Turing instability in reaction-subdiffusion systems

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We determine the conditions for the occurrence of Turing instabilities in activator-inhibitor systems, where one component undergoes subdiffusion and the other normal diffusion. If the subdiffusing species has a nonlinear death rate, then coupling between the nonlinear kinetics and the memory effects of the non-Markovian transport process advances the Turing instability if the inhibitor subdiffuses and delays the Turing instability if the activator subdiffuses. We apply the results of our analysis to the Schnakenberg model, the Gray-Scott model, the Oregonator model of the Belousov-Zhabotinsky reaction, and the Lengyel-Epstein model of the chlorine dioxide–iodine–malonic acid reaction.

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I. INTRODUCTION

Nonequilibrium systems can display a wide variety of spatiotemporal patterns, ranging from traveling waves, fronts, and pulses to stationary and oscillatory spatial structures [1,2]. A particular well-studied class of applications consists of reaction-diffusion systems. When fluctuations can be neglected, they provide a good model for systems of particles or individuals, such as molecules or organisms, that spread spatially through diffusion and interact with each other via local kinetic laws [3–5]. In the standard reaction-diffusion picture, the effects of reaction and diffusion are separable and combine additively to influence the total spatiotemporal evolution of the concentration field of a given species, $\rho(x,t)$, a direct consequence of the Markovian nature of diffusion (Brownian motion) and reaction kinetics. Brownian motion is a memoryless transport process, and diffusion appears as a local operator in a standard reaction-diffusion equation,

$$\frac{\partial \rho(x,t)}{\partial t} = D\nabla^2 \rho(x,t) + f(\rho(x,t)). \tag{1}$$

As pointed out first by Turing [6], the interaction between nonlinear kinetics and diffusion can lead, under certain conditions, to an instability of the homogeneous steady state, a Turing instability, which gives rise to the formation of stationary spatial structures, Turing patterns. Turing patterns have been studied extensively for standard reaction-diffusion systems and are well understood in that context. However, little is known about the interaction between nonlinear kinetics and transport with memory and the effect of the non-Markovian nature of the dispersal process on the Turing instability. In a recent work [7] we derived general kinetic equations that extend the standard reaction-diffusion equations to reacting systems with non-Markovian spatial dispersal. We showed that strong memory effects associated with the transport process lead to the coupling of transport to reactions, in contrast to standard reaction-diffusion equations, where reaction and diffusion processes appear as separate, additive terms. In this paper we focus on studying subdiffusive transport. Not only is subdiffusion a non-Markovian process, and thus a suitable candidate for examining transport with strong memory effects [8], but also has abundant motivating experimental contexts ranging from proteins in cell membranes [9] to transport in media with obstacles or binding sites [10–12].

In [7] we carried out a general stability analysis of the homogeneous steady state of the generalized reaction-diffusion equations with subdiffusive transport and found that the Turing instability persists in the presence of subdiffusion. Moreover, we found that strong memory effects modify the Turing threshold and the characteristics of the band of unstable modes close to the threshold. In this paper we investigate in detail the interaction between the nonlinear kinetics and subdiffusive transport, and its consequence for the Turing instability for several activator-inhibitor models that display this instability in the standard diffusive regime.

II. TURING CONDITION FOR REACTION-SUBDIFFUSION SYSTEMS

In [7] we employed as our starting point a mesoscopic approach, namely, the formalism proposed by Vlad and Ross based on continuous-time random walks (CTRWs) with reactions [13], to arrive at the general kinetic equations governing the evolution of a system with reactions and spatial dispersal in the long-time and large-spatial-scale limit,

$$\frac{\partial \rho_i(x,t)}{\partial t} = R_i^+(\rho(x,t)) - R_i^-(\rho(x,t)) + \sigma_i^2\nabla^2 \times \left[\int_0^t \Theta_i(t-t')\rho_i(x,t') \times \exp\left(- \int_{t'}^t \frac{R_i^-(\rho(x,t''))}{\rho_i(x,t'')} dt'' \right) dt' \right]. \tag{2}$$

Here, $\rho_i(x,t)$ is the concentration field of species i, and the species undergo reactions, i.e., birth and death processes, with a birth rate $R_i^+(\rho(x,t)) \geq 0$ and a death rate $R_i^-(\rho(x,t)) \geq 0$, where $\rho(x,t)=(\rho_1(x,t),\rho_2(x,t),\ldots,\rho_n(x,t))$. The operator $\sigma_i^2\nabla^2$ has its origin in the spatial jump probability distri-

