

Active transport as a mechanism of microphase selection in biomolecular condensates

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The size and organization of biomolecular condensates formed by liquid-liquid phase separation are set by multiple cellular mechanisms that are not yet fully understood. Here, we identify a transport-driven mechanism: Stochastic binding of phase-separating proteins to cytoskeletal motor proteins, followed by active redistribution along filament networks, generates an effective long-range repulsion that arrests coarsening and selects a finite condensate size. A minimal diffusion-transport model, analyzed by linear stability theory and three-dimensional simulations, reveals a transition from macroscopic to microphase separation at remarkably low binding/release fractions, corresponding to minute motor-bound populations. Tuning motor binding rates b or transport velocities enables sublinear control of condensate sizes ($L \sim b^{-1/4}$) from a few hundred nanometers up to the micron scale. The selected length scale is robust to the intrinsic shot noise of the binding-release reactions. In anisotropic cytoskeletal environments, transport asymmetry drives morphological transitions from spherical to cylindrical condensates, independently of the thermodynamic parameters. This mechanism provides a versatile, spatiotemporally programmable route to condensate organization and informs the design of synthetic active emulsions with tunable architectures.

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I. INTRODUCTION

Eukaryotic cells compartmentalize their biochemical activities through both membrane-bound organelles (e.g., mitochondria, lysosomes) and membraneless assemblies (e.g., nucleoli, centrosomes, stress granules). Many of the latter arise via liquid-liquid phase separation (LLPS), forming dynamic, liquidlike condensates composed of proteins and RNA [1–5]. These condensates enable reversible, spatially confined reactions without enclosing membranes. Conversely, dysregulation of condensate dynamics or aging can trigger irreversible amyloid fibril formation [6,7], a hallmark of neurodegenerative diseases such as Alzheimer's, Parkinson's, and Amyotrophic lateral sclerosis (ALS).

Under physiological conditions, dilute and condensed phases coexist near equilibrium [8–10], exhibiting slow coarsening dynamics governed by fusion and Ostwald ripening [11]. Left unchecked, this slow relaxation would ultimately yield a single macroscopic droplet. To arrest coarsening and stabilize finite-sized condensates, cells exploit various nonequilibrium mechanisms [12]. Chemical reaction-diffusion cycles, such as kinase-phosphatase or autocatalytic

conversions, can generate interfacial fluxes that balance diffusive loss and select a finite length scale [13–26]. Diffusiophoretic forces arising from ion gradients [27], macromolecular crowding [28], protein oligomerization [29,30], long-range electrostatic repulsion [31], Pickering emulsion mechanism [32], or elastic confinement by the cytoskeleton [33–36] can also impede droplet growth. More recently, coupling a phase-separating species to a self-straining cytoskeletal network has been shown to arrest coarsening mechanically [37], though the broader role of stochastic motor-mediated transport in regulating condensate sizes remains largely unexplored.

Many proteins and RNA molecules that are constituents of phase-separated condensates are transported by motor proteins to defined subcellular locations [38,39]. The microtubule-associated motor protein dynein colocalizes with stress granules, which help to protect cells during acute stresses [40]. Inhibiting dynein reduces stress granule formation and delays their disassembly once the stress subsides [40]. Knockdown of dynein likewise inhibits stress granule formation, whereas knockdown of kinesin delays their dissolution [41]. A motor-based transport system for RNA protein complexes and protein condensates has been realized in cells and this could be a starting point for understanding how transport affects phase behavior [42].

Here, we show that motor-mediated transport along cytoskeletal filaments is sufficient on its own to arrest condensate coarsening and stabilize finite domain sizes. In contrast to chemical reaction-diffusion mechanisms, motor binding does not alter the chemical properties of proteins and their molecular interactions. Starting from a minimal diffusion-transport model, we derive an analytical criterion for the onset

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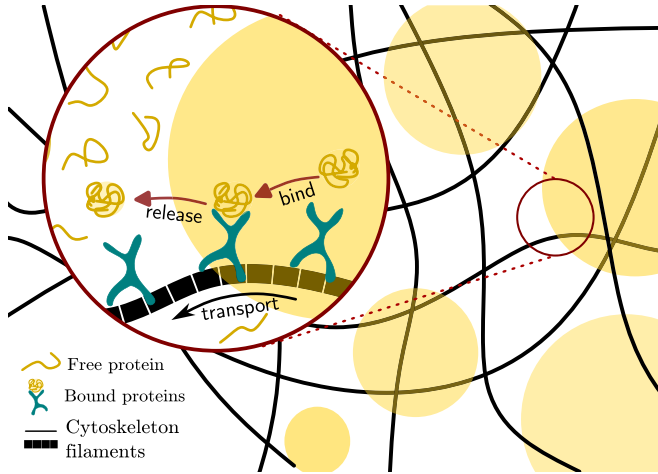


FIG. 1. Schematic cartoon of the proposed active transport mechanism to control droplet sizes. Magnified view: Phase-separating proteins (yellow) exchange between a free diffusive state and a motor-bound transported state at rates b and s . Motor-mediated transport along cytoskeletal filaments (black) spatially redistributes bound proteins, creating an effective long-range repulsion that arrests coarsening at finite length scale.

of microphase separation and obtain a scaling law that directly connects condensate dimensions to motor binding rates and transport velocities. Three-dimensional simulations confirm these predictions across a wide range of noise levels and quench depths, and further reveal that cytoskeletal anisotropy extends this control from size to shape, driving transitions between spherical, cylindrical, and lamellar morphologies. A schematic cartoon is shown in Fig. 1.

II. MODEL AND PHYSICAL INTUITION

Our model is inspired by the Foret model for membrane rafts, which shows how actively driven lipid turnover can confine domain sizes in phase-separating lipid membranes [43]. In that model, lipid molecules are stochastically removed from or inserted into the membranes at fixed rates. This process turns out to be mathematically equivalent to introducing an effective long-range repulsion between like species [44,45], which arrests coarsening and stabilizes microdomains. Here, we develop a three-dimensional bulk analog of this mechanism. Proteins interconvert between a free state, ρ_1 , and a transported state, ρ_2 , in which they are actively carried along cytoskeletal filaments by motor proteins, with binding and release rates b and s . In this context, active transport plays a role analogous to stochastic removal and insertion. Motor-driven redistribution actively homogenizes the bound protein fraction, again generating effective long-range interactions. The coupled dynamical equations are

$$\partial_t \rho_1 = M \nabla^2 \left(\frac{\delta \mathcal{F}}{\delta \rho_1} \right) - b \rho_1 + s \rho_2 + \eta_e, \quad (1)$$

$$\partial_t \rho_2 = \nabla \cdot \Lambda \nabla \rho_2 + b \rho_1 - s \rho_2 - \eta_e. \quad (2)$$

Equation (1) describes the diffusive relaxation of free proteins driven by the chemical potential gradient $\nabla \delta \mathcal{F} / \delta \rho_1$, with Onsager mobility M , together with binding/unbinding

exchange with rates b and s ; the accompanying chemical noise η_e is modeled via Langevin dynamics [46]. Equation (2) captures the active transport of motor-bound proteins. We consider bidirectional random transport along a disordered network of filaments [47,48], which can be coarse grained as rapid, anisotropic diffusion with an effective diffusion tensor Λ [49,50]. In an isotropic cytoskeletal network, we have $\Lambda = \lambda \mathbf{I}$, whereas filament alignment leads to anisotropic transport described by a uniaxial diffusion tensor with eigenvalues $\lambda_{\parallel} > \lambda_{\perp}$, reflecting enhanced mobility along filament bundles [48,50,51]. For analytical tractability, we assume isotropy in the scaling and linear stability analyses, and explore the effects of anisotropic transport by numerical simulations. The free-energy functional, $\mathcal{F}[\rho]$, depends on the total density $\rho = \rho_1 + \rho_2$ via

$$\frac{\mathcal{F}[\rho]}{\rho_0 k_B T} = \int_V d^3 r \left[\frac{\tau_2}{2\rho_0^2} (\rho - \rho_c)^2 + \frac{\tau_4}{4\rho_0^4} (\rho - \rho_c)^4 + \frac{g}{2\rho_0^2} |\nabla \rho|^2 \right], \quad (3)$$

where τ_2 and τ_4 control bulk thermodynamics, $\rho_0 = \bar{\rho}$ and ρ_c are the averaged total density and the critical density, V is the system volume, and g sets the interfacial stiffness. The noise accounts for the discrete nature of stochastic binding/unbinding events and is modeled by chemical Langevin dynamics [46] (see also Appendix A 1)

$$\eta_e(\mathbf{r}, t) = \sqrt{b\rho_1(\mathbf{r})} \zeta_1(\mathbf{r}, t) - \sqrt{s\rho_2(\mathbf{r})} \zeta_2(\mathbf{r}, t), \quad (4)$$

where $\zeta_i(\mathbf{r}, t)$ are uncorrelated white noise fields (Itô interpretation [[52], Sec. 2.3, p. 40]) with $\langle \zeta_i \rangle \equiv 0$ and $\langle \zeta_i(\mathbf{r}, t) \zeta_j(\mathbf{r}', t') \rangle = \delta_{ij} \delta(\mathbf{r} - \mathbf{r}') \delta(t - t')$.

Simple scaling arguments can provide intuition on the characteristic domain size. Balancing binding kinetics (binding-release rate, b and s), free-protein diffusion ($M k_B T / \rho_0$), and transport (λ) yields two distinct regimes: (1) In the *exchange-limited* regime, transport processes are fast enough to be quasi-instantaneous. Molecules are redistributed rapidly and become available to participate in LLPS elsewhere. Thus, domain growth is limited by the competition between binding kinetics and structural relaxation, which is set by the Cahn-Hilliard ∇^4 dissipation. A fluctuation of size L relaxes on a timescale $t_M \sim (L^4 \rho_0) / (M g k_B T)$. Matching the binding time, $t_b \sim 1/b$, gives

$$L \sim l_e := \left(\frac{M k_B T g}{\rho_0 b} \right)^{1/4}. \quad (5)$$

(2) In the *diffusive* regime, transport is too slow to ensure complete redistribution of proteins. If free proteins bind and get released within one droplet, the droplet growth is not arrested. Bound molecules travel a characteristic distance $\Delta l_a = \sqrt{\lambda/s}$. The transition between the two regimes is thus expected at $l_e \sim \Delta l_a$ or

$$\kappa := \frac{l_e}{\Delta l_a} = \left(\frac{M k_B T g s^2}{\rho_0 b \lambda^2} \right)^{1/4} \approx 1. \quad (6)$$

For a more quantitative analysis, we first cast the equations in dimensionless form, using scaled lengths $\hat{r} = r/\ell_0$ and times $\hat{t} = t/t_0$, with $\ell_0 = \sqrt{g}$ and $t_0 = (g \rho_0) / (M k_B T)$

and defining the order parameters, $\phi_1 = (\rho_1 - \bar{\rho}_1)/\rho_0$ and $\phi_2 = (\rho_2 - \bar{\rho}_2)/\rho_0$, with $\bar{\rho}_1 = \rho_0 s/(b+s)$ and $\bar{\rho}_2 = \rho_0 b/(b+s)$. In physical terms, ℓ_0 is on the order of the size of a protein, ℓ_0^2/t_0 is roughly the molecular diffusion constant of free proteins, and $\bar{\rho}_{1,2}$ correspond to the mean concentrations of unbound/bound proteins in the homogeneous fluid.

