

Anomalous diffusion and effective shear modulus in a semisolid membrane

Vikash Pandey ^{1,2,3} and Dhrubaditya Mitra ¹

¹*NORDITA, KTH Royal Institute of Technology and Stockholm University, Hannes Alfvéns väg 12, 10691 Stockholm, Sweden*

²*Department of Molecular, Cellular and Developmental Biology, Yale University, New Haven, Connecticut 06520, USA*

³*Yale Institute for Biospheric Studies, Yale University, New Haven, Connecticut 06520, USA*



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From the perspective of physical properties, the cell membrane is an exotic two-dimensional material that has a dual nature: it exhibits characteristics of fluids, i.e., lipid molecules show lateral diffusion, while also demonstrating properties of solids, evidenced by a nonzero shear modulus. We construct a model for such a *semisolid membrane*. Our model is a fluctuating randomly triangulated mesh with two different kinds of nodes. The solid nodes never change their neighbors, while the fluid nodes do. As the area fraction occupied by the solid nodes (Φ) is increased the motion of fluid nodes transitions from diffusion to localization via subdiffusion. Next, the solid nodes are pinned to mimic the pinning of the plasma membrane to the cytoskeleton. For the pinned membrane, there exists a range of Φ over which the model has both a nonzero shear modulus and a nonzero lateral diffusivity. The bending modulus, measured through the spectrum of height fluctuations, remains unchanged.

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I. INTRODUCTION

The cell membrane forms a barrier that separates the cytoplasm from the external environment. Typically, composition-wise, it is approximately 73% lipid and about 23% proteins [1]. The canonical model of cell membrane is the fluid mosaic model, proposed by Singer and Nicolson [2], who conceptualized it as a fluid membrane with two leaflets made of lipids with diverse inclusions of proteins, carbohydrates, and cholesterol. The fluid membrane is modeled by the Helfrich energy functional [3–5], which has energy cost to bending and change in area but has zero shear elastic modulus. Minimizing this energy functional with the constraint of fixed volume captures the common biconcave shape of red blood cells (RBCs) [6–8]. Such a model correctly interprets the micro-pipette aspiration experiments [5]. From the point of view of material properties, such a membrane is a two-dimensional (2D) fluid. However, to capture all the different shapes a RBC can have (e.g., discocyte, stomatocyte, and echinocyte) *from a single model* it is necessary to consider a composite model [9], “where the plasma membrane contributes bending rigidity and the protein-based membrane skeleton contributes stretch and shear elasticity.”

Furthermore, it is necessary to include shear elasticity in models of RBCs to capture their shapes in microfluidic flows [10] and to interpret the results of mechanical deformation of RBCs in certain optical tweezer experiments [11–14]. Hence

the consensus is that the cell membrane of RBCs, and by extension all cells, have a shear modulus: the membrane is a *two-dimensional solid*. The shear modulus emerges due to coupling of the fluid membrane with the cytoskeleton. In contrast, experiments [15–22] show that the lateral motion of tagged lipid molecules in the cell membrane is diffusive, although the diffusion may be anomalous. This is a typical feature of 2D fluids. Additionally, the fact that the cell membrane forms tethers provides evidence in support of its fluid nature. Thus, the cell membrane is an exotic 2D material which has physical properties akin to both solids and fluids *simultaneously*. At present, there is no model of cell membrane that can capture this dual nature. Here we show how to construct a minimal model that does.

II. MODEL

We start with a model for isotropic and homogeneous 2D elastic material. In the Monge gauge the deformation can be described by an in-plane vector $\mathbf{u}(x_1, x_2)$ and an out-of-plane scalar $f(x_1, x_2)$. The Hamiltonian is given by

$$\mathcal{H} = \int d\mathbf{x} \left[\frac{\kappa}{2} (\nabla^2 f)^2 + \mu s_{\alpha\beta}^2 + \frac{\lambda}{2} s_{\alpha\alpha}^2 \right], \quad (1a)$$

$$\text{where } s_{\alpha\beta} \equiv \frac{1}{2} (\partial_\alpha u_\beta + \partial_\beta u_\alpha + \partial_\alpha f \partial_\beta f) \quad (1b)$$

is the strain tensor and μ and λ are the two Lamé coefficients [23,24]. Monte Carlo modeling of such a membrane [25–29] proceeds as follows. First, generate a random triangulated grid on a flat surface with N nodes; see Fig. 1(a). Second, update the positions of the points ($\mathbf{X}_i$ at node i) by discretizing the Hamiltonian in Eq. (1) over this grid. The energy has

