Theory of microbe motion in a poisoned environment

Christian Hoell and Hartmut Löwen
Institut für Theoretische Physik II: Weiche Materie, Heinrich-Heine-Universität Universitätsstrasse 1, D-40225 Düsseldorf, Germany
(Received 27 July 2011; revised manuscript received 1 September 2011; published 19 October 2011)

The motility of a microorganism which tries to avoid a poisoned environment by chemotaxis is studied within a simple model which couples its velocity to the concentration field of the poison. The latter is time independent but inhomogeneous in space. The presence of the poison is assumed to irreversibly reduce the propulsion speed. The model is solved analytically for different couplings of the total poison dose experienced by the microbe to the propulsion mechanism. In a stationary poison field resulting from a constant emission of a fixed point source, we find a power law for the distance traveled by the microbe as a function of time with a nonuniversal exponent which depends on the coupling in the model. With an inverted sign in the couplings, the acceleration of microbe motion induced by a food field can also be described.

DOI: 10.1103/PhysRevE.84.042903 PACS number(s): 87.17.Jj, 82.70.Dd, 83.10.Pp

I. INTRODUCTION

Understanding the motility of microbes [1–3] and other self-propelled particles in solution [4,5] in terms of the underlying physics [6,7] is a challenging problem. In particular, it is interesting to control the motility by environmental stimuli and conditions [8,9]. In fact, many microorganisms are guided by chemotaxis [10–12]; i.e., they "smell" the concentration of a chemical field and on average swim along its steepest gradient in order to optimize their local environment. If the microbe tries to avoid an unfavorable chemical (which may also be termed "poison" or a chemorepellent field) [13], it will go effectively along the negative steepest gradient of this field, while the opposite is true for a favorable chemical (which may also be termed "food" [14] or a chemoattractant field) [11,15].

Recently, individual models for microbe dynamics have been studied which incorporate the influence of chemoattractants and chemorepellents [16–23]. In the following, the effective averaged speed resulting from the ability of microorganisms to actively move along chemical gradients is referred to as drift velocity. In the models studied hitherto, the drift velocity was modelled to be proportional to the local *gradient*. In this paper, we model a complementary situation where the chemical affects irreversibly the maximal drift velocity.[1] In a dangerous "paralyzing" chemical environment the microbe will try to escape with its maximal drift velocity along the negative steepest gradient of the poison field. If the poison field irreversibly destroys or reduces the ability to move, the microbe slows down accordingly. Then the important questions are: under which conditions can the microbe escape a prescribed poison field? How far can it get until a lethal dose stops its motion completely? Of course, the answer to these questions depends on the detailed destructive action of the poison to the propulsion mechanism of the microbe. Experiments on the motility of *Escherichia coli* in an environment polluted by copper ions [24] point indeed to the idea that the motion of bacteria is controlled by the amount of locally experienced poison. The same was found in more recent investigations on *Hotorhabdus temperata* where the motility depends on the salt concentration of the carrier liquid [25].

In this paper, we propose a simple model for a microbe which tries to avoid a poisoned environment by chemotaxis. The model couples its drift velocity to the concentration field of the poison. The chemical concentration field is assumed to be time independent but inhomogeneous in space. The presence of the poison is supposed to irreversibly reduce the drift velocity, and a hyperbolic coupling is proposed in terms of a simple spring destruction model. The model is solved analytically for a one-dimensional setup (ignoring reorientation of the microbe). Three different static concentration fields are considered explicitly, namely a homogeneous field, a linear gradient field, and a field resulting from a poison point source in a quiescent solvent. For a poison source fixed in space which secretes a chemical at a constant rate, an inverse distance scaling of the concentration field is realized. In this field, the traveled distance is found to scale with a power law as a function of time with a *nonuniversal exponent* which depends on the coupling in the model. With an inverted sign in the couplings, the acceleration of microbe motion induced by a chemoattractant field (food field) can also be described accordingly. Our results may be helpful to discriminate between different mechanisms [26,27] for poison action on the propulsion [28] by using detailed real-space measurements of microorganisms [29,30].