The resulting rescaled dynamic equations [Eqs. (A9) and (A10)] (see Appendix A) reveal that the behavior of the system is governed by four dimensionless quantities: The binding/release ratio $e = b/s$ setting the partitioning of molecules between the two states, the dynamic asymmetry $\hat{\lambda} = (\lambda\rho_0)/(Mk_B T)$ giving the ratio of random transport in the bound state and free diffusion in the unbound state, the scaled release rate $\hat{s} = (sg\rho_0)/(Mk_B T)$, and the noise level parameter $C_0 = g^{-3/4}/\sqrt{\rho_0}$ (see Appendix A 1). In these units, the characteristic quantities introduced above are given by $l_e = (\hat{s}e)^{-1/4} \ell_0$ and $\kappa = (e\hat{\lambda}^2/\hat{s})^{-1/4}$.

III. LINEAR STABILITY ANALYSIS AND PHASE DIAGRAM

Next, we examine the stability of the homogeneous state ($\phi_i \equiv \text{const}$) against small perturbations. It is instructive to consider first a simplified model, in which we assume that the fraction of transported molecules is small and replace $\mathcal{F}[\rho]$ by $\mathcal{F}[\rho_i]$ in the free-energy equation (3). This approximation allows us to map the noise-free part of our model onto an equilibrium model governed by purely diffusive relaxation. The key step is to represent the source and sink terms in Eqs. (1) and (2) as equivalent long-range Coulomb interactions. Similar transformations have been employed to describe arrested phase separation in the Foret model of lipid rafts [44,45] and pattern formation in certain reactive binary mixtures [13,14,53]. We first symmetrize the exchange terms by rescaling the order parameter, $\tilde{\phi} = (\phi_1, \phi_2/\sqrt{e})$. After some mathematical manipulations detailed in Appendix B 1, the dynamical equations can be rewritten as

$$\partial_t \tilde{\phi} = \hat{\nabla}^2 \frac{\delta \hat{\mathcal{F}}_t^{\text{eff}}}{\delta \tilde{\phi}} + \hat{\eta}_e(\mathbf{r}, t) \begin{pmatrix} 1 \\ -1/\sqrt{e} \end{pmatrix} \quad (7)$$

in terms of a virtual effective free-energy functional

$$\hat{\mathcal{F}}_t^{\text{eff}} = \frac{1}{2V} \sum_{\mathbf{q} \neq 0} \tilde{\phi}_{\mathbf{q}}^\dagger \mathbf{B}_{\mathbf{q}} \tilde{\phi}_{\mathbf{q}} + \int_V d^3 \hat{r} \left[-\tau_4 \phi_c \tilde{\phi}_1^3 + \frac{\tau_4}{4} \tilde{\phi}_1^4 \right], \quad (8)$$

$$\text{with } \mathbf{B}_{\mathbf{q}} = \begin{pmatrix} \tau + q^2 + e\hat{s}/q^2 & -\sqrt{e}\hat{s}/q^2 \\ -\sqrt{e}\hat{s}/q^2 & \hat{\lambda} + \hat{s}/q^2 \end{pmatrix}. \quad (9)$$

Here, $\tau = \tau_2 + 3\tau_4\phi_c^2$, with $\phi_c := \rho_c/\rho_0 - 1$, $\hat{V} = V/\ell_0^3$, and $\hat{\eta}_e$ is an uncorrelated noise field with mean zero and variance $C_0^2 \hat{s}e(\tilde{\phi}_1 + \tilde{\phi}_2/\sqrt{e} + 2/(1+e))$ (see Appendix D). In the absence of noise, the homogeneous solution of the dynamical equation (7), $\tilde{\phi} \equiv 0$, is (meta)stable whenever $\mathbf{B}_{\mathbf{q}}$ is positive definite, and becomes unstable when $\det(\mathbf{B}_{\mathbf{q}}) = 0$. This condition yields two branches of the spinodal, $\tau_{c0}(e) = -e\hat{\lambda}$ and $\tau_{c1}(e) = -2\sqrt{e}\hat{s} + \hat{s}/\hat{\lambda}$, which meet at a critical exchange ratio $e_c = \hat{s}/\hat{\lambda}^2$ (see Fig. 2). For $e < e_c$, the unstable mode occurs at $q \rightarrow 0$, corresponding to macroscopic (bulk) phase separation. For $e > e_c$, the instability appears at a finite wave number $q = q_0$, marking the onset of a microphase regime with characteristic domain size $L \sim 2\pi/q_0$.

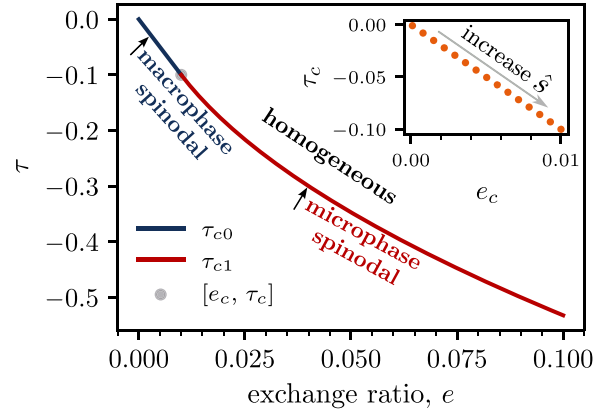


FIG. 2. Stability diagram in the (e, τ) plane. The blue branch $\tau_{c0}(e)$ is the spinodal with respect to macrophase separation, and red branch $\tau_{c1}(e)$ is the spinodal with respect to microphase separation; they meet at the critical point (e_c, τ_c) . The labels refer to the character of the instability at onset, not to a division of the two-phase region. Parameters are $\hat{s} = 1$ and $\hat{\lambda} = 10$. Inset: shift of the critical point as the scaled release rate $\hat{s}$ increases from 0.01 to 1.

The domain size L diverges at the critical point $(e_c, \tau_c) = (\hat{s}/\hat{\lambda}^2, -\hat{s}/\hat{\lambda})$, where the most unstable mode shifts from $q = 0$ to $q > 0$.

At the spinodal $\tau = \tau_{c1}$, the characteristic wave number is given by

$$q_0^2 = \ell_0^{-2}(\sqrt{e}\hat{s} - \hat{s}/\hat{\lambda}). \quad (10)$$

Expressing the domain size in terms of the quantities l_e and κ introduced in Eqs. (5) and (6), one gets

$$L = 2\pi l_e(1 - \kappa^2)^{-1/2}. \quad (11)$$

The domain size diverges for $\kappa \rightarrow 1$ and approaches $2\pi l_e$ for $\kappa \rightarrow 0$, consistent with our earlier scaling arguments. Furthermore, noting that $\kappa = (e_c/e)^{1/4}$, one can extract the power law $L \sim (2\pi l_e \sqrt{2e_c})(e - e_c)^{-1/2}$ describing how the domain size diverges upon approaching e_c .

The structure of the functional $\hat{\mathcal{F}}_t^{\text{eff}}$ in Eq. (7) closely resembles the Ohta-Kawasaki-Ito functional for homopolymer/copolymer blends [54]. Such systems exhibit both Ising-type macrophase separation and Landau-Brazovskii-type [55] microphase separation, separated by a multicritical Lifshitz point where the characteristic domain size diverges—consistent with our stability analysis. It is important to emphasize, however, that the nonequilibrium model defined by Eq. (7) is *not equivalent* to an equilibrium model with free energy $\hat{\mathcal{F}}_t^{\text{eff}}$. The stochastic term violates the fluctuation dissipation relation, meaning that even in the limit $C_0 \rightarrow 0$, globally minimizing $\hat{\mathcal{F}}_t^{\text{eff}}$ will not necessarily yield the correct steady-state phase diagram, particularly in the presence of competing metastable states.

In the full model, $\mathcal{F}[\rho]$ with $\rho = \rho_1 + \rho_2$, the dynamical matrix cannot be symmetrized; therefore, the mapping onto an equilibrium system with an effective free energy $\hat{\mathcal{F}}_t^{\text{eff}}$ of Ohta-Kawasaki type is not possible. Nevertheless, we can still perform a stability analysis. The stability boundary is obtained directly from the condition that the linearized dynamical matrix has a vanishing eigenvalue (see Appendix B 2). The

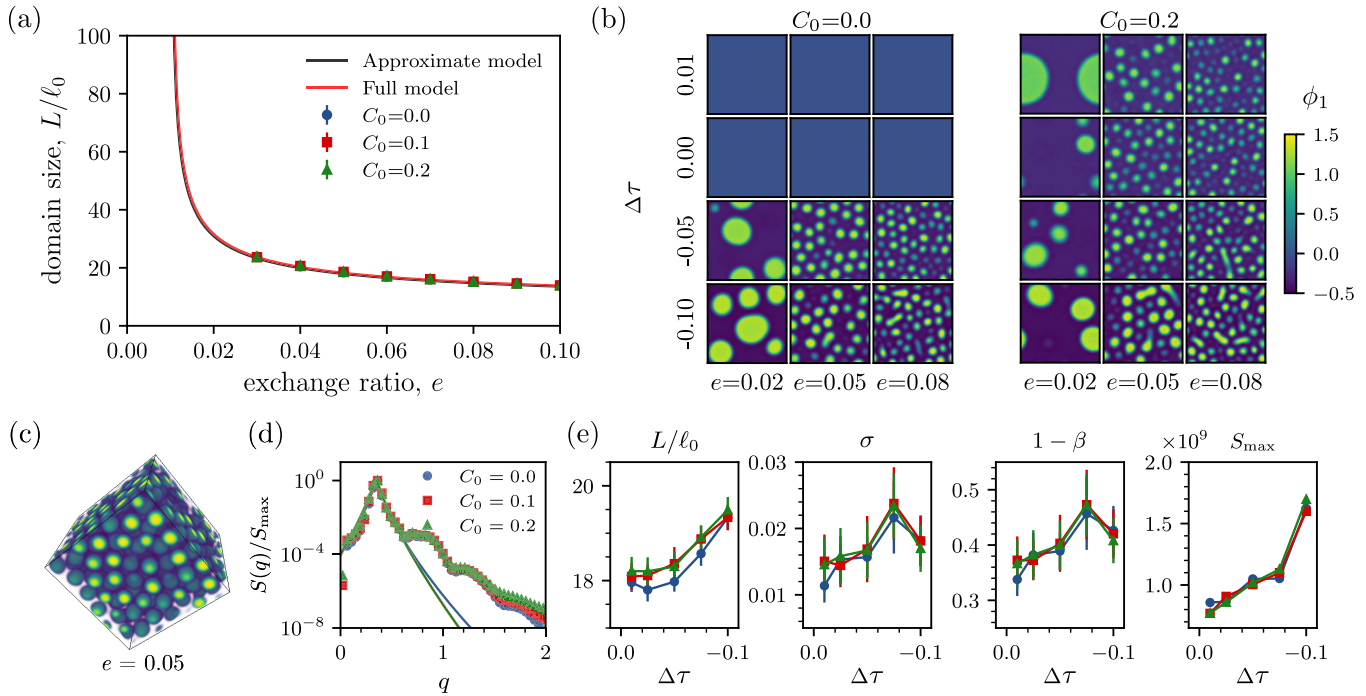


FIG. 3. Transport-driven domain selection in three-dimensional (3D) simulations. (a) Steady-state domain size L/ℓ_0 vs exchange ratio e : theory (black solid line for approximate model and red solid line for full model) and simulations with different noise strength $C_0 = 0, 0.1, 0.2$ for $\Delta\tau := \tau - \tau_{c1}^F(e) = -0.01$ (symbols). For $e = 0.02$, the domain sizes keep growing with a $\hat{t}^{1/3}$ power law at the end of the simulation (see movies in the Supplemental Material [56], and for additional data, see Fig. 8). (b) Midplane ϕ_1 slices for $e = 0.02, 0.05, 0.08$ with and without noise at time $\hat{t} = 5 \times 10^4$. Rows correspond to four values of the quench parameter $\Delta\tau = 0.01, 0.00, -0.05, -0.10$, where $\Delta\tau = 0$ corresponds to the spinodal. (c) 3D snapshot for $e = 0.05$ at time $\hat{t} = 5 \times 10^4$. (d) Angle-averaged structure factor $S(q)$ for $e = 0.05$ at $\Delta\tau = -0.01$ and noise strengths $C_0 = 0, 0.1, 0.2$, plotted in semilog representation. Solid lines are fits to the empirical form $S(q) \sim \exp\{1 - [1 + ((q - q_0)/\sigma)^2]^{1-\beta}\}$. (e) Fitted L , peak width σ , exponent $1 - \beta$, and peak amplitude $S_{\max}$ vs quench depth $\Delta\tau$ for $e = 0.05$. Parameters are $\hat{s} = 1, \hat{\lambda} = 10, \phi_c = 0.4, \tau_4 = 0.4$.

