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Capillary confinement selects the monolayer-to-multilayer transition in bacterial swarms: a theoretical model

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Research Article

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Capillary confinement selects the monolayer-to-multilayer transition in bacterial swarms: a theoretical model

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Abstract

Swarming bacterial populations commonly occupy partially hydrated surfaces fed from beneath agar, and many systems exhibit a spatial architecture in which the advancing edge remains approximately one cell thick while regions farther behind the front become multilayered. Existing explanations for escape from a bacterial monolayer emphasize growth-generated compression, local jamming, motility-induced clustering, and cell-substrate mechanics. Here we develop a complementary theoretical mechanism in which the liquid-air interface supplies a spatially varying vertical confinement. A cell that sits on agar and is partially wetted by water from below creates a meniscus and experiences a normal capillary force. We combine the capillary scaling F_{cap}~γd with a minimal spatial model for interfacial exposure and cell packing to define an extrusion number E, the ratio of active compressive stress to the sum of substrate and capillary restoring stresses. The monolayer is predicted to lose stability where E first reaches unity. For an illustrative Bacillus-scale parameter set, the model predicts a transition approximately 70 μm behind the leading edge. Parameter sweeps show that increasing surface tension widens the monolayer region, reducing edge interfacial exposure shifts the transition toward the edge, and increasing active stress likewise promotes earlier multilayering. These trends persist over alternative capillary exposure laws and under broad parameter uncertainty. The model does not claim experimental validation; rather, it identifies a quantitative, falsifiable mechanism that connects thin-film capillarity to a dimensional transition in active bacterial matter. Direct measurements of film thickness, surface tension, cell density, and the multilayer boundary provide clear tests of the theory.

Keywords: bacterial swarming; capillary forces; thin liquid films; active matter; multilayer transition; Bacillus subtilis; surface tension

Article type: Original theoretical research / modeling study

1 Introduction

Bacterial swarming is a collective mode of surface motility in which large numbers of flagellated cells move rapidly over hydrated substrates. Swarming differs from planktonic swimming because cells move within a geometrically restricted liquid environment and continuously interact with the underlying solid, neighboring cells, and the liquid-air interface. Classic work on surface motility established that swarming requires coordinated changes in cell physiology and extracellular wetting, while later studies emphasized the importance of surfactants, wetting-driven water recruitment, packing, and local mechanical interactions [1-6]. These observations make a purely bulk-fluid description incomplete: the mechanical boundary conditions of the thin film are themselves part of the swarm.

A recurring structural feature is the persistence of a thin, motile peripheral zone while denser regions behind the front can accumulate into multiple layers. Monolayer persistence can be extensive in Bacillus subtilis swarms under suitable conditions [4]. Conversely, increasing local density or obstructing expansion can create jams and multilayer islands [10]. The corresponding question is mechanical as well as biological: what determines the location at which a nominally two-dimensional active population gains access to the third dimension?

Several important mechanisms have already been established. You et al. showed theoretically that a growing bacterial monolayer can undergo a localized rotational instability when growth-induced in-plane compression exceeds a vertical restoring force associated with cell-substrate interactions [11]. Beroz et al. reached a related conclusion for biofilm verticalization, where local compression and adhesion generate threshold reorientation events [12]. In actively moving B. subtilis, Grobas et al. showed that slowing or crowding the swarm can drive a motility- and stress-induced transition to multilayer clusters [10]. These studies make clear that in-plane mechanical loading can drive cells upward. They do not, however, require the vertical restoring force to be spatially uniform or purely substrate-derived.

The liquid-air interface provides an additional restoring mechanism. For an object that protrudes through a liquid film, the interface deforms to satisfy wetting and curvature boundary conditions. The resulting surface-tension and Laplace-pressure contributions can generate substantial normal forces. Gudavadze and Florin solved the Young-Laplace problem for a spherical object protruding through a thin film and showed that capillary confinement becomes important when the film thickness is smaller than the object diameter [8]. More recently, Black et al. directly demonstrated that menisci around bacteria can generate capillary interactions strong enough to reorganize bacterial communities in partially hydrated environments [9]. Surface tension is also known to regulate B. subtilis swarm expansion and to be modified by surfactin [5,6].

These findings motivate a specific hypothesis: the monolayer-to-multilayer boundary in a spreading bacterial swarm can be selected by competition between an inward-growing active stress and an inward-decreasing capillary confinement. Near the leading edge, cells sit on agar and are sufficiently exposed that wetting from below creates a meniscus around each bacterium, while the local compressive load is comparatively small. Farther inward, cell density and mechanical loading increase; in a two-layer region the lower cell becomes fully wetted and the upper cell is the one that forms the meniscus. The dimensional transition should therefore occur at the point where the active stress exceeds the combined capillary and substrate restoring stresses.

The goal of this paper is not to fit a particular unpublished data set. Instead, we construct the minimal quantitative theory required to make the capillary-confinement hypothesis falsifiable. We derive a dimensionless extrusion criterion, explore its predictions numerically, identify robust parameter trends, and state experiments that could distinguish this mechanism from a crowding-only description.

