma_0 \rightarrow 0^+$, the model can be mapped to a Kuramoto model with a bimodal frequency distribution.²³

In this paper, we extend the analysis of the Kuramoto model with coupling through an external medium to include the effects of stochastic resetting. From the perspective of nonequilibrium statistical physics, there is growing interest in understanding the effects of stochastic resetting on interacting particle systems (see the recent review²⁴), which builds upon the original work on single particle resetting.^{25–28} Several recent papers have analyzed the standard Kuramoto model with stochastic resetting,^{29–32} but as far as we are aware, the corresponding model with indirect coupling has not been considered. At the multi-particle level, there is an important distinction between cases where all the particles are simultaneously reset to their initial states (*global resetting*) and cases where each particle independently resets (*local resetting*). Another scenario is the so-called *subsystem resetting* in which a chosen proper subset of particles are simultaneously reset, while the remaining particles continue from their current state (nonresetting population). In a recent study of the classical Kuramoto model with subsystem resetting,³² it was found that for sufficiently fast resetting, even if only a small fraction of the oscillators reset, the nonresetting population also synchronizes (although not necessarily to a steady state). This was established using a combination of numerical simulations and a reduction of the nonresetting dynamics to an OA manifold in the continuum limit.

Following along analogous lines to recent work on the classical Kuramoto model with resetting,²⁹ we consider global phase resetting in an external environment that is not reset. From the perspective of the full dynamical system (phase oscillators and the external medium), this is an example of subsystem resetting. The structure of the paper is as follows. We begin by introducing the Kuramoto model with indirect coupling and stochastic phase resetting (Sec. II). We then derive the continuity equation for the population density in the continuum limit $\mathcal{M} \rightarrow \infty$ (Sec. III). As we have recently shown for the Kuramoto model with direct coupling,³⁰ this is a non-trivial problem since the resulting density equation is itself subject to stochastic resetting. That is, it is not possible to eliminate the stochasticity using a mean field argument. In Sec. IV, we solve the continuity equation by projecting the dynamics onto a corresponding Ott–Antonsen (OA) manifold with $h(\omega)$ taken to be Lorentzian. In contrast to the effective one-dimensional OA manifold found for direct coupling,²² the OA manifold for indirect coupling and stochastic resetting is four-dimensional with coordinates $(r(t), R(t), \phi(t), \Phi(t))$, where $r(t)e^{i\phi(t)}$ is the first circular moment of the phases, and only $r(t)$ and $\phi(t)$ reset (subsystem resetting). The validity of this dimensional reduction was previously established in the absence of resetting¹⁶ and still holds when resetting is included, provided that the set of initial phases also lies on the OA manifold. We numerically solve the stochastic OA dynamics and explore how the behavior depends on the cell density σ_0 and other model parameters. We also show how subsystem resetting can induce a switch in behavior when the deterministic system operates in a bistable regime. Finally, in Sec. V, we perform a slow/fast analysis of the OA dynamics when $\omega_0 = 0$ such that the OA dynamics reduces to a planar system with coordinates $(r(t), R(t))$. In the low density regime ($\sigma_0 \ll 1$), $R(t)$ varies much more slowly than the other coordinate, so we can use a separation of time scales. That is, we first treat $R(t)$ as a constant and calculate the non-equilibrium stationary state (NESS) of the probability density for r . The slow variable $R(t)$ is then determined by averaging r with respect to the NESS. If $R(0) = 0$ and the resetting rate is sufficiently small, then partial synchrony emerges as $R(t)$ slowly increases with t .

II. KURAMOTO MODEL WITH COUPLING THROUGH AN EXTERNAL MEDIUM

Consider a population of $\mathcal{M}$ limit cycle oscillators that are diffusively coupled to an external medium. Each oscillator represents a synthetically engineered bacterial cell that synthesizes two different types of autoinducer that can be exchanged with the extracellular environment via diffusion. (A single autoinducer would not support intrinsic chemical oscillations within each cell.) When the diffusing molecules leave a cell and enter the extracellular environment, their concentration is diluted by a factor $\alpha = V_{\text{int}}/V_{\text{ext}}$, which is the ratio of the cell volume to the total extracellular volume. Let $z_j(t)$, $j = 1, \dots, \mathcal{M}$, denote the complex amplitude of the j th oscillator at time t . In terms of the intracellular concentrations $u_j(t)$ and $v_j(t)$ of the two chemical species, we have $z_j(t) = u_j(t) + iv_j(t)$. The state of the environment is then represented by the complex variable $Z(t) = U(t) + iV(t)$, where $U(t)$ and $V(t)$ are the corresponding extracellular concentrations. (Note that all concentrations are defined relative to some fixed background concentration so that

the variables u_j, v_j, U, V can take negative values.) Let ω_j be the natural frequency of the j -th oscillator. The frequencies ω_j are randomly drawn from a distribution $h(\omega)$, which is taken to be an even function about a mean frequency ω_0 . Finally, introducing the dimensionless cell density $\sigma_0 = \alpha N$ and moving to a rotating frame with frequency ω_0 , the dynamics of the system can be represented by the equations^{15,16}

$$\frac{dz_j}{dt} = (\gamma_0 + i\omega_j - |z_j|^2) z_j - K(z_j - Z), \quad (2.1a)$$

$$\frac{dZ}{dt} = \frac{\sigma_0 K}{\mathcal{M}} \sum_{j=1}^{\mathcal{M}} (z_j - Z) - i\omega_0 Z, \quad (2.1b)$$

where K denotes the strength of diffusive coupling and γ_0 is a positive constant. For simplicity, we take these to be the same for both autoinducers. (In this paper, we also assume that the degradation of autoinducers within the external medium is negligible.) In the rotating frame, the frequencies ω_j are drawn from an even distribution $g(\omega) = h(\omega - \omega_0)$ with zero mean. Note that the density factor σ_0 in Eq. (2.1b) is the origin of the density-dependent collective response of bacterial QS systems. Intuitively speaking, increasing the effective coupling K should favor synchronization, whereas having a wider distribution of frequencies ω_j should favor desynchronization. These opposing effects are well known within the theory of oscillator synchronization.^{13,19}

Suppose that Eq. (2.1a) is rewritten in polar coordinates with $z_j = r_j e^{i\theta_j}$ and $Z = R e^{i\Phi}$,

$$\frac{dr_j}{dt} = (\gamma_0 - K - r_j^2) r_j + KR \cos(\Phi - \theta_j), \quad (2.2a)$$

$$\frac{d\theta_j}{dt} = \omega_j + \frac{KR}{r_j} \sin(\Phi - \theta_j). \quad (2.2b)$$

As shown in Ref. 16, if γ_0 is sufficiently large for fixed coupling K , then the amplitude r_j rapidly converges to the quasi steady-state $r_j \approx \sqrt{\gamma_0}$ for all $j = 1, \dots, N$. (Note that γ_0 and K both have units of inverse time so we have the equivalent dimensionless condition $\gamma_0/K \gg 1$.) Thus, we can effectively eliminate the dynamics of the amplitudes r_j . After performing the rescalings $r_j \rightarrow r_j/\sqrt{\gamma_0}$ and $R \rightarrow R/\sqrt{\gamma_0}$, Eq. (2.1) reduces to

$$\frac{d\theta_j}{dt} = \omega_j + KR \sin(\Phi - \theta_j), \quad (2.3a)$$

$$\frac{dZ}{dt} = \sigma_0 K (z(t) - Z(t)) - i\omega_0 Z(t), \quad (2.3b)$$

with

$$z(t) := \frac{1}{\mathcal{M}} \sum_{j=1}^{\mathcal{M}} e^{i\theta_j(t)} = r(t) e^{i\phi(t)}. \quad (2.4)$$

This is a modified version of the classical Kuramoto model, where Z is identified with the state of an external medium rather than the first circular moment $z(t)$ of the N oscillators. The latter has a geometric interpretation as the centroid of the phases with $\phi(t)$ equal

to the average phase and $r(t)$ a measure of the degree of phase-coherence. A completely incoherent state corresponds to the case $r = 0$, whereas a completely synchronized state satisfies $r = 1$.