remaining algebra is unchanged and yields the spinodal

$$\tau_{c1}^F(e) = -2\sqrt{e\hat{s}} + \frac{\hat{s}(1+e)}{\hat{\lambda}}, \quad (12)$$

with the corresponding wave number $(q_0^F)^2 = \ell_0^{-2}[\sqrt{e\hat{s}} - \hat{s}(1+e)/\hat{\lambda}]$ and domain size $L^F = 2\pi/q_0^F$. Both expressions reduce to those of the simplified model if one replaces $\hat{s}(1+e) \rightarrow \hat{s}$; thus, the entire effect of retaining ϕ_2 is this single substitution. The spinodal of the full model is shifted upward by

$$\delta\tau_{c1}(e) = \tau_{c1}^F(e) - \tau_{c1}(e) = \frac{\hat{s}e}{\hat{\lambda}} > 0. \quad (13)$$

Since the transported proteins also contribute to the driving force for demixing, phase separation sets in slightly earlier. The shift vanishes as $e \rightarrow 0$ and grows linearly with e ; for the parameters used here, it amounts to $\delta\tau_{c1} = 2 \times 10^{-3}$ at $e = 0.02$ and 5×10^{-3} at $e = 0.05$, and the corresponding domain size is larger by $\delta L/L \simeq \hat{s}e/[2\hat{\lambda}(q_0\ell_0)^2] \approx 2\%$ across the range studied in the numerical simulations of the next section. The approximation underlying $\hat{\mathcal{F}}_t^{\text{eff}}$ is therefore very good in the parameter regions considered here, while becoming less so on approaching the transition point, $e = e_c$, where $q_0 \rightarrow 0$.

IV. THREE-DIMENSIONAL SIMULATIONS

To test our analytical predictions and explore the nonlinear regime, we performed direct numerical simulations. We solved the rescaled versions of Eqs. (A9) and (A10) (simulation and numerical details; see Appendix C), fully taking into account the ρ_2 contribution to the free energy $\mathcal{F}$ in Eq. (3). For comparison, we also simulated the approximate model used for the stability analysis; these results are presented in Appendix E.

A. Microphase arrest and robustness

Figure 3(a) shows that the finite- q instability quantitatively predicts the steady-state droplet size L . For large e , coarsening arrests and L remains finite, while for small e , droplets continue growing, following the characteristic $\hat{t}^{1/3}$ growth law during the whole simulation time of up to $\hat{t} = 5 \times 10^4$ (see Fig. 8), which is characteristic of macrophase separation. The transition from microphase to macrophase separation predicted by the linear theory is reproduced by the simulations, but the boundary lies at a slightly higher e than the analytic e_c . The offset is present in the simplified-model simulations as well [see Fig. 6(a)]. This observation is consistent with the phase behavior of the corresponding effective equilibrium system, where Lifshitz points are generally known to be unstable in three dimensions and preempted by a multiphase

equilibrium point in block copolymer/homopolymer mixtures [57].

Panel (b) presents midplane snapshots of ϕ_1 for $\phi_c = 0.4$, three values of e , and four quench depths $\Delta\tau := \tau - \tau_{cl}^F(e)$, comparing simulations without and with noise ($C_0 = 0$ and $C_0 = 0.2$). Without noise, no structure forms at $\Delta\tau = 0$ or above, as expected: At the spinodal, the growth rate of the critical mode vanishes; therefore, an infinitesimal perturbation is not amplified within the simulated time. Upon adding noise, droplets appear both at the spinodal and above it at $\Delta\tau = 0.01$, where the homogeneous state is linearly stable. The morphology of structures already formed at deeper quenches, however, is largely unaffected by the noise. The noise-induced structure formation can again be rationalized based on the phase diagram of the corresponding effective equilibrium system: At $\phi_c = 0.4$, corresponding to the mean density $\rho_0 = 0.71\rho_c$, the order-disorder transition is first order; the spinodal lies inside the ordered region. Therefore, noise can induce microphase separation. Furthermore, the system exhibits hysteresis: Once established, the domains do not disappear even if the noise is turned off, demonstrated for the simplified model in Appendix E2.

To quantify noise effects more systematically, we analyze the structure factor in panels (d) and (e). Panel (d) displays the normalized structure factor $S(q)/S_{\max}$ for $e = 0.05$, revealing a sharp primary peak at q_0 and secondary oscillations at larger q . These postpeak oscillations signal short-range translational order, indicating that the droplets locally adopt a quasiregular arrangement reminiscent of a soft crystal or microphase, as the three-dimensional snapshot in panel (c) illustrates. Introducing noise predominantly increases the spectral weight at large q , effectively lifting the high- q tail of $S(q)$. As shown in the Appendix D, this follows from the noise adding a q^{-4} background of amplitude $\propto C_0^2$, which dominates once the deterministic tail has decayed. Finally, panel (e) shows that deeper quenches ($\Delta\tau < 0$) modestly increase L and drive the exponent $(1 - \beta) \rightarrow 0.5$, indicating a crossover toward large- q exponential decay in $S(q)$. In this regime, noise slightly enlarges L , consistent with enhanced interfacial fluctuations. These trends are nearly independent of noise amplitude, underscoring the robustness of the transport-driven length-scale selection.

B. Anisotropic transport

While intracellular transport may appear disordered, cytoskeletal organization often leads to anisotropy in the effective diffusion of bound proteins. Cytoskeletal filament networks, even when not forming ordered bundles, can exhibit preferred orientations due to centrosomal organization or cell geometry. To investigate how such transport anisotropy affects condensate formation, we replace the scalar diffusion $\hat{\lambda}$ with an anisotropic tensor $\hat{\Lambda} = \text{diag}(\hat{\lambda}_{\parallel}, \hat{\lambda}_{\perp}, \hat{\lambda}_{\perp})$ in numerical simulations, representing enhanced transport along a preferred direction.

Transport anisotropy does not disrupt microphase separation, but can have a profound effect on the morphologies of microphase-separated domains. Figure 4 maps steady-state morphologies across the parameter space of scaled average protein concentration, $\rho_0/\rho_c = 1/(1 + \phi_c)$, and transport

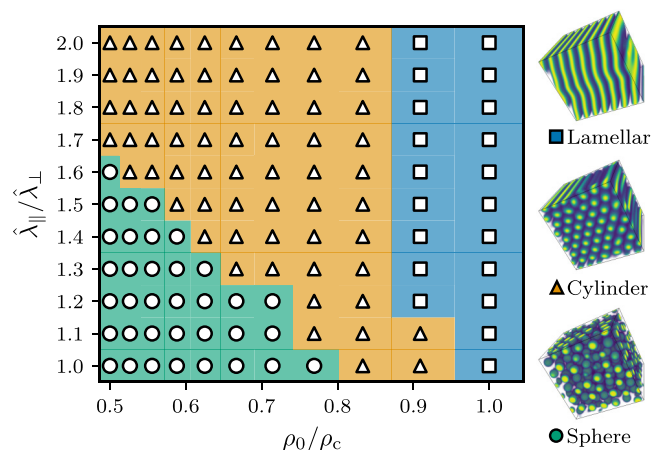


FIG. 4. Morphology phase diagram for anisotropic transport. Steady-state morphologies in $(\rho_0/\rho_c, \hat{\lambda}_{\parallel}/\hat{\lambda}_{\perp})$ space: Lamellar (squares), cylinders (triangles), spheres (circles). Increasing transport anisotropy drives sphere-to-cylinder transitions. Parameters: $\hat{s} = 1$, $\tau_4 = 0.2$, $\hat{\lambda}_{\perp} = 10$, $e = 0.06$, $\Delta\tau = -0.01$, 128^3 grid.

anisotropy, $\hat{\lambda}_{\parallel}/\hat{\lambda}_{\perp}$. The interaction parameters are chosen such that $\Delta\tau = -0.01$, slightly below the (micro)phase separation temperature in the corresponding isotropic system. All simulations were initialized from steady-state spherical droplets (obtained at $\hat{\lambda}_{\parallel} = \hat{\lambda}_{\perp}$ and $\phi_c = 0.4$) and evolved up to the time $\hat{t} = 5 \times 10^4$. We classify final morphologies as follows: (1) spheres if droplets remain approximately round; (2) cylinders if they elongate into rods aligned with the fast transport axis; and (3) lamellar if they reorganize into layered structures. Phase boundaries mark where spherical droplets become visibly elongated.

Already in the isotropic systems, cylindrical and lamellar condensate morphologies appear at near-critical protein concentrations ($\rho_0/\rho_c \sim 1$). This is to be expected, since such morphological transitions are generally observed in Landau-Brazovskii-type models [55,58]. Transport anisotropy shifts these transitions to biologically more relevant regimes of smaller protein concentrations. For example, we observe a sphere-to-cylinder transition for $\rho_0/\rho_c \sim 0.7$ at modest $\hat{\lambda}_{\parallel}/\hat{\lambda}_{\perp} \gtrsim 1.1$, shifting to higher anisotropy values as ρ_0/ρ_c decreases further. The transition mechanism involves a transport-driven morphological instability. Spherical droplets experience enhanced flux along the parallel direction, which amplifies elongation fluctuations aligned with the fast axis. The resulting cylinders minimize interfacial area in slow-transport directions while extending along the fast axis where material redistributes more efficiently. The parameter ρ_0/ρ_c , controlled by the total volume fraction of condensates, modulates these transitions. Lower ρ_0/ρ_c corresponds to greater compositional asymmetry and favors compact spherical domains over thin cylindrical domains, analogous to minority-block spheres in copolymers.

V. BIOLOGICAL AND EXPERIMENTAL IMPLICATIONS

To translate our results into physical units, we identify the molecular scales $\ell_0 = 1$ nm and $t_0 = 1$ μ s, corresponding to a free-protein diffusion constant $\ell_0^2/t_0 = 10^{-12}$ m²/s, and adopt

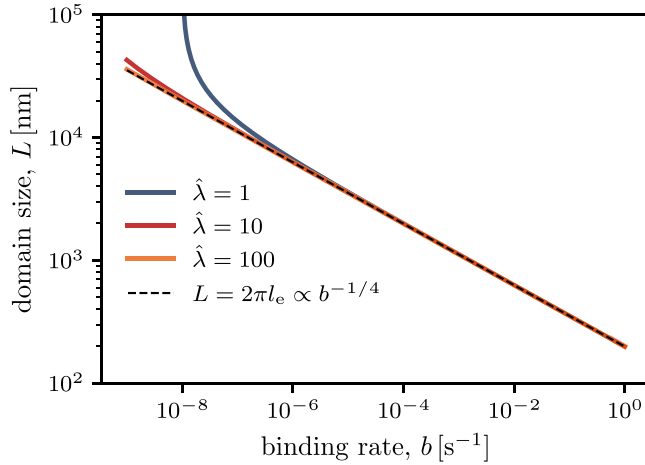


FIG. 5. Domain size L vs the binding rate b for three different values of the dynamical asymmetry $\hat{\lambda}$. The black dashed line gives the exchange-limited domain size.

a typical cargo release rate of 0.1 s^{-1} [59], giving $\hat{s} = 10^{-7}$. The dynamic asymmetry $\hat{\lambda}$ compares the effective transport of motor-bound proteins with the diffusion of free ones. Motor activity in cells generates diffusivelike motion of substantially larger amplitude than thermal diffusion [49,50]; thus, the biologically relevant regime is $\hat{\lambda} > 1$; the precise value depends on motor type and processivity.

