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We propose a minimal model of *locally-activated diffusion*, in which the diffusion coefficient of a one-dimensional Brownian particle is modified in a prescribed way — either increased or decreased — upon each crossing of the origin. Such a local mobility decrease arises in the formation of atherosclerotic plaques due to diffusing macrophage cells accumulating lipid particles. We show that spatially localized mobility perturbations have remarkable consequences on diffusion at all scales, such as the emergence of a non-Gaussian multi-peaked probability distribution and a dynamical transition to an absorbing static state. In the context of atherosclerosis, this dynamical transition can be viewed as a minimal mechanism that causes macrophages to aggregate in lipid-enriched regions and thereby to the formation of atherosclerotic plaques.

Many-particle systems that consume energy for self-propulsion — active particle systems — have received growing attention in the last decade, both because of the new physical phenomena that they display and their wide range of applications. Examples include molecular motors, cell assemblies, and even larger organisms [1]. The intrinsic out-of-equilibrium nature of these systems leads to remarkable effects such as non-Boltzmann distributions [2], long-range order even in low spatial dimensions [3] and spontaneous flows [4].

At the single-particle level, the active forcing of a Brownian particle leads to non-trivial statistics. For example, it has been recently shown [5, 6] that a random walk which is reset to its starting point at a fixed rate has a non-equilibrium stationary state, as opposed to standard Brownian motion. Another example is given by self-propelled Brownian particles [7], which can yield sharply peaked probability densities for the particle velocity.

In this letter, we consider a new class of problems in which the active forcing of a Brownian particle is *localized in space*. While the impact of localized perturbations on random walks has been investigated [8], in part because of its relevance to a wide range of situations, such as localized sources and sinks [9, 10], trapping [11, 12] or diffusion with forbidden [13], hop-over [14] or defective [15] sites, the role of local activation on Brownian-particle dynamics remains open. We present a minimal model of locally activated diffusion, in which the diffusivity of a Brownian particle is modified — either increased or decreased — in a prescribed way upon each crossing of the origin.

A prototypical example is a bacterium in the presence of a localized patch of nutrients, which enhances the ability of the bacterium to move, or, alternatively, toxins that impair bacterial mobility. This type of localized decrease of mobility also underlies the dynamics of a cell
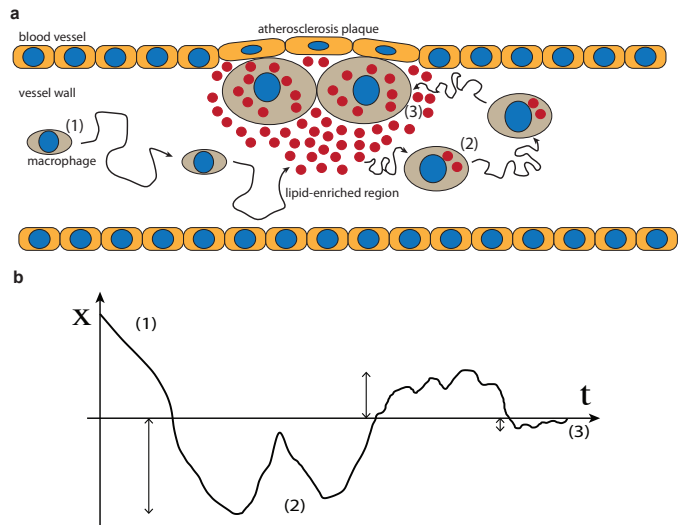

FIG. 1: **a.** Sketch of the different stages of atherosclerosis plaque formation: (1) rapid diffusion of a "free" macrophage cell; (2) upon entering a localized lipid-enriched region, the macrophage accumulates lipids, and thereby grows and becomes less mobile; (3) after many crossings of the lipid-enriched region, the macrophage eventually gets trapped, resulting in the formation of an atherosclerotic plaque. **b.** Sketch of a one-dimensional particle trajectory of the model of locally decelerated random walk.

(e.g., a macrophage) that grows by accumulating smaller and spatially localized particles, such as lipids (Fig. 1) [16, 17]. As the cell grows, its ability to move decreases and the ultimate result is the formation of an atherosclerotic plaque [18]. The spatial localization arises from the presence of lipids at specific points in the arterial network; these lipids can be located, as is now well ac-

cepted, by the properties of the blood flow [19]. Observations show that macrophages that have accumulated lipids move more slowly. Eventually the macrophage stops in an lipid-enriched region, resulting in the formation of an atherosclerotic plaque [20, 21]. Here we propose a simple model to account for this local mobility decrease and address the particular questions of (i) the potential trapping of cells in locally lipid-enriched regions, and (ii) the kinetics of the resulting segregation process when it exists.

