

Confinement Reduces Surface Accumulation of Swimming Bacteria

Da Wei^{1,*}, Shiyuan Hu^{2,3,*}, Tangmiao Tang^{1,4}, Yaochen Yang^{3,4}, Fanlong Meng^{3,4,5,†} and Yi Peng^{1,4,‡}

¹*Beijing National Laboratory for Condensed Matter Physics, Institute of Physics, Chinese Academy of Sciences, Beijing 100190, China*

²*School of Physics, Beihang University, Beijing 100191, China*

³*Institute of Theoretical Physics, Chinese Academy of Sciences, Beijing 100190, China*

⁴*School of Physical Sciences, University of Chinese Academy of Sciences, 19A Yuquan Road, Beijing 100049, China*

⁵*Wenzhou Institute, University of Chinese Academy of Sciences, Wenzhou, Zhejiang 325000, China*



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Many swimming bacteria naturally inhabit confined environments, yet how confinement influences their swimming behaviors remains unclear. Here, we combine experiments, continuum modeling, and particle-based simulations to investigate near-surface bacterial swimming in dilute suspensions under varying confinement. Confinement reduces near-surface accumulation and facilitates bacterial escape. These effects are quantitatively captured by models incorporating the force quadrupole, a higher-order hydrodynamic singularity, that generates a rotational flow reorienting bacteria away from surfaces. Under strong confinement, bacterial trajectories straighten due to the balancing torques exerted by opposing surfaces. These findings highlight the role of hydrodynamic quadrupole interactions in near-surface bacterial motility, with implications for microbial ecology, infection control, and industrial applications.

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Bacterial motility near surfaces is crucial for various microbial processes, including colony growth, biofilm formation, and pathogenic infections. Over the past decades, near-surface swimming behaviors have been extensively studied, primarily in semi-infinite spaces bounded by a single surface [1–14]. Flagellated bacteria accumulate near surfaces due to the interplay of hydrodynamic interactions (HIs) and anisotropic steric interactions. A pioneering study attributed surface accumulation to the leading-order, long-range dipolar flow field [3], while later research highlighted the essential role of direct collisions with the surface [4,10,15]. Moreover, near-field HIs dictate a steady pitching angle that stabilizes surface-adjacent swimming [7,9,13], also contributing to accumulation. On the other hand, bacteria escape surface entrapment through angular diffusion [4] and effective tumbling [12].

While these studies have advanced our understanding of bacterial motility near single surfaces, the accumulation behavior and its governing mechanisms in confined geometries remain less explored. This knowledge gap is significant, as many bacteria inhabit confined spaces in both natural and clinical environments, such as sediment layers [16], urinary tracts [17], and tissue interstices [18]. Boundary element simulations predict that bacteria preferentially swim along the midplane between two parallel

plates when the separation falls below a critical threshold [19]. Bacterial tracking in microfluidic tunnels reveals stable swimming along the central axis in narrow tunnels [20]. However, experiments across various microfluidic channel designs demonstrate a complex response of bacterial motion to confinement, influenced by both bacterial and channel geometry [21].

At the microscale, confinement significantly alters both HIs and steric effects [22–24], reshaping bacterial surface entrapment. When a swimming bacterium is modeled as a collection of flow singularities [7,25], higher-order terms, which are often negligible in unbounded fluids, can become significant under strong confinement. The influence of different singularities on bacterial distribution under varying confinement remains unclear, yet understanding this effect could inform microfluidic designs for controlling microswimmer motility [26–30].

In this Letter, we combine experiments and models to investigate bacterial accumulation between two parallel plates with varying separations. As the plate separation decreases, bacterial accumulation near the surfaces reduces and can even shift into the bulk. Single bacterium tracking reveals that confinement enhances bacterial escape from surface entrapment. Simulations incorporating both HIs and steric interactions demonstrate that a higher-order singularity—the image force quadrupole—is essential to quantitatively reproduce the density profile near surfaces. This quadrupolar term induces a rotational flow, reorienting bacteria away from surface, consistent with experimental observation. While the quadrupole flow decays rapidly with distance from the surface, its rotational effect, coupled

*These authors contributed equally to this work.

†Contact author: fanlong.meng@itp.ac.cn

‡Contact author: pengy@iphy.ac.cn

with bacterial swimming, affects population deep in the bulk even at large plate separation. In strongly confined environments, bacteria follow straighter trajectories rather than circular paths near a single surface, consistent with boundary element simulations.