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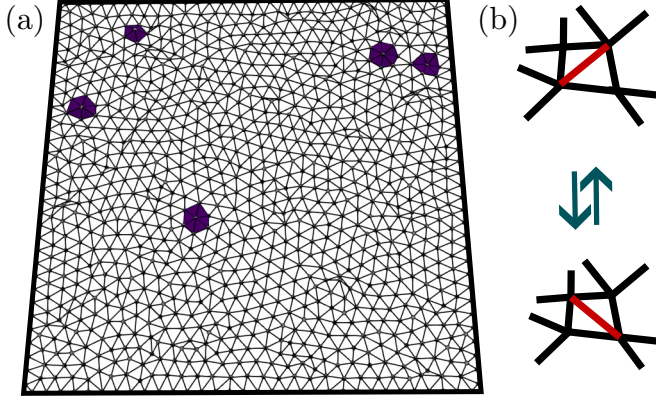


FIG. 1. (a) A representative picture of our model: it is a fluctuating, randomly triangulated surface where the solid regions are colored in purple. It is connected to a frame. (b) An example of bond flips that fluidize our membrane.

two contributions, $E = E_S + E_B$, where the stretching and the bending contributions, respectively, are

$$E_S = \frac{H}{2} \sum_{i,j} (X_{ij} - \ell_{ij}^0)^2, \quad (2a)$$

$$\text{where } X_{ij} \equiv |X_i - X_j| \quad (2b)$$

$$\text{and } E_B = \frac{\kappa}{2} \sum_i \mathcal{A}_i L_i^2. \quad (2c)$$

The spring constant H sets the two Lamé coefficients [26,30], $\mathcal{A}_i$ is the area of the dual Voronoi cell at the node i and

L_i is the discretized Laplacian [25,26,28–31]. For the rest of this paper we shall call such a membrane a *solid membrane* as it has a finite in-plane shear modulus. We use the Metropolis algorithm: always accept a move if it results in lowering the energy of the membrane; a move that increases the energy of the membrane by ΔE is accepted with the probability $\exp[-\Delta E/k_B T]$ where k_B is the Boltzmann constant and T is the temperature. We define a single Monte Carlo step as an attempt to move each of the N nodes at least once, on average. Typically, our production runs use 10^6 Monte Carlo steps.

It is possible to *fluidize* this membrane [see, e.g., 27,32, Sec. 6.2.4]: after every M Monte Carlo steps flip bonds between neighbors—the bond between any two adjacent nodes is broken and a new bond between other points is formed, as shown in Fig. 2(b). This operation is allowed as long as the minimum number of neighbors of a node is greater than four and the length of the new bond is less than a given maximum bond length. The first feature of our model is to randomly assign a fraction of nodes to be *solid* and the rest of them to be *fluid*. The bonds of the solid nodes with their neighbors are never broken while the bonds between two fluid nodes can be removed or added. Physically the fluid nodes are the coarse-grained region with relatively high diffusivity compared to the solid nodes. Changing the fraction Φ from 0 to 1 we go from a fluid membrane to fluid membranes with solid inclusions and finally a solid membrane; see Fig. 2 where the triangles formed by the solid points with their neighbors are colored. The fractions of area of the membrane that is solid is $\Phi \equiv N_s^T/N^T$, where N_s^T is the number of triangles