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bution function (PDF) $\lambda_i(x)$ of the underlying CTRW, having the form $\lambda_i(k)=1-\sigma_i^2k^2$ in conjugate Fourier space. The kernel $\Theta_i(t)$ is related to the waiting time PDF $\phi_i(t)$ of the CTRW in conjugate Laplace space through $\Theta_i(u) \equiv u\phi_i(u)/[1-\phi_i(u)]$. For subdiffusive transport, where the waiting time PDF of species i is given in conjugate Laplace space by $\phi_i(u)=1-(u\eta_i)^{\alpha_i}$, Eq. (2) can be rewritten in the following form, using fractional derivatives, as shown in the Appendix:

$$\frac{\partial \rho_i(x,t)}{\partial t} = R_i^+(\rho(x,t)) - R_i^-(\rho(x,t)) + K_{i;\alpha_i}\nabla^2 \times\left\{ \exp\left(-\int_0^t \frac{R_i^-(\rho(x,t''))}{\rho_i(x,t'')}dt'' \right) D_t^{1-\alpha_i} \times\left[\rho_i(x,t)\exp\left(\int_0^t \frac{R_i^-(\rho(x,t''))}{\rho_i(x,t'')}dt'' \right) \right] \right\}. \quad (3)$$

Here $D_t^{1-\alpha_i}$ is the Grünwald-Letnikov fractional derivative [see Eq. (A5)], and

$$K_{i;\alpha_i} = \frac{\sigma_i^2}{\eta_i^{\alpha_i}}. \quad (4)$$

is the generalized diffusion coefficient; see Ref. [8].

Recently, some confusion has arisen in the literature about the absence of cross-diffusive terms in the generalized reaction-diffusion equations (2) and (3) [14]. These equations are based on a mesoscopic approach, namely, the Vlad-Ross formalism. If reactions locally obey classical kinetic laws, as is the case in porous media for instance [15], they can be modeled at the mesoscopic level by a local birth and death description. (For a discussion of the validity of this description, see, for example, Refs. [3,16].) In other words, reactant particles are destroyed and product particles are created during a reactive event. The reaction changes the waiting times of particles, and new particles are born with zero age [7,13]. Most chemical reactions and processes in population dynamics are of this nature. Exceptions are reactions that amount simply to a change in the state or "color" of the particles [17–19], e.g., isomerization reactions. For such reactions, the particles themselves survive a reactive event and their waiting time is not changed. It is only for this class of reactions that extra cross-diffusive terms appear in the reaction-subdiffusion equations, since the particles "remember" their transport behavior prior to the reaction [14,15,20].

Before discussing the Turing instability of reaction-subdiffusion systems, we briefly summarize the main features of the Turing bifurcation in standard reaction-diffusion systems. Consider the two-species system

$$\frac{\partial \rho_1(x,t)}{\partial t} = f(\rho_1(x,t),\rho_2(x,t)) + D_1\nabla^2\rho_1(x,t), \quad (5a)$$

$$\frac{\partial \rho_2(x,t)}{\partial t} = g(\rho_1(x,t),\rho_2(x,t)) + D_2\nabla^2\rho_2(x,t), \quad (5b)$$

where the total reaction terms f and g are defined as

$$f(\rho_1(x,t),\rho_2(x,t)) \equiv R_1^+(\rho(x,t)) - R_1^-(\rho(x,t)), \quad (6a)$$

$$g(\rho_1(x,t),\rho_2(x,t)) \equiv R_2^+(\rho(x,t)) - R_2^-(\rho(x,t)), \quad (6b)$$

with $\rho(x,t)=(\rho_1(x,t),\rho_2(x,t))$. The homogeneous steady state $(\rho_1^0(x),\rho_2^0(x))=(\rho_1^0,\rho_2^0)$ of the system (5) is given by

$$f(\rho_1^0,\rho_2^0) = g(\rho_1^0,\rho_2^0) = 0. \quad (7)$$

It is stable against homogeneous perturbations, i.e., spatial perturbations with wave number $k=0$, if

$$f_1 + g_2 < 0, \quad (8)$$

$$f_1g_2 - f_2g_1 > 0, \quad (9)$$

where $f_i \equiv (\partial f/\partial \rho_i)|_{\rho^0}$ and $g_i \equiv (\partial g/\partial \rho_i)|_{\rho^0}$. In the following we assume that species 1 is an activator and species 2 an inhibitor, i.e.,

$$f_1 > 0, \quad g_2 < 0. \quad (10)$$

Let θ_0 denote the ratio of the diffusion coefficients of the inhibitor and the activator, $\theta_0=D_2/D_1$. A Turing bifurcation to a stationary pattern occurs when the homogeneous steady state becomes unstable to a spatial perturbation with wave number $k_c \neq 0$, which requires that

$$(-f_1 + D_1k^2)(-g_2 + D_2k^2) - g_1f_2 = 0. \quad (11)$$

Equation (11) is known as the classical Turing condition. It implies that the homogeneous steady state becomes unstable to spatial perturbations at the critical ratio of diffusion coefficients

$$\theta_{0,c} = \left(\frac{1}{f_1}(\sqrt{f_1g_2 - f_2g_1} + \sqrt{-f_2g_1}) \right)^2. \quad (12)$$

It is easily verified, using inequalities (8) and (9), that $\theta_{0,c} > 1$. In a standard reaction-diffusion system, a Turing instability can occur only if the diffusion coefficient of the inhibitor exceeds the diffusion coefficient of the activator by a certain amount. This is the well-known principle of short-range activation and long-range inhibition.