Our formalism allows us to describe both the situations of decreased and increased localized mobility changes. We show that this type of perturbation has remarkable consequences on the diffusion process at all scales. We stress that the diffusion coefficient of the active particle at any time depends on the entire history of the trajectory. Thus the evolution of the particle position is intrinsically non-Markovian [22–25]. Our main findings are: (i) The probability distribution of the position has a non-Gaussian tail. (ii) For local acceleration, a diffusing particle is repelled from the origin, so that the maximum in the probability distribution is at non-zero displacement. (iii) For local deceleration, a dynamical transition to an absorbing state occurs. For sufficiently strong deceleration, the particle can get trapped at the origin at a finite time. The exact time dependence for the particle survival probability is determined explicitly. Conversely, if the deceleration process is sufficiently weak, the particle never gets trapped. This dynamical transition to an absorbing state provides a minimal mechanism that could help understand the formation kinetics of atherosclerotic plaques.

The Model. A one-dimensional diffusing particle is accelerated or decelerated whenever it crosses the origin $x = 0$ according to the following Langevin equations:

$$\dot{x} = \sqrt{2D}\,\xi(t)\,, \qquad \dot{D} = f(D)\,\delta(x)\,, \qquad (1)$$

where ξ is a Gaussian white noise of intensity one, D the particle diffusion coefficient, x the particle position, $\delta(x)$ the Dirac distribution, and $f(D)$ an arbitrary prescribed function that accounts for the local activation. For simplicity, we assume that the particle is initially at $x = 0$ with $D = D_0 > 0$. Note that: (i) both the position x and the diffusion coefficient D are random variables; (ii) as mentioned previously, the evolutions of x or D alone are non-Markovian; (iii) the function $f(D)$ can be positive (local acceleration) or negative (local deceleration), but with $f(0) = 0$ so that D remains non negative.

Following standard steps, the corresponding Fokker-Planck equation [26] for the joint distribution of position x and diffusion coefficient D at time t, $P(x, D, t)$, is:

$$\frac{\partial P}{\partial t} = D\frac{\partial^2 P}{\partial x^2} - \delta(x)\frac{\partial[f(D)P]}{\partial D} - \lambda(t)\delta(x)\delta(D), \quad (2)$$

where the last term of the right side accounts for the absorbing state at $(x = 0, D = 0)$. The explicit expression for $\lambda(t)$ is determined demanding that P is normalized,

from which we obtain

$$\lambda(t) = \lim_{D\to 0}\big[f(D)P(0, D, t)\big]. \qquad (3)$$

When $f(D)$ is positive, then D is always non zero. In this case, the particle is never trapped and $\lambda(t) = 0$ at all times. While intuitively obvious for local acceleration ($f(D) > 0$), we show below that $\lambda(t)$ can equal zero for local deceleration processes.

Local Acceleration, $f(D) > 0$. Laplace transforming Eq. (2) gives

$$-s\widehat{P} + D\frac{\partial^2 \widehat{P}}{\partial x^2} = \delta(x)\Big[\frac{\partial(f\widehat{P})}{\partial D} - \delta(D - D_0)\Big], \qquad (4)$$

where $\widehat{P} = \widehat{P}(x, D, s)$ is the Laplace transform of the probability distribution. For $x \neq 0$, the solution is

$$\widehat{P}(x, D, s) = A(D, s)\,e^{-|x|\sqrt{s/D}}\,, \qquad (5)$$

where the coefficient $A(D, s)$ is determined by integrating Eq. (4) across $x = 0$ to obtain the jump of the first derivative of $\widehat{P}$ with respect to x at this point:

$$D\left[\frac{\partial\widehat{P}}{\partial x}\Big|_{x=0+} - \frac{\partial\widehat{P}}{\partial x}\Big|_{x=0-}\right] = \frac{\partial[f\widehat{P}(x=0)]}{\partial D} - \delta(D-D_0)\,.$$

Using Eq. (5), we have

$$f\frac{\partial A}{\partial D} + \Big[f' + 2\sqrt{sD}\Big]A = \delta(D - D_0). \qquad (6)$$

When $f(D)$ is positive, then $A(D, s) = 0$ for $D < D_0$, while for $D > D_0$ the solution to (6) is

$$A = B(s)\,\frac{f(D_0)}{f(D)}\,e^{-\sqrt{4s}\,F}\,, \qquad (7)$$

with

$$F(D) \equiv \int_{D_0}^{D} \frac{\sqrt{D'}}{|f(D')|}\,\mathrm{d}D'\,.$$

The unknown function $B(s)$ is determined by the jump of A at D_0:

$$A(D_0^+, s) - A(D_0^-, s) = \frac{1}{f(D_0)}\,,$$

which finally yields

$$\widehat{P}(x, D, s) = \Theta(D - D_0)\frac{1}{f(D)}\,e^{-Z\sqrt{s}}\,, \qquad (8)$$

where Θ is the Heaviside step function and

$$Z \equiv \frac{|x|}{\sqrt{D}} + 2F(D)\,.$$

Laplace inverting this expression, we obtain the joint distribution

$$P(x, D, t) = \Theta(D - D_0)\frac{Z}{f(D)\sqrt{4\pi t^3}}\,e^{-Z^2/4t}\,. \qquad (9)$$