Figure 5 shows the steady-state domain size L as a function of the binding rate b for three values of the dynamic asymmetry $\hat{\lambda}$. In the exchange-limited regime (high $\hat{\lambda}$), binding-release kinetics set the scale and $L \propto b^{-1/4}$. Because b is governed by motor abundance and cargo affinity [59,60], a hundredfold change in b shifts L by only $100^{1/4} \approx 3.2$, yet sweeping b across its physiological range moves L continuously over a broad range up to the micron scale. This sublinear sensitivity makes the selected size robust to fluctuations in motor expression while keeping it broadly tunable.

The dynamic asymmetry determines where in parameter space this control operates. Because $e_c = \hat{s}/\hat{\lambda}^2$, larger dynamic asymmetry $\hat{\lambda}$ lowers the critical exchange ratio and shifts the transition to macrophase separation to smaller binding rates. As $\hat{\lambda}$ decreases, for instance, when motor activity is reduced [61,62], the system leaves the exchange-limited regime and condensates grow larger, recovering bulk phase separation once transport becomes negligible. Size control thus rests on two distinct handles: The binding rate b sets the domain size within a given transport regime, while the dynamic asymmetry $\hat{\lambda}$ fixes the location of the critical point e_c and hence the threshold binding rate $b_c = e_c \cdot s$ above which microphase separation occurs.

The resulting sizes span the range of biomolecular condensates observed in cells, from neuronal RNA transport granules of a few hundred nanometers [63] to micron-scale bodies such as nucleoli and germline P granules [38]. The largest values in Fig. 5, of order tens of microns, reflect the critical divergence as $e \rightarrow e_c$: On approach to the critical point L grows as $(e - e_c)^{-1/2}$, which is why the $\hat{\lambda} = 1$ branch turns upward at small b . Micron-scale domains, in contrast, lie well inside the exchange-limited regime and follow the $b^{-1/4}$ law.

TABLE I. Summary of key parameters and results.

Parameter	Definition	Description
$\hat{s}$	$s g \rho_0 / (M k_B T)$	(Scaled release rate)
$\hat{\lambda}$	$\lambda \rho_0 / (M k_B T)$	(Dynamic asymmetry)
e	b/s	(Binding-release ratio)
e_c	$\hat{s}/\hat{\lambda}^2$	(Critical e for microphase onset)
l_e	$(M g k_B T / b \rho_0)^{1/4}$	(Exchange-limited domain size)
L	$2\pi l_e (1 - \sqrt{e_c/e})^{-1/2}$	(General domain size)

The mechanism thus enables both small and large condensates within a single framework.

It should be noted that Fig. 5 must be interpreted as an upper bound for domain sizes if active transport takes place, the actual condensate sizes can be lower. Domain sizes of tens of microns, as calculated for the lowest binding rates, are not possible in most cells. In this regime, the actual sizes will be constrained by additional factors such as geometry or the amount of available material.

VI. DISCUSSION

We have demonstrated that unbiased motor-mediated protein transport along cytoskeletal filaments acts as an effective long-range repulsion between proteins, shifting the LLPS spinodal and arresting coarsening at a tunable length scale. The physical origin of this repulsion is the nonlocal redistribution of motor-bound proteins: Whenever a condensate grows beyond the transport length $\Delta l_a = \sqrt{\lambda/s}$, bound proteins are carried away faster than diffusion can replenish them, creating a flux that opposes further growth. This mechanism is captured by the simple scaling law $L \propto b^{-1/4}$ (Table I), which holds across a wide range of exchange ratios and is robust to the presence of noise.

The defining feature of this mechanism is that it leaves the proteins untouched. Reaction-diffusion models of coarsening arrest [12] control size by chemically converting phase-separating molecules into a soluble, noncondensing species; here, a motor-bound protein still retains its chemical identity, fully capable of condensation once released. Size control therefore requires no engineered interaction domains or enzymatic modifications, and can in principle act on any condensate whose components can be recruited to a motor. The threshold for this regime is strikingly low. Because the critical exchange ratio $e_c = \hat{s}/\hat{\lambda}^2$ scales as the inverse square of the dynamic asymmetry between transport and free diffusion, and motors move cargo orders of magnitude faster than proteins diffuse, only a minute motor-bound fraction is needed to arrest coarsening. The accompanying sublinear sensitivity (a hundredfold change in binding rate shifts L by just a factor of ~ 3) makes the resulting size robust to fluctuations in motor expression and ATP availability.

Transport also controls shape, not just size. In a polarized cytoskeleton, anisotropic redistribution elongates spherical condensates into cylinders and lamellae once the anisotropy exceeds a modest threshold, with no change to the underlying phase diagram. This separation of geometric from thermodynamic control is, to our knowledge, distinctive: A cell

could in principle reprogram condensate morphology purely by remodeling its filament network. The prediction is directly testable, since recruiting phase-separating proteins to motors or tuning motor activity [42,61,62,64] should shift both size and shape in the predicted directions, and polarized cell types such as neurons offer a natural setting in which to look for transport-shaped condensates. The same principles suggest a design strategy for synthetic active emulsions [65], where pairing a phase-separating scaffold with a tunable motor module would yield condensates whose architecture is programmable independently of the thermodynamics.

Our work establishes a potential mechanism of size selection in biomolecular condensates: Unbiased motor-driven transport is by itself sufficient to arrest coarsening and select a finite condensate size, from a few hundred nanometers up to the micron scale. Whether some cells actually exploit it is an open question, but given the simplicity of the mechanism it seems not unlikely, and the predictions above, the size law $L \propto b^{-1/4}$, the onset threshold e_c , and the anisotropy-driven shape transitions can be tested *in vitro*. The mechanism is particularly suited to stabilize larger domains. If active, it sets an upper limit to the size of condensates, and it may act in concert with other local geometric factors or size control mechanisms that provide further constraints.

Our model isolates the transport mechanism in its simplest form, treating redistribution as effective diffusion along a disordered network. This makes the scaling laws transparent, and it also identifies the ingredients whose interplay with transport would be interesting to explore next. Directed transport, filament architecture [66], and motor processivity [67] all shape how cargo redistributes in structured cytoskeletons, and should modify the selected size and morphology in ways the present framework can accommodate with some adjustments, such as adding convective flow terms. Collective active flows from cytoskeletal contraction and microtubule sliding generate large-scale advection in the cytoplasm [68] that may reinforce or compete with the redistribution described here, and hydrodynamic coupling between condensates [27,69] becomes relevant at high volume fractions. The scaling laws derived here provide a quantitative baseline against which such effects can be measured, and a starting point for studying specific cellular implementations of transport-controlled condensate organization.

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The authors declare no competing interests.

DATA AVAILABILITY

The data that support the findings of this article are openly available in Ref. [70].

APPENDIX A: THEORETICAL MODEL

Rescaled dimensionless equations

We write the total protein concentration at position $\mathbf{r}$ as

$$\rho(\mathbf{r}, t) = \rho_1(\mathbf{r}, t) + \rho_2(\mathbf{r}, t), \quad \rho_0 = \bar{\rho} = \text{const.} \quad (\text{A1})$$

In the homogeneous steady state, all gradient terms vanish and the dynamics reduce to the reaction part, so the stationary condition is $b\bar{\rho}_1 = s\bar{\rho}_2$ (from $\partial_t \rho_i = -b\rho_i + s\rho_2 = 0$ in the uniform case). Together with the conserved total density $\bar{\rho}_1 + \bar{\rho}_2 = \rho_0$, this fixes the concentrations of the two species uniquely,

$$\bar{\rho}_1 = \rho_0 \frac{s}{b+s}, \quad \bar{\rho}_2 = \rho_0 \frac{b}{b+s}. \quad (\text{A2})$$

The coupled dynamics in the ρ variables are Eqs. (1) and (2), with the free energy $\mathcal{F}[\rho]$ of Eq. (3) and the active exchange noise

$$\eta_e(\mathbf{r}, t) = \sqrt{b\rho_1} \zeta_1(\mathbf{r}, t) - \sqrt{s\rho_2} \zeta_2(\mathbf{r}, t), \quad (\text{A3})$$

where the $\zeta_i(\mathbf{r}, t)$ are Gaussian white noise fields satisfying

$$\langle \zeta_i(\mathbf{r}, t) \rangle = 0, \quad \langle \zeta_i(\mathbf{r}, t) \zeta_j(\mathbf{r}', t') \rangle = \delta_{ij} \delta(\mathbf{r} - \mathbf{r}') \delta(t - t'). \quad (\text{A4})$$

This exchange noise is the chemical-Langevin (shot) noise of the binding/release reactions, constructed following Gillespie's chemical Langevin equation [46], and is interpreted in the Itô sense [[52], Sec. 2.3, p. 40]. It accounts for the stochastic character of binding/release events and becomes important if the concentrations of bound or free species are small, such that a treatment in terms of deterministically evolving continuous fields is no longer appropriate. The precise form was derived by Gillespie for the case of bulk reactions [46], based on the assumption that individual reactions are independent from each other. When applied to spatially varying concentration fields, the same assumption leads to the spatial delta correlation, $\delta(\mathbf{r} - \mathbf{r}')$, in Eq. (A4). We will see below that the amplitude of the noise scales with $1/\sqrt{\rho_0}$ in appropriately reduced units, such that the deterministic limit is reached at $\rho_0 \rightarrow \infty$.

Equations (1) and (2) do not include conserved noise: We retain only the athermal exchange noise, so that $\rho_0 \rightarrow \infty$ defines the deterministic baseline against which the stochastic contribution is measured. This distinction matters for Appendix D, where the fact that η_e enters outside the Laplacian is precisely what produces the q^{-4} tail, in contrast to the q^{-2} that conserved noise would give.