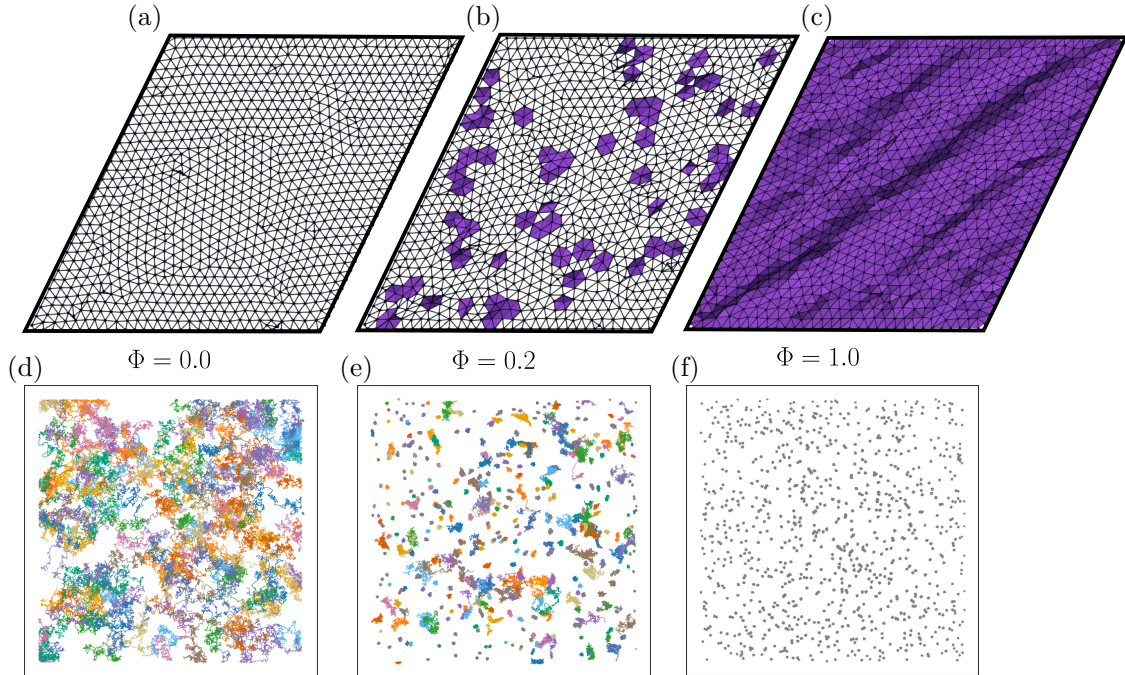


FIG. 2. (Top panel) Typical snapshots of sheared membranes from our simulations. The area fraction occupied by solid inclusion is Φ . From left to right: (a) $\Phi = 0$ (fluid membrane), (b) $\Phi = 0.2$ (fluid with solid inclusions), and (c) $\Phi = 1$ (solid membrane) which shows ridges typical of sheared fabric. The purple patches in the middle figure represent the solid regions. (Bottom panel) Representative trajectories of the nodes for the same three values of Φ .

TABLE I. The parameters used in our simulations: N is the total number of nodes; H is the spring constant of the harmonic spring that connects to neighboring nodes; κ is the bending modulus; Φ is the area fraction occupied by the solid nodes; S is the externally imposed strain on the frame; and K is the spring constant of the harmonic pinning potential. Our production runs are typically for 10^6 Monte Carlo steps. The length of a side of the square frame is $\sqrt{N}b$ where b , the average bond length, is our unit of length. The parameters, H , κ , and K are measured in the unit of $k_B T$; N , Φ , and S are dimensionless.

N	65 536 (diffusion expt.) 16 384 (shear expt.)
H	10^4
κ	4.0
Φ	0.0, 0.002, 0.017, 0.034, 0.051, 0.068, 0.094, 0.12, 0.14, 0.17, 0.21, 0.31, 0.35, 0.48
S	0.04, 0.08, 0.12, 0.16, 0.22, 0.28, 0.34, 0.40, 0.48
K	200

associated with the solid points and N^T is the total number of triangles.

The second feature of our model is to add pinning of the solid nodes, i.e., we run two kind of simulations.

(1) The solid nodes and the patches around them move around.

(2) The solid nodes are pinned.

We call the former unpinned (or unanchored) and the latter pinned. For all the cases where Φ is not equal to zero or unity; i.e., for the cases where there are both solid and fluid nodes in the model, we introduce a harmonic pinning potential that pins (anchors) only the solid points to the x_1 - x_2 plane. The additional term in the energy of the membrane is

$$E_{\text{pin}} = \sum_{i=s} \frac{K}{2} (X_i - X_i^0)^2, \quad (3)$$

where the suffix "s" denotes the solid points. The vector X_i^0 is the initial position of the i th node and K the spring constant of the pinning potential. The goal is to mimic the attachment of the membrane to the cytoskeleton.