In this paper, we consider a stochastic version of the Kuramoto model (2.3) in which each oscillator simultaneously resets to its initial phase θ_{0j} at a fixed Poisson rate λ . On the other hand, the external complex amplitude $Z(t)$ does not reset. Following Refs. 24 and 30, we distinguish this global update rule from a local resetting scheme in which the oscillators independently reset. Let T_n denote the n th resetting time with $n \geq 1$ and $T_0 = 0$. The inter-reset times $\tau_n = T_n - T_{n-1}$ are exponentially distributed with

$$\mathbb{P}[\tau_n \in [s, s + ds]] = \lambda e^{-\lambda s} ds. \quad (2.5)$$

In addition, the number of resets occurring in the time interval $[0, t]$ is given by the Poisson process

$$N(t) = n, \quad T_n \leq t < T_{n+1}, \quad \mathbb{P}[N(t) = n] = \frac{(\lambda t)^n e^{-\lambda t}}{n!}, \quad (2.6)$$

with $\mathbb{E}[N(t)] = \lambda t = \text{Var}[N(t)]$. Note that $N(t)$ is defined to be right-continuous: $N(T_n^-) = n - 1$, whereas $N(T_n) = n$.

As we have discussed elsewhere,³⁰ instantaneous global resetting can be implemented using the stochastic calculus of jump processes. In particular, introducing the differential

$$dN(t) = h(t)dt, \quad h(t) = \sum_{n \geq 1} \delta(t - T_n). \quad (2.7)$$

Equation (2.3) becomes

$$\frac{d\theta_j(t)}{dt} = \omega_j + KR(t) \sin(\Phi(t) - \theta_j(t)) + h(t)(\theta_{j,0} - \theta_j(t^-)), \quad (2.8a)$$

$$\frac{dZ}{dt} = \sigma_0 K(z(t) - Z(t)) - i\omega_0 Z(t). \quad (2.8b)$$

Integrating Eq. (2.8a) with respect to t implies that

$$\begin{aligned} \theta_j(t) &= \theta_{j,0} + \omega_j t + K \int_0^t R(s) \sin(\Phi(s) - \theta_j(s)) ds \\ &\quad + \sum_{m=1}^{N(t)} (\theta_{j,0} - \theta_j(T_m^-)). \end{aligned} \quad (2.9)$$

Setting $t = T_n$ and $t = T_n^-$, respectively, and subtracting the resulting pair of equations shows that $\theta_j(T_n) = \theta_j(T_n^-) + \theta_{j,0} - \theta_j(T_n^-) = \theta_{j,0}$. Hence, we recover instantaneous resetting. A crucial element in establishing this equivalence is the right-continuity of the Poisson process $N(t)$.

An alternative subsystem resetting protocol involves resetting the state of the external medium rather than the phase oscillators, $Z(T_n^-) \rightarrow Z_0$. (One could also consider a combination of both resetting protocols.) Equations (2.8a) and (2.8b) then become

$$\frac{d\theta_j(t)}{dt} = \omega_j + KR(t) \sin(\Phi(t) - \theta_j(t)), \quad (2.10a)$$

$$\frac{dZ}{dt} = \sigma_0 K(z(t) - Z(t)) - i\omega_0 Z(t) + h(t)(Z_0 - Z(t^-)). \quad (2.10b)$$

There are noticeable differences between Eqs. (2.8) and (2.10) with regard to their dependence on the cell density σ_0 . In the high-density

limit $\sigma_0 \rightarrow \infty$, we find that $R(t) \approx r(t)$ between resetting events. Plugging $R(t) = r(t)$ into Eqs. (2.8a) and (2.10a), then gives the classical Kuramoto with and without resetting, respectively. On the other hand, in the low density limit $\sigma_0 \rightarrow 0$ with $\omega_0 = 0$, we see that $Z(t) \approx Z_0$ in Eq. (2.10b), whereas $Z(t)$ is determined by the relatively fast dynamics of $z(t)$ in Eq. (2.8b). In both of these limits, phase resetting exhibits more complicated dynamics than resetting of the external medium. Therefore, in this paper, we focus on global phase resetting, which is also the paradigm considered in Ref. 29.

III. DERIVATION OF GLOBAL DENSITY EQUATION

A well-known method for analyzing the classical Kuramoto model is to consider the PDE for the joint phase and natural frequency density in the continuum limit $\mathcal{M} \rightarrow \infty$.^{20,33–36} In most cases, the density equation is simply written down in the form of a Liouville equation, or a Fokker–Planck equation, in the presence of phase fluctuations. However, there is an implicit assumption that the continuum or mean field limit exists. The latter has been proven rigorously by treating the noisy Kuramoto model as a system of Brownian particles on an $\mathcal{M}$ -torus,³⁷ and this result has recently been extended to a wider class of interacting particle systems on the torus.^{38,39} In a previous paper, we exploited the connection between the classical Kuramoto model and systems of interacting Brownian particles on a torus to incorporate the effects of stochastic resetting into the corresponding continuum model.³⁰ In this section, we use an analogous construction to incorporate global resetting into the density equation for the Kuramoto model coupled via an external medium.

Introduce the global density or empirical measure

$$\rho^{(\mathcal{M})}(\theta, t, \omega) \equiv \frac{1}{\mathcal{M}} \sum_{j=1}^{\mathcal{M}} \rho_j(\theta, t, \omega) = \frac{1}{\mathcal{M}} \sum_{j=1}^{\mathcal{M}} \delta(\theta - \theta_j(t)) \delta(\omega - \omega_j). \quad (3.11)$$

Consider an arbitrary smooth test function $f: [0, 2\pi] \times \mathbb{R} \rightarrow \mathbb{R}$. Setting

$$\frac{1}{\mathcal{M}} \sum_{j=1}^{\mathcal{M}} f(\theta_j(t), \omega_j) = \int_0^{2\pi} d\theta \int_{\mathbb{R}} d\omega \rho^{(\mathcal{M})}(\theta, t, \omega) f(\theta, \omega), \quad (3.12)$$

we have

$$\int_0^{2\pi} d\theta \int_{\mathbb{R}} d\omega f(\theta, \omega) \frac{\partial \rho^{(\mathcal{M})}(\theta, t, \omega)}{\partial t} = \frac{1}{\mathcal{M}} \sum_{j=1}^{\mathcal{M}} \frac{df(\theta_j(t), \omega_j)}{dt}, \quad (3.13)$$

with

$$\begin{aligned} \frac{df}{dt}(\theta_j(t), \omega_j) &= \frac{\partial f}{\partial \theta_j}(\theta_j(t), \omega_j) \frac{d\theta_j(t)}{dt} \\ &= \left[\omega_j + KR(t) \sin(\Phi(t) - \theta_j(t)) \right] \frac{\partial f}{\partial \theta_j}(\theta_j(t), \omega_j) \\ &\quad + h(t) \left[f(\theta_{j,0}, \omega_j) - f(\theta_j(t^-), \omega_j) \right]. \end{aligned} \quad (3.14)$$

Using the definition of $\rho^{(\mathcal{M})}$, we have

$$\begin{aligned} & \int_0^{2\pi} d\theta \int_{\mathbb{R}} d\omega f(\theta, \omega) \frac{\partial \rho^{(\mathcal{M})}(\theta, t, \omega)}{\partial t} \\ &= \int_0^{2\pi} d\theta \int_{\mathbb{R}} d\omega \rho^{(\mathcal{M})}(\theta, t, \omega) \left[\mathcal{V}[\theta, \omega, Z] \partial_{\theta} f(\theta, \omega) \right] \\ &+ h(t) \int_0^{2\pi} d\theta \int_{\mathbb{R}} d\omega [\rho_0^{(\mathcal{M})}(\theta, \omega) - \rho(\theta, \omega, t^-)] f(\theta, \omega), \end{aligned} \quad (3.15)$$

where

$$\mathcal{V}[\theta, \omega, Z] = \omega + \frac{K}{2i} (Z(t)e^{-i\theta} - Z^*(t)e^{i\theta}) \quad (3.17)$$

and

$$\rho_0^{(\mathcal{M})}(\theta, \omega) = \frac{1}{\mathcal{M}} \sum_{j=1}^{\mathcal{M}} \delta(\theta - \theta_{0,j}) \delta(\omega - \omega_j). \quad (3.18)$$

Integrating by parts the various terms involving derivatives of f and using the fact that f is arbitrary yields the following equation for ρ (in the weak sense):

$$\begin{aligned} \frac{\partial \rho^{(\mathcal{M})}(\theta, t, \omega)}{\partial t} &= -\frac{\partial}{\partial \theta} \left[\rho^{(\mathcal{M})}(\theta, t, \omega) \mathcal{V}[\theta, \omega, Z] \right] \\ &+ h(t) \left[\rho_0^{(\mathcal{M})}(\theta, \omega) - \rho^{(\mathcal{M})}(\theta, t^-, \omega) \right], \end{aligned} \quad (3.19)$$

where $Z(t)$ evolves according to Eq. (2.8b) with

$$z(t) = \int_{-\infty}^{\infty} \int_0^{2\pi} \rho^{(\mathcal{M})}(\theta, \omega, t) e^{i\theta} d\theta d\omega. \quad (3.20)$$