The marginal distribution with respect to x, that is, the probability distribution of positions, is obtained by integrating Eq. (9) over all D in the range $[D_0, \infty]$. While it does not seem possible to evaluate this integral analytically, the large-x behavior can be obtained by the Laplace method. For the illustrative case where $f(D)$ is a constant (that we define as a), this method gives

$$P(x,t) \sim \frac{1}{t} \sqrt{\frac{|x|}{3a}} \exp\left[-\frac{8|x|^{3/2}}{9\sqrt{a}\,t}\right] \qquad x \to \infty, \quad (10)$$

which we numerically checked is close to the exact value $P(x,t)$ over a wide spatial range. We wish to emphasize two important features of this result for $P(x,t)$ that are in marked contrast with the Gaussian propagator of the usual Brownian motion: (i) $P(x,t)$ generally has a non-Gaussian tail; (ii) $P(x,t)$ reaches its maximum at a *non-zero* displacement. Equation (10) shows that the location of this maximum asymptotically grows as $t^{2/3}$ when $f(D) = a$. Thus local acceleration pushes a diffusing particle away from the origin.

From the general expression (9), the marginal distribution with respect to D can also be easily obtained by integration over x. We find

$$P(D,t) = \Theta(D - D_0) \frac{2\sqrt{D}}{f(D)\sqrt{\pi t}} \, e^{-F^2/t}. \quad (11)$$

In the particular case of $f(D) = a$, Eq. (11) shows that the diffusion coefficient of the particle asymptotically grows as $t^{1/3}$.

As a byproduct, Eq. (11) also provides the distribution of the local time $\tau(t)$ spent by the particle in the active zone (the origin for the present case) up to time t. Using the second of Eqs. (1), this basic observable in the theory of diffusion, which has dimension of time per unit of length [27], is related to the diffusion coefficient at time t by

$$\tau(t) \equiv \int_0^t \delta(x(t'))\,dt' = \int_{D_0}^D \frac{dD'}{f(D')}\,. \quad (12)$$

Thus the distribution of the local time, defined as $\mathcal{P}(\tau,t)$, is given by $\mathcal{P}(\tau,t) = f(D)P(D,t)$, with $P(D,t)$ given by Eq. (11) and D implicitly defined as a function of τ in Eq. (12). For the illustrative case of $f(D) = a$, the distribution of the local time at time t therefore is

$$\mathcal{P}(\tau,t) = \frac{2\sqrt{a\tau + D_0}}{\sqrt{\pi t}} \exp\left\{-\frac{4\left[(a\tau+D_0)^{3/2} - D_0^{3/2}\right]^2}{9a^2 t}\right\}. \quad (13)$$

This result strongly contrasts with the Gaussian distribution that arises in the case of Brownian motion, which can be recovered from Eq. (13) in the limit $a \to 0$:

$$\mathcal{P}_{BM}(\tau,t) = \frac{2\sqrt{D_0}}{\sqrt{\pi t}} \, e^{-D_0\tau^2/t}\,. \quad (14)$$

Notice, in particular, the typical local time for an accelerated particle with $f(D) = a$ grows as $t^{1/3}$ instead of $t^{1/2}$ in the case of Brownian motion.

It is worth noting an intriguing dichotomy with a discrete-time version of local acceleration — the "greedy" random walk [28]. In this discrete model, the step length ℓ_k after the kth return of a random walk to the origin is given by $\ell_k = k^\alpha$. To match with the continuous model with $f(D) = D^\alpha$, one must choose the value $\alpha = 1/2$. With this choice, Eq. (13) of [28] gives, ignoring all multiplicative factors, $P(x,t) \propto x^{1/3}/t \exp[-x^{4/3}/t]$, which is different from (10). The source of this discrepancy is that the probability of being at the origin is not affected by the enhancement mechanism of greedy walks [28], while this return probability is fundamentally modified in the case of locally-activated random walks, as seen explicitly from the distribution of the local time (13). Thus our locally activated diffusion model cannot be viewed as the continuous limit of the greedy random walk. However, it can be shown that it is the continuous limit of a discrete space and continuous time random walk whose jump frequency is modified at each visit of the active site, which is intrinsically different from the greedy random walk.