We attach the nodes lying on the boundary of the membrane to a frame which does not undergo any Monte Carlo move. By straining the frame we can impose an external strain on the membrane and calculate its response thereby calculating effective elastic moduli. Note that we do not include the triangles formed with the points on the frame in N^T .

All the parameters used in this study are given in Table I. To estimate the error we use BAGging (Bootstrap AGgregation) [33], and the data (Γ) are divided into $n = 40$ subsets $\{\Gamma_1, \dots, \Gamma_n\}$. For any quantity, e.g., the total energy E , we calculate n mean values, where the k th value is calculated by averaging over the data in Γ_k . The standard deviation calculated over all the sets is our error estimate.

We use the mean bond length for the initial grid, $b \equiv \langle \ell_{ij}^0 \rangle$ as our unit of length. We use the number of Monte Carlo steps as time. Energy is measured in the unit of $k_B T$.

III. RESULTS

Let us first consider the unpinned membrane. In Figs. 2(a) to 2(c) we show typical snapshots of strained membrane from our simulations. Figure 2(a) is for the fluid membrane ($\Phi = 0$), and Fig. 2(c) is for the solid membrane ($\Phi = 1$). Figure 2(b) is for an intermediate value, $\Phi = 0.2$. The simulation of the solid membrane shows typical ridges that appear in sheared solid membranes, e.g., fabrics [34]. In Fig. 2(d) to Fig. 2(e) we plot the trajectories of a (fraction of) nodes. In Fig. 2(d) all the nodes are fluid. The nodes move by a large amount. From Fig. 2(d) to Fig. 2(e) the nodes get more and more localized. In Fig. 2(e) the membrane is solid—the nodes are completely localized.

A. Emergence of anomalous diffusion

In Fig. 3(a) we plot the mean-square displacement (MSD) of the nodes as a function of time (t),

$$\langle R^2(t) \rangle \equiv \langle [X(t) - X(0)]^2 \rangle, \quad (4)$$

where the symbol $\langle \cdot \rangle$ denotes an average over the nodes. The fluid membrane, which corresponds to the top line, shows $\langle R^2 \rangle \sim t$, i.e., the nodes show diffusion. As the area fraction Φ increases we move from diffusion to subdiffusion ($\langle R^2 \rangle \sim t^\beta$ with $\beta < 1$) to the case of solid membrane where $\langle R^2 \rangle$ is constant at late times, a typical signature of localization. We note that for the fluid membrane, scaling persists for three decades.

Experimentally, lateral motion of tagged molecules on the plasma membrane [15–21, 35] and on giant unilamellar vesicles which are reinforced with a network of polymeric filaments (e.g., actin filaments) [36–38] have been studied extensively. The general consensus is that tagged molecules show (anomalous) diffusion the mechanism of which can be a combination of continuous time random walk (CTRW) and fractional Brownian motion (fBM). Transmembrane protein molecules which can be attached to the cytoskeleton can remain trapped at a position: their motion can be described by CTRW. Otherwise, molecules (both proteins and lipids) show random walk in a crowded environment. Our model is at a length scale much larger than individual molecules; nevertheless, it behooves us to ask what is the likely mechanism behind the anomalous diffusion observed in our simulations. A possible way to distinguish between CTRW and fBM is to study the probability distribution function (PDF) of the first passage time of the diffusing node. In CTRW this PDF has a power-law tail, but in our simulations we observe an exponential one. Thus we can conclude that CTRW is not the mechanism anomalous diffusion seen in our simulations.

