

Self-organized anomalous aggregation of particles performing nonlinear and non-Markovian random walks

Sergei Fedotov and Nickolay Korabel

School of Mathematics, The University of Manchester, Manchester M13 9PL, United Kingdom

(Received 12 May 2015; revised manuscript received 24 August 2015; published 16 December 2015)

We present a nonlinear and non-Markovian random walks model for stochastic movement and the spatial aggregation of living organisms that have the ability to sense population density. We take into account social crowding effects for which the dispersal rate is a decreasing function of the population density and residence time. We perform stochastic simulations of random walks and discover the phenomenon of *self-organized anomaly* (SOA), which leads to a collapse of stationary aggregation pattern. This anomalous regime is self-organized and arises without the need for a heavy tailed waiting time distribution from the inception. Conditions have been found under which the nonlinear random walk evolves into anomalous state when all particles aggregate inside a tiny domain (anomalous aggregation). We obtain power-law stationary density-dependent survival function and define the critical condition for SOA as the divergence of mean residence time. The role of the initial conditions in different SOA scenarios is discussed. We observe phenomenon of transient anomalous bimodal aggregation.

DOI: [10.1103/PhysRevE.92.062127](https://doi.org/10.1103/PhysRevE.92.062127)

PACS number(s): 05.40.Fb, 05.65.+b, 87.10.Mn, 87.18.-h

I. INTRODUCTION

Aggregation of motile organisms is an example of ecological spatial self-organization due to direct or indirect interactions between individuals [1]. Many organisms (cells, birds, mammals) have the ability to sense a population density, which leads to density-dependent dispersal [2,3]. This dependence can be explained by various mechanisms including (i) competition that forces an individual to emigrate (positive density-dependence), (ii) avoidance of crowded areas by individuals (positive density-dependence), (iii) social crowding effects when certain areas are attractive to many conspecifics (negative density-dependence) [3]. Density-dependent dispersal can be regarded as a behavioral response in which an individual changes its rate of jump due to sensing the mean population density [4,5]. To model the density-dependent dispersal and aggregation, various nonlinear local and nonlocal advection-diffusion equations have been used in the literature [5–10]. Some living organisms like amoeboid microorganism *Dicystelium discoideum* can interact indirectly. They secrete a diffusible attractant (signaling molecules) to which individuals respond chemotactically. They move toward regions of high concentration of attractant and aggregate into a mound. On the population level, the standard model for chemical interaction of species is a pair of the advection-diffusion-reaction equations for the average population density and concentration of signaling molecules. There is a huge body of literature on chemotactic aggregation modeling (see, for example, Refs. [11–16]).

The microscopic theory of actively moving organisms is based on various random walk models [4–14]. One of the main characteristics of the continuous-time random walk (position jump walk) is the escape rate \mathbb{T} from the point x . For density-dependent dispersal models involving direct interaction between individuals this rate depends on the population density [5]. Indirect interaction can be modeled by the dependence of \mathbb{T} on concentration of signaling molecules produced by individuals [11–14]. An important feature of such random walk models is that they are Markovian. However, many transport processes are non-Markovian for which the

transport operators are nonlocal in time. Examples include the Lévy walk that may accelerate aggregation [17], slow subdiffusive transport that may lead to the phenomenon of anomalous aggregation [18]. The challenge is to take into account both nonlinear density-dependent dispersal and non-Markovian anomalous behavior [19–21]. Although some research has been done to address the interplay between nonlinearity and non-Markovian effects [23], it is still an open problem.

In this paper we consider nonlinear and non-Markovian random walks and propose an alternative mechanism of aggregation, which we call the regime of *self-organized anomaly* (SOA). It leads to the collapse of a standard stationary aggregation pattern and development of nonstationary anomalous aggregation. By using Monte Carlo simulations we show that under certain conditions particles performing a non-Markovian random walk with crowding effects aggregate inside a tiny domain (anomalous aggregation). An important fact is that this anomalous regime is self-organized and arises spontaneously without the need for a heavy-tailed waiting time distribution with an infinite mean time from the inception.

II. NONLINEAR AND NON-MARKOVIAN RANDOM WALKS

In this section we formulate our nonlinear and non-Markovian continuous-time random walks model. Instead of the waiting time probability density function (PDF) we use the escape rate $\mathbb{T}(\tau, \rho)$, which depends on the residence time τ and the density of particles ρ . Due to the dependence of the rate \mathbb{T} on the residence time τ , our model is non-Markovian and should involve memory effects. Our intention is to take into account nonlinear social crowding effects and non-Markovian negative aging. We assume that the random walker has the ability to sense the population density ρ . We model the escape rate \mathbb{T} as a decreasing function of the density $\rho(x, t)$ [3,5],