Confinement reduces surface accumulation—We employ *E. coli* as our model bacteria, composed of a 3 μm -long rod-shaped body and 10 μm -long flagellar bundle. The bacteria exhibit wild-type run-and-tumble behaviors and express green fluorescent protein. Cell concentrations ranges from 0.1 to 1 n_0 , where $n_0 = 8 \times 10^8 \text{ mL}^{-1}$. Bacterial suspensions are loaded into closed chambers formed by two horizontal parallel plates with separation H ranging from 5 to 160 μm . In these chambers, bacteria swim at $v_0 \approx 15 \mu\text{m/s}$ during measurements. We use spinning-disk confocal microscopy to image *E. coli* swimming with high vertical spatial resolution (see Supplemental Material [31] for details). For a given H , we measure the vertical density profile $\Psi(z) = A(z) / \int_0^H A(z') dz'$, where z is the height from the bottom plate and $A(z)$ is the area occupied by bacterial bodies at z [Figs. 1(a) and 1(b)]. The profile $\Psi(z)$ varies progressively from surface accumulation in thick chambers, consistent with previous studies [1–4,6,8,10,11], to bulk accumulation under strong confinement.

The density peaks z_{peak} occur at a finite distance from the surfaces, consistent with prior observations with high resolution in the z direction [33,34] and similar to behavior in synthetic rodlike microswimmers [35]. The nonzero distance results from bacterial rotation and interactions with the surface, with also possible contributions from cell-cell HIs [36], which lift cells away from the surface. As H decreases, z_{peak} remains 4 μm for $H \gtrsim 20 \mu\text{m}$, but drops for $H \lesssim 20 \mu\text{m}$. The decrease primarily arises from confinement-induced suppression of bacterial rotation.

To quantify the effect of confinement on surface accumulation, we calculate the ratio of bacterial density at the midplane to its peak value, $\Psi_{\text{mid}}/\Psi_{\text{peak}}$, as a function of H [Fig. 1(d)]. For $H > 40 \mu\text{m}$, $\Psi_{\text{mid}}/\Psi_{\text{peak}}$ plateaus at ~ 0.4 , indicating sustained surface accumulation as in semi-infinite systems. As H decreases below 40 μm , $\Psi_{\text{mid}}/\Psi_{\text{peak}}$ rises sharply, reflecting reduced surface accumulation. For $H \lesssim 10 \mu\text{m}$, $\Psi(z)$ shows a single peak at midplane [Figs. 1(a) and 1(b)], with $\Psi_{\text{mid}}/\Psi_{\text{peak}} \approx 1$, confirming the prediction from boundary element simulations [19].

To investigate how confinement reduces surface accumulation, we develop a continuum model that incorporates HIs and steric interactions between bacteria and confining surfaces. A multipolar representation of the flow field is employed to quantify bacterium-surface HIs [7]. By modeling the bacterium as a spheroidal body with a slender rodlike flagellum (hereafter, the rod-spheroid model), we derive the strengths of the force dipole D and force quadrupole Q [31],