To understand the effect of anomalous diffusion on the Turing instability, we have examined the linear stability of a reaction-subdiffusion system consisting of two species obeying Eq. (2) [7], where species 1 (activator) undergoes subdiffusion, i.e., its waiting time PDF is $\phi_1(u)=1-(u\eta_1)^{\alpha}$ in Laplace space, while species 2 (inhibitor) undergoes normal diffusion. If conditions (8) and (9) are satisified, the homogeneous steady state undergoes a Turing instability when

$$[(-f_1 + \sigma_1^2k^2p^{1-\alpha}/\eta_1^{\alpha})(-g_2 + \sigma_2^2k^2/\eta_2) - g_1f_2] + \sigma_1^2k^2\rho_1^0p^{-\alpha} \times \eta_1^{-\alpha}(\alpha-1)(A_2g_1 - A_1g_2 + A_1\sigma_2^2k^2/\eta_2) = 0. \quad (13)$$

Here,

$$A_1 = \frac{\partial}{\partial \rho_1}\left(\frac{R_1^-[\rho(x,t)]}{\rho_1(x,t)} \right)_{(\rho_1^0,\rho_2^0)}, \quad (14a)$$

$$A_2 = \frac{\partial}{\partial \rho_2}\left(\frac{R_1^-[\rho(x,t)]}{\rho_1(x,t)} \right)_{(\rho_1^0,\rho_2^0)}, \quad (14b)$$

and $p=R_1^-(\rho^0)/\rho_1^0 > 0$, where ρ^0 is the vector of steady state values of all pertinent fields.

Using the generalized diffusion coefficient $K_{\alpha;1}$ [see Eq. (4)] and the regular diffusion coefficient $D_2 \equiv \sigma_2^2/\eta_2$, we write the Turing condition as

$$[(-f_1 + K_{\alpha;1}k^2p^{1-\alpha})(-g_2 + D_2k^2) - g_1f_2] + [K_{\alpha;1}k^2\rho_1^0p^{-\alpha}(\alpha-1)](A_2g_1 - A_1g_2 + A_1D_2k^2) = 0. \tag{15}$$

Equation (15) expresses the Turing condition purely in terms of a generalized diffusion constant. This is a remarkable result since Eq. (15) is independent of the parameters σ_1 and η_1, and thus has a much broader physical meaning, independent of the microscopic details of the random walk used in its derivation. In practice, one may directly measure the generalized diffusion coefficient [21], and utilize it in Eq. (15) to study the Turing instability in the subdiffusive regime.

Rewriting Eq. (15) in the form

$$k^4 + c_2k^2 + c_4 = 0, \tag{16}$$

with

$$c_2 = \frac{-D_2f_1p^\alpha + (\alpha-1)A_2g_1K_{\alpha;1}\rho_1^0 - g_2K_{\alpha;1}[p + (\alpha-1)A_1\rho_1^0]}{D_2K_{\alpha;1}[p + (\alpha-1)A_1\rho_1^0]} \tag{17}$$

and

$$c_4 = \frac{(f_1g_2 - f_2g_1)p^\alpha}{D_2K_{\alpha;1}[p + (\alpha-1)A_1\rho_1^0]} \tag{18}$$

furnishes the upper and lower cutoffs of the band of unstable modes,

$$\frac{(-c_2 - \sqrt{c_2^2 - 4c_4})}{2} < k^2 < \frac{(-c_2 + \sqrt{c_2^2 - 4c_4})}{2}, \tag{19}$$

provided $c_2 < 0$, and $c_2^2 - 4c_4 > 0$. At the Turing threshold, $c_2^2 - 4c_4 = 0$, and the critical wave number is $k_c^2 = -c_2/2$.

III. TURING INSTABILITY FOR SUBDIFFUSING ACTIVATOR

In Ref. [7] we analyzed the Turing condition (15) for model reaction schemes where the death rate of the subdiffusing species 1 is linear. In this section, the effects of a subdiffusing activator on the Turing instability are examined in detail for two experimentally relevant activator-inhibitor models. The kinetics for the activator exhibit a nonlinear death rate in these models. Studies of standard reaction-diffusion systems have shown that the combination of local activation and long-range inhibition, which implies a larger diffusion constant for the inhibitor species as compared to the activator, promotes spatially inhomogeneous patterns [5]. This is quantitatively expressed by the critical ratio of diffusion coefficients given by Eq. (12). The latter is equivalent to the ratio of the chemical length scales squared of the inhibitor and the activator. This observation provides the means to achieve an appropriate generalization of the ratio of diffusion coefficients to situations where the two species diffuse with a different characteristic exponent α_i. Define the effective diffusion constant for species i by