If $\mathcal{M}$ is finite, then Eq. (3.19) is highly singular since the solution is a sum of Dirac delta functions. There are two ways to obtain a non-singular PDE. The first is to take expectations with respect to the Poisson process $N(t)$ that generates the sequence of resetting times. However, as we show below, this leads to a moment closure problem even in the continuum limit. The second is to take the continuum limit but keep stochastic resetting, which is the approach we take in Sec. IV. In order to understand how a smooth density can arise in the continuum limit, consider an arbitrary smooth function $f(\omega)$ and the identities

$$\frac{1}{\mathcal{M}} \sum_{j=1}^{\mathcal{M}} f(\omega_j) = \int_{\mathbb{R}} \rho^{(\mathcal{M})}(\omega) f(\omega) d\omega,$$

where $\rho^{(\mathcal{M})}(\omega) = \mathcal{M}^{-1} \sum_{j=1}^{\mathcal{M}} \delta(\omega - \omega_j)$ and

$$\lim_{\mathcal{M} \rightarrow \infty} \frac{1}{\mathcal{M}} \sum_{j=1}^{\mathcal{M}} f(\omega_j) = \int_{\mathbb{R}} g(\omega) f(\omega) d\omega.$$

It follows from the arbitrariness of f that

$$\lim_{\mathcal{M} \rightarrow \infty} \rho^{(\mathcal{M})}(\omega) = g(\omega)$$

in the sense of distributions.

In order to take expectations of Eq. (3.19) with respect to $N(t)$, we use the following results. Since $\rho^{(\mathcal{M})}(\theta, T_n^-, \omega)$ only depends on jump times prior to the n th resetting, we have

$$\begin{aligned} & \mathbb{E} \left[h(t) \left[\rho_0^{(\mathcal{M})}(\theta, \omega) - \rho^{(\mathcal{M})}(\theta, t^-, \omega) \right] \right] \\ &= \mathbb{E} \left[\rho_0^{(\mathcal{M})}(\theta, \omega) - \rho^{(\mathcal{M})}(\theta, t^-, \omega) \right] \mathbb{E}[h(t)] \\ &= \left[\rho_0^{(\mathcal{M})}(\theta, \omega) - p^{(\mathcal{M})}(\theta, t, \omega) \right] \mathbb{E}[h(t)], \end{aligned} \quad (3.21)$$

where $p^{(\mathcal{M})}(\theta, t, \omega) = \mathbb{E}[\rho^{(\mathcal{M})}(\theta, t, \omega)]$. Moreover, $N(t) - N(\tau) = \int_{\tau}^t dN(s)$ and

$$\int_{\tau}^t \mathbb{E}[dN(s)] = \mathbb{E}[N(t) - N(\tau)] = \lambda(t - \tau). \quad (3.22)$$

That is,

$$\mathbb{E}[h(t)] dt \equiv \mathbb{E}[dN(t)] = \lambda dt. \quad (3.23)$$

Hence, substituting for $\mathcal{V}$ using Eq. (3.17) and then taking expectations of Eq. (3.19) gives

$$\begin{aligned} \frac{\partial p^{(\mathcal{M})}(\theta, t, \omega)}{\partial t} &= \lambda [\rho_0^{(\mathcal{M})}(\theta, \omega) - p^{(\mathcal{M})}(\theta, t, \omega)] \\ &- \frac{\partial}{\partial \theta} \left\{ \omega p^{(\mathcal{M})}(\theta, t, \omega) + \frac{K}{2i} e^{-i\theta} \mathbb{E}[\rho^{(\mathcal{M})}(\theta, t, \omega) Z(t)] \right. \\ &\left. - \frac{K}{2i} e^{i\theta} \mathbb{E}[\rho^{(\mathcal{M})}(\theta, t, \omega) \bar{Z}(t)] \right\}. \end{aligned} \quad (3.24a)$$

On the other hand, taking expectations of Eqs. (2.8b) and (3.20), we have

$$\frac{dZ}{dt} = \sigma_0 K (\mathbb{E}[z(t)] - Z(t)) - i\omega_0 Z(t), \quad (3.24b)$$

where $Z(t) = \mathbb{E}[Z(t)]$ and

$$\mathbb{E}[z(t)] = \int_{-\infty}^{\infty} \int_0^{2\pi} p^{(\mathcal{M})}(\theta, \omega, t) e^{i\theta} d\theta d\omega. \quad (3.24c)$$

Equation (3.24a) does not form a closed system since the one-point density $p^{(\mathcal{M})}(\theta, t, \omega) = \mathbb{E}[\rho^{(\mathcal{M})}(\theta, t, \omega)]$ couples to the two-point density $\mathbb{E}[\rho^{(\mathcal{M})}(\theta, t, \omega) Z(t)]$ and its complex conjugate. We are using the fact that $Z(t)$ is a linear functional of $\rho^{(\mathcal{M})}(\theta, t, \omega)$, which follows from Eqs. (2.8b) and (3.20). Unfortunately, we cannot appeal to a mean field limit in which $\mathbb{E}[\rho^{(\mathcal{M})}(\theta, t, \omega) Z(t)] \rightarrow p^{(\mathcal{M})}(\theta, t, \omega) Z(t)$, which means that we cannot eliminate the stochasticity by averaging. Hence, we follow the alternative approach to obtaining a nonsingular PDE, which is based on taking the continuum limit of Eq. (3.19). This gives the stochastic PDE

$$\begin{aligned} \frac{\partial \rho(\theta, t, \omega)}{\partial t} &= -\frac{\partial}{\partial \theta} \left[\rho(\theta, t, \omega) \mathcal{V}[\theta, \omega, Z] \right] \\ &+ h(t) [\rho_0(\theta, \omega) - \rho(\theta, t^-, \omega)], \end{aligned} \quad (3.25)$$

with $\rho(\theta, \omega) = \lim_{\mathcal{M} \rightarrow \infty} \rho^{(\mathcal{M})}(\theta, \omega)$, etc.

IV. STOCHASTIC DYNAMICS ON THE OTT-ANTONSEN MANIFOLD

The next step in our analysis is to project Eq. (3.25) onto the OA manifold along analogous lines to the deterministic model of Ref. 16. First, note that the continuum density satisfies the normalization condition

$$\int_0^{2\pi} \rho(\theta, \omega, t) d\theta = \lim_{\mathcal{M} \rightarrow \infty} \frac{1}{\mathcal{M}} \sum_{j=1}^{\mathcal{M}} \delta(\omega - \omega_j) = g(\omega). \quad (4.1)$$

Hence, ρ can be expanded as a Fourier series in θ ,

$$\rho(\theta, \omega, t) = \frac{g(\omega)}{2\pi} \left(1 + \sum_{n=1}^{\infty} [\rho_n(\omega, t) e^{in\theta} + \text{c. c.}] \right). \quad (4.2)$$

An analogous Fourier expansion holds for the reset density,

$$\rho_0(\theta, \omega) = \frac{g(\omega)}{2\pi} \left(1 + \sum_{n=1}^{\infty} [\rho_{0,n}(\omega) e^{in\theta} + \text{c. c.}] \right). \quad (4.3)$$

As it stands, solving the initial value problem for ρ involves solving an infinite hierarchy of equations for the coefficients ρ_n . However, a major simplification can be achieved by applying the OA Ansatz $\rho_n = \eta^n$ so that

$$\rho(\theta, \omega, t) = \frac{g(\omega)}{2\pi} \left(1 + \sum_{n=1}^{\infty} [\eta^n(\omega, t) e^{in\theta} + \text{c. c.}] \right). \quad (4.4)$$

Numerical simulations of the full system in the absence of resetting indicate that the dynamics is captured by such a reduction in the sense that the OA manifold is an attractor.¹⁶ In order to project the dynamics onto the attracting OA manifold in the presence of resetting, we require that the reset density also lies on the OA manifold,

$$\rho_0(\theta, \omega) = \frac{g(\omega)}{2\pi} \left(1 + \sum_{n=1}^{\infty} [r_0^n e^{in\theta} + \text{c. c.}] \right). \quad (4.5)$$