To derive the rescaled equations, we define the normalized order parameters

$$\phi_i(\mathbf{r}, t) = \frac{\rho_i(\mathbf{r}, t) - \bar{\rho}_i}{\rho_0}, \quad \bar{\phi}_i = 0. \quad (\text{A5})$$

Since $\partial_t \phi_i = \rho_0^{-1} \partial_t \rho_i$, replacing every ρ_i in Eqs. (1) and (2) yields

$$\partial_t \phi_1 = \frac{M}{\rho_0^2} \nabla^2 \frac{\delta \mathcal{F}}{\delta \phi_1} - b \phi_1 + s \phi_2 + \frac{\eta_e}{\rho_0}, \quad (\text{A6})$$

$$\partial_t \phi_2 = \nabla \cdot \mathbf{A} \nabla \phi_2 + b \phi_1 - s \phi_2 - \frac{\eta_e}{\rho_0}. \quad (\text{A7})$$

The noise rescales as

$$\frac{\eta_e(\mathbf{r}, t)}{\rho_0} = \frac{1}{\sqrt{\rho_0}} \left[\sqrt{b\phi_1 + \frac{bs}{b+s}} \zeta_1(\mathbf{r}, t) - \sqrt{s\phi_2 + \frac{bs}{b+s}} \zeta_2(\mathbf{r}, t) \right]. \quad (\text{A8})$$

We now rewrite these equations in terms of rescaled lengths $\hat{r} = r/\ell_0$ and times $\hat{t} = t/t_0$ with $\ell_0 = \sqrt{g}$ and $t_0 = (g\rho_0)/(Mk_B T)$. Further defining the dimensionless parameters (see main text) $e = b/s$, $\hat{\lambda} = (\lambda\rho_0)/(Mk_B T)$, $\hat{s} = (sg\rho_0)/(Mk_B T)$, $C_0 = g^{-3/4}/\sqrt{\rho_0}$, and $\phi_c = \rho_c/\rho_0 - 1$, the coupled dynamical equations simplify to

$$\partial_t \phi_1 = \hat{\nabla}^2 \frac{\delta \hat{\mathcal{F}}[\phi]}{\delta \phi_1} - e \hat{s} \phi_1 + \hat{s} \phi_2 + \hat{\eta}_e, \quad (\text{A9})$$

$$\partial_t \phi_2 = \hat{\nabla} \cdot \hat{\Lambda} \hat{\nabla} \phi_2 + e \hat{s} \phi_1 - \hat{s} \phi_2 - \hat{\eta}_e, \quad (\text{A10})$$

where

$$\phi := \phi_1 + \alpha \phi_2, \quad \alpha = \begin{cases} 0, & \text{simplified model, } \hat{\mathcal{F}}[\phi_1], \\ 1, & \text{full model, } \hat{\mathcal{F}}[\phi_1 + \phi_2], \end{cases} \quad (\text{A11})$$

is the total order parameter. All variables are dimensionless; hatted symbols denote rescaled quantities. Here, $\hat{\Lambda}$ is the rescaled transport tensor: In the isotropic limit, where the cytoskeleton forms a disordered meshwork with no preferred orientation, $\hat{\Lambda} = \hat{\lambda} \mathbf{I}$ (with $\mathbf{I}$ the identity tensor); anisotropic transport arising from aligned filament architectures is modeled by the diagonal tensor $\hat{\Lambda} = \text{diag}(\hat{\lambda}_{\parallel}, \hat{\lambda}_{\perp}, \hat{\lambda}_{\perp})$ with $\hat{\lambda}_{\parallel} > \hat{\lambda}_{\perp}$. The rescaled free-energy functional reads

$$\begin{aligned} \hat{\mathcal{F}}[\phi] &= \frac{\mathcal{F}[\rho_0(1+\phi)]}{\rho_0 \ell_0^3 k_B T} \\ &= \int_{\hat{V}} d^3 \hat{r} \left[\frac{\tau_2}{2} (\phi - \phi_c)^2 + \frac{\tau_4}{4} (\phi - \phi_c)^4 + \frac{1}{2} |\hat{\nabla} \phi|^2 \right], \end{aligned} \quad (\text{A12})$$

where τ_2 and τ_4 control the bulk thermodynamics, ϕ_c sets the critical composition, and the gradient term penalizes interfaces. Its derivative is

$$\frac{\delta \hat{\mathcal{F}}[\phi]}{\delta \phi_1} = \tau_2 (\phi - \phi_c) + \tau_4 (\phi - \phi_c)^3 - \hat{\nabla}^2 \phi. \quad (\text{A13})$$

The rescaled noise is given by

$$\begin{aligned} \hat{\eta}_e(\hat{\mathbf{r}}, \hat{t}) &= C_0 \sqrt{\hat{s}} \left(\sqrt{e\phi_1 + \frac{e}{1+e}} \hat{\zeta}_1(\hat{\mathbf{r}}, \hat{t}) \right. \\ &\quad \left. - \sqrt{\phi_2 + \frac{e}{1+e}} \hat{\zeta}_2(\hat{\mathbf{r}}, \hat{t}) \right), \end{aligned} \quad (\text{A14})$$

with $\langle \hat{\zeta}_i(\hat{\mathbf{r}}, \hat{t}) \rangle \equiv 0$ and $\langle \hat{\zeta}_i(\hat{\mathbf{r}}, \hat{t}) \hat{\zeta}_j(\hat{\mathbf{r}}', \hat{t}') \rangle = \delta_{ij} \delta(\hat{\mathbf{r}} - \hat{\mathbf{r}}') \delta(\hat{t} - \hat{t}')$. The noise amplitude $C_0 = g^{-3/4}/\sqrt{\rho_0}$ sets the strength of the fluctuations. Equations (A9)–(A14) are the equations solved in our 3D simulations.

APPENDIX B: LINEAR STABILITY ANALYSIS

1. Effective free energy for the simplified model

The dynamics of Eqs. (A9) and (A10) are not of relaxational form, because the exchange terms are not derivable from a free energy. If the transported density, ρ_2 , is omitted from the free energy, however, they can be mapped onto an effective relaxational model, which is what connects the system to the Ohta-Kawasaki class and yields the closed-form spinodal quoted in the main text. We construct that mapping here; the resulting functional is also the starting point for the analysis of the noise in Appendix D. Throughout linear stability analysis, we take the transport to be isotropic $\hat{\Lambda} = \hat{\lambda} \mathbf{I}$, so that $\hat{\lambda}$ enters as a scalar; the anisotropic case of Sec. IV B is treated only numerically.

Omitting ρ_2 from the free energy gives, in rescaled units,

$$\hat{\mathcal{F}}_1[\phi_1] = \int_{\hat{V}} d^3 \hat{r} \left[\frac{\tau_2}{2} (\phi_1 - \phi_c)^2 + \frac{\tau_4}{4} (\phi_1 - \phi_c)^4 + \frac{1}{2} |\hat{\nabla} \phi_1|^2 \right]. \quad (\text{B1})$$

Defining in addition

$$\hat{\mathcal{F}}_2[\phi_2] = \int_{\hat{V}} d^3 \hat{r} \frac{\hat{\lambda}}{2e} \phi_2^2, \quad \hat{\mathcal{F}}_t = \hat{\mathcal{F}}_1[\phi_1] + \hat{\mathcal{F}}_2[\phi_2], \quad (\text{B2})$$

and rescaling the second field as $\tilde{\phi} = (\tilde{\phi}_1, \tilde{\phi}_2) = (\phi_1, \phi_2/\sqrt{e})$ so as to symmetrize the exchange terms, Eqs. (A9) and (A10) take the matrix form

$$\partial_t \tilde{\phi} = \hat{\nabla}^2 \frac{\delta \hat{\mathcal{F}}_t}{\delta \tilde{\phi}} + \mathbf{A} \tilde{\phi} + \hat{\eta}_e \begin{pmatrix} 1 \\ -1/\sqrt{e} \end{pmatrix}, \quad \mathbf{A} = \hat{s} \begin{pmatrix} -e & \sqrt{e} \\ \sqrt{e} & -1 \end{pmatrix}. \quad (\text{B3})$$

The factor $1/e$ in $\hat{\mathcal{F}}_2$ is required by this rescaling: The functional derivative in Eq. (B3) is taken with respect to $\tilde{\phi}$, and $\hat{\nabla}^2 \delta \hat{\mathcal{F}}_2 / \delta \tilde{\phi}_2 = \hat{\lambda} \hat{\nabla}^2 \tilde{\phi}_2$ reproduces the transport term of Eq. (A10) only with this coefficient. Thus, $\hat{\mathcal{F}}_2$ encodes the transport term rather than a thermodynamic free energy. In these variables, one gets

$$\begin{aligned} \hat{\mathcal{F}}_t[\tilde{\phi}] &= \int_{\hat{V}} d^3 \hat{r} \left[-\phi_c (\tau_2 + \tau_4 \phi_c^2) \tilde{\phi}_1 + \frac{1}{2} (\tau_2 + 3\tau_4 \phi_c^2) \tilde{\phi}_1^2 \right. \\ &\quad \left. - \tau_4 \phi_c \tilde{\phi}_1^3 + \frac{\tau_4}{4} \tilde{\phi}_1^4 + \frac{1}{2} |\hat{\nabla} \tilde{\phi}_1|^2 + \frac{\hat{\lambda}}{2} \tilde{\phi}_2^2 \right] + \text{const.} \end{aligned} \quad (\text{B4})$$

The terms that are constant or linear in $\tilde{\phi}_1$ do not contribute to the dynamical equations and can be omitted. Using a Fourier representation for the quadratic terms, $\hat{\mathcal{F}}_t[\tilde{\phi}]$ can equivalently be replaced by

$$\begin{aligned} \hat{\mathcal{F}}_t[\tilde{\phi}] &= \frac{1}{2\hat{V}} \sum_{\mathbf{q} \neq 0} [(\tau_2 + 3\tau_4 \phi_c^2 + q^2) |\tilde{\phi}_1(\mathbf{q})|^2 + \hat{\lambda} |\tilde{\phi}_2(\mathbf{q})|^2] \\ &\quad + \int_{\hat{V}} d^3 \hat{r} \left[-\tau_4 \phi_c \tilde{\phi}_1^3 + \frac{\tau_4}{4} \tilde{\phi}_1^4 \right]. \end{aligned} \quad (\text{B5})$$

Here, the Fourier transform is defined via $\tilde{\phi}_i(\mathbf{q}) = \int_{\hat{V}} d^3 \hat{r} \tilde{\phi}_i(\hat{\mathbf{r}}) e^{-i\mathbf{q} \cdot \hat{\mathbf{r}}}$, which implies $\tilde{\phi}_i(\hat{\mathbf{r}}) = \hat{V}^{-1} \sum_{\mathbf{q}} \tilde{\phi}_i(\mathbf{q}) e^{i\mathbf{q} \cdot \hat{\mathbf{r}}}$, and we have used Parseval's theorem with $\hat{V} \rightarrow i\mathbf{q}$ producing

the q^2 term. Next, we exploit the identity

$$\mathbf{A} \tilde{\phi} = \hat{\nabla}^2 \frac{\delta \hat{\mathcal{F}}_{\text{Coul}}}{\delta \tilde{\phi}}, \quad (\text{B6})$$

where

$$\begin{aligned} \hat{\mathcal{F}}_{\text{Coul}}[\tilde{\phi}] &= -\frac{1}{2} \iint \tilde{\phi}^\top(\mathbf{r}') \mathbf{A} G(\mathbf{r} - \mathbf{r}') \tilde{\phi}(\mathbf{r}) d^3\mathbf{r} d^3\mathbf{r}' \\ &= -\frac{1}{2\hat{V}} \sum_{\mathbf{q} \neq 0} \tilde{\phi}_{\mathbf{q}}^\dagger \mathbf{A} G_{\mathbf{q}} \tilde{\phi}_{\mathbf{q}}, \end{aligned} \quad (\text{B7})$$

with G the Green's function of the Laplacian, $\hat{\nabla}^2 G(\mathbf{r} - \mathbf{r}') = -\delta(\mathbf{r} - \mathbf{r}')$, i.e., $G_{\mathbf{q}} = 1/q^2$. The $1/q^2$ kernel is the long-range Coulomb-like interaction generated by the exchange. Equation (B3) therefore becomes

$$\partial_t \tilde{\phi} = \hat{\nabla}^2 \frac{\delta \hat{\mathcal{F}}_t^{\text{eff}}}{\delta \tilde{\phi}} + \hat{\eta}_e \begin{pmatrix} 1 \\ -1/\sqrt{e} \end{pmatrix}, \quad (\text{B8})$$

with the virtual free energy $\hat{\mathcal{F}}_t^{\text{eff}} = \hat{\mathcal{F}}_t' + \hat{\mathcal{F}}_{\text{Coul}}$,

$$\hat{\mathcal{F}}_t^{\text{eff}} = \frac{1}{2\hat{V}} \sum_{\mathbf{q} \neq 0} \tilde{\phi}_{\mathbf{q}}^\dagger \mathbf{B}_{\mathbf{q}} \tilde{\phi}_{\mathbf{q}} + \int_{\hat{V}} d^3\mathbf{r} \left[-\tau_4 \phi_c \tilde{\phi}_1^3 + \frac{\tau_4}{4} \tilde{\phi}_1^4 \right], \quad (\text{B9})$$

with

$$\mathbf{B}_{\mathbf{q}} = \begin{pmatrix} \tau_2 + 3\tau_4 \phi_c^2 + q^2 + e\hat{s}/q^2 & -\sqrt{e}\hat{s}/q^2 \\ -\sqrt{e}\hat{s}/q^2 & \hat{\lambda} + \hat{s}/q^2 \end{pmatrix}. \quad (\text{B10})$$

We stress that Eq. (B8) is not an equilibrium model with free energy $\hat{\mathcal{F}}_t^{\text{eff}}$: The noise violates the fluctuation-dissipation relation, so minimizing $\hat{\mathcal{F}}_t^{\text{eff}}$ need not give the correct steady state.