$$\hat{D}_{\alpha_i;i} = K_{\alpha_i;i}p^{1-\alpha_i}, \tag{20}$$

which for normal diffusive behavior reduces to the standard diffusion constant D_i. Further, the mean squared chemical length of species i is given by $\langle x_i^2(\tau_{\rm ch})\rangle$. The chemical time scale $\tau_{\rm ch}$ is determined by the characteristic lifetime of the activator, namely, $\tau_{\rm ch} = p^{-1}$. The mean squared length for subdiffusing particles is given by [8]

$$\langle x_i^2(t)\rangle = \frac{2}{\Gamma(1+\alpha_i)}K_{\alpha_i}t^{\alpha_i}. \tag{21}$$

We obtain for the ratio of the mean squared chemical lengths of the inhibitor and the activator

$$\frac{\langle x_2^2(\tau)\rangle}{\langle x_1^2(\tau)\rangle} = \frac{\Gamma(1+\alpha_1)\hat{D}_{\alpha_2;2}}{\Gamma(1+\alpha_2)\hat{D}_{\alpha_1;1}}. \tag{22}$$

For the case considered here that the activator undergoes subdiffusion, $\alpha_1 = \alpha$, and the inhibitor normal diffusion, $\alpha_2 = 1$, Eq. (22) reads

$$\frac{\langle x_2^2(\tau)\rangle}{\langle x_1^2(\tau)\rangle} = \Gamma(1+\alpha)\frac{D_2}{\hat{D}_{\alpha_1;1}}. \tag{23}$$

Since the factor $\Gamma(1+\alpha)$ is close to 1 for $0 < \alpha \leqslant 1$, the result implies that the ratio $\theta_{\gamma,c} = (D_2/\hat{D}_{\alpha;1})_c$, the ratio of the diffusion constant of the inhibitor and the effective diffusion constant of the activator at the Turing threshold, is the appropriate generalization of Eq. (12) to reaction-subdiffusion systems. We have set $\gamma = 1-\alpha$ for convenience. The Turing threshold condition $c_2^2 - 4c_4 = 0$ provides two solutions for the ratio $\theta_{\gamma,c}$. To choose the physically meaningful solution, we recall that $\theta_{0,c} = (D_2/D_1)_c$ should always be greater than unity for standard diffusion ($\gamma = 0$) [5]. This criterion leads to the choice

$$\theta_{\gamma,c} = \frac{1}{f_1^2p^2}\{-2f_2g_1p^2 + f_1g_2p^2 - A_2f_1g_1p\rho_1^0\gamma + 2A_1f_2g_1p\rho_1^0\gamma - A_1f_1g_2p\rho_1^0\gamma + 2\sqrt{g_1(f_2g_1 - f_1g_2)p^2(p - A_1\rho_1^0\gamma)[A_2f_1\rho_1^0\gamma + f_2(p - A_1\rho_1^0\gamma)]}\}, \tag{24}$$

which reduces to $\theta_{0,c}$ [see Eq. (12)], for $\gamma=0$.

We apply the result Eq. (24) to the Oregonator model of the Belousov-Zhabotinsky (BZ) reaction [22,23] and the Lengyel-Epstein model [24,25] of the chlorine dioxide–iodine–malonic acid (CDIMA) reaction. Turing instabilities have been observed in experimental systems with normal diffusive behavior of the activator and inhibitor for both reactions. The kinetic terms of the two-variable Oregonator model read

$$f(\rho_1,\rho_2)=\frac{1}{\epsilon}\left(\rho_1-\rho_1^2-h\rho_2\frac{\rho_1-q}{\rho_1+q}\right), \tag{25a}$$

$$g(\rho_1,\rho_2)=\rho_1-\rho_2. \tag{25b}$$

The variables ρ_1 and ρ_2 correspond to the dimensionless concentrations of the autocatalytic species (activator) $HBrO_2$ and the inhibitor, the oxidized catalyst, respectively. Here ϵ and q are scaling parameters, and h is an adjustable stoichiometric parameter. The nontrivial homogeneous steady state of the Oregonator model is given by

$$\rho_1^0=\frac{1-h-q+\sqrt{1-2h+h^2+2q+6hq+q^2}}{2}, \tag{26a}$$

$$\rho_2^0=\rho_1^0. \tag{26b}$$

Equation (24) can of course be evaluated explicitly for any kinetic scheme using symbolic computation software. However, the resulting expression for the Oregonator model is very lengthy and not enlightening at all. It will therefore not be displayed here. We will instead illustrate the behavior by choosing specific values for the kinetic parameters, namely, $q=0.005$, $h=1.9$, and $\epsilon=0.3$. For these values, conditions (8)–(10) are satisfied, and

$$\theta_{\gamma,c}=6.8012+6.1183\gamma + 5.8167\sqrt{(0.985\,39+\gamma)(1.3513+\gamma)}. \tag{27}$$