For simplicity, the OA coefficient r_0 is taken to be real and independent of ω . It then follows that $\rho_0(\theta, \omega) = g(\omega)p_0(\theta)$, where $p_0(\theta)$ is the ω -independent distribution of initial phases,

$$\begin{aligned} p_0(\theta) &\equiv \lim_{\mathcal{M} \rightarrow \infty} \frac{1}{\mathcal{M}} \sum_{j=1}^{\mathcal{M}} \delta(\theta - \theta_{0,j}) \\ &= \frac{1}{2\pi} \left(-1 + \sum_{n=0}^{\infty} [r_0^n e^{in\theta} + \text{c. c.}] \right) \\ &= \frac{1}{2\pi} \left(-1 + \frac{1}{1 - r_0 e^{i\theta}} + \frac{1}{1 - r_0 e^{-i\theta}} \right) \\ &= \frac{1}{2\pi} \left(-1 + \frac{2 - 2r_0 \cos \theta}{1 - 2r_0 \cos \theta + r_0^2} \right) \\ &= \frac{1}{2\pi} \left(\frac{1 - r_0^2}{1 - 2r_0 \cos \theta + r_0^2} \right). \end{aligned} \quad (4.6) \quad (4.7)$$

Implementing such a resetting density is non-trivial both numerically and experimentally. Therefore, in the examples, we will consider the special case $r_0 = 1$ for which

$$p_0(\theta) = \delta(\theta). \quad (4.8)$$

In terms of dynamical QS with $z_j(t) = u_j(t) + iv_j(t) \approx \sqrt{\gamma_0} e^{i\theta_j(t)}$, we have the resetting rule $\theta_j(t) \rightarrow 0$ for all j , which is equivalent to rapidly resetting the concentration of the second chemical component within each cell.

Substituting Eqs. (4.4) and (4.5) into the continuity equation (3.19) gives

$$\left[\frac{\partial \eta}{\partial t} + i\omega \eta + \frac{K}{2} (Z\eta^2 - Z^*) \right] = h(t)[r_0 - \eta(\omega, t^-)], \quad (4.9)$$

with $Z(t)$ evolving according to Eq. (2.8b). In particular,

$$\eta(\omega, T_n^-) \rightarrow r_0 \quad (4.10)$$

at the reset times T_n , $n \geq 1$. Moreover, Eq. (3.20) implies that on the OA manifold

$$z(t) = \int_{-\infty}^{\infty} g(\omega) \eta^*(\omega, t) d\omega. \quad (4.11)$$

Further simplification can be achieved in the case of a Lorentzian distribution of frequencies

$$g(\omega) = \frac{1}{\pi} \frac{\Delta}{\omega^2 + \Delta^2}. \quad (4.12)$$

Substituting (4.12) into Eq. (4.11) and evaluating the resulting contour integral establishes that

$$z(t) = \eta^*(-i\Delta, t).$$

The resetting rule then becomes $z(t) \rightarrow r_0$. Hence, setting $\omega = -i\Delta$ in Eq. (4.9) and taking the complex conjugate yields the following equations for z and Z between resetting events:

$$\frac{dz}{dt} + \Delta z + \frac{K}{2} (Z^* z^2 - Z) = h(t)[r_0 - z(t^-)], \quad (4.13a)$$

$$\frac{dZ}{dt} = \sigma_0 K (z - Z) - i\omega_0 Z. \quad (4.13b)$$

Introducing the planar coordinates $Z(t) = R(t)e^{i\Phi(t)}$ and $z(t) = r(t)e^{i\phi(t)}$ gives

$$\begin{aligned} \frac{dr}{dt} + ir \frac{d\phi}{dt} + \Delta r + \frac{KR}{2} (r^2 e^{i[\phi - \Phi]} - e^{-i[\phi - \Phi]}) \\ = h(t) [r_0 - r(t^-) e^{i\phi(t^-)}] \end{aligned} \quad (4.14a)$$

and

$$\frac{dR}{dt} + iR \frac{d\Phi}{dt} = \sigma_0 K (r e^{i[\phi - \Phi]} - R) - i\omega_0 R. \quad (4.14b)$$

Equating real and imaginary parts yields

$$\frac{dr}{dt} = -\Delta r + \frac{KR \cos[\Phi - \phi]}{2} (1 - r^2) + h(t)[r_0 - r(t^-)], \quad (4.15a)$$

$$\frac{dR}{dt} = \sigma_0 K r \cos[\Phi - \phi] - \sigma_0 K R \quad (4.15b)$$

and

$$r \frac{d\phi}{dt} = \frac{KR \sin(\Phi - \phi)}{2} (1 + r^2) - h(t)\phi(t^-), \quad (4.15c)$$

$$R \frac{d\Phi}{dt} = -\sigma_0 K r \sin(\Phi - \phi) - \omega_0 R. \quad (4.15d)$$

Equation (4.15) form a system of stochastic differential equations on a four-dimensional OA manifold with coordinates $(r(t), R(t), \phi(t), \Phi(t))$. Each realization of the stochastic dynamics involves a random sequence of resetting times T_n generated from a Poisson process $N(t)$ with $\mathbb{E}[N(t)] = \lambda t$ and $h(t) = \sum_{n \geq 1} \delta(t - T_n)$. Under the additional constraints $\omega_0 = 0$ and $\phi(0) = \Phi(0) = 0$, we have $\phi(t) = \Phi(t)$ for all t . Equations (4.15a) and (4.15b) then reduce to the planar dynamical system

$$\frac{dr}{dt} = -\Delta r + \frac{KR}{2} (1 - r^2) + h(t)[r_0 - r(t^-)], \quad (4.16a)$$

$$\frac{dR}{dt} = \sigma_0 K [r - R]. \quad (4.16b)$$

In the absence of resetting ($h(t) = 0$), Eq. (4.16) have the fixed points $(\bar{r}, \bar{R})$ with $\bar{R} = \bar{r}$ and $\bar{r}(K - 2\Delta - K\bar{r}^2) = 0$. The latter condition is identical to the fixed point condition obtained for the classical Kuramoto model. That is, a stable incoherent state $\bar{r} = 0$ loses stability at the critical coupling $K_c = 2\Delta$ and a stable partially coherent state coexist when $K > K_c$. The more general deterministic dynamics on the OA manifold for $\omega_0 \neq 0$ and the associated phase diagrams were analyzed extensively in Ref. 16.

Example plots of solutions to Eqs. (4.16a) and (4.16b) with resetting are shown in Figs. 3 and 4. We use the same random reset times in each plot but vary σ_0 and K while the other parameters are fixed. In particular, $r_0 = 1$ and $R(0) = 0$. Figure 4 shows the case $K < K_c$ for which the steady-state $\bar{r} = 0 < r_0$ in the absence

of resetting. In the high density regime ($\sigma_0 K \gg \Delta$), we see that $R(t) \approx r(t)$ between resetting events and $R(t) \rightarrow r_0$ almost instantaneously following each reset. Reducing $\sigma_0 K$ to values of $O(\Delta)$ significantly alters the dynamics of $R(t)$ but only weakly modifies $r(t)$. On the other hand, in the low density regime $\sigma_0 K \ll \Delta$, there is a more significant decrease in $r(t)$ following each resetting event due to the fact that $R(t)$ slowly increases from its initial value $R(0) = 0$. The latter effect is particularly noticeable when $K > K_c$, see Fig. 4.

If $\omega_0 \neq 0$, then it is necessary to solve the full system of Eq. (4.15). In the absence of resetting, one finds numerically that the low density regime exhibits bistability between either (i) an incoherent state and a coherent state or (ii) two coherent states with different amplitudes R .¹⁶ Such bistability persists in the presence of resetting. However, in certain cases, the stochasticity due to subsystem resetting can induce a state transition, which is illustrated in Fig. 5. We choose parameter values that lie in one of the bistability regions displayed in Fig. 1 of Ref. 16. Figure 5 shows noise-induced transitions between two coherent states with $R \approx 0.8$ and $R \approx 0.1$. Meanwhile, the environmental phase varies as $\Phi(t) \approx \omega_0 t$, whereas $\phi(t)$ resets to zero. The direction of the transition depends on the initial condition $R(0)$.

The noise-induced transitions shown in Fig. 5 are significant in the sense that the underlying mechanism is based on subsystem resetting alone. The standard paradigm is to consider how stochastic resetting affects noise-induced escape or first passage time events that are driven by an additional external noise source such as Brownian motion.²⁸ Here, there are no additional sources of noise. The crucial observation is that only a subset of the variables, namely, $(r(t), \phi(t))$, are reset. If all of the variables reset to their initial conditions, then there would be no mechanism for the system to jump from one basin of attraction to another. On the other hand, under subsystem resetting, we can interpret the state $(r_0, R(T_n), \phi_0, \Phi(T_n))$ immediately following the n th resetting event as a new initial condition.