$$\mathbb{T}(\tau, \rho) = \frac{\mu(\tau)}{1 + A\rho(x, t)}, \quad (1)$$

where A is a positive parameter. This nonlinear function describes the phenomenon of conspecific attraction: the rate at which individuals emigrate from the point x is reduced due to the presence of many conspecifics. This negative density-dependence can be explained by the various benefits of social aggregation like mating, antipredator aggregation, etc. [3]. The rate parameter $\mu(\tau)$ is a decreasing function of the residence time (negative aging):

$$\mu(\tau) = \frac{\mu_0}{\tau_0 + \tau}, \quad (2)$$

where μ_0 and τ_0 are positive parameters. This particular choice of the rate parameter $\mu(\tau)$ has been motivated by non-Markovian crowding: the longer the living organisms stay in a particular site, the smaller becomes the escape probability to another site. Since the escape rate $\mathbb{T}(\tau, \rho)$ depends on both residence time τ and t (indirectly through ρ), we cannot define the waiting (residence) time PDF. It can be done only for the linear case when $A = 0$. In this case the waiting time PDF $\psi(\tau)$ can be defined in the standard way: $\psi(\tau) = \mathbb{T}(\tau, 0) \exp[-\int_0^\tau \mathbb{T}(\tau, 0) d\tau]$ [24]. The particular choice Eq. (2) generates the power law distribution:

$$\psi(\tau) = \frac{\mu_0 \tau_0^{\mu_0}}{(\tau_0 + \tau)^{1+\mu_0}}. \quad (3)$$

For the exponent $\mu_0 < 1$, this waiting time probability density function has infinite first moment which corresponds to anomalous subdiffusion [19–21]. In this paper we choose μ_0 as

$$\mu_0 > 1, \quad (4)$$

for which the mean waiting time is finite for the linear case ($A = 0$). We do not introduce the anomalous effects from the inception as it is done for a classical theory of subdiffusive transport [19–21].

Regarding the space dynamics, we consider the random walks in the stationary external field $S(x)$ on the one-dimensional lattice with the step size a . We should note that the extension for the two and three dimensions is pretty straightforward. When the walker escapes from the point x with the rate $\mathbb{T}(\tau, \rho)$, it jumps to $x + a$ with the probability $p_+(x)$, and it jumps to $x - a$, with the probability $p_-(x)$. For the standard chemotaxis models [22], it is assumed that the jumping probabilities are determined by the chemoattractant concentration $S(x)$ on both sides of the point x as

$$p_\pm(x) = \frac{e^{\beta S(x \pm a)}}{e^{\beta S(x-a)} + e^{\beta S(x+a)}}. \quad (5)$$

In this way we introduce the bias of the random walk in the direction of the increase of the external field $S(x)$. The positive parameter $\beta > 0$ is the measure of the strength of the bias. For small step size a , one can obtain the expressions for $p_\pm(x)$ in the continuous case

$$p_\pm(x) = \frac{1}{2} \pm \frac{\beta}{2} \frac{\partial S}{\partial x} a + o(a). \quad (6)$$

Because of the dependence of \mathbb{T} on the residence time τ , it is convenient to define the structured density of particles $\xi(x, \tau, t)$ at time t such that $\xi(x, \tau, t) \Delta x \Delta \tau$ gives the number of particles in the space interval $(x, x + \Delta x)$ whose residence

time lies in $(\tau, \tau + \Delta \tau)$ [23–25]. We consider initial conditions $\xi(x, \tau, 0) = \rho_0(x) \delta(\tau)$, for which all particles have zero residence time at $t = 0$. The total density $\rho(x, t)$ is defined in the standard way:

$$\rho(x, t) = \int_0^t \xi(x, \tau, t) d\tau. \quad (7)$$

The balance equation for the density $\xi(x, \tau, t)$ for $\tau > 0$ takes the Markovian form

$$\xi(x, \tau + \Delta \tau, t + \Delta t) = \xi(x, \tau, t) (1 - \mathbb{T}(\tau, \rho) \Delta \tau) + o(\Delta t),$$

where $1 - \mathbb{T}(\tau, \rho) \Delta \tau$ is the survival probability during $\Delta \tau$ at point x . Since $d\tau/dt = 1$, in the limit $\Delta t \rightarrow 0$ we obtain the following equation:

$$\frac{\partial \xi}{\partial t} + \frac{\partial \xi}{\partial \tau} = -\mathbb{T}(\tau, \rho) \xi. \quad (8)$$

In what follows we use this equation to determine the conditions for the self-organized anomalous regime. The master equation for ρ can be written as [4]

$$\begin{aligned} \frac{\partial \rho}{\partial t} = & -i(x, t) + p_+(x - a) i(x - a, t) \\ & + p_-(x + a) i(x + a, t), \end{aligned} \quad (9)$$

where $i(x, t) = \int_0^t \mathbb{T}(\tau, \rho) \xi(x, \tau, t) d\tau$. In general, the expression for the total escape rate $i(x, t)$ is not known. In the Markovian case, when the rate \mathbb{T} is independent of τ , using Eq. (7) we obtain

$$i(x, t) = \mathbb{T}(\rho) \rho(x, t). \quad (10)$$

For $\mu_0 < 1$ and only for linear case ($A = 0$), the total escape rate takes the form [18]

$$i(x, t) = (\Gamma(1 - \mu_0) \tau_0^{\mu_0})^{-1} D_t^{1-\mu_0} \rho(x, t), \quad (11)$$

where $D_t^{1-\mu_0}$ is the Riemann-Liouville fractional derivative.