There has been many attempts to model the stochastic lateral motion of tagged molecules: at one end lies molecular dynamics simulations of lipid membranes [39, 40] which can cover a very small patch on the membrane; at the other end lies random walk in a medium with fixed obstacles [22, 37, 41–44]. An important member of the second class of models is

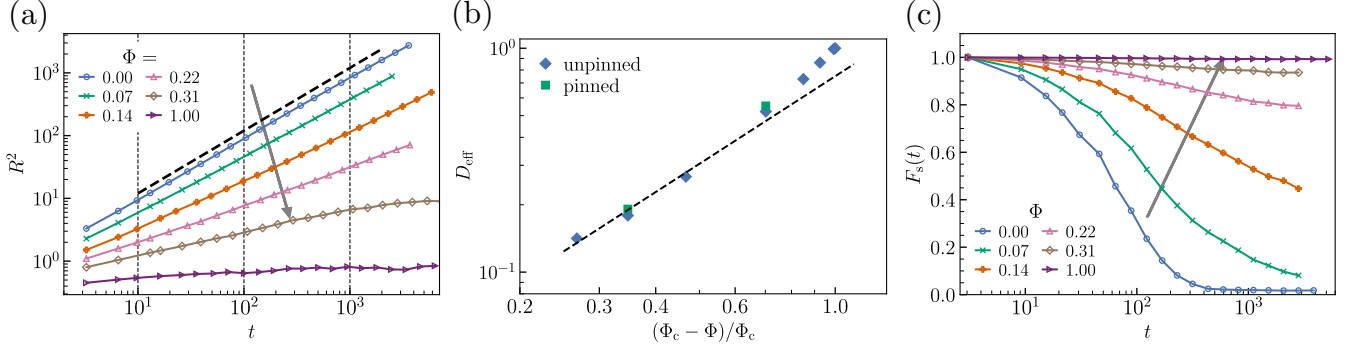


FIG. 3. (1) Log-log plot of the MSD $\langle R^2 \rangle$ as a function of t . Our unit of length is $b \equiv \langle \ell_{ij}^0 \rangle$, and unit of time is $\tau \equiv b^2/D(\Phi = 0)$, where $D(\Phi = 0)$ is the diffusion constant defined in (5). (b) Log-log plot of D_{eff} versus $(\Phi_c - \Phi)/\Phi_c$. The dashed line, with an exponent 1.33 is the corresponding scaling relation for the Lorentz model. (c) The self-intermediate scattering function F_s as a function of time t . The arrow points to the direction of increase in Φ .

the Lorentz model, in which the obstacles are randomly distributed. As the number density of the obstacles is increased the walker goes from diffusion to subdiffusion for intermediate times. At very late times the subdiffusive trajectories either become diffusive or get localized ($D_{\text{eff}} \rightarrow 0$) [41,42]. There are similarities between these models and ours with the exception that in our model the solid patches are not fixed but can also move around. In fact, for $\Phi < \Phi_c$ the solid patches also show subdiffusion but with an exponent smaller than the fluid ones.

We interpret the data of $\langle R^2(t) \rangle$ in two ways. One, we consider the transition from diffusion to subdiffusion as Φ , the area fraction occupied by the solid patches, is changed. We fit the MSD (as a function of time) with $\langle R^2 \rangle \sim t^\beta$, where $\beta = 1$ (for $\Phi = 0$) corresponds to diffusion. As Φ increases β decreases, down to approximately 0.75: this behavior is somewhat similar to what has been observed in Lorentz model [41,43]. Beyond a critical value of Φ , Φ_c , the trajectories are localized, i.e., $\langle R^2 \rangle$ goes to a constant at late times. For $\Phi > \Phi_c$ the trajectories can no longer be described by a diffusive process. From our data we estimate $\Phi_c \approx 0.23$ (it is difficult to determine Φ_c accurately as the diffusion is limited to a fixed box).

The second way to interpret the data of $\langle R^2(t) \rangle$ is to calculate a Φ -dependent diffusivity

$$D(\Phi) \equiv \lim_{t \rightarrow \infty} \frac{\langle R^2 \rangle}{t} \quad (5a)$$

$$\text{and } D_{\text{eff}} \equiv \frac{D(\Phi)}{D(\Phi = 0)} \quad (5b)$$

the effective diffusivity. We can introduce a new timescale $\tau \equiv b^2/D(\Phi = 0)$. With τ as our unit of time, the diffusion constant for the fluid membrane, $D(\Phi = 0) = 1$. For $\Phi > \Phi_c$ the diffusion coefficient $D(\Phi) \approx 0$. For the Lorentz model,

$$D_{\text{eff}} \sim \left(\frac{\Phi_c - \Phi}{\Phi_c} \right)^\gamma \quad (6)$$

with $\gamma \approx 1.33$ [42]. We see a similar trend in our simulations too, although the data are not sufficient to determine the precise dependence of D_{eff} near $\Phi \approx \Phi_c$. This question is not central to our work.