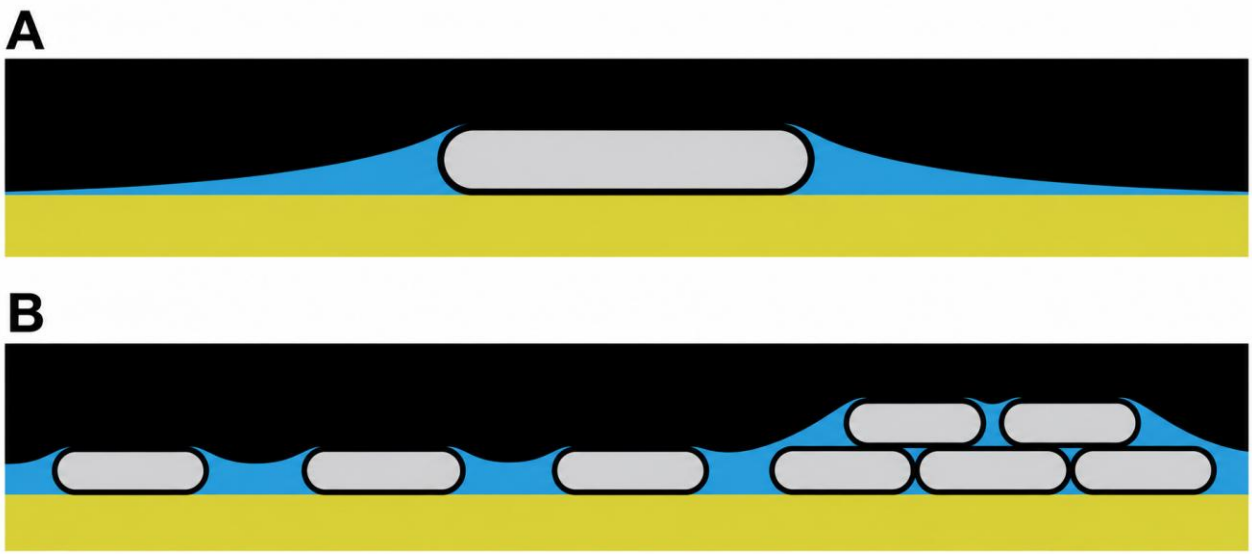


Figure 1. Proposed capillary geometry. (A) A bacterium rests on the agar gel while water supplied through the gel wets the cell and forms a local liquid-air meniscus; the far-field free-surface reference is z = 0. (B) The left side shows the monolayer edge, where each exposed cell rests on agar and forms a local meniscus. The right side shows a two-layer region, where lower cells are wetted and crowded by neighboring cells while the upper exposed cells form the menisci. The schematic is conceptual and not experimental data.

2 Theoretical model

2.1 Geometry and spatial fields

We use a one-dimensional coordinate x measured inward from the instantaneous swarm edge. The model is intended as a local description of a slowly curved front, so radial curvature is neglected over the width of the peripheral monolayer. Let d denote the characteristic cell diameter, l the cell length, η(x) a reduced interfacial-exposure field for the meniscus-forming cell, and φ(x) the local two-dimensional packing fraction of cells in the surface layer. Here η=0 denotes vanishing far-field exposure and larger η denotes stronger meniscus-forming exposure.

Published swarming studies show that hydration, interfacial contact, and density can vary over tens to hundreds of micrometers near the advancing front [7]. To avoid building conclusions around a specific functional form, we use smooth monotonic profiles that represent only the existence of such gradients. Because water is supplied from beneath the agar by wetting, we do not assume a continuous overlying film; instead the reduced interfacial-exposure field is written

$$\eta(x) = \eta_e e^{-x/L_h} \tag{1}$$

and the packing fraction is written

$$\phi(x) = \phi_e + (\phi_\infty - \phi_e)\left[1 - e^{-x/L_\rho}\right] \tag{2}$$

Here η_e is the edge value, η_∞=0 by construction, and L_h and L_ρ are spatial relaxation lengths. The packing fraction increases from φ_e toward φ_∞. These equations are constitutive profiles rather than fitted measurements; alternative monotonic profiles produce the same qualitative transition as long as interfacial exposure decreases and/or packing increases systematically with distance from the edge.

2.2 Capillary confinement stress

For a bacterium that is partially wetted and meniscus-forming, the characteristic magnitude of a capillary force is set by surface tension multiplied by a contact-line length. For a cell cross-section of diameter d, this gives F_{cap} on the order of πdγ, where γ is the liquid-air surface tension. Exact values depend on contact angle, interface shape, and cell geometry and can be obtained from a Young-Laplace calculation [8,13]. Because the present paper focuses on the competition that selects the spatial transition rather than on repeating the full Young-Laplace solution, we write the normal confinement in reduced form

$$F_{\mathrm{cap}}(x) = \pi d\gamma\, w\, \Phi[\eta(x)] \tag{3}$$

where 0<w≤1 is an effective wetting/geometric factor and Φ describes how strongly a cell intersects the interface. Dividing by a projected cell area of order ld yields a capillary stress scale

$$\sigma_\gamma(x) = \frac{\pi\gamma}{l}\, w\, \Phi[\eta(x)] \tag{4}$$

The key physical requirement is Φ→0 when the cell no longer appreciably forms a meniscus and Φ increases with stronger meniscus-forming exposure. For numerical calculations we use the minimal exposure law

$$\Phi(\eta) = \eta^m, \qquad 0 \le \eta \le 1 \tag{5}$$

with baseline exponent m=1 and sensitivity calculations spanning m=0.5-2. This function is not presented as a closed-form Young-Laplace solution. It is a reduced constitutive law that preserves the experimentally and theoretically established dependence of capillary confinement on immersion. The use of several m values tests whether predictions depend on the exact interpolation.