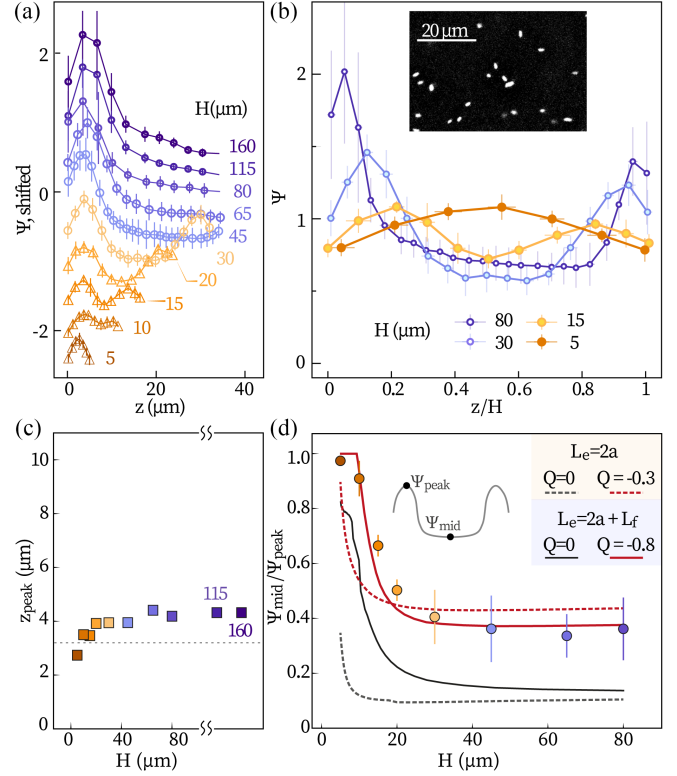


FIG. 1. Experimental measurements of bacterial density profiles. (a) Probability distribution function (PDF) $\Psi(z)$ for various confinement height H . The PDFs are vertically shifted for clarity. (b) $\Psi(z)$ plotted against scaled height z/H . Inset: contrast-enhanced confocal image near the bottom plate; bright regions indicate cell bodies. (c) Peak location of $\Psi(z)$, z_{peak} and (d) ratio $\Psi_{\text{mid}}/\Psi_{\text{peak}}$ as functions of H . Symbols and error bars denote mean $\pm$ SD over 3–4 experiments. Lines in (d) show predictions of the Smoluchowski model with fixed dipole strength $D = 0.6 \text{ pN}\mu\text{m}$, varying exclusion length L_e , and quadrupole strength Q (in $\text{pN}\mu\text{m}^2$).

$$D = \frac{1}{2} F_b (2a + L_h), \quad \text{with} \quad F_b = \zeta_{\parallel} v_0,$$

$$Q = -\frac{1}{6} F_b \left[L_h^2 + 3aL_h + a^2(3 - e^2) \right], \quad (1)$$

where F_b is the drag force on the cell body, $\zeta_{\parallel}$ the drag coefficient along the major axis, a the semimajor axis length, e the eccentricity, and L_h the hydrodynamic flagellum length. Using parameters from Ref. [6], we estimate $L_h \approx 2.5 \mu\text{m}$, which is smaller than the geometric length of flagellar bundle L_f . Thus, we estimate $D \approx 0.6 \text{ pN}\mu\text{m}$, and $Q \approx -0.8 \text{ pN}\mu\text{m}^2$. The corresponding source dipole strength is $S \approx -0.02 \text{ pN}\mu\text{m}^2$ [31]. For bacteria with rod-shaped bodies, the source dipole S is negligible compared to the force quadrupole Q , unlike in squirmer models [37].

The surface-induced flow field at the height of the cell-body center z is approximated as

$$\mathbf{u}(z, \mathbf{p}) \approx [DG_D^*(\mathbf{r}, \mathbf{p}) + Q(\mathbf{p} \cdot \nabla)G_D^*(\mathbf{r}, \mathbf{p})]_{\mathbf{r}=\mathbf{z}\hat{\mathbf{z}}} \cdot \mathbf{p}, \quad (2)$$

where the image force dipole $G_D^*(\mathbf{r}, \mathbf{p}) = (\mathbf{p} \cdot \nabla)G^*(\mathbf{r}, \mathbf{r})$, with G^* being the image system for a Stokeslet placed between two parallel plates, and the swimming direction $\mathbf{p} = (\cos \phi \sin \theta, \sin \phi \sin \theta, \cos \theta)$. We construct $\mathbf{u}(z, \mathbf{p})$ numerically using the exact solution of G^* given in Ref. [38], which enforces the no-slip condition on both plates when superposed with the free-space Stokeslet. Figures 2(a) and 2(b) show the image flow fields of the force dipole and quadrupole, respectively, for bacteria swimming parallel to the plates. The dipolar flow field generates a drift that pulls the bacterium toward the nearest surface. In contrast, the quadrupolar flow induces no drift toward the surface but a nonzero vorticity that reorients the bacterium to swim away from the surface.

We then model bacteria as elongated spheroidal particles with major axis L_e and calculate the density profile by solving the Smoluchowski equation. The probability density function $\Psi(z, \mathbf{p}, t)$ of finding a particle with height z and orientation $\mathbf{p}$ at time t is governed by [39,40]