A plot of this curve, $\theta_{\gamma,c}$ versus γ, is shown in Fig. 1. The

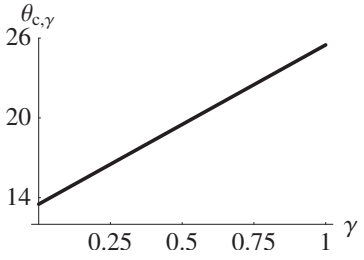

FIG. 1. $\theta_{\gamma,c}$ for the Oregonator model. The parameters are $q=0.005$, $h=1.9$, and $\epsilon=0.3$.

ratio of the diffusion constant of the inhibitor and the effective diffusion constant of the activator at the Turing threshold increases as the motion becomes more subdiffusive, $\gamma\to 1$.

Since we are interested in the effect of subdiffusive motion of the activator on Turing instabilities, we focus on this mechanism exclusively and consider the Lengyel-Epstein (LE) model in its original form [24,25],

$$f(\rho_1,\rho_2)=a-\rho_1-4\frac{\rho_1\rho_2}{1+\rho_1^2}, \tag{28a}$$

$$g(\rho_1,\rho_2)=b\left(\rho_1-\frac{\rho_1\rho_2}{1+\rho_1^2}\right), \tag{28b}$$

i.e., without a substrate. Turing patterns are realized in experimental studies of the CDIMA reaction with normal diffusive behavior by introducing a substrate into the reactor. The activator binds reversibly to this substrate, which lowers its effective diffusion constant. The combined effect of subdiffusive motion of the activator and reversible binding to a substrate will be considered elsewhere. The variables ρ_1 and ρ_2 correspond to the dimensionless concentrations of the activator I^- and the inhibitor ClO_2^- respectively, and the parameters a and b depend on the concentrations of the reactants. The LE model has a unique homogeneous steady state, $(\rho_1^0,\rho_2^0)=(a/5,1+a^2/25)$.

Equation (24) results in the following expression:

$$\theta_{\gamma,c}=\frac{1}{(125-3a^2)^2}\left(625ab+65a^3b+\frac{128a^5b\gamma}{25+a^2}+4\sqrt{10}\sqrt{\frac{a^4b^2(125+5a^2+8a^2\gamma)(125-125\gamma+5a^2+11a^2\gamma)}{25+a^2}}\right). \tag{29}$$

We again illustrate the behavior of the critical ratio $\theta_{\gamma,c}$ by choosing specific values for the kinetic parameters, namely, $a=50.0$ and $b=40.0$. For these values, conditions (8)–(10) are satisfied, and

$$\theta_{\gamma,c}=5.9983+11.650\gamma + 10.829\sqrt{(0.461\,19+\gamma)(0.631\,25+\gamma)}. \tag{30}$$

A plot of this curve is shown in Fig. 2. As for the Oregonator model, the ratio of the diffusion constant of the inhibitor and the effective diffusion constant of the activator at the Turing threshold increases as the motion becomes more subdiffusive, $\gamma\to 1$.

It is worth noting that the nonlinear death rate for the subdiffusing species, present in the two models analyzed above, is responsible for the increase of the ratio $\theta_{c,\gamma}$ with an increase in γ. The ratio $\theta_{c,\gamma}$ remains unchanged on changing

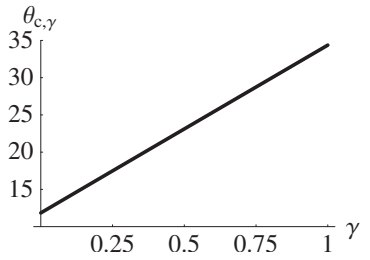

FIG. 2. $\theta_{c,\gamma}$ for the Lengyel-Epstein model. The parameters are $a=50.0$ and $b=40.0$.

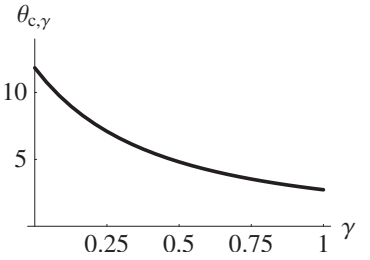

FIG. 3. $\theta_{c,\gamma}$ for the Lengyel-Epstein model with a subdiffusing inhibitor. The parameters are $a=50.0$ and $b=40.0$.

γ, if the subdiffusing species has a linear death rate, since in this case $A_1=A_2=0$.