2.3 Active stress and extrusion criterion

Cells in a swarming monolayer generate and transmit in-plane stress through growth, collisions, motility, transient jamming, and constraints on expansion. Rather than assign these contributions separately, we define an effective active compressive stress σ_a that increases once the local packing fraction exceeds a threshold φ_0. A convenient form is

$$\sigma_a(x) = \sigma_{a,\infty}\left[\frac{\max(\phi(x)-\phi_0,0)}{\phi_\infty-\phi_0}\right]^n \tag{6}$$

where $\sigma_{a,\infty}$ is the interior active-stress scale and n controls how sharply stress rises with crowding. A baseline non-capillary restoring stress σ_0 represents cell-substrate adhesion, substrate deformation, and other vertical constraints that remain even when the interface no longer contacts the cell.

We define the extrusion number

$$E(x) = \frac{\sigma_a(x)}{\sigma_0+\sigma_\gamma(x)} \tag{7}$$

The monolayer is mechanically favored for E<1. The first position at which the active load reaches the restoring scale defines the predicted onset of multilayering

$$x^* = \inf\{x \geq 0 : E(x) \geq 1\} \tag{8}$$

This criterion is a continuum counterpart of the torque-balance picture used for single-cell verticalization. In a small-angle description, an in-plane force produces a lifting torque proportional to the load and cell length, whereas substrate and capillary interactions provide restoring stiffness. Adding capillary confinement therefore raises the critical load for rotation near a sufficiently thin interface. Equation (7) expresses the same competition at coarse-grained stress level without assuming a particular microscopic adhesion law.

2.4 Nondimensional form

Dividing Eq. (7) by the asymptotic active stress highlights the controlling groups. We define

$$\Pi_0 = \frac{\sigma_0}{\sigma_{a,\infty}} \qquad \Pi_\gamma = \frac{\pi\gamma}{l\sigma_{a,\infty}}$$

$$E(x) = \frac{G[\phi(x)]}{\Pi_0 + \Pi_\gamma w \Phi[\eta(x)]} \tag{9}$$

where G is the dimensionless crowding function in Eq. (6). The ratio Π_γ compares the capillary stress scale with the maximum active stress. The theory predicts that monolayer stabilization is strongest when Π_γ is large, the edge is underfilled (η_e<1), and active compression grows slowly with x.

2.5 Numerical implementation and parameter choices

Equations (1)-(9) were evaluated on a one-dimensional grid using Python/NumPy. No experimental observations were fit. The baseline parameters were chosen to be physically plausible for micron-scale rod-shaped bacteria and to place the transition within a near-edge region that can be imaged experimentally. Accordingly, absolute values should be interpreted as an illustrative prediction rather than a measurement. The more important results are the direction and magnitude of parameter dependencies.

Parameter	Baseline	Explored range	Interpretation
Cell length, l	5 μm	3-8 μm	Sets conversion of γ to capillary stress
Surface tension, γ	35 mN m−1	15-75 mN m−1	Capillary stress amplitude
Wetting/geometric factor, w	0.80	0.45-1.0	Fraction of capillary force projected into confinement
Edge interfacial exposure, η_e	0.85	0.35-1.00	Strong meniscus-forming

Parameter	Baseline	Explored range	Interpretation
			exposure near edge
Interior interfacial exposure, η_∞	0.00	fixed	Far-field interface reference
Exposure-decay length, Lh	80 µm	40-150 µm	Spatial decay of meniscus-forming exposure
Edge packing fraction, φ_e	0.40	0.3-0.5	Low-density edge
Interior packing fraction, φ_∞	0.90	0.75-0.95	Crowded interior
Stress-onset packing, φ_0	0.52	0.45-0.65	Onset of crowding stress
Active stress, $\sigma_{a,\infty}$	35 kPa	20-70 kPa	Maximum in-plane loading
Baseline restoring stress, σ_0	4 kPa	2-10 kPa	Non-capillary confinement
Capillary exponent, m	1.0	0.5-2.0	Shape of immersion dependence
Active exponent, n	1.5	1-3	Nonlinearity of crowding stress

Table 1. Baseline values and ranges used for theoretical exploration. Values are not fitted to new experimental data.

3 Results

3.1 A capillary-stabilized peripheral monolayer emerges from competing gradients

The baseline calculation produces three distinct regions. At the edge, η<1 and the capillary contribution is approximately 11 kPa, while the active stress is negligible because packing remains below the crowding threshold. Consequently E is far below unity and the model strongly favors a monolayer. Moving inward, φ increases and σ_a rises, while η approaches unity and σ_γ decreases. The two trends act cooperatively to increase E.