Local Deceleration, $f(D) < 0$. Following the same analysis as that used for local acceleration, the Laplace transform of the joint distribution is

$$\widehat{P} = \frac{\Theta(D_0 - D)}{|f(D)|} e^{-Z\sqrt{s}} - \frac{\widehat{\lambda}(s)}{s}\delta(x)\,\delta(D)\,, \quad (15)$$

where $\widehat{\lambda}$ is the Laplace transform of $\lambda(t)$ defined in Eqs. (2) and (3). Using these defining relations for $\lambda(t)$, Eq. (15) gives

$$\widehat{\lambda}(s) = \lim_{D \to 0}\left[f(D)\,\widehat{P}(0,D,s)\right] = -e^{-\sqrt{4s}\,\overline{F}}\,, \quad (16)$$

where we define

$$\overline{F}(D) \equiv \int_0^{D_0} \frac{\sqrt{D'}}{|f(D')|}\,dD'\,.$$

In this result for $\widehat{\lambda}$, we have used $\delta(D)f(D) = 0$, since $f(0) = 0$ by the definition of our model. The important feature of Eq. (16) is that $\widehat{\lambda}(s) = 0$ as soon as $\overline{F}$ diverges.

Thus our final result is

$$\widehat{P}(x,D,s) = \frac{\Theta(D_0 - D)}{|f(D)|} e^{-Z\sqrt{s}} + \frac{\delta(x)\delta(D)}{s} e^{-\sqrt{4s}\,\overline{F}}\,, \quad (17)$$

which gives, after Laplace inversion,

$$P(x,D,t) = \frac{\Theta(D-D_0)}{|f(D)|} \frac{Z\,e^{-Z^2/4t}}{\sqrt{4\pi t^3}} + T(t)\delta(x)\delta(D). \quad (18)$$

Here

$$T(t) = \text{erfc}\left(\frac{1}{\sqrt{t}} \int_0^{D_0} \frac{\sqrt{D'}}{|f(D')|}\,dD'\right) \quad (19)$$

is the trapping probability, namely, the probability that the particle becomes stuck at $x = 0$ by time t because

the diffusion coefficient has reached zero. As a corollary, the survival probability is given by $S(t) = 1 - T(t)$, and we have obtained this quantity for an explicitly non-Markovian process. We also mention that, as in the case of local acceleration, the joint distribution easily gives the marginal distributions of the position and the diffusion coefficient, as well as the local time.

A fundamental consequence of the local deceleration of a Brownian particle is that two different dynamical regimes emerge. We illustrate these regimes for the particular case where $f(D) = -D^\beta$ as $D \to 0$. If the deceleration is sufficiently strong, which occurs when $\beta < 3/2$, there is a non-zero probability for the particle to get trapped at the origin. More precisely, the survival probability has the asymptotic behavior

$$S(t) \sim \frac{4 D_0^{3/2-\beta}}{\sqrt{\pi t}\,(3 - 2\,\beta)} \to 0 \qquad t \to \infty\,. \qquad (20)$$

Thus in this regime of strong deceleration, the survival probability has the same scaling with time as in the case of a usual Brownian particle in the presence of a perfect trap. In the opposite case of $\beta \geq 3/2$, then $S(t) = 1$ for all $t > 0$ and the particle never gets trapped at the origin. Thus a locally decelerated Brownian particle undergoes a dynamical transition to the absorbing state $(x = 0, D = 0)$ as the deceleration strength increases. Mathematically, this transition occurs at the point where $\overline{F}$ is no longer divergent.

In conclusion, we introduced a minimal model of locally activated diffusion, in which the diffusion coefficient of a Brownian particle is modified in a prescribed way at each crossing of the origin. In one dimension, a purely diffusing particle hits the origin of the order of $\sqrt{t}$ times after a time t. Consequently, the local activation mechanism is repeatedly invoked during the trajectory of a Brownian particle. Thus the asymptotic dynamics of a Brownian particle is globally affected, leading to markedly different behavior than that of pure diffusion. Since the unusual properties of local activation rely on the recurrence of Brownian motion, we anticipate that qualitatively similar, but quantitatively distinct, behavior would arise in two dimensions.

Our model encompasses both the situations where the Brownian particle is locally accelerated or decelerated. For local acceleration, the probability distribution is non-Gaussian and multi-peaked, with maxima away from the origin no matter how weak the acceleration. For sufficiently weak local deceleration, a Brownian particle manages to avoid getting trapped at the origin in spite of its recurrence. However, for strong deceleration, there is a dynamical transition to an absorbing state in which the particle ultimately gets trapped at the origin.

In the context of atherosclerosis mentioned initially, the dynamical transition to an absorbing state can be viewed as a minimal mechanism that leads to the segregation of macrophages in lipid-enriched regions and thus to the formation of atherosclerotic plaques. Our model suggests that even in absence of chemical signals (such as chemokines or cytokines) that can bias the motion of cells, there exists a critical intensity of the mobility decrease, which depends on the local lipid concentration, beyond which an atherosclerotic plaque will occur. Our model can also help understand the kinetics of this plaque formation.

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