III. STOCHASTIC SIMULATIONS AND SELF-ORGANIZED ANOMALY

In this section we present the stochastic simulations of nonlinear and non-Markovian continuous time random walks along a one-dimensional lattice. Because of the density-dependent dispersal we intend to model, we cannot introduce the power-law probability density function for the random residence time as it is done for the classical continuous time random walk (CTRW) [19–21]. Therefore, we cannot apply the standard Gillespie algorithm [26]. In this paper, we simulate the random walks in a domain $[0, L]$, which is divided into M boxes of length $a = L/M$. To calculate the escape rate Eq. (1) from the box, we approximate the density of walkers ρ in each box as the number of walkers in this box divided by the size of the box and the total number of particles. Each walker has its own residence time τ , which determines its escape rate Eq. (1). First, we consider a stationary linear profile

$$S(x) = \alpha x, \quad (12)$$

where α is the chemotactic strength parameter. In Fig. 1 we present a convergence of the population density profile $\rho(x, t)$ to the stationary distribution $\rho_{st}(x)$ on the interval $[0, 4]$ in

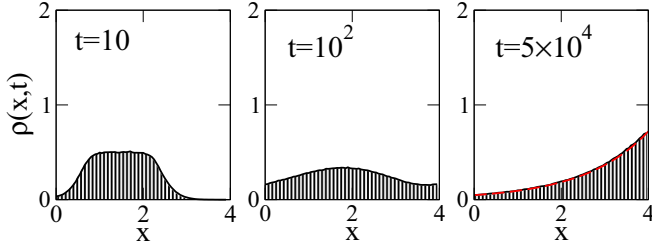


FIG. 1. (Color online) Population density profiles in linear regime $A = 0$. External field (chemoattractant concentration) is linear: $S(x) = \alpha x$. Parameters are $\tau_0 = 1$, $\beta = 1$, $\alpha = 0.34$, and $\mu_0 = 4$. We consider $L = 4$, and $M = 40$, so that $a = 0.1$. The dashed line in the rightmost panel (coincides with the profile) represents the stationary Boltzmann distribution. We have used ensemble of 10^6 walkers uniformly distributed at time $t = 0$ on the interval $[0.5, 2.5]$ with zero residence time ($\tau = 0$). The boundaries at $x = 0$ and $x = 4$ are assumed to be reflecting.

the linear case ($A = 0$) for which the walkers only sense the external field (concentration of signaling molecules) $S(x)$ and not the density of particles $\rho(x, t)$. One can see that the standard aggregation pattern develops. It is easy to show that in the continuous case this distribution is the stationary solution of the standard Fokker-Planck equation: $\rho_{st}(x) \sim \exp[2\beta S(x)]$.

The striking feature of our random walk is that the interplay of nonlinearity Eq. (1) and non-Markovian aging effect Eq. (2) leads to nonequilibrium phase transition. In the nonlinear case ($A > 0$), when the chemotactic strength parameter α is smaller than a critical value α_{cr} , the population density converges to the stationary aggregation profile. When the chemotactic strength parameter α is greater than α_{cr} , crowding effects induce a collapse of the stationary aggregation pattern. The nonlinear random walk evolves into the *self-organized anomaly*. In this regime, the stationary density profile does not exist and all walkers tend to aggregate at the point x_m where the subcritical ($\alpha < \alpha_{cr}$) stationary density $\rho_{st}(x)$ takes the maximum value $\rho_{st}(x_m)$. The initial conditions are chosen such that $\rho_0(x) < \rho_{st}(x_m)$. We discuss the role of other initial conditions in Sec. V. Stochastic simulations of the nonlinear and non-Markovian random walks are presented in Fig. 2. The top row shows the convergence of the population density to the stationary aggregation profile for $\alpha = 0.34$. When $\alpha < \alpha_{cr} \simeq 0.345$, we observe a development of a stationary aggregation with a continuous increase in the maximum value of population density at the point $x_m = 4$ as the parameter α increases up to α_{cr} . The value of α_{cr} depends on the properties of the random walk, system size, and the boundary conditions. Here we do not study this dependence. The observed profile is similar to that of Fig. 1. The only difference is that the nonlinear crowding effects make the value of $\rho_{st}(4)$ greater. However, the drastic change happens, when the value of α exceeds α_{cr} . The dashed line in Fig. 2 represents $\rho_{st}(4) = 1.5$ given by Eq. (28) below. Our nonlinear random walk evolves into the nonstationary SOA, which becomes an attractor for random dynamics. In this regime all particles eventually concentrate in the vicinity of $x_m = 4$. The bottom row in Fig. 2 shows this collapse of the density for $\alpha = 1$. Note that similar phenomenon occurs as a result of chemotaxis, the so-called *chemotactic collapse* when all cells aggregate at some point. Our explanation of