For the baseline parameter set the active and restoring stresses cross at x^*=70 µm. The interface is still partially coupled to the cells at the crossing (η≈0.82), which is important: the transition does not require capillary confinement to vanish completely. It occurs when the relative confinement is no longer sufficient to resist the growing in-plane load. Farther inward, η exceeds unity in the reduced model, σ_γ falls to zero, and the multilayered state is favored by the remaining imbalance between active and baseline restoring stresses.

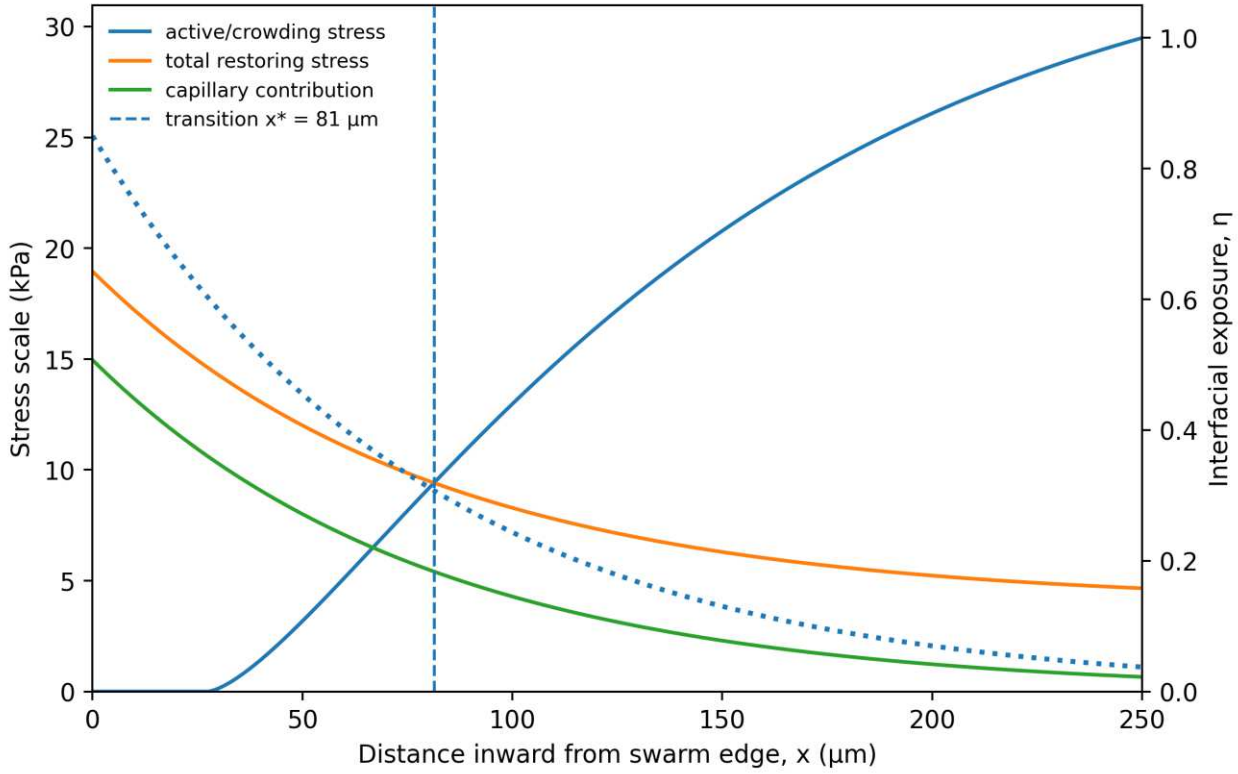


Figure 2. Baseline spatial profiles. Active/crowding stress rises inward while capillary confinement decreases as the normalized film thickness $\frac{h}{d}$ increases. The total restoring stress is $\sigma_0 + \sigma_\gamma$. Their first crossing defines the predicted transition x^* (approximately 70 μm for the baseline parameter set).