II. THE MODEL AND ITS ANALYTICAL SOLUTION

In our model, the microbe performs a completely overdamped self-propelled one-dimensional motion in a viscous solvent [6,31]. As long as the microbe has not encountered poison, it moves with a constant drift velocity v_0. Now the microbe has a sensing of chemical poison concentration as a mean of avoiding poison. In analogy to previous work on chemotaxis [20–23] we assume that the microbe responds by directing its motion in the direction of the steepest decline (the

[1]This is different from the short-time memory many microbes feature in their chemotaxis. For appropriate modeling in that case, e.g., see N. Vladimirov and V. Sourjik, Biol. Chem. **390**, 1097 (2009); N. Bournaveas and V. Calvez, Kinetic and Related Models **1**, 29 (2008).

negative gradient) of poison concentration[2] which is assumed to be a prescribed time-independent field $c(x)$.[3] At time $t = 0$, the microbe is at position $x(0) = x_0$ and is moving with its original maximal velocity $\dot{x}(0) = v(0) = v_0$.

The poison concentration field attacks the motion apparatus by rendering parts of it unusable, as detailed below. Recovery and adaptation processes are not considered, but rather the actual magnitude of the drift velocity v responds to the *total dose* of poison $y = \int_0^t c\,[x(t')]\,dt'$ the microbe has encountered since the time it was first exposed to poison. The coupling between $c(x)$ and v is modelled as $v = G\{\int_0^t c\,[x(t')]\,dt'\}$, where the *coupling function* $G(y)$ is monotonic decreasing in its argument y since the poison reduces the motion. Furthermore, according to our initial conditions, $G(0) = v_0$. The resulting integrodifferential equation for the microbe motion

$$\dot{x}(t) = G\left\{\int_0^t c\,[x(t')]\,dt'\right\} \tag{1}$$

can be solved analytically. The solution $x(t)$ is implicitly given by solving the algebraic equation

$$t = \int_{x_0}^{x(t)} \frac{dx'}{H^{-1}\left[\int_{x_0}^{x'} dx'' c(x'')\right]}. \tag{2}$$

Here, the function $H(y)$ is given by

$$H(y) = \int_{v_0}^{y} dy' y' \frac{d(G^{-1})}{dy'}(y'), \tag{3}$$

and $f^{-1}(y)$ denotes the inverse function to $f(y)$.

Most of the physics is contained in the explicit form of the coupling function $G(y)$, which describes the irreversible weakening of the propulsion if the microbe is exposed to a static poison field $c(x)$. In the following we motivate a *hyperbolic* and a *linear form* for $G(y)$ by using a simple spring model for the coupling.

III. HYPERBOLIC COUPLING

For many microswimmers [32] the propulsion and (hence the drift) velocity is coupled to material elasticity. We model the latter by an effective spring assuming a linear relation between the spring constant and the drift velocity v. The effective spring is serially composed of N individual springs of spring constant D_0 so that the total spring constant equals D_0/N. The presence of a poison particle now irreversibly weakens the elasticity of one spring by reducing its spring constant from D_0 to $D_1 < D_0$ (see also Fig. 1). After $K \leqslant N$ of such incidents, the reduced effective spring constant is $D^{(K)} = 1/[(N-K)/D_0 + K/D_1] = 1/[N/D_0 + K(1/D_1 - 1/D_0)]$.

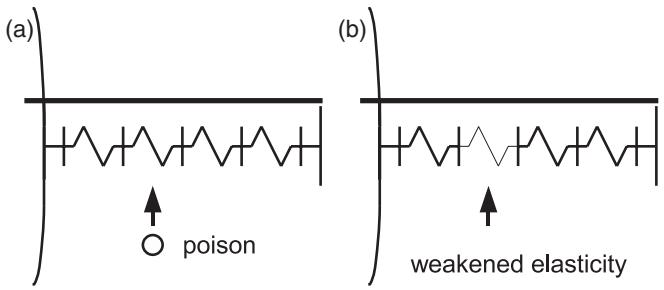

FIG. 1. Sketch of poison action: a poison particle weakens the elasticity of one of several serial coupled springs.

Going over to continuous concentrations leads to a hyperbolic coupling of

$$G(y) = \frac{v_0}{1 + \lambda y}. \tag{4}$$

Here, λ denotes a coupling constant modeling the strength of poison action. Equation (4) guarantees monotonicity as well as the condition $G(0) = v_0$.