As mentioned earlier, Turing patterns arise from a competition between local activation and long-range inhibition. For the kinetic schemes studied in this section, the interaction between the nonlinear kinetics, especially the nonlinear death rate, and the memory effects of the transport, as explicitly displayed in the generalized reaction-diffusion equation (2) by the presence of both the kernel $\Theta_i(t-t')$, related to the waiting time PDF of the CTRW, and the death rate $R_i^-(\rho(x,t))$ in the memory term, leads to an enhanced local activation. For the Oregonator and Lengyel-Epstein models and for the parameter values considered here, the specific death rate of the activator, $R_1^-(\rho)/\rho_1$, decreases as the concentration ρ_1 increases away from the steady state value. The increased survival rate in regions with positive fluctuations in the activator concentration, combined with the fact that subdiffusive activator particles stay longer in a given region than normally diffusing particles, leads to an increase of the local autocatalytic effect. This effect becomes more pronounced as the dispersion of the activator becomes more subdiffusive, $\gamma\to 1$. A Turing instability can occur only if the enhanced local autocatalytic effect is countered by a longer-ranged inhibitory effect, and consequently $\theta_{c,\gamma}$ increases as γ increases.

IV. TURING INSTABILITY FOR SUBDIFFUSING INHIBITOR

Having examined the consequences of a subdiffusing activator on the Turing instability in the pervious section, we turn now to the opposite case of a subdiffusing inhibitor. We explore whether the coupling between a nonlinear specific death rate and subdiffusion can similarly enhance the inhibitory effect and result in conditions that are more favorable for the formation of Turing patterns. The Turing condition Eq. (15) is valid under the assumption that species 1 undergoes subdiffusion. In the previous section, ρ_1 and ρ_2 were the concentrations of the activator and inhibitor, respectively, which is the usual convention. To be able to utilize Eq. (15) in studying the role of a subdiffusing inhibitor, we reverse the convention by setting species 1 as the inhibitor and species 2 as the activator. It is straightforward to show by symmetry arguments that the critical ratio of the effective diffusion constant for the subdiffusive inhibitor and the standard diffusion constant for the normally diffusing activator is given by

$$\theta_{c,\gamma}=\left(\frac{1}{f_1^2p^2}\{-2f_2g_1p^2+f_1g_2p^2-A_2f_1g_1p\rho_1^0\gamma+2A_1f_2g_1p\rho_1^0\gamma-A_1f_1g_2p\rho_1^0\gamma-2\sqrt{g_1(f_2g_1-f_1g_2)p^2(p-A_1\rho_1^0\gamma)[A_2f_1\rho_1^0\gamma+f_2(p-A_1\rho_1^0\gamma)]}\}\right)^{-1}. \tag{31}$$

Previously, the physically meaningful solution branch for $\theta_{c,\gamma}$, given by Eq. (24), was obtained by requiring that $\theta_{c,0}>1$. Reversing the labels of the activator and inhibitor amounts to considering the inverse of the conjugate solution to Eq. (24), which is provided by Eq. (31).

The death rate of the inhibitor in the Oregonator model is linear. Subdiffusive motion of this species leaves the critical ratio $\theta_{c,\gamma}$ unchanged as γ increases, $\theta_{c,\gamma}=\theta_{0,c}$. In the following we consider the Lengyel-Epstein model and two model schemes frequently used in theoretical studies, namely, the Schnakenberg model [26] and the Gray-Scott model [27]. We will not display the explicit expression (31) for $\theta_{c,\gamma}$ for each model, but rather illustrate the behavior by a plot of $\theta_{c,\gamma}$ versus γ for a typical set of parameter values.

For the Lengyel-Epstein model with a subdiffusing inhibitor, a typical plot of $\theta_{c,\gamma}$, obtained from Eq. (31), is shown in Fig. 3. In our reversed convention, the nonlinearities are given by

$$f(\rho_1,\rho_2)=b\left(\rho_2-\frac{\rho_1\rho_2}{1+\rho_2^2}\right), \tag{32a}$$

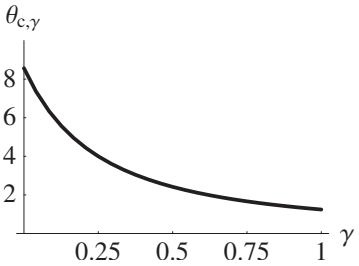

FIG. 4. $\theta_{c,\gamma}$ for the Schnakenberg model with a subdiffusing inhibitor. The parameters are $a=0.1$ and $b=0.9$.

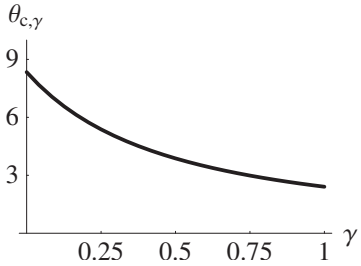

FIG. 5. $\theta_{c,\gamma}$ for the Gray-Scott model with a subdiffusing inhibitor. The parameters are $q=0.15$ and $h=0.03$.

$$g(\rho_1,\rho_2) = a - \rho_2 - 4\frac{\rho_1\rho_2}{1+\rho_2^2}, \tag{32b}$$

and the unique homogeneous steady state is $(\rho_1^0,\rho_2^0)=(1+a^2/25, a/5)$.