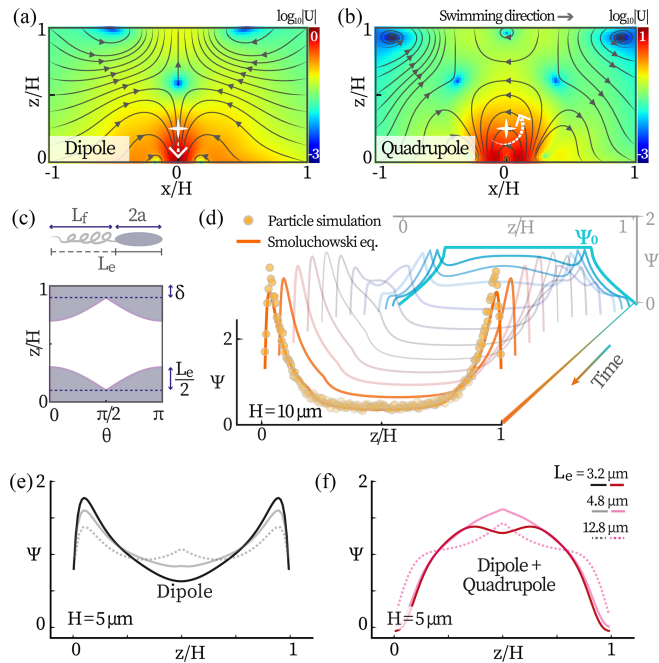


FIG. 2. The effect of quadrupole on bacterial population distribution. (a),(b) Image flow fields induced by a (a) force dipole and (b) force quadrupole. White crosses mark the positions of point singularities; white arrows indicate bacterial motion under image flows. (c) Model schematic of the bacterial body plan (top) and the accessible configuration space constrained by steric exclusion (bottom, white region). The exclusion length (L_e) is tested to span merely the bacterial body ($2a$) and the entire bacterium ($2a + L_f$). (d) Time evolution of $\Psi(z, t)$ from the Smoluchowski model (lines) and the particle simulations (circles), initialized with Ψ_0 . (e) Steady-state $\Psi(z)$ for $D = 0.6 \text{ pN}\mu\text{m}$ and $Q = 0$. (f) Steady-state $\Psi(z)$ for $D = 0.6 \text{ pN}\mu\text{m}$ and $Q = -0.5 \text{ pN}\mu\text{m}^2$.

$$\partial\Psi/\partial t + \partial(v_z\Psi)/\partial z + \nabla_{\mathbf{p}} \cdot (\dot{\mathbf{p}}\Psi) = 0, \quad (3)$$

where $\nabla_{\mathbf{p}}$ is the gradient operator on a unit sphere. The flux velocities v_z and $\dot{\mathbf{p}}$ include swimming, image flow $\mathbf{u}(z, \mathbf{p})$ [Eq. (2)], and thermal diffusion: $v_z = [v_0\mathbf{p} + \mathbf{u}(z, \mathbf{p}) - \mathbf{D}_t \partial \ln \Psi / \partial z] \cdot \hat{\mathbf{z}}$ and $\dot{\mathbf{p}} = \mathbf{\Omega} \times \mathbf{p} - D_r \nabla_{\mathbf{p}} \ln \Psi$, where $\mathbf{D}_t$ is the matrix of translational diffusion coefficients, D_r is the rotational diffusion coefficient around the short axis, and $\mathbf{\Omega}$ the angular velocity given by Jeffery's equation. Both $\mathbf{D}_t$ and D_r are assumed to be independent of z .

Steric exclusion enforces a geometric constraint by preventing particles from penetrating the solid plates [41]. Near the bottom plate, the allowed range of θ is given by

$$\cos^{-1}[2(z - \delta)/L_e] \leq \theta \leq \cos^{-1}[-2(z - \delta)/L_e], \quad (4)$$

for $\delta \leq z \leq L_e/2 + \delta$ [Fig. 2(c)]. Here, δ denotes the minimum distance between the particle and the surface, typically on the order of the particle's semiminor axis. A particle with center close to the plate can only orient in parallel to it. A similar constraint is applied at the top plate; otherwise, $0 \leq \theta \leq \pi$. Steric exclusion is imposed by setting the probability flux normal to the boundaries defined in Eq. (4) to zero. Because the allowed range of θ depends on z , integrating a uniform density $\Psi_0(z, \mathbf{p})$ over $\mathbf{p}$ yields a nonuniform profile $\Psi_0(z)$, exhibiting depletion layers near the plates. This baseline distribution represents the equilibrium population distribution of nonmotile cells.

We solve Eq. (3) numerically using a finite-volume method [31,41]. Figure 2(d) shows the evolution of the marginal distribution $\Psi(z, t)$ for $D = 0.6 \text{ pN}\mu\text{m}$ and $H = 10 \mu\text{m}$, initialized from $\Psi_0(z, \mathbf{p})$. Despite accumulation near surfaces, the depletion layers persist over time, consistency with experimental observations [Figs. 1(a) and 1(b)]. Notably, steric exclusion and force dipole are insufficient to generate a central density peak, as shown by the distributions for various body length [Fig. 2(e)]. Bulk accumulation under strong confinement emerges only when the quadrupole is included [Fig. 2(f)].