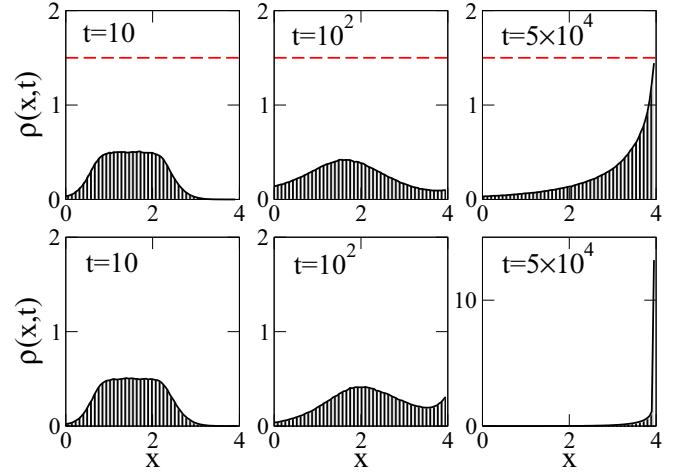


FIG. 2. (Color online) Transition to self-organized anomalous regime for the linear density of external field $S(x) = \alpha x$. Transition from stationary density to collapsed density occurs when signaling strength α exceeds critical value α_{cr} , or in other words when the maximum of the stationary density, $\rho_{st}(x_m)$, exceeds the critical value $\rho_{cr} = 1.5$ given by Eq. (28). Here $x_m = 4$ and $\mu_0 = 4$, $A = 2$. Other parameters, initial and boundary conditions are the same as in Fig. 1. The top row shows the formation of stationary distribution for $\alpha = 0.34 < \alpha_{cr}$. The bottom row illustrates the density collapse that takes place for $\alpha = 1 > \alpha_{cr}$. The critical strength of signaling is estimated numerically as $\alpha_{cr} \simeq 0.345$.

this collapse is different from the classical Patlak-Keller-Segel theory in which the growth of cell density to infinity happens in finite time [11,15,16].

We should note that the *self-organized anomaly* is an universal effect that can occur for any nonuniform external field $S(x)$. To demonstrate that the boundary effects are irrelevant and to show that the SOA does not depend on the form of $S(x)$, in particular on the derivative of $S(x)$ at the point x_m , we consider quadratic external field with the maximum at the center of the domain $[0, 4]$:

$$S(x) = -\sigma(x - 2)^2/2. \quad (13)$$

Here $\sigma > 0$ is the strength parameter. Figure 3 illustrates the phenomenon of the density collapse that takes place at the point $x_m = 2$. This shows that SOA is not a boundary effect.

IV. NONLINEAR MARKOVIAN MODEL

We should stress the fact that the self-organized anomalous regime occurs only for the non-Markovian case. In this section we show that there is no anomalous collapse for the Markovian nonlinear dynamics when the escape rate \mathbb{T} is independent of the residence time τ :

$$\mathbb{T}(\rho) = \frac{\mu_0}{\tau_0(1 + A\rho)}. \quad (14)$$

We start with the Markovian master equation for the total density ρ . Using Eq. (9), we have

$$\begin{aligned} \frac{\partial \rho}{\partial t} = & -\mathbb{T}(\rho)\rho(x, t) + p_+(x - a)\mathbb{T}(\rho(x - a, t))\rho(x - a, t) \\ & + p_-(x + a)\mathbb{T}(\rho(x + a, t))\rho(x + a, t), \end{aligned} \quad (15)$$

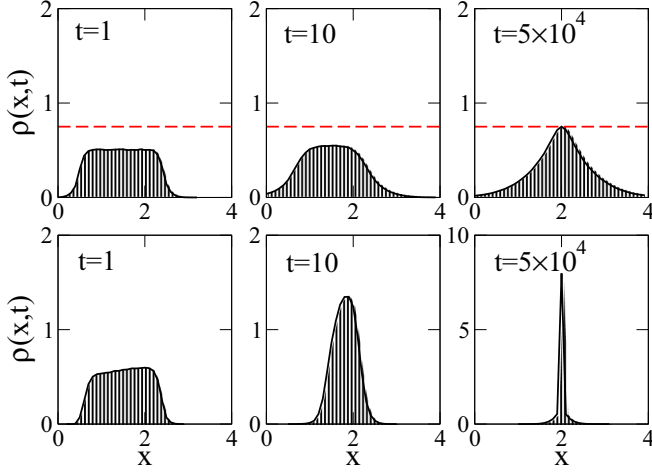


FIG. 3. (Color online) Same as in Fig. 2 but for quadratic external field $S(x) = -\sigma(x-2)^2/2$. The upper row shows the formation of stationary distribution for $\sigma = 0.64 < \sigma_{cr}$. The bottom row illustrates the density collapse that takes place for $\sigma = 10 > \sigma_{cr}$. Here we consider parameters $A = 4$ and $\mu_0 = 4$. Other parameters, initial and boundary conditions are the same as in Fig. 2. The critical value of σ is estimated numerically to be $\sigma_{cr} \simeq 0.65$. Dashed lines represent critical value of the density $\rho_{cr} = 3/4$ given by Eq. (28). Notice that in this case the collapse takes place at $x = 2$.