Typical plots of $\theta_{c,\gamma}$ for the Schnakenberg model with the kinetic terms

$$f(\rho_1,\rho_2) = b - \rho_1\rho_2^2, \tag{33a}$$

$$g(\rho_1,\rho_2) = a - \rho_2 + \rho_1\rho_2^2, \tag{33b}$$

and steady state $(\rho_1^0,\rho_2^0)=(b/(a+b)^2, a+b)$, and for the Gray-Scott model with the kinetic terms

$$f(\rho_1,\rho_2) = -\rho_1\rho_2^2 + q(1-\rho_1), \tag{34a}$$

$$g(\rho_1,\rho_2) = \rho_1\rho_2^2 - (h+q)\rho_2, \tag{34b}$$

and steady state

$$(\rho_1^0,\rho_2^0) = \left(1 - \frac{(h+q)(-q-\sqrt{-4h^2q+q^2-8hq^2-4q^3})}{-2q(h+q)}, \right. \tag{35}$$

$$\left. \frac{-q-\sqrt{-4h^2q+q^2-8hq^2-4q^3}}{-2q(h+q)}\right),$$

are shown in Figs. 4 and 5, respectively.

A subdiffusing inhibitor with a nonlinear death term results in a decreasing $\theta_{c,\gamma}$ as γ increases, in contrast to the opposite case of a subdiffusing activator examined in the previous section. Again, the interaction between the nonlinear kinetics, especially the nonlinear death rate, and the memory effects of the transport enhances the effectiveness of the inhibitor. This effect becomes more pronounced as the dispersion of the activator becomes more subdiffusive, $\gamma \to 1$, and makes it easier for a Turing instability to arise. Consequently $\theta_{c,\gamma}$ decreases as γ increases.

V. CONCLUSIONS

For reacting and subdiffusing systems we need to distinguish three cases [28–30]. (i) The waiting or trapped particles undergo reactions, i.e., birth and death processes, that obey classical kinetics. This case is described by Eq. (2) and (3) and is relevant for reactions in porous media and for applications in hydrology. It also covers many biological and ecological situations. (ii) The chemical reactions correspond simply to a change in state or color of the particles; the reactive event does not affect the age of a particle (see Sec. II). (iii) The waiting particles are protected from reactions, and reactions occur only at the end of each waiting period with a certain probability [31].

We have studied Turing instabilities for case (i). Specifically, we have investigated the stability behavior of two experimentally relevant activator-inhibitor models, namely, the Oregonator model for the BZ reaction and the Lengyel-Epstein model for the CDIMA reaction, as well as two commonly studied theoretical models, where one species undergoes subdiffusion and the other normal diffusion. If the species that subdiffuses has a nonlinear death rate, then interaction between the nonlinear kinetics and the memory effects of subdiffusion enhances the action of that species. If the activator undergoes subdiffusion, the onset of the Turing instability is delayed. The reaction-subdiffusion system is more stable against spatially inhomogeneous perturbations than the corresponding standard reaction-diffusion system. On the other hand, inhibitor subdiffusion facilitates the onset of the Turing instability; the reaction-subdiffusion system is less stable than the corresponding standard reaction-diffusion system.

Turing instabilities for case (ii) were studied by Nec and Nepomnyashchy [30]. Since reactions do not change the waiting time PDF in this case, both the activator and the inhibitor subdiffuse with the same characteristic exponent α. The authors find that activator-inhibitor systems with anomalous diffusion are more stable than the corresponding normal reaction-diffusion system. Turing instabilities for case (iii) were investigated by Langlands and co-workers [32]. They also considered the case that both species exhibit subdiffusion with the same value of α. The authors find that for this case the critical ratio of diffusion constants is exactly the same as for standard reaction-diffusion systems.

We conclude by mentioning an open problem that deserves further attention, namely, determination of the spatial pattern that is formed as the system is pushed beyond the Turing threshold. This problem can be addressed by a weakly nonlinear analysis of the dynamics in the vicinity of the Turing instability and by numerical methods.

The first approach is a highly nontrivial problem because of the nonlocal character of the generalized reaction-diffusion equation (2) and (3). For standard reaction-diffusion systems, where memory is unimportant, the under-

lying operators in its physical description are local in time. This implies that all the slow eigenmodes of motion close to an instability are themselves local, and it is well understood how to construct a system of evolution equations describing their evolution and interaction, i.e., a weakly nonlinear theory [1,33]. In the case of reaction-subdiffusion systems, the presence of memory implies the nonlocality of the slow eigenmodes, and their growth or decay is not characterized by pure exponentials. Precisely how these nonlocal eigenmodes interact close to an instability is a nontrivial, open problem beyond the scope of this present work.