We compare the numerical solutions of Eq. (3) with experiments to investigate the roles of interactions in surface accumulation under confinement [Fig. 1(d)]. For $D = 0.6 \text{ pN}\mu\text{m}$ and $Q = 0$, simulated $\Psi_{\text{mid}}/\Psi_{\text{peak}}$ remains significantly below experimental measurements across all H . Increasing Q rises $\Psi_{\text{mid}}/\Psi_{\text{peak}}$. Steric exclusion contributes to the rapid increase in $\Psi_{\text{mid}}/\Psi_{\text{peak}}$ for $H \lesssim L_e$. Using the bacterial body length $2a = 3.2 \mu\text{m}$ as L_e , $Q \approx -0.3 \text{ pN}\mu\text{m}^2$ fits the experimental data at large H , but exhibits an offset at small H . With $L_e = 2a + L_f = 12.8 \mu\text{m}$, approximately the full bacterium length, and using the estimated quadrupole $Q \approx -0.8 \text{ pN}\mu\text{m}^2$ [Eq. (1)], the experimental data is accurately reproduced across all H without introducing fitting parameters. Comparisons of the full bacterial distributions between experiments and models are provided in

Supplemental Material [31]. These results highlight the essential roles of force quadrupole and flagellar steric interaction in shaping the density profile.

Quadrupole-enhanced bacterium escape—To elucidate the role of the quadrupole in bacterium-surface interaction, we track swimming bacteria in a horizontal plane as they collide with a vertical wall [Fig. 3(a)]. The observation plane is positioned more than $30\ \mu\text{m}$ away from the top and bottom surfaces, where the influence of both plates is negligible. To enhance tracking accuracy, we use cephalixin-treated cells with elongated cell bodies $2a \approx 6.0\ \mu\text{m}$ rather than wild-type cells [42]. We identify incident events, in which bacteria approach and become trapped near the wall, and outgoing events, in which they escape from the entrapped region. As most trajectories do not include both incident and outgoing events, we analyze their angular distributions separately [31]. Figure 3(b) shows the representative tracks, whose closest approaching points to the wall are shifted and aligned at the origin. Clearly, the outgoing angle α_{out} exhibits a narrower distribution than the incident angle α_{in} .

To resolve the collision dynamics with the wall, we simulate bacteria as active Brownian particles [31]. Our simulations validate that, in the two-plate setup, the steady-state distribution $\Psi(z)$ from particle simulations agrees with the continuum model [Fig. 2(d)]. We then simulate

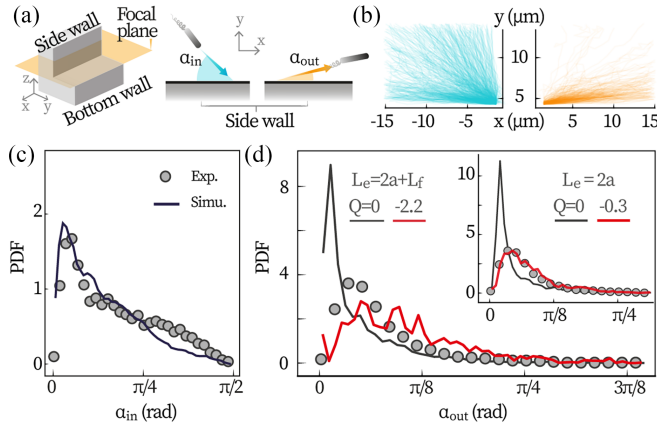


FIG. 3. Side-wall collision dynamics and the role of force quadrupole. (a) Left: schematic of the side-wall experiment. Right: definition of incident and outgoing angles, α_{in} and α_{out} . (b) Experimentally measured incident and outgoing trajectories. Each trajectory is translated such that the closest approach to the wall aligns with the origin. (c) Distributions of α_{in} measured experimentally (circles) and imposed in particle-based simulations (line). (d) Distribution of α_{out} from experiment (circles) and simulations (line) with dipole strength $D = 1.2\ \text{pN}\ \mu\text{m}$, varying bacterial exclusion length L_e , and quadrupole strength Q (in $\text{pN}\ \mu\text{m}^2$). The value $Q = -2.2\ \text{pN}\ \mu\text{m}^2$ is calculated for an elongated bacterium from the rod-spheroid model, rather than a fitting parameter. Inset: $Q = -0.3\ \text{pN}\ \mu\text{m}^2$ gives the best fit for $L_e = 2a$. Simulations use a rotational diffusion coefficient $D_r = 0.06\ \text{rad}^2/\text{s}$, consistent with Ref. [6].