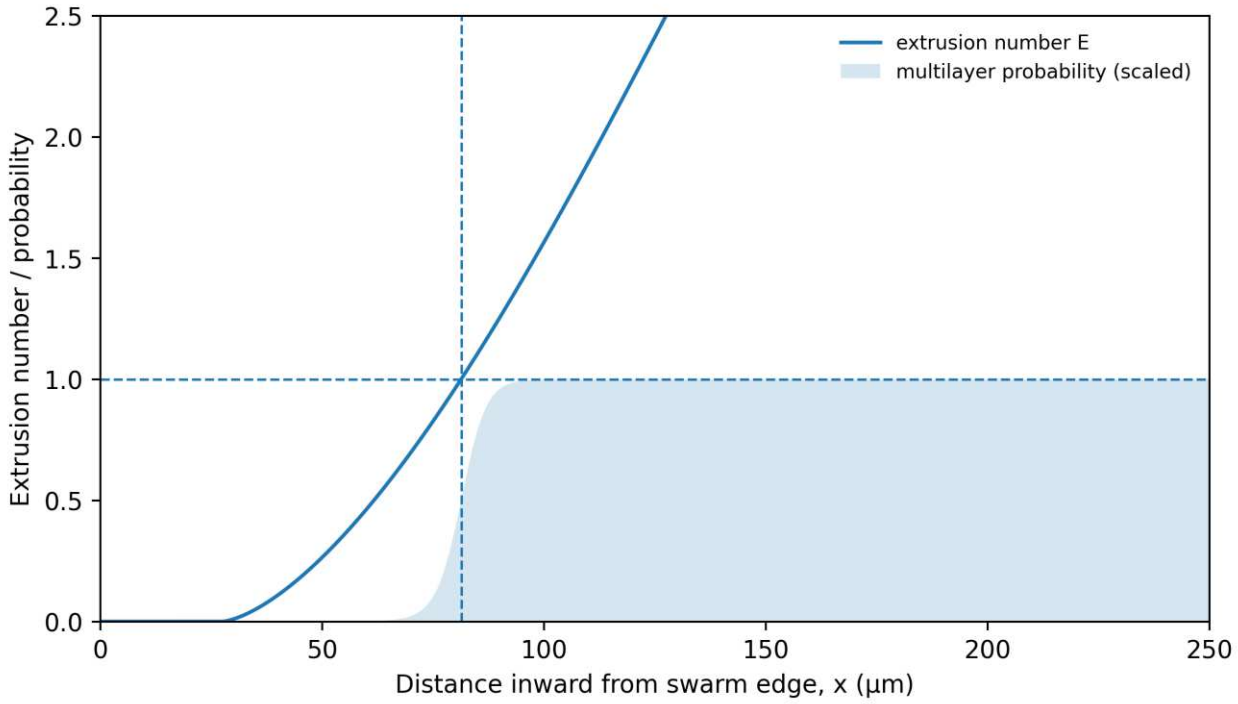


Figure 3. Extrusion number $E = \frac{\sigma_a}{\sigma_0 + \sigma_\gamma}$ for the baseline simulation. The monolayer is favored for $E < 1$ and multilayering for $E > 1$. The smooth multilayer probability is a visualization of heterogeneous thresholds, implemented as a logistic function centered on $E = 1$; it is not fitted to data.

3.2 Surface tension and hydration move the transition in opposite directions

The model makes a direct prediction for interfacial perturbations. Increasing γ strengthens the capillary stress at every partially immersed location, so a larger inward active stress is required before E reaches unity. At fixed hydration, raising γ from 20 to 70 mN m−1 moves the predicted transition from about 65 to 80 μm behind the edge. Thus higher surface tension should widen the monolayer annulus, all else equal.

Hydration has the opposite effect. Increasing η_∞ thickens the film and reduces the interval over which cells significantly intersect the liquid-air interface. In the baseline family of profiles, increasing η_∞ from 0.8 to 1.5 moves x^* from about 86 to 59 μm. The combined surface-tension/hydration phase map shows that a given monolayer width can arise from different combinations of γ and η_∞, but the directions of the two controls are distinct. This provides an experimental route to separate capillary confinement from purely density-driven multilayering: surface-tension perturbation should move the boundary even at matched cell density profiles.