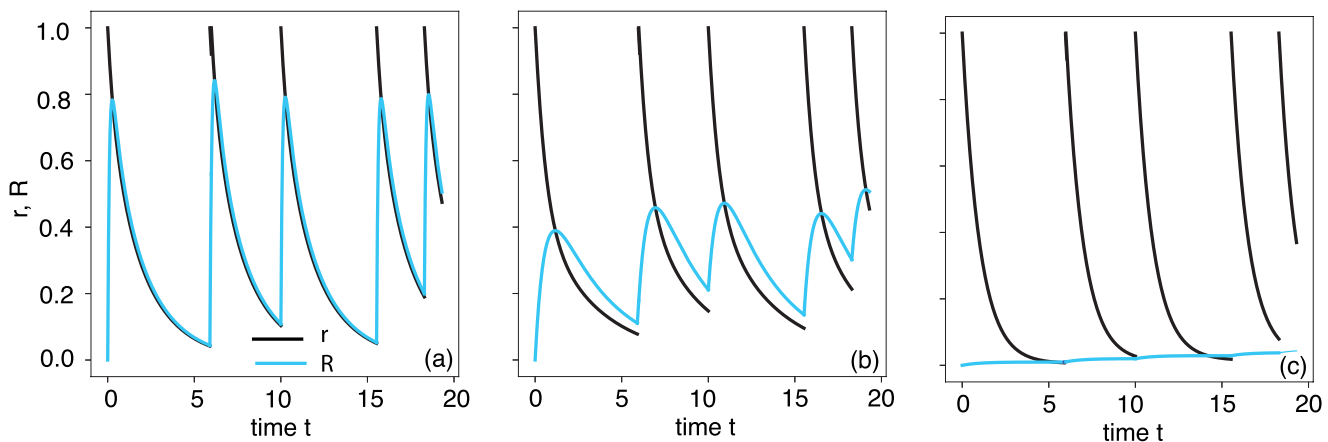


FIG. 3. Planar dynamics (4.16) on the OA manifold with resetting for $\omega_0 = 0$ and different values of the density: (a) $\sigma_0 K = 10$, (b) $\sigma_0 K = 1$, (c) $\sigma_0 K = 0.01$. Other parameters are $r_0 = 1.0$, $\lambda = 0.25$, $\Delta = 1$, and $K = 1$. Black (dark) curves show $r(t)$ and blue (light) curves show $R(t)$.

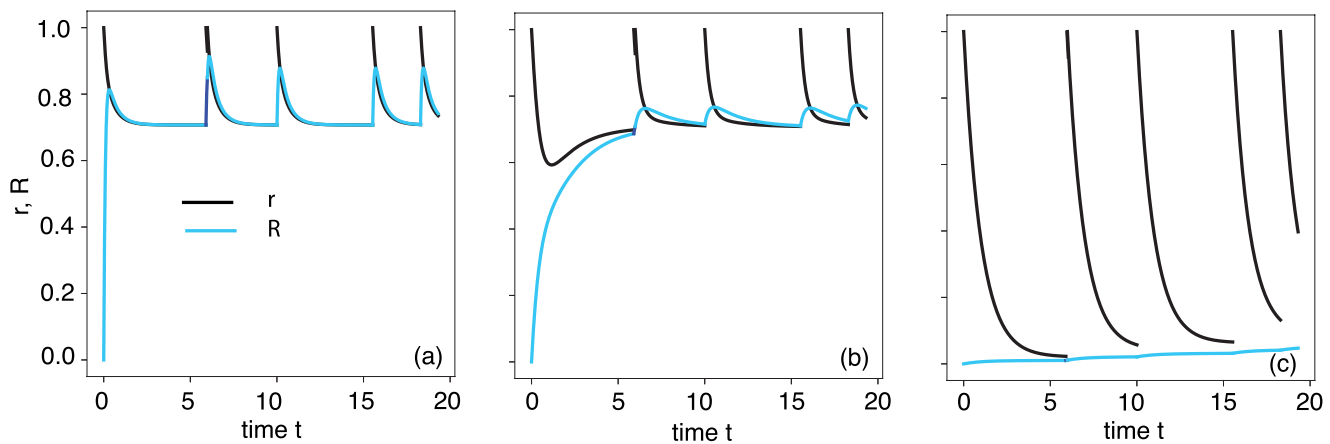


FIG. 4. Same as Fig. 4 except that $K = 10$.

V. SLOW/FAST ANALYSIS OF THE OA DYNAMICS

Further analysis can be performed on the planar system (4.16) in the low density regime using a slow/fast analysis.

A. Low density regime

In the low density limit $\sigma_0 \rightarrow 0^+$, the external amplitude $R(t)$ is a slow variable, whereas the order parameter $r(t)$ is a fast variable. [An analogous separation of time scales would not be possible if the external medium reset rather than the phase oscillators, since $R(t)$ would still have a fast dynamical component due to resetting.] As a first approximation, we can treat the factor R appearing in Eq. (4.16a) as a constant R_0 so that we obtain

$$\frac{dr}{dt} = -f(r) + h(t)[r_0 - r(t^-)], \quad (5.1)$$

with

$$f(r) = \Delta r - \frac{KR_0}{2}(1 - r^2) = \frac{KR_0}{2}(r^2 + 2\Lambda r - 1), \quad \Lambda = \frac{\Delta}{KR_0}. \quad (5.2)$$

The probability density $q(r, t)$ corresponding to a piecewise deterministic equation of the form (5.1) satisfies the PDE

$$\frac{\partial q(r, t)}{\partial t} = \frac{\partial [f(r)q(r, t)]}{\partial r} - \lambda q(r, t) + \lambda \delta(r - r_0). \quad (5.3)$$

There exists a unique nonequilibrium stationary state (NESS), $q^*(r) = \lim_{t \rightarrow \infty} q(r, t)$ with

$$f(r) \frac{dq^*(r)}{dr} + (f'(r) - \lambda)q^*(r) = -\lambda \delta(r - r_0). \quad (5.4)$$

Solving Eq. (5.4) on either side of r_0 gives

$$q^*(r) = \begin{cases} \frac{A_-}{F(r)} & r < r_0 \\ \frac{A_+}{F(r)} & r > r_0 \end{cases}, \quad (5.5)$$

where $F(r)$ is the integrating factor

$$F(r) := \exp\left(\int \frac{f'(s) - \lambda}{f(s)} ds\right) = f(r) \exp\left(-\int \frac{\lambda}{f(s)} ds\right). \quad (5.6)$$

Second, integrating Eq. (5.4) over the interval $[r_0 - \epsilon, r_0 + \epsilon]$ and taking the limit $\epsilon \rightarrow 0^+$ implies that

$$A_+ - A_- = -\frac{\lambda F(r_0)}{f(r_0)}. \quad (5.7)$$

This equation together with the normalization condition

$$\int_0^1 q^*(r) dr = 1 \quad (5.8)$$

determine the unknown coefficients $A_{\pm}$.

First, consider the case of no resetting. Factorizing $f(r)$, we have the deterministic equation

$$\frac{dr}{dt} = -\frac{KR_0}{2}(r - r_+)(r - r_-), \quad (5.9)$$

with

$$r_{\pm} = -\Lambda \pm \sqrt{\Lambda^2 + 1}. \quad (5.10)$$

Since $r_- < 0$ and $0 < r_+ < 1$, it follows that there is a unique fixed point, which is stable, so that $r(t) \rightarrow r_+$ as $t \rightarrow \infty$. Including the resetting condition $r(t) \rightarrow r_0$ then implies that $r(t) \in [r_0, r_+]$ if $r_0 < r_+$ and $r(t) \in [r_+, r_0]$ when $r_0 > r_+$. Substituting Eq. (5.2) into Eq. (5.6) with

$$\begin{aligned} \int \frac{ds}{f(s)} &= \frac{1}{KR_0 \sqrt{\Lambda^2 + 1}} \int \left[\frac{ds}{s - r_+} - \frac{ds}{s - r_-} \right] \\ &= \frac{1}{KR_0 \sqrt{\Lambda^2 + 1}} [\ln |r - r_+| - \ln(r - r_-)], \end{aligned} \quad (5.11)$$