Turning now to the case where the solid nodes are pinned we find that there is almost no change to the behavior of the fluid nodes compared to the unpinned case.

B. Self-intermediate scattering function

Localization phenomena in glasses is often studied through the self-intermediate scattering function [45,46],

$$F_s(t) \equiv \langle \exp[i\mathbf{k} \cdot \mathbf{X}(t)] \rangle, \quad (7)$$

which we plot in Fig. 3(c) as a function of time for $|\mathbf{k}| = \pi/(8b)$. The average is performed over all the angles of the wavevector $\mathbf{k}$ and over all the nodes. For a fluid $F_s(t) \rightarrow 0$ exponentially fast at late times; for a solid $F_s(t)$ goes to a nonzero constant at late times. Again, we find a transition from fluid to a solid at a critical value $\Phi = \Phi_c$. On one hand, our model of solid and fluid nodes are similar to a binary mixture with large and small particles. In such binary mixtures arrested states, where the MSD is a constant, can be found. We obtain similar behavior for $\Phi > \Phi_c$. On the other hand, in glassy systems a long plateau is typically observed in the self-intermediate scattering function (F_s). In our system, such a plateau is absent. We note that our simulations are of a 2D surface embedded in three dimensions, thus it cannot be directly connected to either 2D or three-dimensional simulations of binary liquids.

In summary, from observation of anomalous diffusion and a self-intermediate scattering function we conclude that our model shows a transition from a fluid to a disordered solid, in two dimensions, as $\Phi \rightarrow \Phi_c$.

C. Shear modulus

The key aspect where our model is superior to the Brownian walk-based models is that ours allows, in addition, calculation of elastic properties of the membrane. We calculate the response of the membrane to imposed linear shear strain. We deform the frame such that a point at location x, y on the frame is displaced by $Sy, 0$. The strain of the frame is given by S . In Fig. 4(a) we plot the change in energy of the membrane ΔE as a function of time for different values of Φ and for a single strain. The energy is maximum at $t = 0$. As time progresses the energy relaxes and at late times reaches a constant value ΔE_∞ , with fluctuations. For $\Phi < \Phi_c$ we find

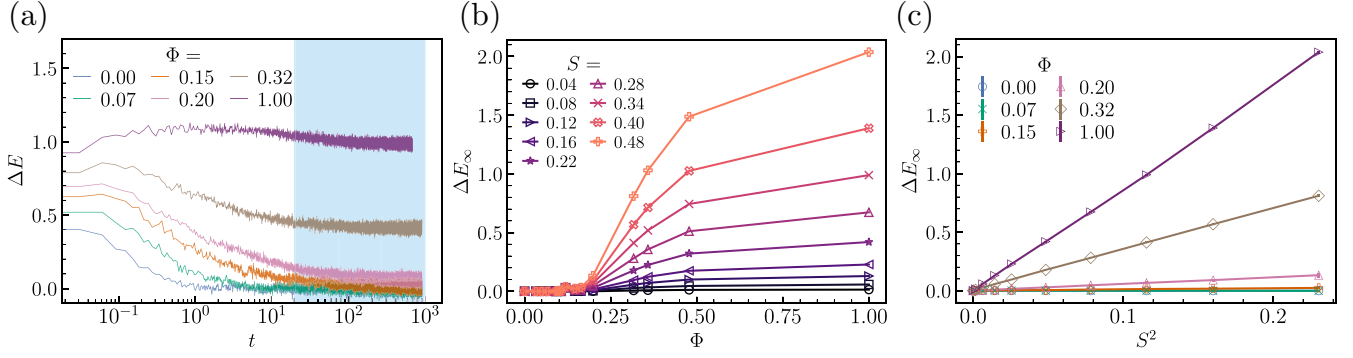


FIG. 4. (a) The change in energy, ΔE , of the sheared membrane as a function of nondimensional time, for different values of Φ and constant shear strain $S = 0.34$. The area fraction Φ increases from bottom to top. The mean energy at late times is ΔE_∞ (calculated as an average over the shaded region), which is a function of both S and Φ . (b) The change in energy at late times, ΔE_∞ , as a function of Φ for different values of S . For Φ smaller than certain critical value $\Phi_c \approx 0.23$, $\Delta E_\infty = 0$ for all values of S . (c) The change in energy at late times, ΔE_∞ , as a function of S^2 for different values of Φ . For $\Phi < \Phi_c$, $\Delta E_\infty = 0$. The slopes of these lines are the effective shear modulus $\mu(\Phi)$. The errorbars in (b) and (c) are smaller than the size of the symbols.