side-wall collisions by constructing the image flow field [Eq. (2)] using the Blake tensor [43]. For elongated bacteria, we estimate the dipole and quadrupole strengths using the rod-spheroid model [Eq. (1)] as $D \approx 1.2\ \text{pN}\ \mu\text{m}$ and $Q \approx -2.2\ \text{pN}\ \mu\text{m}^2$, respectively. In simulations, particles are initialized near the surface, with incident angles α_{in} sampled from the experimental distribution [Fig. 3(c)]. When the quadrupole is neglected ($Q = 0$), the distribution of α_{out} displays a sharp peak at a smaller angle than observed experimentally [Fig. 3(d)]. In contrast, incorporating the quadrupole term ($Q = -2.2\ \text{pN}\ \mu\text{m}^2$) flattens the distribution of α_{out} that matches the experimental data without fitting parameters. The minor deviation may arise from the flexible joint between the flagella and the cell body [44], which reduces the effective bacterial length L_e . Using the lower bound $L_e = 2a$, we find that a nontrivial quadrupole term is required to fit the experimental data.

In addition to confocal imaging, we use defocused fluorescent microscopy to track 3D bacterial motion [45]. By focusing on the top surface of the chamber, out-of-focus bacteria appear as diffraction rings. The ring radius is proportional to the distance between the bacterium and the focal plane. An escape event is defined as a near-surface bacterium swimming more than $5\ \mu\text{m}$ away from the surface. The surface escape rate is the number of escape events per cell per unit time [14,31]. In an $80\ \mu\text{m}$ -thick chamber, we measure an escape rate of $0.038 \pm 0.003\ \text{s}^{-1}$, corresponding to a mean trapping time $\tau \approx 26\ \text{s}$, consistent with the previously reported value of $21\ \text{s}$ [12]. As H decreases to $30\ \mu\text{m}$, the escape rate increases by 46%, indicating enhanced bacterial escape from surface entrapment under confinement. Simulations incorporating a force quadrupole predict a $\approx 50\%$ increase in escape rate as H decreases from 80 to $30\ \mu\text{m}$, compared to $< 30\%$ in simulations neglecting the quadrupole. These results demonstrate that the force quadrupole enhances bacterial escape from surfaces, thereby reducing surface accumulation.

Bacterial trajectories—Flagellated bacteria are known to swim in circles near solid surfaces, driven by a surface-induced hydrodynamic torque acting on the cells [2]. To probe the effect of confinement on circular swimming, we use confocal microscopy to track two-dimensional bacterial trajectories near the bottom plate in chambers of varying height H . Circular motion is characterized by the trajectory curvature κ . The probability distribution of κ peaks near $0.042\ \mu\text{m}^{-1}$ and displays a long tail for $H > 15\ \mu\text{m}$ [Fig. 4(a)]. As H decreases below $15\ \mu\text{m}$, the peak position κ_{peak} shifts to lower values and the tail shortens, indicating straighter trajectories under confinement.

We perform numerical simulations using the boundary element method (BEM) [31,38,46], yielding results in quantitative agreement with experimental observations [Fig. 4(b)]. Near a surface, circular motion arises from an effective hydrodynamic torque generated by interactions between the surface and the rotating flagella and cell body

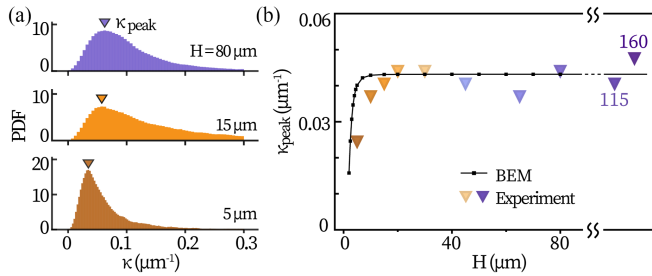


FIG. 4. Curvature of bacterial swimming trajectories under confinement. (a) Probability distributions of trajectory curvature κ measured experimentally for various H . Inverted triangles mark the most probable curvatures κ_{peak} . (b) κ_{peak} as a functions of H . Solid line shows prediction from BEM simulations.

[2]. In confined environments, the opposing surfaces exert hydrodynamic torques in opposite directions, canceling each other [38]. As a result, under strong confinement, both the net torque and trajectory curvature decrease with decreasing H . The circular swimming arises to leading order from the image flow of the rotlet dipole. Since near a single plate $\kappa \sim 1/z^4$ [47], the top plate starts to affect κ only when it is sufficiently close to the bacteria, resulting in a sharp variation of κ at small H [Fig. 4(b)].