where $p_{\pm}(x)$ is defined by Eq. (5). We use the Taylor series in Eq. (15) expanding the righthand side in the small a and truncate the series at the second term. The equation for ρ takes the form

$$\frac{\partial \rho}{\partial t} = -2\beta \frac{\partial}{\partial x} \left[\frac{\partial S}{\partial x} D(\rho) \rho \right] + \frac{\partial^2}{\partial x^2} [D(\rho) \rho], \quad (16)$$

with the nonlinear diffusion coefficient

$$D(\rho) = \frac{a^2 \mathbb{T}(\rho)}{2} = \frac{a^2 \mu_0}{2\tau_0(1+A\rho)}. \quad (17)$$

There is no anomalous collapse in this model for any form of the signaling concentration $S(x)$. The nonuniform stationary solution of Eqs. (16) with (17) gives us a population aggregation profile. In the linear case $A = 0$ we have a classical Fokker-Planck equation involving the external field $S(x)$. We should note that in this paper we do not consider the aggregation process due to negative diffusion coefficient [6]. This effect might occur for some negative dependence of the escape rate $\mathbb{T}(\rho)$ on the density ρ . For our model with Eq. (14) the last term in Eq. (16) can be modified in a such way that Eq. (16) takes the form

$$\frac{\partial \rho}{\partial t} = -2\beta \frac{\partial}{\partial x} \left[\frac{\partial S}{\partial x} D(\rho) \rho \right] + \frac{\partial}{\partial x} \left[D_m(\rho) \frac{\partial \rho}{\partial x} \right],$$

with

$$D_m(\rho) = \frac{a^2 \mu_0}{2\tau_0(1+A\rho)^2} > 0. \quad (18)$$

Clearly, the modified nonlinear diffusion coefficient $D_m(\rho)$ is positive.

V. THE UNDERLING MECHANISM FOR THE SELF-ORGANIZED ANOMALOUS REGIME

A. Stationary aggregation profile

Can we understand the underlying mechanism for the SOA observed in our Monte Carlo simulations? They show that in the self-organized anomalous regime, the standard stationary density profile (aggregation pattern) does not exist. So, it is natural first to find a stationary solution $\xi_{st}(x, \tau)$ to the Eq. (8) from

$$\frac{\partial \xi_{st}}{\partial \tau} = -\mathbb{T}(\tau, \rho_{st}(x)) \xi_{st}.$$

Using the escape rate Eq. (2) we obtain

$$\xi_{st}(x, \tau) = \xi_{st}(x, 0) \left(\frac{\tau_0}{\tau_0 + \tau} \right)^{\frac{\mu_0}{1+A\rho_{st}(x)}}.$$

This can be rewritten in terms of the *density-dependent* power-law survival function

$$\Psi(\tau, \rho_{st}) = \left(\frac{\tau_0}{\tau_0 + \tau} \right)^{\frac{\mu_0}{1+A\rho_{st}(x)}} \quad (19)$$

and the stationary arrival rate $j_{st}(x) = \xi_{st}(x, 0)$ as

$$\xi_{st}(x, \tau) = j_{st}(x) \Psi(\tau, \rho_{st}). \quad (20)$$

In the stationary case the arrival rate of particles $j_{st}(x)$ to the point x and the escape rate of particles from the point x are the same: $j_{st}(x) = i_{st}(x)$. The stationary density $\rho_{st}(x)$ can be obtained from Eqs. (7) and (20) in the limit $t \rightarrow \infty$:

$$\rho_{st}(x) = \int_0^{\infty} \xi_{st}(x, \tau) d\tau = i_{st}(x) \int_0^{\infty} \Psi(\tau, \rho_{st}) d\tau. \quad (21)$$

Note that

$$\bar{T}(\rho_{st}(x)) = \int_0^{\infty} \Psi(\tau, \rho_{st}) d\tau \quad (22)$$

can be interpreted as the expected value of the random residence time T whose survival function is given by Eq. (19). It should be emphasized that we cannot introduce the power-law survival function as the function of nonstationary population density $\rho(x, t)$. It follows from Eqs. (21) and (22) that the stationary escape rate $i_{st}(x)$ can be written in the standard Markovian form

$$i_{st}(x) = \frac{1}{\bar{T}[\rho_{st}(x)]} \rho_{st}(x). \quad (23)$$

Using Eq. (9), we write the stationary master equation:

$$-i_{st}(x) + p_+(x-a)i_{st}(x-a) + p_-(x+a)i_{st}(x+a) = 0. \quad (24)$$

Using probabilities Eq. (5), in the limit $a \rightarrow 0$, we obtain from Eqs. (23) and (24) the nonlinear stationary advection-diffusion equation for the population density $\rho_{st}(x)$:

$$-2\beta \frac{\partial}{\partial x} \left\{ \frac{S'(x)\rho_{st}(x)}{\bar{T}[\rho_{st}(x)]} \right\} + \frac{\partial^2}{\partial x^2} \left\{ \frac{\rho_{st}(x)}{\bar{T}[\rho_{st}(x)]} \right\} = 0. \quad (25)$$