2. Spinodal of both models

Neglecting the noise, the homogeneous solution of Eq. (B8) is $\tilde{\phi} \equiv 0$. We now determine when it first becomes unstable, treating the simplified and the full model together by carrying the parameter α of Eq. (A11) through the calculation. Linearizing the chemical potential of Eq. (A13) about the homogeneous state,

$$\frac{\delta \hat{\mathcal{F}}}{\delta \phi_1} \approx (\tau - \hat{\nabla}^2) \phi, \quad \tau := \tau_2 + 3\tau_4 \phi_c^2, \quad (\text{B11})$$

and Fourier transforming, Eqs. (A9) and (A10) become $\partial_t \phi_{\mathbf{q}} = -\mathbf{M}_{\mathbf{q}} \phi_{\mathbf{q}}$, with $\phi_{\mathbf{q}} = (\phi_1, \phi_2)^\top$ and

$$\mathbf{M}_{\mathbf{q}} = q^2 \begin{pmatrix} (\tau + q^2) + e\hat{s}/q^2 & \alpha(\tau + q^2) - \hat{s}/q^2 \\ -e\hat{s}/q^2 & \hat{\lambda} + \hat{s}/q^2 \end{pmatrix}. \quad (\text{B12})$$

For $\alpha = 0$, the rescaling of Appendix B 1 renders $\mathbf{M}_{\mathbf{q}}/q^2$ symmetric and one recovers $\mathbf{B}_{\mathbf{q}}$ of Eq. (B10). For $\alpha = 1$, no rescaling achieves this, because M_{12} carries the free-energy term $q^2(\tau + q^2)$ while M_{21} does not: The transported density enters $\hat{\mathcal{F}}$ and hence drives ϕ_1 , but $\hat{\mathcal{F}}$ does not drive ϕ_2 , which moves by motor transport rather than by a chemical-potential gradient. No effective free energy exists in that case, providing a precise sense in which the full model lies further from equilibrium.

The stability criterion itself requires no symmetry. For a 2×2 system, the eigenvalues satisfy $\mu_+ \mu_- = \det \mathbf{M}_{\mathbf{q}}$ and

$\mu_+ + \mu_- = \text{tr} \mathbf{M}_{\mathbf{q}}$, and both have positive real part if and only if $\det \mathbf{M}_{\mathbf{q}} > 0$ and $\text{tr} \mathbf{M}_{\mathbf{q}} > 0$. The α dependence enters only through $\det \mathbf{M}_{\mathbf{q}}$, and hence, on the spinodal, only through the relation $\tau + q^2 = -e\hat{s}\hat{\lambda}/[\hat{\lambda}q^2 + \hat{s}(1 + \alpha e)]$; the diagonal element M_{11} carries $e\hat{s}$ for both models. Evaluating the trace on the spinodal therefore gives

$$\text{tr} \mathbf{M}_{\mathbf{q}} = \hat{\lambda}q^2 + \hat{s}(1 + e) - \frac{e\hat{s}\hat{\lambda}q^2}{\hat{\lambda}q^2 + \hat{s}(1 + \alpha e)} > 0 \quad (\text{B13})$$

for the parameters used. Thus, no oscillatory (Hopf) instability occurs and the marginal-stability condition is $\det \mathbf{M}_{\mathbf{q}} = 0$ in both models. For $\alpha = 0$, this is equivalent to $\det \mathbf{B}_{\mathbf{q}} = 0$, since $\det \mathbf{B}_{\mathbf{q}} = \det \mathbf{M}_{\mathbf{q}}/q^4$; explicitly, the q^{-4} terms cancel and

$$\det \mathbf{B}_{\mathbf{q}} = \hat{\lambda}q^2 + (\tau\hat{\lambda} + \hat{s}) + \frac{\hat{s}(\tau + e\hat{\lambda})}{q^2}. \quad (\text{B14})$$

Writing $X := q^2(\tau + q^2)$, the determinant is

$$\det \mathbf{M}_{\mathbf{q}} = X[\hat{\lambda}q^2 + \hat{s}(1 + \alpha e)] + e\hat{s}\hat{\lambda}q^2. \quad (\text{B15})$$

The entire α dependence thus enters through the single substitution $\hat{s} \rightarrow \hat{s}(1 + \alpha e)$. Solving $\det \mathbf{M}_{\mathbf{q}} = 0$ for τ gives $\tau(q) = -q^2 - e\hat{s}\hat{\lambda}/[\hat{\lambda}q^2 + \hat{s}(1 + \alpha e)]$; its $q \rightarrow 0$ limit is the macrophase branch, and its maximum over q is the microphase branch together with the wave number selected there,

$$\tau_{c0}(e) = -\frac{e\hat{\lambda}}{1 + \alpha e}, \quad (\text{B16})$$

$$\tau_{c1}(e) = -2\sqrt{e\hat{s}} + \frac{\hat{s}(1 + \alpha e)}{\hat{\lambda}}, \quad (\text{B17})$$

$$q_0^2(e) = \sqrt{e\hat{s}} - \frac{\hat{s}(1 + \alpha e)}{\hat{\lambda}}. \quad (\text{B18})$$

If $\alpha < \hat{\lambda}^2/4\hat{s}$, the two branches meet at a transition point e_c , defined via the implicit equation

$$1 + \alpha e_c = \sqrt{e_c} \hat{\lambda}/\sqrt{\hat{s}}, \quad (\text{B19})$$

where $q_0 \rightarrow 0$ and the characteristic domain size diverges; of its two roots, we take the one continuous with the $\alpha = 0$ case. For e below that point, only macrophase separation is possible [$q_0^2(e)$ becomes negative]. Above it, the finite- q (microphase) mode is crossed first as τ is lowered, and the system microphase separates.

3. Comparison of the two models

For the simplified model ($\alpha = 0$), Eqs. (B16)–(B18) reduce to $\tau_{c0} = -e\hat{\lambda}$ and $\tau_{c1} = \hat{s}/\hat{\lambda} - 2\sqrt{e\hat{s}}$, meeting at $e_c = \hat{s}/\hat{\lambda}^2$ and $\tau_c = -\hat{s}/\hat{\lambda}$. In unrescaled units, Eq. (B18) reads $q_0^2 = \ell_0^{-2}(\sqrt{e\hat{s}} - \hat{s}/\hat{\lambda})$, the characteristic wave number quoted in the main text.

For the full model ($\alpha = 1$), microphase separation is only possible if $\hat{s}/\hat{\lambda}^2 < 1/4$. The transition point e_c shifts upward, with the exact relation

$$\sqrt{e_c}|_{\alpha=1} - \sqrt{e_c}|_{\alpha=0} = \frac{\sqrt{\hat{s}}}{\hat{\lambda}} e_c|_{\alpha=1}. \quad (\text{B20})$$

For $\hat{s} = 1$ and $\hat{\lambda} = 10$, this gives $e_c = 0.0102$ instead of 0.01. The spinodals are shifted upward by

$$\Delta_0 := \tau_{c0}|_{\alpha=1} - \tau_{c0}|_{\alpha=0} = \frac{e^2 \hat{\lambda}}{1+e} > 0, \quad (\text{B21})$$

$$\Delta_1 := \tau_{c1}|_{\alpha=1} - \tau_{c1}|_{\alpha=0} = \frac{\hat{s} e}{\hat{\lambda}} > 0, \quad (\text{B22})$$

so microphase separation sets in slightly earlier when ϕ_2 is retained, since the transported proteins then also contribute to the driving force for demixing. The shift vanishes as $e \rightarrow 0$ and grows linearly with e : For the parameters used, $\Delta_1 = 2 \times 10^{-3}$ at $e = 0.02$ and 5×10^{-3} at $e = 0.05$. The selected q_0^2 is smaller by the same amount, $\delta q_0^2 = -\Delta_1$, and since $L = 2\pi/q_0$ implies $\delta L/L = -\frac{1}{2} \delta(q_0^2)/q_0^2$, the relative domain size increases according to

$$\frac{\delta L}{L} \simeq \frac{\hat{s} e}{2\hat{\lambda} q_0^2|_{\alpha=0}} = \frac{\hat{s} e}{2\hat{\lambda}(\sqrt{e\hat{s}} - \hat{s}/\hat{\lambda})}, \quad (\text{B23})$$

which is about 2% over the range studied in the simulations. The approximation underlying $\hat{\mathcal{F}}_i^{\text{eff}}$ is therefore quantitatively controlled at the parameters considered in this work, and becomes least accurate on approaching the transition point $e = e_c$, where $q_0 \rightarrow 0$ and the relative shift diverges.

APPENDIX C: SIMULATION AND NUMERICAL DETAILS

We solve Eqs. (A9) and (A10) using a semiimplicit pseudospectral (FFT-based) method with periodic boundary conditions. On a cubic grid of spacing $\Delta \hat{h} = 1$, time step $\Delta \hat{t}$, and volume per cell $\Delta \hat{V} = 1$, we discretize in Fourier space, treating the stiff k -dependent linear terms implicitly and the remaining linear, nonlinear, and noise terms explicitly. Let $\Phi_i^n(\mathbf{k})$ be the Fourier transform of $\phi_i^n(\hat{\mathbf{r}})$, with $\hat{\nabla}^2 \rightarrow -k^2$ and $\hat{\nabla}^4 \rightarrow k^4$. Writing $\mathcal{N}^n \equiv \text{FFT}[(\phi_1^n + \phi_2^n - \phi_c)^3]$, $\mathcal{Z}^n \equiv \text{FFT}[\zeta_e^n]$, $D_1 \equiv 1 + \Delta \hat{t}(k^4 + \tau_2 k^2)$, and $D_2 \equiv 1 + \Delta \hat{t} \mathbf{k} \cdot \hat{\mathbf{A}} \mathbf{k}$, the updates for the full model are

$$\Phi_1^{n+1} = D_1^{-1} \{ \Phi_1^n + \Delta \hat{t} [-e\hat{s} \Phi_1^n + (\hat{s} - k^2 \tau_2 - k^4) \Phi_2^n - k^2 \tau_4 \mathcal{N}^n + \mathcal{Z}^n] \}, \quad (\text{C1})$$

$$\Phi_2^{n+1} = D_2^{-1} \{ \Phi_2^n + \Delta \hat{t} [e\hat{s} \Phi_1^n - \hat{s} \Phi_2^n - \mathcal{Z}^n] \}. \quad (\text{C2})$$

The transport tensor enters only through D_2 : For isotropic transport, $\mathbf{k} \cdot \hat{\mathbf{A}} \mathbf{k} = \hat{\lambda} k^2$, while for the anisotropic runs of Sec. IV B it is $\hat{\lambda}_\parallel k_x^2 + \hat{\lambda}_\perp (k_y^2 + k_z^2)$, with x the fast transport axis. The operator acting on ϕ_1 is isotropic in both cases, so D_1 is unchanged. Finally, invert the FFT to obtain $\phi_i^{n+1}(\hat{\mathbf{r}})$ and repeat. In the simplified model, where ϕ_2 is neglected in the free energy, $\mathcal{N}^n$ reduces to $\text{FFT}[(\phi_1^n - \phi_c)^3]$ and the Φ_1 update becomes

$$\Phi_1^{n+1} = D_1^{-1} \{ \Phi_1^n + \Delta \hat{t} [\hat{s} \Phi_2^n - e\hat{s} \Phi_1^n - k^2 \tau_4 \mathcal{N}^n + \mathcal{Z}^n] \}, \quad (\text{C3})$$

while Φ_2 is updated as in Eq. (C2).