that the membrane relaxes such that at late time $\Delta E_\infty = 0$. In other words, there is no energy cost to shear strain. These are examples of a 2D fluid membrane. But for $\Phi > \Phi_c$ at late times the ΔE_∞ reaches a constant nonzero value (with thermal fluctuations). This is a feature of solid membranes which can sustain shear strain. In Fig. 4(b) we plot the change in energy at late times, ΔE_∞ , as a function of Φ for different values of strain, S . For $\Phi < \Phi_c$, ΔE_∞ is zero for all strains. For $\Phi > \Phi_c$, ΔE_∞ is finite and also increases with S . In Fig. 4(c) we plot ΔE_∞ as a function of S^2 for different values of Φ . We obtain $\Delta E_\infty \propto S^2$, the constant of proportionality is the effective shear modulus μ which depends on Φ . In Fig. 5(a) we plot both the diffusivity obtained from Fig. 3(a) and the shear modulus μ obtained from Fig. 4(c) as a function of the area fraction Φ . We measure D in units of $D(\Phi = 0)$ and measure μ in units of $\mu_s \equiv \mu(\Phi = 1)$ —the shear modulus of the solid membrane. As Φ increases D decreases with $D \approx 0$ for $\Phi > \Phi_c$. Simultaneously $\mu \approx 0$ for $\Phi < \Phi_c$. The shear modulus μ increases as Φ increases beyond Φ_c . There is a

very tiny range of Φ around Φ_c for which both μ and D are nonzero although both are much smaller than unity. We conclude that the structural transition of the membrane from fluid to solid is accompanied by a rigidity transition, as shown by its shear elastic modulus, at $\Phi = \Phi_c$. The membrane is either a solid ($\Phi > \Phi_c$) or a fluid ($\Phi < \Phi_c$), it does not simultaneously have the dual nature (both a solid and a fluid) — exhibited by cell membrane.

D. Rigidity transition in the pinned Membrane

We now turn to the rigidity transition in the pinned membrane. In Fig. 5(b) we show a typical snapshot of the sheared *pinned* membrane. We mentioned already that the pinning has no effect on $\langle R^2 \rangle$, it is indistinguishable from $\langle R^2 \rangle$ of the unpinned membrane. Consequently the lateral diffusion coefficient depends on Φ alone; it is independent of whether the solid points are pinned or not. But pinning significantly changes the shear modulus μ . To Fig. 5(a) we now add the