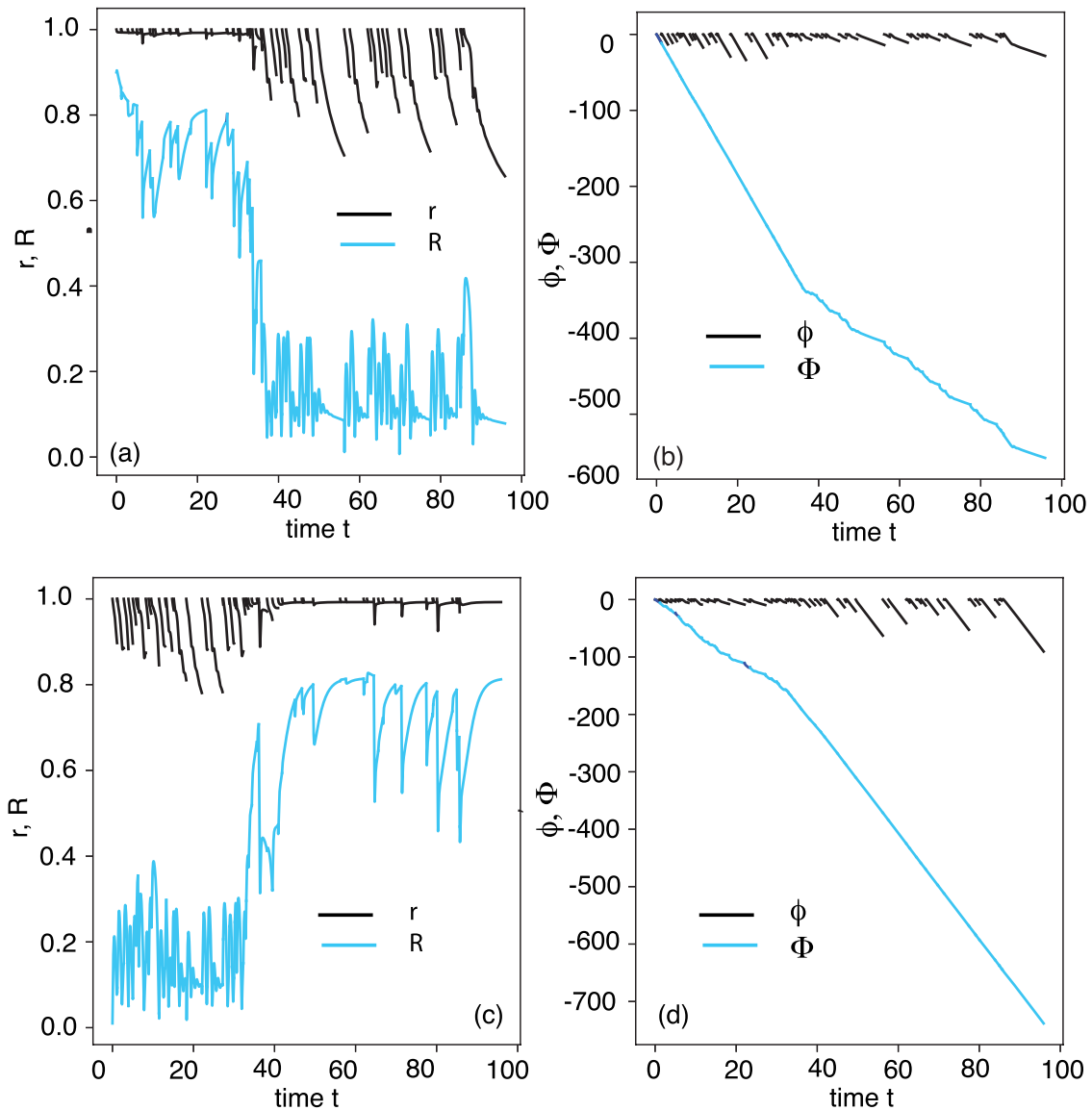


FIG. 5. Noise-induced transition between a pair of coherent states for the full OA system given by Eqs. (4.15a)–(4.15d) with $r_0 = 1$. Other parameter values are $\omega_0 = 10$, $\Delta = 0.1$, $K = 20$, $\lambda = 0.5$, and $\sigma_0 = 0.05$. (a) and (c) Plots of $r(t)$ (dark curves) and $R(t)$ (light curves) as a function of t for $R(0) = 0.1$ and $R(0) = 0.9$, respectively. (b) and (d) Corresponding plots of $\phi(t)$ (dark curves) and $\Phi(t)$ (light curves).

we obtain the result

$$F(r) = \frac{KR_0}{2} |r - r_+|^{1-\beta} (r - r_-)^{1+\beta} \quad (5.12)$$

with

$$\beta = \frac{\lambda}{KR_0 \sqrt{\Delta^2 + 1}}. \quad (5.13)$$

We then have two distinct cases. If $r_0 < r_+$, then $A_- = 0$, whereas if $r_0 > r_+$ then $A_+ = 0$. Hence, the solution in either case can be

written as

$$q^*(r; R_0) = \frac{2\lambda}{KR_0} \left(\left| \frac{r - r_+}{r_0 - r_+} \right| \right)^{\beta-1} \left(\frac{r - r_-}{r_0 - r_-} \right)^{-1-\beta} \times \frac{1}{|r_+ - r_0|(r_0 - r_-)}, \quad r \in [r_{\min}, r_{\max}], \quad (5.14)$$

with $r_{\min} = \min\{r_0, r_+\}$ and $r_{\max} = \max\{r_0, r_+\}$. Note that q^* is parameterized by R_0 . Using the identities

$$\frac{d}{dr} \left(\frac{r-r_+}{r-r_-} \right)^\beta = \frac{\beta(r_+-r_-)}{(r-r_+)(r-r_-)} \left(\frac{r-r_+}{r-r_-} \right)^\beta \quad (5.15)$$

and

$$\beta(r_+-r_-) = 2\sqrt{1+\Lambda^2} \frac{\lambda}{KR_0\sqrt{1+\Lambda^2}} = \frac{2\lambda}{KR_0}, \quad (5.16)$$

we see that for $r_0 > r_+$,

$$q^*(r; R_0) = \left(\frac{r_0-r_-}{r_0-r_+} \right)^\beta \frac{d}{dr} \left(\frac{r-r_+}{r-r_-} \right)^\beta \quad (5.17)$$

and hence,

$$\int_{r_+}^{r_0} q^*(r; R_0) dr = 1. \quad (5.18)$$

The same normalization condition holds when $r_0 < r_+$. It also follows from Eq. (5.14) that $q^*(r)$ is singular at $r = r_+$ when $\beta < 1$, which is equivalent to the condition

$$\lambda < \lambda_c = KR_0\sqrt{1+\Lambda^2}. \quad (5.19)$$

This is illustrated in Fig. 6, which shows example plots of the NESS $q^*(x)$ for various values of R_0 and λ with $r_0 = 1$. (In this case, $r_+ < r_0$.) Note that if $R_0 \ll 1$ then the NESS $q^*(r)$ is localized around 0^+ indicating that the oscillators are in an incoherent state. This is consistent with the behavior shown in Fig. 4.

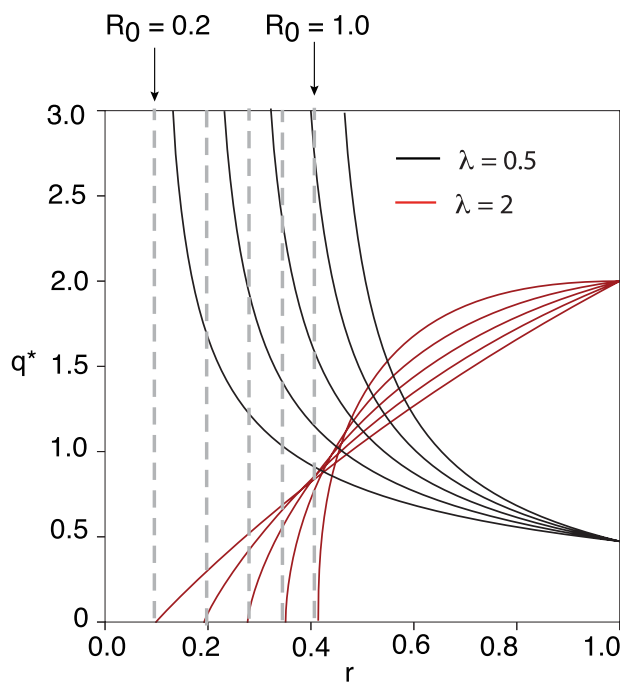


FIG. 6. Plots of the NESS $q^*(r)$ given by Eq. (5.14) as a function of the order parameter r for different values of R_0 and $r_0 = 1.0$ for $R_0 = 0.2, 0.4, 0.6, 0.8, 1.0$ and $\lambda = 0.5$ (black curves) or $\lambda = 2$ (red curves). Other parameters are $\Delta = 1$ and $K = 1$.