It follows from Eq. (25) that the steady profile $\rho_{st}(x)$ on the interval $[0, L]$ with the reflecting boundaries can be found from the nonlinear equation $\rho_{st}(x) = N^{-1} \bar{T}(\rho_{st}) \exp[2\beta S(x)]$,

where N is determined by the normalization condition $N = \int_0^L \bar{T}(\rho_{st}(x)) \exp[2\beta S(x)] dx$. This stationary profile $\rho_{st}(x)$ is illustrated in Fig. 2 for the linear external field Eq. (12) and in Fig. 3 for quadratic field Eq. (13) (see rightmost profiles in the top rows).

B. Conditions for self-organized anomaly

The question arises why the increase of the parameter α for the linear external field Eq. (12) or σ for the quadratic field Eq. (13) above the corresponding critical value α_{cr} or σ_{cr} leads to a collapse of stationary aggregation pattern (see bottom rows in Figs. 2 and 3). Our main idea is that when α or σ gets bigger it leads to the increase of the population density at the point x_m where density $\rho_{st}(x)$ takes the maximum value and the divergence of the integral

$$\int_0^\infty \Psi(\tau, \rho_{st}(x_m)) d\tau. \quad (26)$$

In another words: the *self-organized anomaly* occurs when the effective mean residence time $\bar{T}(x_m) = \int_0^\infty \Psi(\tau, \rho_{st}(x_m)) d\tau$ becomes infinite and the stationary Eq. (25) breaks down. The reason why we call this regime anomalous is that the divergence of the mean waiting time explains anomalous subdiffusive behavior of the random walkers [19–21]. The essential difference to the standard CTRW theory is that we use the stationary density-dependent power-law survival function. Although SOA is similar to the phenomenon of anomalous aggregation [18] or the accumulation of subdiffusive particles in one of two infinite domains with two different values of anomalous exponents [27], it is essentially different. Anomalous conditions are not imposed by power law waiting time PDF Eq. (3) with the anomalous exponent $\mu_0 < 1$. *Anomalous regime is self-organized* for $\mu_0 > 1$ through the nonlinear interactions of random walkers due to social crowding effects described by Eq. (1).

Substitution of the survival function Eq. (19) into Eq. (26) gives

$$\bar{T}(x_m) = \frac{\tau_0[1 + A\rho_{st}(x_m)]}{\mu_0 - 1 - A\rho_{st}(x_m)}. \quad (27)$$

The divergence of $\bar{T}(x_m)$ gives the critical density ρ_{cr} :

$$\rho_{cr} = \frac{\mu_0 - 1}{A}. \quad (28)$$

One can also write the critical condition as $\gamma = 1$, where

$$\gamma = \frac{\mu_0}{1 + A\rho_{st}(x_m)}, \quad \mu_0 > 1. \quad (29)$$

In particular, for the linear external field $S(x) = \alpha x$ in the interval $[0, 4]$ the stationary density $\rho_{st}(x)$ has a maximum value at the point $x_m = 4$. We can find the critical value α_{cr} as

$$\lim_{\alpha \rightarrow \alpha_{cr}} \frac{\mu_0}{1 + A\rho_{st}(x_m)} = 1, \quad \mu_0 > 1. \quad (30)$$

Numerical simulations presented in Figs. 2 and 3 are in excellent agreement with Eq. (28). In Fig. 4 we illustrate the time evolution of the population density $\rho(x_m, t)$ at the boundary $x_m = 4$ for linear field $S(x)$ with different strength of the signaling α . Transition to self-organized anomalous regime is observed by the transition of the density through the

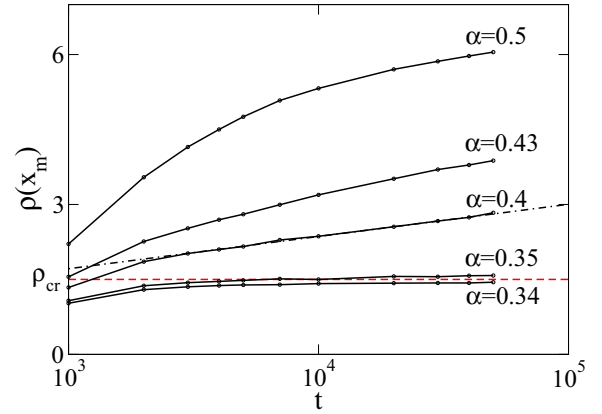


FIG. 4. (Color online) Dependence of the population density at the boundary $x_m = 4$, $\rho(x_m, t)$, for linear field $S(x) = \alpha x$. Parameters are the same as in Fig. 2. Dashed line mark critical values of $\rho_{cr} = 1.5$ given by Eq. (28). Dashed-dotted curve is the best fit for intermediate value of $\alpha = 0.4$ by logarithmic function $\rho(x_m, t) \sim 0.28 \ln(t) - 0.22$.

critical value given by Eq. (28). For intermediate values of α , we find that the density grows as $\ln t$; see Fig. 4.