Simulations of reaction-subdiffusion systems can be carried out at two levels. One can simulate a CTRW with reactions, or one can use our integro-differential formalism of reaction-subdiffusion, Eq. (2) and (3), as a starting point and integrate them numerically. Both these approaches present significant challenges. Previous attempts at simulating CTRWs with reaction were somewhat inconclusive as the number of particles used in the simulations was rather small, and thus fluctuations played an important role [34]. The challenge therefore is to ensure that the number of particles is sufficiently large to render effects of fluctuations negligible. The questions of stability and accuracy for nonlocal operators are central to successful numerical integration of our integro-differential reaction-subdiffusion equations (2) and (3). Work on this topic is currently in progress, and preliminary studies reveal it to be a nontrivial and highly challenging problem.

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APPENDIX: REACTION-SUBDIFFUSION EQUATION

To cast the generalized reaction-diffusion equation (2) in the form of a fractional generalized reaction-diffusion equation for the case of subdiffusive transport, start with Eq. (26) in [7],

$$\frac{\partial \rho_i(x,t)}{\partial t} = R_i^+(\rho(x,t)) - R_i^-(\rho(x,t)) + \sigma_i^2 \nabla^2 \times \left[\exp\left(- \int_0^t \frac{R_i^-(\rho(x,t''))}{\rho_i(x,t'')} dt'' \right) \int_0^t \phi_i(t-t') \times Z_i(x,t') \exp\left(\int_0^{t'} \frac{R_i^-(\rho(x,t''))}{\rho_i(x,t'')} dt'' \right) dt' \right], \quad \text{(A1)}$$

and Eq. (24) in [7],

$$\frac{u\phi_i(u)}{1-\phi_i(u)} L\left[\rho_i(x,t) \exp\left(\int_0^t \frac{R_i^-(\rho(x,t''))}{\rho_i(x,t'')} dt'' \right) \right] = \phi_i(u) L\left[Z_i(x,t') \exp\left(\int_0^{t'} \frac{R_i^-(\rho(x,t''))}{\rho_i(x,t'')} dt'' \right) \right]. \quad \text{(A2)}$$

For subdiffusive transport

$$\frac{u\phi_i(u)}{1-\phi_i(u)} = \Theta_i(u) = \eta_i^{-\alpha_i} u^{1-\alpha_i}. \quad \text{(A3)}$$

Thus

$$\eta_i^{-\alpha_i} u^{1-\alpha_i} L\left[\rho_i(x,t) \exp\left(\int_0^t \frac{R_i^-(\rho(x,t''))}{\rho_i(x,t'')} dt'' \right) \right] = \phi_i(u) L\left[Z_i(x,t') \exp\left(\int_0^{t'} \frac{R_i^-(\rho(x,t''))}{\rho_i(x,t'')} dt'' \right) \right]. \quad \text{(A4)}$$

For $0<p<1$, the Grünwald-Letnikov derivative is defined by [35]

$$D_t^p f(t) \equiv \lim_{\substack{h\to 0 \\ nh=t}} h^{-p} \sum_{r=0}^{n} (-1)^r \binom{p}{r} f(t-rh) = \frac{f(0)t^{-p}}{\Gamma(1-p)} + \frac{1}{\Gamma(1-p)} \int_0^t \frac{f'(s)}{(t-s)^p} ds. \quad \text{(A5)}$$

The Laplace transform of the Grünwald-Letnikov derivative is given by [35]

$$L[D_t^p f(t);u] = u^p f(u). \quad \text{(A6)}$$

We inverse Laplace transform Eq. (A4), using Eq. (A6), to obtain

$$\eta_i^{-\alpha_i} D_t^{1-\alpha_i} \left[\rho_i(x,t) \exp\left(\int_0^t \frac{R_i^-(\rho(x,t''))}{\rho_i(x,t'')} dt'' \right) \right] = \int_0^t \phi_i(t-t') Z_i(x,t') \exp\left(\int_0^{t'} \frac{R_i^-(\rho(x,t''))}{\rho_i(x,t'')} dt'' \right) dt'. \quad \text{(A7)}$$

Substituting Eq. (A7) into Eq. (A1) yields

$$\frac{\partial \rho_i(x,t)}{\partial t} = R_i^+(\rho(x,t)) - R_i^-(\rho(x,t)) + K_{i;\alpha_i} \nabla^2 \times \left\{ \exp\left(- \int_0^t \frac{R_i^-(\rho(x,t''))}{\rho_i(x,t'')} dt'' \right) \times D_t^{1-\alpha_i} \left[\rho_i(x,t) \exp\left(\int_0^t \frac{R_i^-(\rho(x,t''))}{\rho_i(x,t'')} dt'' \right) \right] \right\} \quad \text{(A8)}$$

with

$$K_{i;\alpha_i} = \frac{\sigma_i^2}{\eta_i^{\alpha_i}}. \quad \text{(A9)}$$

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