So far, we have taken the external amplitude to be fixed at a value R_0 . However, we can use the NESS for r to obtain an approximate solution for the slow variable $R(t)$ by applying an averaging theorem to Eq. (4.16b) and assuming that the stochastic dynamics of $r(t)$ is ergodic. More specifically, we replace Eq. (4.16b) by the averaged system

$$\frac{dR}{d\tau} = \mathbb{E}[r|R] - R, \quad (5.20)$$

where $\tau = \epsilon t$, $\epsilon = \sigma_0 K \ll 1$, and

$$\mathbb{E}[r|R_0] = \lim_{T \rightarrow \infty} \frac{1}{T} \int_0^T r(t) dt = \int_0^1 q^*(r; R_0) r dr. \quad (5.21)$$

Assuming that $R(\tau) \rightarrow R^*$ in the limit $t \rightarrow \infty$, we deduce that if the averaging theorem holds, then the asymptote R^* should approximately satisfy the self-consistency condition

$$R^* = \mathbb{E}[r|R^*]. \quad (5.22)$$

The conditional expectation can be determined using the solution (5.14). For the sake of illustration, suppose that $r_0 > r_+$. Then,

$$\begin{aligned} \mathbb{E}[r|R_0] &= \left(\frac{r_0-r_-}{r_0-r_+} \right)^\beta \int_{r_+}^{r_0} r \frac{d}{dr} \left(\frac{r-r_+}{r-r_-} \right)^\beta dr \\ &= r_0 - \left(\frac{r_0-r_-}{r_0-r_+} \right)^\beta \int_{r_+}^{r_0} \left(\frac{r-r_+}{r-r_-} \right)^\beta dr, \end{aligned} \quad (5.23)$$

after integrating by parts. [Recall from Eq. (5.10) that $r_\pm$ depends on R_0 .] The remaining integral can be evaluated using quadrature. Example plots of $\mathbb{E}[r|R_0]$ as a function of R_0 are shown in Fig. 7 for various resetting rates λ . The intercept of each curve with the straight line $y = R_0$ then determines the asymptote R^* for each λ . For example, we find that $\mathbb{E}[r|R^*] \approx 0.444$ for $\lambda = 0.5$, $\mathbb{E}[r|R^*] \approx 0.589$ for $\lambda = 1$, $\mathbb{E}[r|R^*] \approx 0.669$ for $\lambda = 1.5$, and $\mathbb{E}[r|R^*] \approx 0.721$ for $\lambda = 2$. We can confirm the validity of the slow/fast

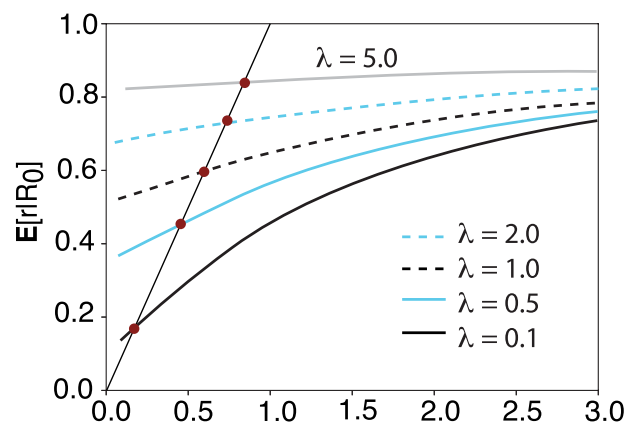


FIG. 7. Slow/fast analysis in the low density regime. Example plots of the average $\mathbb{E}[r|R_0]$ as a function of R_0 are shown for various resetting rates λ . Other parameters are $R(0) = 0.1$, $\Delta = 1$, $K = 1$, $r_0 = 1$, and $\sigma_0 = 0.01$. The intercepts with the straight line $y = R_0$ determine the asymptotic values R^* according to Eq. (5.22.).

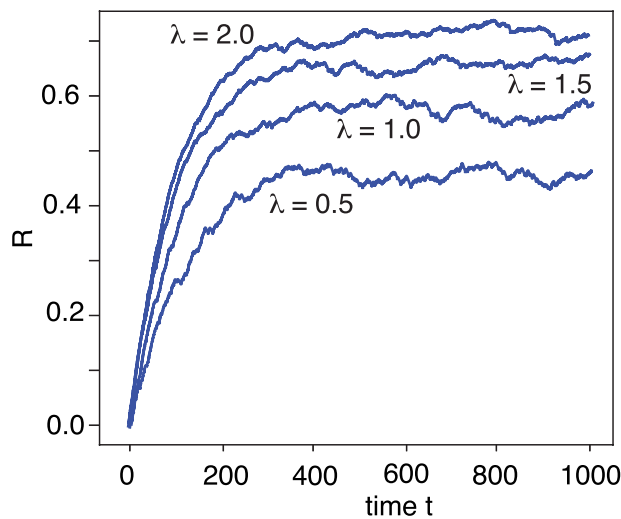


FIG. 8. Plot of the slow amplitude $R(t)$ in the low density regime for $\lambda = 0.5, 1.0, 1.5, 2.0$. Other parameters are $R(0) = 0$, $\Delta = 1$, $K = 1$, $r_0 = 1$, and $\sigma_0 = 0.01$.

analysis by numerically solving the planar system (4.16a) and (4.16b) in the low density regime. Example plots are shown in Fig. 8. It can be seen that asymptotes R^* are consistent with our analytical results.

B. High density regime

In the high density regime ($\sigma_0 K \gg \Delta$), the amplitude $R(t)$ is the fast variable and $r(t)$ is the slow variable. In particular, taking $\sigma_0 \rightarrow \infty$, we can set $R(t) = r(t)$ in Eq. (4.16a) to give Eq. (5.1) with

$$f(r) = \frac{Kr}{2}(r^2 - \Gamma), \quad \Gamma = 1 - \frac{2\Delta}{K}. \quad (5.24)$$

Equations (5.1) and (5.24) are identical in form to the stochastic dynamics of the order parameter for the classical Kuramoto model with global resetting, see Refs. 29 and 30 for further details.

C. Numerical simulations of the full system

So far, we have considered the effects of global phase resetting on the Kuramoto model with indirect coupling by projecting the dynamics onto an OA manifold. In Ref. 16, it was shown that in the absence of resetting, the OA dynamics captures very well the dynamics of the full system for a large but finite number of oscillators. This suggests that the OA manifold is a stable attractor of the dynamics in the mean field limit. In this paper, we take the resetting point to lie on the OA manifold, which means that the OA manifold remains invariant under resetting. However, this does not guarantee that it remains a stable attractor of the full network dynamics with resetting. One of the main predictions of our analysis (for $\omega_0 = 0$ and $K < K_c$) is that in the mean field limit and at low cell densities, the system converges to a state characterized by a constant environmental amplitude R^* that increases with the resetting rate λ , see Fig. 8. (In the absence of resetting, we have $R^* = 0$ for $K < K_c$.) Therefore, a useful check of the validity of our analysis is to determine

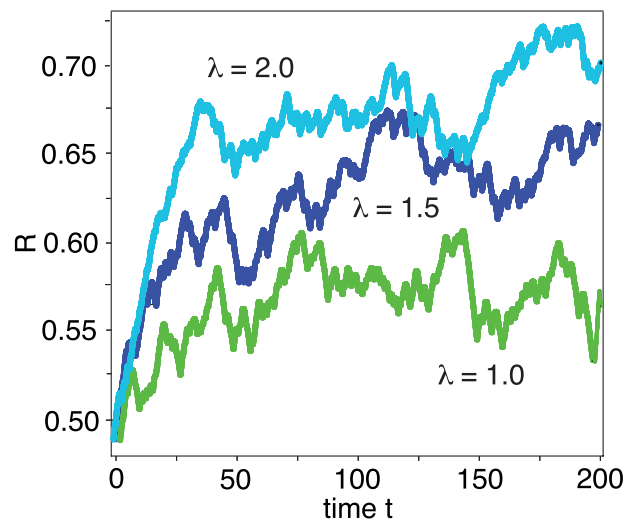


FIG. 9. Numerical simulations of the full system given by Eq. (2.3) for $N = 100$ oscillators and $\lambda = 1, 1.5, 2$. Other parameters are $R(0) = 0.5$, $\Delta = 1$, $K = 1$, $r_0 = 1$, and $\sigma_0 = 0.01$.

whether such a prediction holds for the full system (2.3). Interestingly, we find that even for a relatively small network ($N = 100$), there is good qualitative agreement between the slow/fast analysis on the OA manifold and the dynamics of the full system. This is illustrated in Fig. 9 for $N = 100$ oscillators. Although the plots are clearly more noisy than those shown in Fig. 8, the same asymptotic trends can be seen [We take $R(0) = 0.5$ in order to reach the asymptotic regime more quickly.]