C. The role of the initial conditions

In our numerical simulations we have used the initial conditions for which all walkers have zero residence time (no aging effects) and the maximum value of the initial density $\rho_0(x)$ obeys the inequality

$$\max \rho_0(x) < \rho_{cr}, \quad (31)$$

where ρ_{cr} is given by Eq. (28). However, when this condition is violated the SOA regime could have different scenarios. In Fig. 5 some preliminary results of numerical simulations are shown. We observe three scenarios depending on the strength of the external field σ . The left most panel shows the anomalous aggregation of the walkers in the region of nonzero initial density $[0.5, 0.6]$. The peak of the density in this region is infinitely increasing (this increase is not captured in Fig. 5,

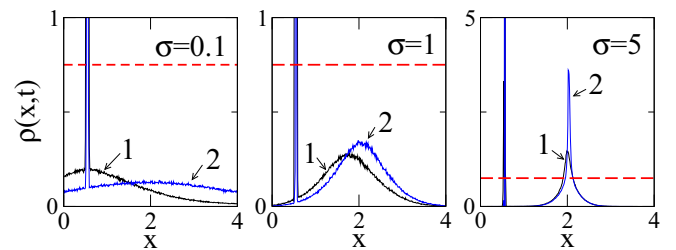


FIG. 5. (Color online) The role of the initial conditions for the quadratic external field $S(x)$ with different field strength σ . We have considered parameters $\tau_0 = 1$, $\beta = 1$, $A = 4$, $\mu_0 = 4$, $L = 4$, and $M = 400$, so that $a = 0.01$. In contrast to Fig. 3, the initial distribution of particles is chosen to violate the condition in Eq. (31). We have used ensemble of 10^6 walkers uniformly distributed at time $t = 0$ on the interval $[0.5, 0.6]$ with zero residence time ($\tau = 0$). The boundaries at $x = 0$ and $x = 4$ are assumed to be reflecting. The dashed lines correspond to the critical value of the density $\rho_{cr} = 3/4$; see Eq. (28). Curve 1 corresponds to $t = 10^4$ and curve 2 to $t = 7 \times 10^4$.

only the peak is visible). The density in the other region reaches a quasistationary form approximately represented by curve 2 for $t = 7 \times 10^4$. Note, however, that it is not a true stationary state since the particles continue to accumulate in the interval $[0.5, 0.6]$. The middle panel demonstrates similar scenario for $\sigma = 1$. However, now the field is strong enough to temporarily accumulate particles at the critical point $x_m = 2$. Our assumption is that similarly to the previous case, in the long time limit these particles will be concentrated in the region of nonzero initial conditions. A different scenario occurs for large strength of the external field. The rightmost panel shows this situation for $\sigma = 5$. In this case the external field is able to strongly attract particles to its critical point $x_m = 2$. At some time the density at this point exceed the critical value Eq. (28). Therefore, in this case the anomalous aggregation of the density is observed at two places, at the region of nonzero initial conditions and at the critical point $x_m = 2$. We call this phenomenon as transient anomalous bimodal aggregation.

VI. CONCLUSIONS

We have discovered the phenomenon of SOA, which takes place in a population of particles performing nonlinear and non-Markovian random walks. The random walks involve social-crowding effects for which the dispersal rate of particles is a decreasing function of the population density and residence time [3,5]. Monte Carlo simulations shows that the regime of *self-organized anomaly* leads to a collapse of a stationary aggregation pattern when all particles concentrate inside a tiny region of space and form a nonstationary high-density cluster. The maximum population density slowly increases

with time as $\ln t$. We should note that the anomalous regime is self-organized and arises spontaneously without the need for introduction of the power-law waiting time distribution with infinite mean time. Only in a stationary case one can obtain a power-law density-dependent survival function and define the critical condition as the divergence of mean residence time. SOA gives a new possible mechanism for chemotactic collapse in a population of living organisms as an alternative to the celebrated Patlak-Keller-Segel theory [11,15,16]. The crossover from the standard stationary aggregation pattern to a nonstationary anomalous aggregation as the strength of chemotactic force increases can be interpreted as nonequilibrium phase transition. Our theory can be used to explain various anomalous aggregation phenomenon including accumulation of phagotrophic protists in attractive patches where they become almost immobile [28]. The phenomenon of *self-organized anomaly* was not known before and therefore new experiments need to be done particularly measurements of the dependence of the rates of movements of the living organisms on residence time in order to prove our theory. The task of our paper is to stimulate these new studies and provide theoretical background. It would be interesting (i) to analyze extensions of our model by taking into account additional effects like volume feeling preventing unlimited density growth [23] and (ii) apply our model for the analysis of how self-organization and an anomalous cooperative effect arise in social systems [29].