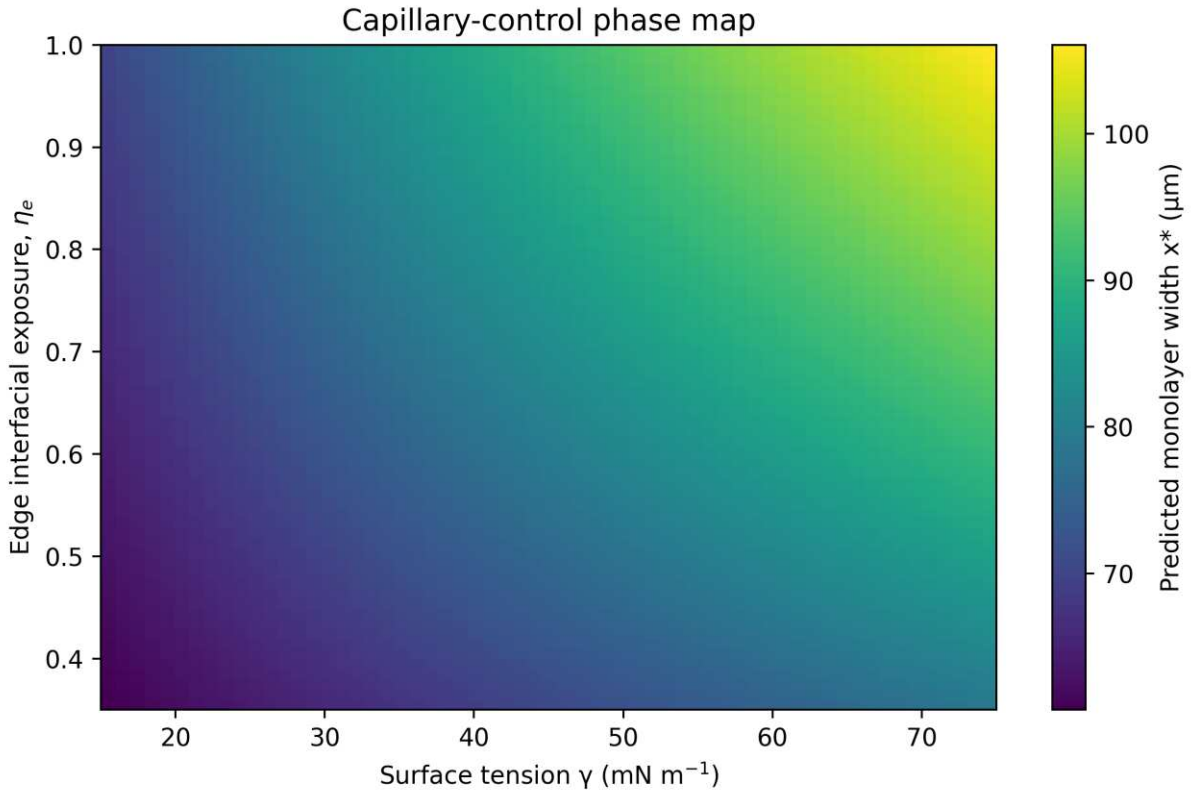


Figure 4. Predicted monolayer width x^* as a function of surface tension and interior normalized film thickness. Higher surface tension stabilizes the monolayer, whereas a thicker film weakens interfacial confinement and shifts the multilayer boundary toward the edge.

3.3 Increased active stress promotes earlier vertical escape

Increasing the asymptotic active stress shifts the transition toward the edge. In the simulations, increasing $\sigma_{a,\infty}$ from 20 to 70 kPa moves x^* from approximately 88 to 55 μm. This is consistent with observations that local obstruction, slowing, or crowding can trigger multilayer islands in swarms [10], but the capillary theory adds a further prediction: the magnitude of that shift should depend on hydration and surface tension.

The dependence on the capillary exposure exponent m is quantitative rather than qualitative. Over m=0.5-2, x^* spans approximately 86-59 μm while preserving the same ordering of surface-tension, hydration, and active-stress effects. The transition therefore does not rely on the particular linear exposure function used in the baseline model.

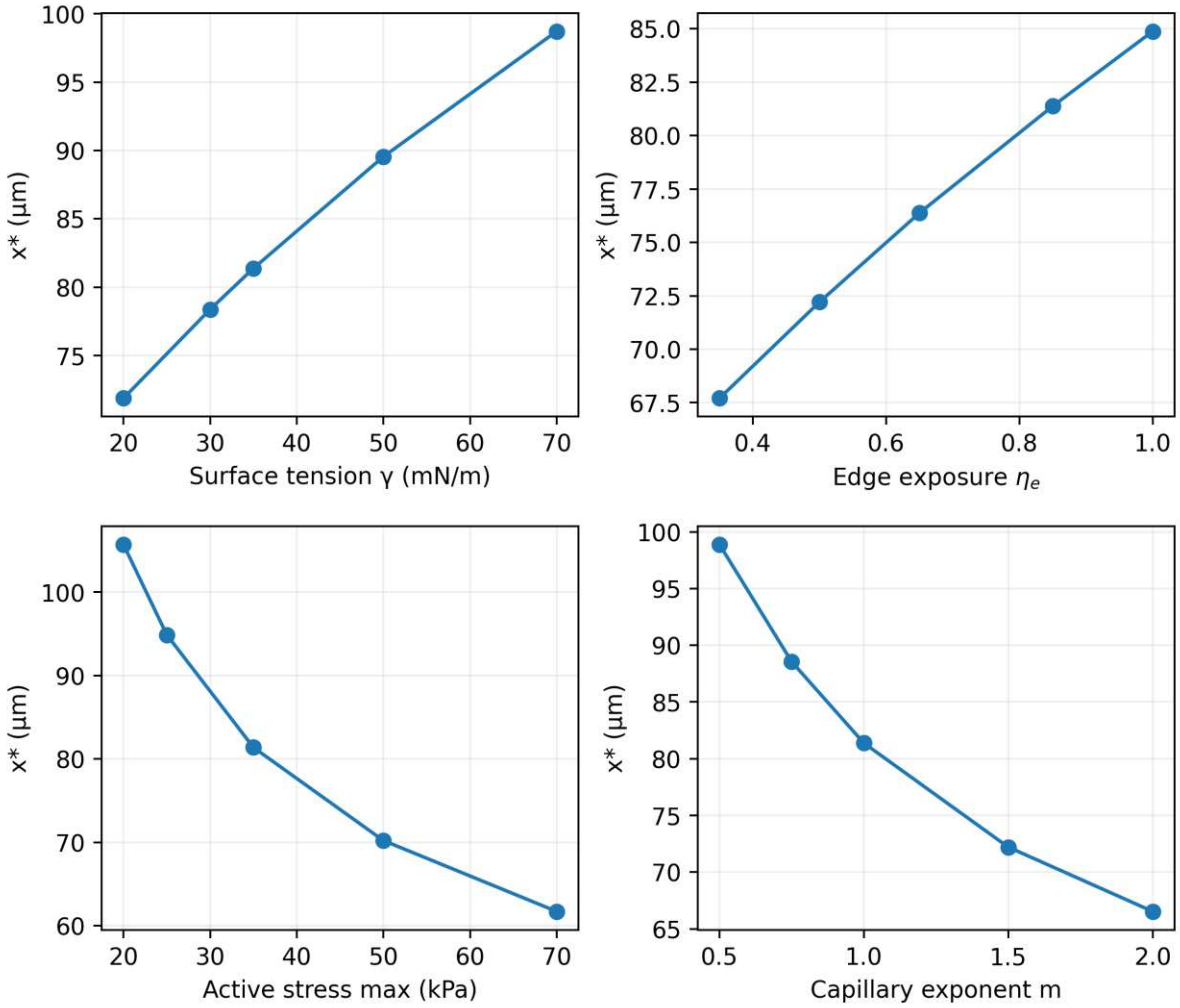


Figure 5. Sensitivity of the predicted transition position to surface tension, edge interfacial exposure, maximum active stress, and the exponent m controlling the reduced capillary exposure law. The directions of the first three effects are robust across the tested range.