For the hyperbolic coupling, Eq. (2) is readily evaluated to give

$$\frac{t}{\tau} = \frac{1}{l}\int_{x_0}^{x(t)} dx' \exp\left[l^2 \int_{x_0}^{x'} dx'' c(x'')\right], \tag{5}$$

where $l = \sqrt{\lambda/v_0}$ and $\tau = l/v_0$ are characteristic length and time scales. If $\int_{x_0}^{\infty} dx\, c(x)$ exists, the microbe escapes the poison field retaining a finite terminal velocity of $v_0 \exp[-l^2 \int_{x_0}^{\infty} dx\, c(x)]$.

We now discuss the solution $x(t)$ for three special cases. First, for a homogeneous concentration field $c(x) = C = const$, the microbe trajectory is given by $x(t) = x_0 + 1/(l^2 C)\log(1 + l^3 C t/\tau)$. Second, for a constant gradient field $c(x) = C_1 x + C$ with $C_1 > 0$ for $x > x_e = -C/C_1$ and $c(x) = 0$ elsewhere, we can assume without loss of generality $x_0 = 0$. Then the solution is implicitly given by $t/\tau = \exp(\beta^2/2)\sqrt{\pi/2}[\Phi(\beta) - \Phi(\sqrt{C_1} l x + \beta)]$ with $\beta = lC/\sqrt{C_1}$ and the probability integral $\Phi(x) = 2/\sqrt{\pi}\int_0^x \exp(-t^2)\,dt$. The microbe escapes the poison field at position x_e after a time $t_e = \tau \exp(\beta^2/2)\sqrt{\pi/2}\Phi(\beta)$ where it has still kept the finite escape velocity $v_e = v_0 \exp(-\beta^2/2)$.

Third, for a point source at the origin which is emitting poison with a constant rate in a quiescent solvent, the steady-state solution of the three-dimensional diffusion equation for the poison concentration field is [19,20] $c(\vec{r}) = A/|\vec{r}|$ with an amplitude A. Anticipating that the microbe tends to escape the poison field, the microbe motion will be one-dimensional in the radial direction with an effective concentration field $c(x) = A/x$ leading to

$$x(t) = x_0\left[1 + (1 + l^2 A)\frac{l}{x_0}\frac{t}{\tau}\right]^{\alpha}, \tag{6}$$

with the exponent

$$0 < \alpha = \frac{1}{1 + l^2 A} < 1. \tag{7}$$

[2]This has to be understood as a dynamically coarse-grained (averaged) effective description of a drift velocity. On shorter time scales the microbe typically performs individual sensing motions [11,15,38–40] which are not considered explicitly here.

[3]We remark that the gradient coupling leads to a quasi-one-dimensional trajectory for a concentration field $c(\vec{r})$ in three spatial dimensions. The curve $\vec{r}(s)$ parameterized by the arc length s, along which microbe motion occurs, is uniquely defined for each starting point by the solution of $d\vec{r}/ds = -\vec{\nabla}c[\vec{r}(s)]/|\vec{\nabla}c[\vec{r}(s)]|$.

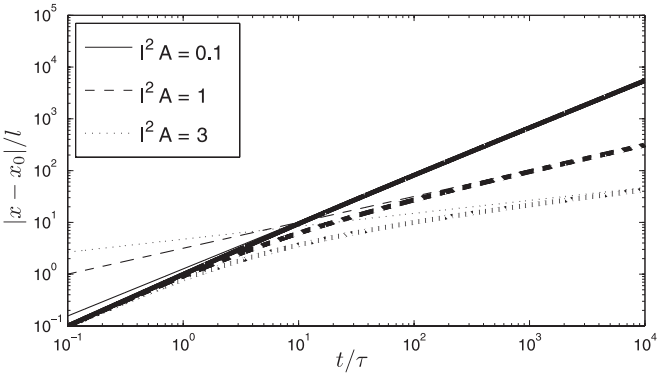

FIG. 2. Logarithmic plot of the reduced displacement $|x - x_0|/l$ vs reduced time t/τ with a starting position $x_0 = 3l$ for different amplitudes A with $l^2A = 0.1$, 1, and 3, leading to exponents $\alpha = 0.\overline{90}$, 0.5, and 0.25. Asymptotic power laws are plotted as light lines.