ACKNOWLEDGMENTS

This work was funded by EPSRC Grant No. EP/J019526/1. The authors thank Steve Falconer for the helpful suggestions.

-
- [1] S. Camazine, J.-L. Deneubourg, N. R. Franks, J. Sneyd, G. Theraula, and E. Bonabeau, *Self-Organization in Biological Systems* (Princeton University Press, Princeton, NJ, 2003).
 - [2] A. Okubo, *Diffusion and Ecological Problems: Mathematical Models* (Springer-Verlag, Berlin, 1980).
 - [3] E. Matthysen, *J. Ecology* **28**, 403 (2005).
 - [4] V. Mendez, D. Campos, and F. Bartumeus, *Stochastic Foundations in Movement Ecology: Anomalous Diffusion, Front Propagation and Random Searches* (Springer Science & Business Media, Berlin, 2013).
 - [5] V. Mendez, D. Campos, and I. Pagonabarraga, and S. Fedotov, *J. Theor. Biol.* **309**, 113 (2012).
 - [6] P. Turchin, *J. Animal Ecol.* **58**, 75 (1989)
 - [7] S. V. Petrovskii and B.-L. Li, *Math. Biosci.* **186**, 79 (2003).
 - [8] F. Sanchez-Garduno, P. K. Maini, and J. Perez-Velazquez, *Discrete Continuous Dyn. Sys. B* **13**, 455 (2010).
 - [9] M. E. Cates, D. Marenduzzo, I. Pagonabarraga, and J. Tailleur, *Proc. Natl. Acad. Sci. USA* **107**, 11715 (2010).
 - [10] Q.-X. Liu, A. Doelman, V. Rottschäfer, M. de Jager, P. M. J. Herman, M. Rietkerk, and J. van de Koppel, *Proc. Natl. Acad. Sci. USA* **110**, 11905 (2013).
 - [11] H. G. Othmer and A. Stevens, *SIAM J. Appl. Math.* **57**, 1044 (1997).
 - [12] R. Erban and H. Othmer, *SIAM J. Appl. Math.* **65**, 361 (2004).
 - [13] D. Horstmann, K. J. Painter, and H. G. Othmer, *Euro. J. Appl. Math.* **15**, 545 (2004).
 - [14] R. E. Baker, Ch. A. Yates, and R. Erban, *Bull. Math. Biol.* **72**, 719 (2010).
 - [15] M. P. Brenner, L. S. Levitov, and E. O. Budrene, *Biophys. J.* **74**, 1677 (1998).
 - [16] I. Theurkauff, C. Cottin-Bizonne, J. Palacci, C. Ybert, and L. Bocquet, *Phys. Rev. Lett.* **108**, 268303 (2012).
 - [17] M. De Jager, F. J. Weissing, P. M. J. Herman, B. A. Nolet, and J. Van de Koppel, *Science* **332**, 1551 (2011).
 - [18] S. Fedotov, *Phys. Rev. E* **83**, 021110 (2011); S. Fedotov and S. Falconer, *ibid.* **85**, 031132 (2012).
 - [19] R. Metzler and J. Klafter, *Phys. Rep.* **339**, 1 (2000).
 - [20] R. Klages, G. Radons, and I. M. Sokolov, eds., *Anomalous Transport: Foundations and Applications* (Wiley-VCH, Weinheim, 2008).
 - [21] V. Mendez, S. Fedotov, and W. Horsthemke, *Reaction-Transport Systems: Mesoscopic Foundations, Fronts, and Spatial Instabilities*, Springer Series in Synergetics (Springer, Berlin, 2010).
 - [22] A. Stevens, *SIAM J. Appl. Math.* **61**, 172 (2000); T. A. M. Langlands and B. I. Henry, *Phys. Rev. E* **81**, 051102 (2010).
 - [23] S. Fedotov, *Phys. Rev. E* **88**, 032104 (2013); P. Straka and S. Fedotov, *J. Theor. Biol.* **366**, 71 (2015).

- [24] D. R. Cox and H. D. Miller, *The Theory of Stochastic Processes* (Methuen, London, 1965).
- [25] M. O. Vlad and J. Ross, *Phys. Rev. E* **66**, 061908 (2002)
- [26] D. T. Gillespie, *J. Phys. Chem.* **81**, 2340 (1977).
- [27] N. Korabel and E. Barkai, *Phys. Rev. Lett.* **104**, 170603 (2010).
- [28] T. Fenchel and N. Blackburn, *Protist* **150**, 325 (1999).
- [29] R. Axelrod, *The Complexity of Cooperation* (Princeton University Press, Princeton, NJ, 1997).