3.4 Parameter uncertainty broadens but does not eliminate the predicted transition

To test whether the baseline crossing is a fine-tuned outcome, we sampled 1,200 parameter sets with broad uncertainty in γ, η_∞, $\sigma_{a,\infty}$, w, and m. The transition remained finite for the sampled cases. The median x^* was 69.7 μm, with a 5th-95th percentile interval of approximately 58.0-94.6 μm. The distribution is asymmetric because strong capillary confinement can delay the crossing more effectively than weak confinement can move it toward x=0 once the edge remains below the packing threshold. This calculation should not be interpreted as a statistical confidence interval for a real biological system; it is a robustness analysis of the model structure.

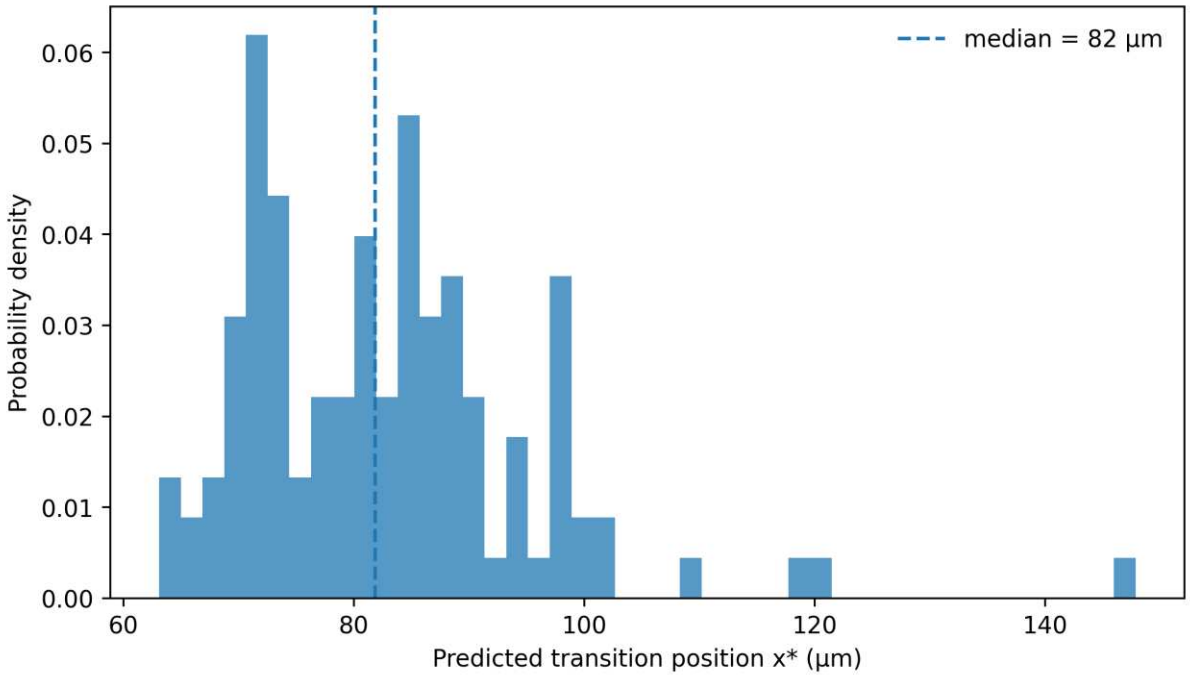


Figure 6. Monte Carlo robustness analysis for 1,200 theoretical parameter sets. The distribution shows the model-predicted transition position under broad parameter uncertainty; it is not an experimental confidence interval.

4 Experimentally testable predictions

A useful theoretical mechanism must generate predictions that distinguish it from alternatives. The capillary-confinement model makes the following tests particularly direct.

1. **Surface-tension perturbation at fixed hydration.** Reducing γ should shift the multilayer boundary toward the edge, while increasing γ should widen the monolayer region. A crowding-only model has no direct γ dependence unless γ changes motility or density indirectly.
2. Hydration perturbation at matched cell density. Increasing film thickness should weaken confinement and move x^* toward the edge. The key comparison is at similar surface coverage, because hydration can also alter motility.
3. **Local rather than global control.** A spatially patterned surfactant or wetting perturbation should locally deform the multilayer boundary. The boundary should follow the imposed interfacial field rather than remain strictly concentric.
4. **Coupled measurement of $\frac{h}{d}$ and x^*.** If the capillary mechanism is important, the transition should preferentially occur where a top exposed cell still forms a measurable meniscus, not only after capillary contact has already vanished.
5. Rescue by surface tension. A crowding-induced shift of x^* toward the edge should be partially reversed by increasing interfacial tension, provided the film remains thin enough for cells to contact the interface.
6. **Species/environment dependence.** Systems that are fully submerged should lose the capillary term and recover a substrate/crowding-controlled verticalization criterion, whereas partially hydrated systems should exhibit stronger sensitivity to wetting and surface tension.