Clearly, for large times, this solution approaches the power law $x(t) \propto t^\alpha$ (see the plots in Fig. 2). The exponent α is nonuniversal; i.e., it depends on the model parameters.

As a further application, we consider a three-dimensional point source in a homogeneous solvent flow. The flow is along the positive x direction with a velocity u. For this setup, the poison concentration field is [19]

$$c(\vec{r}) = \frac{A}{|\vec{r}|} \exp\left[-\frac{u\,(|\vec{r}| - x)}{2D_p}\right], \tag{8}$$

with the diffusion coefficient D_p of the poison in the solvent and $\vec{r} = (x, y, z)$, which reduces obviously to the static situation for vanishing flow velocity, $u = 0$. Due to Galilean invariance, the same expression is gained for a point source moving with speed $-u$ in a quiescent solvent. Interestingly, the concentration field decays algebraically as the inverse distance in the "wake" of the flow, i.e., in the positive x direction, while it decays exponentially in the distance to the origin for all other directions. In the solvent flow, the microbe is dragged along so that the flow velocity has to be added to its velocity. The best strategy for the microbe to escape the poison field is to get away into a direction different than the positive x axis. The major difference is that an escape along the wake along the positive x axis would result in a vanishing self-propulsion of the microbe [an algebraic decay of $c(x)$] while it would still retain a finite self-propulsion velocity in the other directions [an exponential decay of $c(x)$] as the inner integral in Eq. (5) remains finite.

IV. LINEAR COUPLING

We now present the solution for another *linear* coupling function $G(y)$ where

$$G(y) = \begin{cases} v_0(1 - \lambda y), & \text{for } 0 \leqslant y \leqslant 1/\lambda \\ 0, & \text{else} \end{cases}, \tag{9}$$

with a different coupling constant λ. Again, $G(y)$ is monotonic and the condition $G(0) = v_0$ is fulfilled by construction. This expression contains a *lethal dose* of poison $y = y_0 = 1/\lambda$ where the mobility is zero. The linear coupling can be derived from a simple spring model in a similar way as before but now for a system of parallel springs which are affected by the poison

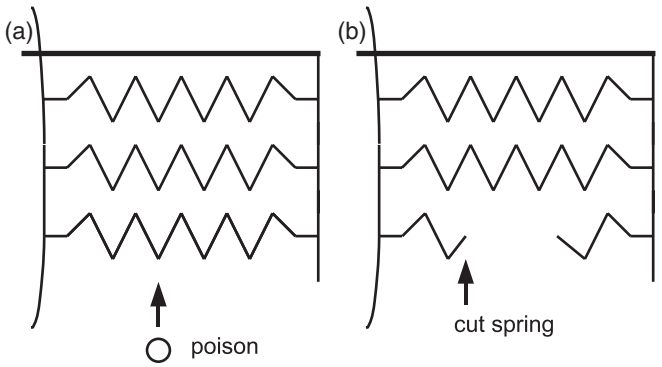

FIG. 3. Action of poison for linear coupling: a poison particle cuts one of several parallel coupled springs.

(see Fig. 3). Now we consider N identical parallel coupled springs with total spring constant ND_0 and assume that the poison will irreversibly cut the springs. After $K \leqslant N$ of such incidents, the new spring constant is $D^{(K)} = D_0\,(N - K)$. So the effective driving force is $v = v_0\,(1 - K/N)$ and the continuum expression yields Eq. (9).

Due to the lethal dose, the microbe can stop completely after a time t_c, i.e., $\dot{x}(t_c) = 0$. Then it has traveled a maximal distance $x_c - x_0$ from its initial position. For a prescribed poison field, $x_c - x_0$ is obtained from the condition $\int_{x_0}^{x_c} dx' c(x') = 1/(2l^2)$ and the corresponding time t_c for immobilization is given by $t_c = \tau/l \int_{x_0}^{x_c} dx'[1 - 2l^2 \int_{x_0}^{x'} dx'' c(x'')]^{-1/2}$.