Discussion—Combining experiments, continuum theory, and particle-based simulations, we show that swimming bacteria tend to escape surface entrapment and accumulate near the midplane in confined geometries, which originates from fluid flows induced by force quadrupole. The force quadrupole introduces a new mechanism facilitating detachment from surfaces, which can act in parallel with the tumble-mediated escape [12]. These findings advance the understanding of microswimmer surface accumulation under confinement and highlight the fundamental role of force-quadrupole hydrodynamics, often neglected in prior studies.

We use spheroidal particles to approximate the bacterial dynamics in the image flow field. Despite its simplicity, this model quantitatively reproduces experimental observations merely using parameters derived from bacterial geometry and motility. The strengths of hydrodynamic singularities can be tuned by altering bacterial geometries and motility, suggesting a potential route to control microswimmer distributions in confinement [5,7,21].

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Data availability—The data that support the findings of this article are openly available [48].

- [1] P. D. Frymier, R. M. Ford, H. C. Berg, and P. T. Cummings, *Proc. Natl. Acad. Sci. U.S.A.* **92**, 6195 (1995).
- [2] E. Lauga, W. R. DiLuzio, G. M. Whitesides, and H. L. Stone, *Biophys. J.* **90**, 400 (2006).
- [3] A. P. Berke, L. Turner, H. C. Berg, and E. Lauga, *Phys. Rev. Lett.* **101**, 038102 (2008).
- [4] G. Li and J. X. Tang, *Phys. Rev. Lett.* **103**, 078101 (2009).
- [5] H. Shum, E. A. Gaffney, and D. J. Smith, *Proc. R. Soc. A* **466**, 1725 (2010).
- [6] K. Drescher, J. Dunkel, L. H. Cisneros, S. Ganguly, and R. E. Goldstein, *Proc. Natl. Acad. Sci. U.S.A.* **108**, 10940 (2011).
- [7] S. E. Spagnolie and E. Lauga, *J. Fluid Mech.* **700**, 105 (2012).
- [8] M. Molaei, M. Barry, R. Stocker, and J. Sheng, *Phys. Rev. Lett.* **113**, 068103 (2014).
- [9] O. Sipos, K. Nagy, R. Di Leonardo, and P. Galajda, *Phys. Rev. Lett.* **114**, 258104 (2015).
- [10] S. Bianchi, F. Saglimbeni, and R. Di Leonardo, *Phys. Rev. X* **7**, 011010 (2017).
- [11] E. Perez Ipiña, S. Otte, R. Pontier-Bres, D. Czerucka, and F. Peruani, *Nat. Phys.* **15**, 610 (2019).
- [12] G. Junot, T. Darnige, A. Lindner, V. A. Martinez, J. Arlt, A. Dawson, W. C. K. Poon, H. Auradou, and E. Clément, *Phys. Rev. Lett.* **128**, 248101 (2022).
- [13] P. Leishangthem and X. Xu, *Phys. Rev. Lett.* **132**, 238302 (2024).
- [14] D. Cao, M. Dvoriashyna, S. Liu, E. Lauga, and Y. Wu, *Proc. Natl. Acad. Sci. U.S.A.* **119**, e2212078119 (2022).
- [15] S. Bianchi, F. Saglimbeni, G. Frangipane, D. Dell’Arciprete, and R. Di Leonardo, *Soft Matter* **15**, 3397 (2019).
- [16] I. Hewson, M. E. Jacobson/Meyers, and J. A. Fuhrman, *Environ. Microbiol.* **9**, 923 (2007).
- [17] A. L. Flores-Mireles, J. N. Walker, M. Caparon, and S. J. Hultgren, *Nat. Rev. Microbiol.* **13**, 269 (2015).
- [18] M. Kim, H. Ashida, M. Ogawa, Y. Yoshikawa, H. Mimuro, and C. Sasakawa, *Cell Host Microbe* **8**, 20 (2010).
- [19] H. Shum and E. A. Gaffney, *Phys. Rev. E* **91**, 033012 (2015).
- [20] G. Vizsnyiczai, G. Frangipane, S. Bianchi, F. Saglimbeni, D. Dell’Arciprete, and R. Di Leonardo, *Nat. Commun.* **11**, 2340 (2020).
- [21] V. Tokárová, A. S. Perumal, M. Nayak, H. Shum, O. Kašpar, K. Rajendran, M. Mohammadi, C. Tremblay, E. A. Gaffney, S. Martel, D. V. Nicolau, and D. V. Nicolau, *Proc. Natl. Acad. Sci. U.S.A.* **118**, e2013925118 (2021).
- [22] O. Sano and H. Hashimoto, *J. Phys. Soc. Jpn.* **48**, 1763 (1980).
- [23] R. Jeanneret, D. O. Pushkin, and M. Polin, *Phys. Rev. Lett.* **123**, 248102 (2019).
- [24] D. Ghosh and X. Cheng, *Phys. Rev. Res.* **4**, 023105 (2022).
- [25] A. T. Chwang and T. Y.-T. Wu, *J. Fluid Mech.* **67**, 787 (1975).
- [26] W. Duan, W. Wang, S. Das, V. Yadav, T. E. Mallouk, and A. Sen, *Annu. Rev. Anal. Chem.* **8**, 311 (2015).