5 Discussion

The central proposal is that the liquid-air interface should be treated as a mechanical boundary condition for a bacterial swarm rather than merely as the upper boundary of a lubricating liquid film. This distinction matters whenever the film is comparable to or thinner than a bacterial diameter. In that regime, interfacial deformation can generate normal forces on the same qualitative scale as other micron-scale mechanical interactions [8,9]. Because hydration changes with position and time, the vertical restoring force can itself be spatially structured.

The model complements, rather than replaces, stress-driven theories of multilayering. In the framework of You et al. [11] and Beroz et al. [12], in-plane compression provides the destabilizing load and cell-surface interactions provide restoring mechanics. Capillary confinement adds a second, environment-dependent component to that restoring term. In the framework of Grobas et al. [10], local slowing and density accumulation increase the active load. The present theory predicts when that load is sufficient to produce vertical escape in the presence of a nearby free interface. These mechanisms can therefore act simultaneously.

The model also connects two bodies of literature that are often treated separately: capillarity in thin bacterial films and mechanical verticalization of dense bacterial populations. Gudavadze and Florin [8] established how normal capillary force depends on immersion in a thin film, while Black et al. [9] demonstrated that bacterial menisci can reorganize collective states through capillary interactions. The new step here is to use capillary confinement as a spatially varying threshold term that can select where a moving swarm changes dimensionality.

One consequence is that surfactin can have mechanically competing effects. Lowering surface tension assists wetting and spreading [5,6], but in the present model the same reduction in γ weakens the normal capillary barrier to vertical displacement. The net effect on multilayering therefore depends on whether surfactin-driven spreading reduces density faster than reduced γ lowers confinement. This provides a natural explanation for why a single biochemical control variable can influence both lateral expansion and vertical architecture without requiring a dedicated verticalization program.

The dimensionless group $\Pi_\gamma = \frac{\pi\gamma}{l\sigma_{a,\infty}}$ provides a useful organizing parameter. Large Π_γ favors two-dimensional motion in partially exposed films, whereas small Π_γ favors escape into the third dimension once crowding develops. Because Π_γ decreases with increasing cell length, the model also predicts that longer cells are less strongly confined in stress units at fixed γ, an effect that can interact with the geometry-dependent verticalization pathways reported in biofilms [12].

6 Limitations

The model is intentionally minimal and should not be confused with a validated quantitative description of a specific B. subtilis strain. First, the capillary exposure function Φ is a reduced law rather than a fresh solution of the Young-Laplace equation. A higher-fidelity paper could replace Eq. (5) with tabulated forces computed directly for a rod or spherocylinder using measured contact angles and film thicknesses. Second, cell density, liquid thickness, and surface tension are prescribed fields rather than dynamically coupled variables. In real swarms, surfactant production, wetting-driven water recruitment, motility, and density feed back on one another [5,6]. Third, the active stress is phenomenological and subsumes growth, motility, collisions, and confinement. Fourth, the model predicts the onset of vertical escape, not the subsequent three-dimensional architecture.

These limitations are also a guide to validation. The theory can be rejected if measured x^* fails to respond to γ or $\frac{h}{d}$ after controlling for density, if multilayering begins where cells are fully submerged and capillarity is negligible, or if measured capillary stresses are far below the relevant mechanical loads. Conversely, simultaneous measurements of interfacial exposure $\eta(x)$, $\gamma(x)$, $\varphi(x)$, and x^* would make the theory substantially more constrained and could convert the present scaling model into a predictive one.

7 Conclusions

A spatial monolayer-to-multilayer transition in bacterial swarms can arise naturally from competition between increasing active compression and decreasing capillary confinement. The resulting extrusion number $E = \frac{\sigma_a}{\sigma_0 + \sigma_\gamma}$ provides a compact criterion for when cells should escape from a quasi-two-dimensional state. The theory predicts that higher surface tension stabilizes a wider monolayer, greater hydration promotes earlier multilayering, and stronger active stress moves the transition toward the advancing edge. These predictions are robust to uncertainty in the exact capillary exposure law and can be tested directly with controlled wetting and surface-tension perturbations. The main contribution is therefore a falsifiable bridge between thin-film capillarity and the dimensionality of active bacterial matter.

Declarations

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Competing interests: The author declares no competing interests.

Ethics approval: Not applicable. No human participants, animals, or new biological experiments were used.

Data availability: No new experimental data were generated or analyzed. Numerical outputs are generated from the equations and parameter ranges reported in the manuscript.

Code availability: Python code reproducing the theoretical figures and parameter sweeps is supplied as Supplementary Code S1 with the manuscript.

Author contributions: I.G. conceived the theoretical framework, developed the model, performed the numerical analysis, prepared the figures, and wrote the manuscript.

Clinical trial number: Not applicable.

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Supplementary material description

Supplementary Code S1 (capillary_confinement_model.py) reproduces Figures 1-6 and exports the Monte Carlo transition-position sample. The code uses only NumPy and Matplotlib and is deterministic apart from the explicitly seeded uncertainty analysis. No external or proprietary data are required.