VI. DISCUSSION

In this paper, we studied the dynamics of a Kuramoto model with global phase resetting and indirect coupling mediated by an external medium. We first derived a continuum equation for the population density that stochastically resets to the initial distribution of phases, see Eq. (3.25). We then projected the infinite-dimensional system onto a four-dimensional OA manifold in which only half the variables reset (subsystem resetting), see Eq. (4.15). Finally, we investigated the stochastic dynamics on the OA manifold using a combination of numerical simulations and slow/fast analysis. Similar to the case without resetting,¹⁶ we found that the OA dynamics is sensitive to the cell density parameter σ_0 . When the oscillators have zero mean frequency ($\omega_0 = 0$), the dynamics on the OA manifold reduces to the planar system (4.16). The latter interpolates between the classical Kuramoto model with global resetting^{29,30} at high densities and the slow/fast system analyzed in Sec. V at low densities. On the other hand, when $\omega_0 \neq 0$, the bistability of the deterministic model persists in the presence of resetting with the additional feature that subsystem resetting can induce a state transition.

To what extent our theoretical results concerning dynamic QS with resetting can be applied to an experimentally realizable system remains unclear. In the case of the classical Kuramoto model

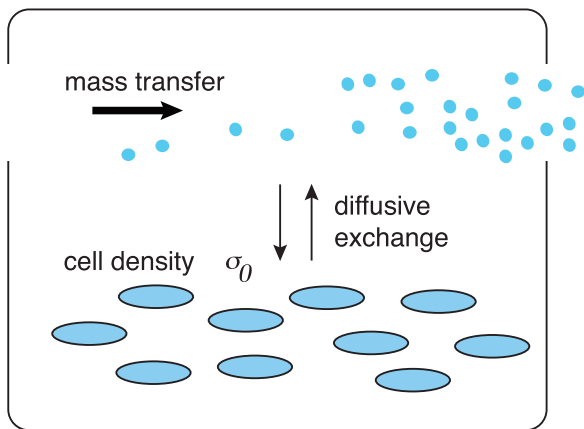


FIG. 10. A schematic illustration of mass transfer as a confounding factor in autoinducer sensing in wild-type bacterial QS.

with global resetting, one could consider a network of coupled neural oscillators that is subject to a global pulse-like electrical current stimulus.²⁹ However, identifying an analogous stimulus for intracellular autoinducer concentrations in dynamics QS is less straightforward. One alternative would be to reset the extracellular autoinducer concentrations using mass transfer away from the cell population. Interestingly, the issue of mass transfer also arises in the case of wild-type QS. That is, the “decision” of a bacterium to alter behavior when a critical population density has been reached is not based on perfect information. In particular, mass transfer can modify the autoinducer concentration that acts as a proxy for cell density, see Fig. 10. This observation has led to the proposal that QS is in fact diffusion sensing (DS).⁴⁰ That is, the function of secreted autoinducers is to determine whether or not secreted effector molecules (implementing the response) would rapidly diffuse and/or advect away from the cell. This would allow an individual bacterium to identify situations in which the probability that effector molecules disappear is low so that effector secretion is efficient. It has also been hypothesized that DS and QS have distinct evolutionary paths. A basic feature of QS is that bacteria sense their population density in order to engage in social behavior, which suggests that sensing evolved due to group benefits. On the other hand, DS proposes that sensing is a basic activity of single cells in order to detect mass-transfer constraints on effective secretion. Thus, DS assumes that sensing evolved because of a direct fitness benefit for individuals.

Beyond the particular application to the Kuramoto model of dynamic QS, our analysis reveals two general features of piecewise deterministic ODEs with subsystem resetting, see Fig. 11, that warrant further study.

(i) *Multistability.* Suppose that a deterministic system $\dot{\mathbf{x}} = \mathbf{f}(\mathbf{x})$, $\mathbf{x} \in \mathbb{R}^{\mathcal{M}}$, is multistable. This means that the long-time behavior of the system depends on the basin of attraction of the initial state $\mathbf{x}(0) = \mathbf{x}_0$. Under the total resetting rule $\mathbf{x}(T_n^-) \rightarrow \mathbf{x}_0$, for $n \geq 1$, the system remains in the same basin of attraction for all $t > 0$ and thus cannot transition to another stable state. On the other hand, if only a subset of variables reset, then

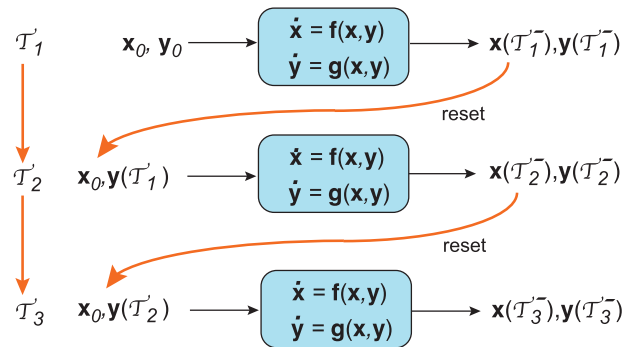


FIG. 11. Schematic diagram showing a piecewise deterministic dynamical system with subsystem resetting. Only the first few resetting events are shown.

noise-induced transitions can occur even though there are no other sources of noise. For the sake of illustration, consider the subsystem resetting rule $x_j(t) \rightarrow x_{j,0}$ for $j = 1, \dots, \mathcal{M}'$ with $\mathcal{M}' < \mathcal{M}$. After each resetting event, we have a new random initial condition $(x_{1,0}, \dots, x_{\mathcal{M}',0}, x_{\mathcal{M}'+1}(T_n), \dots, x_{\mathcal{M}}(T_n))$. This new initial condition could, in principle, lie in a different basin of attraction, thus resulting in a noise-induced transition.

(ii) *Slow/fast analysis.* Consider a slow/fast dynamical system of the form

$$\frac{d\mathbf{x}}{dt} = \mathbf{f}(\mathbf{x}, \mathbf{y}), \quad (6.1a)$$

$$\frac{d\mathbf{y}}{dt} = \epsilon \mathbf{g}(\mathbf{x}, \mathbf{y}), \quad (6.1b)$$

with $0 < \epsilon \ll 1$. Suppose that only the fast variables reset, $\mathbf{x}(T_n^-) \rightarrow \mathbf{x}_0$. In the limit $\epsilon \rightarrow 0$, we can treat the slow variables as fixed and set $\mathbf{y}(\mathbf{y}) = \mathbf{y}_0$ in Eq. (6.1a). We thus have the following reduced system with resetting:

$$\frac{d\mathbf{x}}{dt} = \mathbf{f}(\mathbf{x}, \mathbf{y}_0) + h(t)[\mathbf{x}(t^-) - \mathbf{x}_0], \quad (6.2)$$

with $\mathbf{y}_0$ treated as a set of parameters. Let $p(\mathbf{x}, t; \mathbf{y}_0)$ denote the corresponding parameterized probability density, which evolves according to

$$\frac{\partial p}{\partial t} = -\nabla_{\mathbf{x}} \cdot [\mathbf{f}p] - rp + r\delta(\mathbf{x} - \mathbf{x}_0). \quad (6.3)$$

Suppose that there exists a unique NESS $p^*(\mathbf{x}; \mathbf{y}_0)$, which is ergodic. We then define the averaged equation

$$\frac{d\mathbf{y}}{d\tau} = \mathbf{g}(\langle \mathbf{x}(\tau) \rangle, \mathbf{y}(\tau)), \quad \langle \mathbf{x}(\tau) \rangle = \int \mathbf{x} p^*(\mathbf{x}, \mathbf{y}(\tau)) d\mathbf{x}, \quad (6.4)$$

with $\tau = \epsilon t$. For a specific model, it would be necessary to determine how well the solution of the averaged system approximates the solution of the full system (6.1) over a given time interval.

AUTHOR DECLARATIONS

Conflict of Interest

The authors have no conflicts to disclose.

Author Contributions

Paul C. Bressloff: Conceptualization (lead); Formal analysis (lead); Investigation (lead); Writing – original draft (lead); Writing – review & editing (lead).

DATA AVAILABILITY

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

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