On each grid cell of volume $\Delta \hat{V} = 1$ and time step $\Delta \hat{t}$, the unit white noises of Eq. (A14) are realized as

$$\hat{\zeta}_i^n(\hat{\mathbf{r}}) = \sqrt{\frac{1}{\Delta \hat{t} \Delta \hat{V}}} \Omega_i(\hat{\mathbf{r}}, n), \quad i = 1, 2, \quad (\text{C4})$$

where $\Omega_i(\hat{\mathbf{r}}, n)$ are independent standard Gaussians (zero mean, unit variance) at each cell and time, the prefactor being the lattice representation of $\delta(\hat{\mathbf{r}} - \hat{\mathbf{r}}') \delta(\hat{t} - \hat{t}')$. The exchange noise entering the updates above is then assembled exactly as in Eq. (A14),

$$\zeta_e^n(\hat{\mathbf{r}}) = C_0 \sqrt{\hat{s}} \left(\sqrt{e \phi_1^n + \frac{e}{1+e}} \hat{\zeta}_1^n(\hat{\mathbf{r}}) - \sqrt{\phi_2^n + \frac{e}{1+e}} \hat{\zeta}_2^n(\hat{\mathbf{r}}) \right). \quad (\text{C5})$$

The amplitude C_0 appears here only, through Eq. (A14), and must not be included a second time in $\hat{\zeta}_i^n$.

Unless stated otherwise, we use a cubic computational domain of side $128 \ell_0$ with grid spacing $h = \ell_0$, yielding 128^3 lattice points. Time is discretized with fixed step $\Delta \hat{t} = 0.5$. Initial conditions are spatially homogeneous with small random perturbations: $\phi_i(\hat{\mathbf{r}}, 0) \in [-10^{-3}, 10^{-3}]$ drawn from a uniform distribution, with mean $\langle \phi_i \rangle = 0$. Unless stated otherwise, we fix $\phi_c = 0.4$ and $\tau_4 = 0.4$, and vary τ_2 to control the quench depth $\Delta \tau := \tau - \tau_{c1}^F(e)$ (where $\tau = \tau_2 + 3\tau_4 \phi_c^2$), measured against the spinodal of the full model; for the simplified-model runs of Appendix E, it is measured against $\tau_{c1}(e)$ instead, so that $\Delta \tau = 0$ always denotes the marginal state of the model being simulated. For the isotropic transport simulations (Fig. 3), we set $\hat{s} = 1$ and $\hat{\lambda} = 10$, corresponding to a critical exchange ratio $e_c = 0.0102$ for the full model ($e_c = \hat{s}/\hat{\lambda}^2 = 0.01$ for the simplified one). The noise amplitude C_0 is varied in $\{0, 0.1, 0.2\}$ throughout, except in Appendix E and movies in the Supplemental Material [56], where values up to $C_0 = 0.5$ are used. Simulations are run up to $\hat{t} = 5 \times 10^4$ to ensure steady-state domain statistics or confirm coarsening behavior.

To quantify the characteristic length scale, we compute the structure factor $S(\mathbf{q}) = \langle |\hat{\phi}_1(\mathbf{q})|^2 \rangle$ from the Fourier transform of the free-protein density field and perform angular averaging to obtain $S(q) = \langle S(\mathbf{q}) \rangle_{|\mathbf{q}|=q}$. The dominant length scale L is extracted by fitting $S(q)$ to the empirical peak form

$$S(q) = S_{\max} \exp\{1 - [1 + ((q - q_0)/\sigma)^2]^{1-\beta}\},$$

introduced in Ref. [71], which captures both the central peak at wave number q_0 and the high- q power law or exponential decay. The domain size is then $L = 2\pi/q_0$. Fits are performed over a wave number window that includes the peak and at least one oscillation; uncertainty estimates reflect the covariance of the fit parameters. Time-dependent domain-size evolution (Fig. 8) confirms $L(t) \propto \hat{t}^{1/3}$ for macrophase coarsening and plateau behavior for microphase arrest.

APPENDIX D: EFFECT OF NOISE ON THE LARGE- q TAIL OF $S(q)$

The exchange noise leaves the position and width of the peak in $S(q)$ essentially unchanged but lifts its large- q tail. We show here that this follows from a linear analysis, and that the lift grows as C_0^2 .

We first need the amplitude of the noise in the homogeneous state. From Eq. (A14), with $\hat{\zeta}_1, \hat{\zeta}_2$ independent unit white noises, the cross term drops from $\langle \hat{\eta}_e^2 \rangle$ and the two

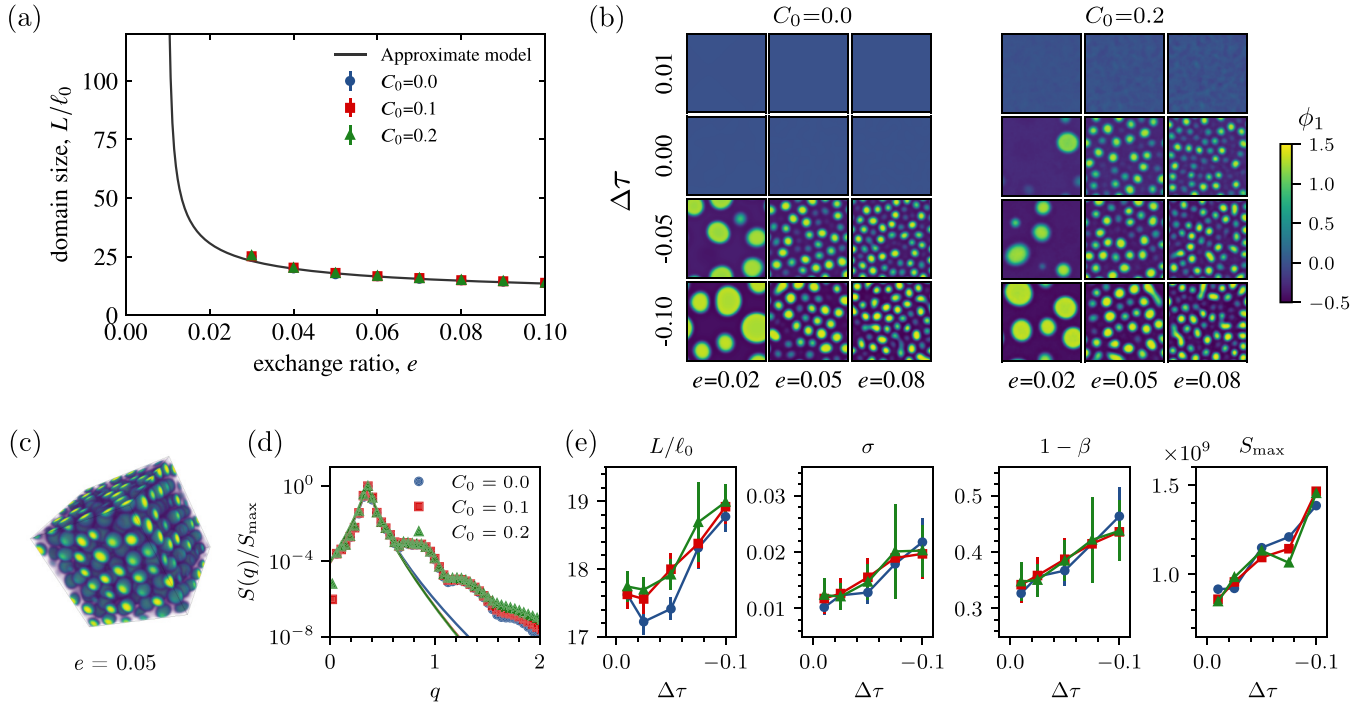


FIG. 6. Simulation results for the simplified two-field model (omitting ϕ_2 from the free-energy functional), to be compared with the full model (Sec. IV, Fig. 3). The quench depth is measured against the spinodal of this model, $\Delta\tau := \tau - \tau_{c1}(e)$, so that $\Delta\tau = 0$ lies on the spinodal. (a) Steady-state domain size L/ℓ_0 vs exchange ratio e : theory (solid line) and simulations at three noise strengths $C_0 = 0, 0.1, 0.2$ for $\Delta\tau = -0.01$. (b) Midplane ϕ_1 slices for $e = 0.02, 0.05, 0.08$ without and with noise ($C_0 = 0$ and $C_0 = 0.2$) at time $\hat{t} = 5 \times 10^4$; rows correspond to four quench depths $\Delta\tau = 0.01, 0.00, -0.05, -0.10$. (c) 3D snapshot for $e = 0.05$ at $\hat{t} = 5 \times 10^4$. (d) Angle-averaged structure factor $S(q)$ for $e = 0.05$ at $\Delta\tau = -0.01$ and the three noise strengths, in semilog representation; solid lines are fits to the empirical form $S(q) \sim \exp\{1 - [1 + ((q - q_0)/\sigma)^2]^{1-\beta}\}$. (e) Fitted domain size L , peak width σ , exponent $1 - \beta$, and peak amplitude $S_{\max}$ vs quench depth $\Delta\tau$ for $e = 0.05$. Parameters are $\hat{s} = 1, \hat{\lambda} = 10, \phi_c = 0.4, \tau_4 = 0.4$.

variances add,

$$\langle \hat{n}_e^2 \rangle = C_0^2 \hat{s} \left[\left(e\phi_1 + \frac{e}{1+e} \right) + \left(\phi_2 + \frac{e}{1+e} \right) \right] \\ \xrightarrow{\phi_i \rightarrow 0} \sigma_0^2 = \frac{2C_0^2 \hat{s} e}{1+e} \neq 0. \quad (\text{D1})$$

The mean exchange flux vanishes in the homogeneous state, since binding and release balance, $b\bar{\rho}_1 = s\bar{\rho}_2$; the fluctuations do not, because the two reactions are independent stochastic events. Two independent event streams of equal rate have zero mean difference but a variance equal to the sum of the rates, which is the factor 2 in Eq. (D1).