We now give analytical expressions for the three special concentration fields discussed above. In a homogeneous field $c(x) = C$, the microbe trajectory is a constant deceleration $x(t) = x_0 + lt/\tau - (l^4C/2)(t/\tau)^2$ with $x_c = x_0 + 1/(l^2C)$ and $t_c = \tau/(l^3C)$. Second, for a gradient field $c(x) = C_1x + C$ vanishing for $x < x_e = -C/C_1$, we obtain for $x_0 = 0$ $x(t) = \beta/(l\sqrt{C_1})[\cosh(\nu t) - 1] - 1/(l\sqrt{C_1})\sinh(\nu t)$ with $\nu = l^2\sqrt{C_1}/\tau$. For $\beta > 1$, the microbe stops at $x_c = x_e(1 - \sqrt{1 - \beta^{-2}})$ after a time $t_c = \nu^{-1}\mathrm{artanh}(\beta^{-1})$, while it escapes for $\beta < 1$ after a time $t_e = \nu^{-1}\mathrm{artanh}(\beta)$, keeping an escape velocity of $v_e = v_0\sqrt{1 - \beta^2}$. For the special case $\beta = 1$, it approaches x_e exponentially as $x(t) = x_e[1 - \exp(-\nu t)]$. Third, in the field $c(x) = A/x$ of a point source, the microbe's motion stops at $x(t) = x_0 \exp[1/(2l^2A)]$ after a time $t_c = \tau(x_c/l)\sqrt{\pi/(2l^2A)}\Phi(1/\sqrt{2l^2A})$.

V. FOOD FIELDS

Our model can be extended to describe an increase in propulsion due to the microbe's ingestion of nutrients. In order to do so, the sign of the coupling parameter λ which couples the dose of the nutrient to the propulsion velocity needs to be inverted, and the motion of the microbe is now along the positive gradient of the concentration field $c(x)$.

Similar analytical solutions as in the previous sections are possible. For hyperbolic coupling and a static point source of food where $c(x) = A/x$, there is a scaling $x(t) = x_0(1 - t/\tau_0)^\alpha$ of the distance $x(t)$ of the microbe toward the origin with time t with the same exponent Eq. (7). The associated time constant is $\tau_0 = \alpha\tau x_0/l$.

VI. CONCLUSIONS

In conclusion, we have modelled microbe motion in a poisoned environment by assuming a spatially varying poison concentration field which irreversibly reduces the motion of the microbe. For a hyperbolic coupling in a static poison source, the distance achieved by the microbe scales with a power law in time, the associated exponent $\alpha < 1$ being nonuniversal [5]. This result is obtained for hyperbolic coupling in the static poison concentration field of a constantly emitting point source. The model can serve to classify different bacterial modes of propagation in poison and food environments.

Future work should incorporate more details of microbe motion, including the tumbling process [33,34]. In particular, the coupling assumed here needs certainly more microscopic background modeling and understanding in terms of more details of the microbe propagation mechanism [35]. Furthermore, it would also be interesting to apply the theory to time-dependent poison fields and to include a recovery of the microbe after the action of the poison field. Moreover, additional Brownian motion should be considered. The model could also be expanded to include the influence of other stimuli, like phototaxis. Finally, the collective and swarming behavior of many microbes in a poisoned environment is expected to give rich new nonequilibrium physics [36,37]. This needs, however, a nontrivial generalization of our toy model, including hydrodynamical interactions.

ACKNOWLEDGMENTS

We thank Jakob Schluck, Sven van Teeffelen, and Wilson C. K. Poon for helpful discussions. This work was supported by the Deutsche Forschungsgemeinschaft (DFG) within the SFB TR6 (Project No. D3).