- [27] C. Bechinger, R. Di Leonardo, H. Löwen, C. Reichhardt, G. Volpe, and G. Volpe, *Rev. Mod. Phys.* **88**, 045006 (2016).
- [28] Q. Brosseau, F. B. Usabiaga, E. Lushi, Y. Wu, L. Ristroph, J. Zhang, M. Ward, and M. J. Shelley, *Phys. Rev. Lett.* **123**, 178004 (2019).
- [29] L. Yuan, H. Straub, L. Shishaeva, and Q. Ren, *Annu. Rev. Anal. Chem.* **16**, 139 (2023).
- [30] Y. Chen, J. Ao, J. Zhang, J. Gao, L. Hao, R. Jiang, Z. Zhang, Z. Liu, J. Zhao, and L. Ren, *Mater. Today* **67**, 468 (2023).
- [31] See Supplemental Material at <http://link.aps.org/supplemental/10.1103/dvc8-tlh1>, which includes Ref. [32], for additional information about the experimental methods, theoretical and simulation details, and extended simulation results.
- [32] J.-Y. Tinevez, N. Perry, J. Schindelin, G. M. Hoopes, G. D. Reynolds, E. Laplantine, S. Y. Bednarek, S. L. Shorte, and K. W. Eliceiri, *Methods* **115**, 80 (2017).
- [33] I. D. Vladescu, E. J. Marsden, J. Schwarz-Linek, V. A. Martinez, J. Arlt, A. N. Morozov, D. Marenduzzo, M. E. Cates, and W. C. K. Poon, *Phys. Rev. Lett.* **113**, 268101 (2014).
- [34] S. Espada Burriel and R. Colin, *PRX Life* **2**, 023002 (2024).
- [35] D. Takagi, J. Palacci, A. B. Braunschweig, M. J. Shelley, and J. Zhang, *Soft Matter* **10**, 1784 (2014).
- [36] P. H. Htet, D. Das, and E. Lauga, *Phys. Rev. Res.* **6**, L032070 (2024).
- [37] T. J. Pedley, *IMA J. Appl. Math.* **81**, 488 (2016).
- [38] N. Liron and S. Mochon, *J. Eng. Math.* **10**, 287 (1976).
- [39] M. Doi and S. F. Edwards, *The Theory of Polymer Dynamics* (Clarendon Press, Oxford, 1986).
- [40] D. Saintillan and M. J. Shelley, *Phys. Rev. Lett.* **100**, 178103 (2008).
- [41] B. Ezhilan and D. Saintillan, *J. Fluid Mech.* **777**, 482 (2015).
- [42] D. Wei, Y. Yang, X. Wei, R. Golestanian, M. Li, F. Meng, and Y. Peng, *Adv. Sci. Lett.* **11**, 2402643 (2024).
- [43] J. R. Blake, *Math. Proc. Cambridge Philos. Soc.* **70**, 303 (1971).
- [44] A. L. Nord, A. Biquet-Bisquert, M. Abkarian, T. Pigaglio, F. Seduk, A. Magalon, and F. Pedaci, *Nat. Commun.* **13**, 2925 (2022).
- [45] M. Wu, J. W. Roberts, and M. Buckley, *Exp. Fluids* **38**, 461 (2005).
- [46] D. J. Smith, *Proc. R. Soc. A* **465**, 3605 (2009).
- [47] E. Lauga, *The Fluid Dynamics of Cell Motility* (Cambridge University Press, Cambridge, England, 2020), Vol. 62.
- [48] D. Wei, S. Hu, F. Meng, and Y. Peng, Dataset underlying “confinement reduces surface accumulation of swimming bacteria,” (2025), [10.6084/m9.figshare.30305500](https://doi.org/10.6084/m9.figshare.30305500).