At large q , the off-diagonal elements of $\mathbf{B}_q$ [Eq. (B10)] vanish as q^{-2} , so the two fields decouple and $\tilde{\phi}_1$ obeys a scalar Ornstein-Uhlenbeck equation,

$$\partial_{\hat{t}} \tilde{\phi}_1(\mathbf{q}) = -\Gamma(q) \tilde{\phi}_1(\mathbf{q}) + \hat{\eta}_q(\hat{t}), \quad \Gamma(q) = q^2(\tau + q^2) \simeq q^4. \quad (\text{D2})$$

Its stationary variance is $\langle x^2 \rangle = \sigma^2/2\Gamma$, so the structure factor $S(q) = \langle |\tilde{\phi}_1(\mathbf{q})|^2 \rangle$ is

$$S(q) \simeq \frac{\sigma_0^2}{2\Gamma(q)} = \frac{C_0^2 \hat{s} e}{(1+e)q^4}, \quad q^2 \gg \hat{s}/\hat{\lambda} \quad \text{and} \quad q^2 \gg |\tau|. \quad (\text{D3})$$

The interpretation is that the drive σ_0^2 is the same at every wave number, because the noise is spatially white and enters outside the Laplacian, whereas the damping grows with q^4 . Short-wavelength modes are therefore forced as strongly as all the others but relax faster, settling at a small residual level that decays only as a power law. The deterministic tail, in contrast, decays exponentially (the fitted exponent $1 - \beta \rightarrow 0.5$), such that the noise background dominates beyond a crossover wave number. Since $S_{\max}$ is set by the deterministic pattern and is essentially independent of C_0 , the normalized tail rises in proportion to $\sigma_0^2 \propto C_0^2$ while the peak is unchanged, as observed.

APPENDIX E: APPROXIMATE MODEL AND EFFECT OF NOISE

1. Comparison with the full model

Figure 6 shows simulation results for the simplified two-field model (omitting ϕ_2 from the free energy) used in the linear stability analysis. Here, the quench depth is measured against the spinodal of each model, so that $\Delta\tau = 0$ corresponds in both cases to the respective marginal state. Compared in this way, the two models agree closely: The predicted domain sizes are indistinguishable on the scale of panel (a), and at $\Delta\tau = 0$ without noise neither model develops structure within the simulated time, as expected since the critical mode is marginal in both.

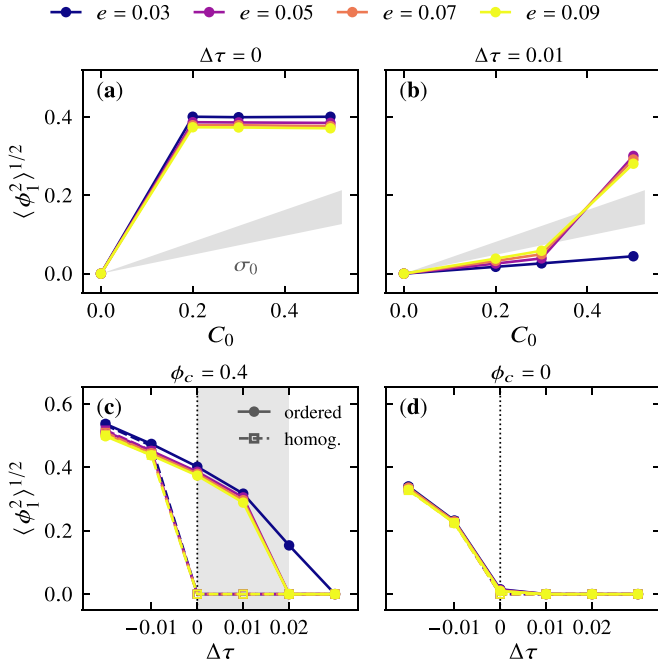


FIG. 7. Effect of the exchange noise at and above the spinodal for the simplified model (ϕ_2 omitted from the free energy). Color encodes the exchange ratio e in all panels. (a), (b) Root-mean-square (RMS) amplitude $\langle \phi_1^2 \rangle^{1/2}$ at $\hat{t} = 5 \times 10^4$ as a function of the noise strength C_0 , for quench depths $\Delta\tau = 0$ (on the spinodal) and $\Delta\tau = 0.01$ (above it). All runs start from a homogeneous state with a random perturbation of amplitude 10^{-3} , with $\phi_c = 0.4$. The gray band shows the noise amplitude $\sigma_0 = C_0 \sqrt{2\hat{s}e/(1+e)}$ over the range of e studied, which sets the scale of the fluctuations sustained in the absence of structure; amplitudes far above it indicate genuine microphase separation. Without noise, no structure forms at either quench depth within the simulated time. (c), (d) Amplitude vs quench depth with the noise switched off ($C_0 = 0$), starting either from a structured configuration (solid lines, filled symbols) or from a homogeneous state (dashed lines, open symbols), for (c) $\phi_c = 0.4$ and (d) $\phi_c = 0$. The shaded region marks the range $\Delta\tau \geq 0$ over which the two initial conditions relax to different steady states, i.e., where ordered and homogeneous states coexist. Hysteresis is present for $\phi_c = 0.4$, where the effective free energy carries a cubic term, and absent for $\phi_c = 0$, where it does not. Parameters are $\hat{s} = 1$, $\hat{\lambda} = 10$, $\tau_4 = 0.4$, on a 128^3 grid.

The residual difference is the shift of the spinodal derived in Appendix B 3, $\delta\tau_{c1} = \hat{s}e/\hat{\lambda}$, which also shifts the selected wave number and increases the domain sizes in the full model by about 2% [Eq. (B23)]. This is consistent with the fitted domain sizes at $\Delta\tau = -0.1$, $L/\ell_0 \simeq 18.9$ in panel (e) versus 19.3 in Fig. 3(e).

The two models differ more visibly in their response to noise just above the spinodal. At $\Delta\tau = +0.01$ with $C_0 = 0.2$ [Fig. 6(b)], the full model develops well-defined droplets, whereas the simplified model shows only weak fluctuations. Since both are the same distance above their respective spinodals, this difference does not reflect the linear onset but the nonlinear terms, in which the cubic contribution acts on $\phi_1 + \phi_2$ rather than on ϕ_1 alone. We examine the underlying mechanism in Fig. 7.

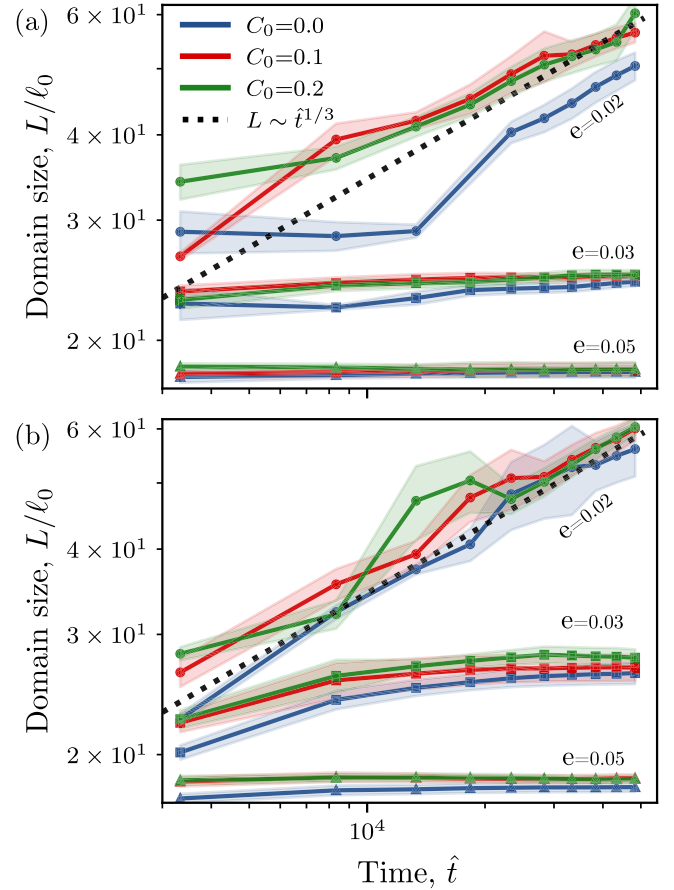


FIG. 8. Time evolution of domain size in 3D phase separation with active noise. Domain size L/ℓ_0 as a function of time $\hat{t}$ for different exchange ratios ($e = 0.02, 0.03, 0.05$, represented by $\bullet$, $\blacksquare$, and $\blacktriangle$, respectively) and noise amplitudes ($C_0 = 0.0, 0.1, 0.2$, shown in blue, red, and green, respectively). Panels show results for quench depths (a) $\Delta\tau = -0.01$ and (b) $\Delta\tau = -0.05$. System parameters: $L_x = L_y = L_z = 128 \ell_0$. Shaded regions indicate uncertainty from the fitting procedure. The black dotted line represents the classical coarsening scaling law $L \sim \hat{t}^{1/3}$, characteristic of diffusion-limited domain growth.

2. Effect of the exchange noise

Figure 7 examines the role of the noise directly. Panels (a) and (b) show the RMS amplitude $\langle \phi_1^2 \rangle^{1/2}$ as a function of noise strength at $\Delta\tau = 0$ and $\Delta\tau = 0.01$, i.e., on and above the spinodal of the simplified model. Without noise, no structure develops at either quench depth: At $\Delta\tau = 0$, the growth rate of the critical mode vanishes; thus, an infinitesimal perturbation is not amplified within the simulated time, and at $\Delta\tau > 0$ the homogeneous state is linearly stable. At $\Delta\tau = 0$, the amplitude jumps at $C_0 = 0.2$ to a plateau that is nearly independent of e and lies a factor of about 5–8 above σ_0 , the scale of the fluctuations, which the noise alone would sustain in a structureless system; this indicates genuine microphase separation rather than driven fluctuations, and structure therefore appears in a parameter range where linear stability analysis predicts none. Above the spinodal, at $\Delta\tau = 0.01$, the amplitude remains at the fluctuation level up to $C_0 = 0.3$ and rises only at $C_0 = 0.5$, and then only for the

larger exchange ratios, consistent with the subcritical barrier being crossed more readily at large e , where σ_0 is larger.

Panels (c) and (d) identify the mechanism. Switching the noise off and following the system from either a structured or a homogeneous initial condition, we find that for $\phi_c = 0.4$ the structured state remains stable above the linear instability: Over a finite range $\Delta\tau \gtrsim 0$, the two initial conditions lead to different steady states; i.e., the ordered and homogeneous states coexist. For $\phi_c = 0$, they do not, and the structured state decays for all $\Delta\tau \geq 0$. Since ϕ_c is the only parameter that was varied between the two panels, and it enters the effective free energy through the cubic term $-\tau_4\phi_c\tilde{\phi}_1^3$ of Eq. (B9), we can attribute the coexistence to that term: The transition to the microphase is subcritical. This is consistent with the generic behavior of the Landau-Brazovskii model, the equilibrium counterpart at $\alpha = 0$, which exhibits a first-order transition in the presence of a cubic term in the free energy [55].

This provides the physical interpretation of the observed noise-induced structure formation. Because the transition is subcritical, a finite-amplitude perturbation is required to reach the ordered state, and the exchange noise supplies it, whereas the infinitesimal numerical seed does not. The noise thus

determines whether the ordered state is reached within an accessible time, not whether it exists. We emphasize that this concerns the approach to the ordered state, not the scaling laws reported in the main text: The domain size selected below the spinodal is set by q_0 and is insensitive to C_0 [Fig. 6(a)], so the results of Sec. IV are unaffected. The one exception is the large- q tail of $S(q)$, which the noise lifts by adding the background of Eq. (D3).

APPENDIX F: TIME EVOLUTION OF DOMAIN SIZES

Figure 8 shows how the exchange ratio and the noise amplitude influence the coarsening dynamics. To exclude early transient behavior, data analysis begins from $\hat{t} \approx 3.3 \times 10^3$. For $e < 0.03$, the domain size continues to grow during the whole simulation time following the classical scaling law $L \sim \hat{t}^{1/3}$, characteristic of diffusion-limited coarsening, and in this regime higher noise levels lead to enhanced domain growth. In contrast, for $e \geq 0.03$, domains reach a steady-state size that is insensitive to C_0 . We provide four movies showing droplet evolution at $e = 0.02$ and $e = 0.05$ for quench depth $\Delta\tau = -0.05$ with and without noise ($C_0 = 0$ and 0.5). Movies are available in Supplemental Material [56].

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