[1] R. M. Macnab and S.-I. Aizawa, Ann. Rev. Biophys. Bioeng. **13**, 51 (1984).
[2] H. Stolp, *Microbial Ecology: Organisms, Habitats, Activities* (Cambridge University Press, Cambridge, 1988).
[3] D. B. Kearns, Nat. Rev. Microbiol. **8**, 634 (2010).
[4] J. Toner, Y. Tu, and S. Ramaswamy, Ann. Phys. (NY) **318**, 170 (2005).
[5] R. Golestanian, Phys. Rev. Lett. **102**, 188305 (2009).
[6] E. M. Purcell, Am. J. Phys. **45**, 3 (1977).
[7] W. C. K. Poon, in *Soft Matter: From Synthetic to Biological Materials*, Vol. D11 (Forschungszentrum Julich GmbH, Germany, 2008), pp. 1–15.
[8] R. M. Ford and R. W. Harvey, Advances in Water Resources **30**, 1608 (2007).
[9] A. Celani and M. Vergassola, Proc. Natl. Acad. Sci. USA **107**, 1391 (2010).
[10] M. Kollmann, L. Lovdok, K. Bartholome, J. Timmer, and V. Sourjik, Nature (London) **438**, 504 (2005).
[11] U. B. Kaupp, N. D. Kashikar, and I. Weyand, Annu. Rev. Physiol. **70**, 93 (2008).
[12] M. Eisenbach, J. Cell. Physiol. **213**, 574 (2007).
[13] M. T. Keating and J. T. Bonner, J. Bacteriol. **130**, 144 (1977).
[14] B. Speranza, M. R. Corbo, and M. Sinigaglia, J. Food Sci. **76**, M12 (2011).
[15] B. M. Friedrich and F. Jülicher, New J. Phys. **10**, 123025 (2008).
[16] A. Czirok, E. Ben-Jacob, I. Cohen, and T. Vicsek, Phys. Rev. E **54**, 1791 (1996).
[17] P. Romanczuk, U. Erdmann, H. Engel, and L. Schimansky-Geier, Eur. Phys. J. (Special Topics) **157**, 61 (2008).
[18] F. Schweitzer and L. Schimansky-Geier, Physica A **206**, 359 (1994).
[19] A. Sengupta, T. Kruppa, and H. Löwen, Phys. Rev. E **83**, 031914 (2011).
[20] Y. Tsori and P.-G. de Gennes, Europhys. Lett. **66**, 599 (2004).
[21] R. Grima, Phys. Rev. Lett. **95**, 128103 (2005).
[22] R. Grima, Phys. Rev. E **74**, 011125 (2006).
[23] A. Sengupta, S. van Teeffelen, and H. Löwen, Phys. Rev. E **80**, 031122 (2009).
[24] J. Adler and B. Templeton, J. Gen. Microbiol. **46**, 175 (1967).
[25] B. Michaels and L. S. Tisa, Can. J. Microbiol. **57**, 196 (2011).
[26] T. Mora, H. Yu, and N. S. Wingreen, Phys. Rev. Lett. **103**, 248102 (2009).
[27] C. A. Brackley, M. C. Romano, C. Grebogi, and M. Thiel, Phys. Rev. Lett. **105**, 078102 (2010).
[28] B. Behkam and M. Sitti, Appl. Phys. Lett. **90**, 023902 (2007).
[29] H. Jeon, Y. Lee, S. Jin, S. Koo, C.-S. Lee, and J. Y. Yoo, Biomed. Microdevices **11**, 1135 (2009).
[30] L. G. Wilson, V. A. Martinez, J. Schwarz-Linek, J. Tailleur, P. N. Pusey, G. Bryant, and W. C. K. Poon, Phys. Rev. Lett. **106**, 018101 (2011).
[31] A. Shapere and F. Wilczek, Phys. Rev. Lett. **58**, 2051 (1987).
[32] E. Gauger and H. Stark, Phys. Rev. E **74**, 021907 (2006).
[33] J. Tailleur and M. E. Cates, Phys. Rev. Lett. **100**, 218103 (2008).
[34] R. W. Nash, R. Adhikari, J. Tailleur, and M. E. Cates, Phys. Rev. Lett. **104**, 258101 (2010).
[35] Y. Z. Yang, J. Elgeti, and G. Gompper, Phys. Rev. E **78**, 061903 (2008).
[36] H. H. Wensink and H. Löwen, Phys. Rev. E **78**, 031409 (2008).
[37] Y. Z. Yang, V. Marceau, and G. Gompper, Phys. Rev. E **82**, 031904 (2010).
[38] H. C. Berg, *E. Coli in Motion* (Springer-Verlag, New York, 2004).
[39] H. C. Crenshaw and L. Edelstein-Keshet, Bull. Math. Biol. **55**, 213 (1993).
[40] A. Guerrero, T. Nishigaki, J. Carneiro, Y. Tatsu, C. D. Wood, and A. Darszon, Dev. Biol. **344**, 52 (2010).