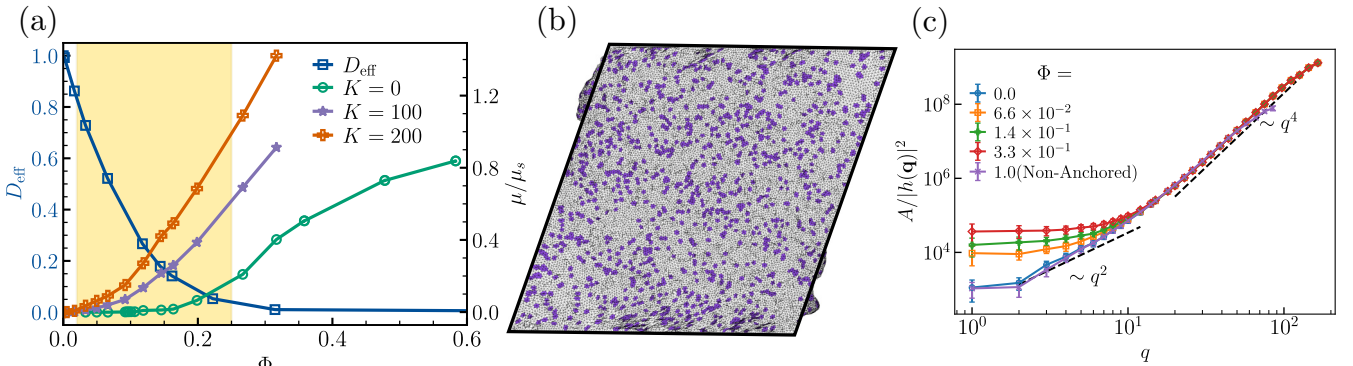


FIG. 5. (a) The diffusion coefficient D , (left axis) and the effective shear modulus μ (right axis) as a function of area fraction Φ . The coefficient D is normalized by the diffusivity of the fluid membrane, $D(0)$. The shear modulus μ is normalized by the shear modulus of the solid membrane μ_s . The diffusion coefficient is zero for $\Phi > \Phi_c$, with $\Phi_c \approx 0.23$. The shear modulus is zero for $\Phi < \Phi_c$. Both of them show a rigidity transition at $\Phi = \Phi_c$. The diffusivity of the pinned membrane (not shown here) is equal (within error bars) to the nonpinned one. The orange curve shows μ/μ_s for the pinned membrane, for which $\mu > 0$ for $\Phi \gtrsim 0.04$. Thus for $0.04 < \Phi < \Phi_c$, the shaded region, the pinned membrane has physical properties akin to both solids and fluids. The error bars in the data are smaller than the size of the symbols. (b) A typical snapshot of the strained pinned membrane for $\Phi = 0.2$ and $S = 0.34$. (c) Log-log plot of the power spectra of the height fluctuations. The black dashed line shows the σq^2 and the κq^4 scaling is shown using a continuous line of the same color.

plot of μ [normalized by $\mu(\Phi = 1)$] for the pinned membrane as a function of Φ . For $\Phi > 0.04$ the pinned membrane has a nonzero μ . The shear modulus of the pinned membrane is always higher than the unpinned one. Remarkably there is a large range of values of Φ , $0.04 < \Phi < 0.2$ over which the membrane is a fluid, characterized by the diffusivity of its nodes, but also solid if the shear elastic modulus μ is measured. This range of Φ is shaded in Fig. 5(a). We have now reached our goal, a minimal model of the cell membrane that can capture both its solid and fluid aspects.

E. Fluctuation spectra

We calculate the spectra for the out-of-plane fluctuations $f(\mathbf{x})$ where the in-plane coordinates $\mathbf{x} = (x_1, x_2)$. For small fluctuations, the power spectra for the fluctuations, $G(q)$, is given by

$$G(q) = \langle f(\mathbf{q})f(-\mathbf{q}) \rangle = \frac{Ak_B T}{(\kappa q^4 + \sigma q^2)}, \quad (8)$$

where A is the area of the projection of the membrane on to the $x_1 - x_2$ plane, κ is the bending modulus and $\sigma = (\mu + \lambda)([A_i/A_i(t=0) - 1])$ is the effective tension [5]. We show the fluctuation spectra from our simulations in Fig. 5(c). For both $\Phi = 0$ (fluid membrane) and $\Phi = 1$ (solid membrane) we can identify both the κq^4 at large q and σq^2 at small q . The bending modulus κ is independent of Φ [Fig. 5(c)]. The area modulus can be written as a function of the two Lamé coefficients: $K_A = 2(\mu + \lambda)$, of which we have already calculated μ . To calculate λ we deform our frame such that its shape remains the same (a square) but the area changes. The change in energy of the membrane at late times gives λ . As expected, we find that both the (unpinned) solid and fluid membrane shows the same response to stretching; pinning increases K_A .

IV. CONCLUSION

In summary, in this paper we introduce a model of membrane that has physical properties akin to both solids and fluids. In particular, within a certain parameter range, the fluid nodes of the membrane show (anomalous) diffusion while the membrane as a whole has a nonzero elastic shear modulus. The key contribution to the elastic shear modulus comes from the pinning of the solid nodes to a flat surface. From the point of view of soft matter physics, our pinned model describes an exotic 2D material with physical properties akin to both solids and fluids. We find a structural transition from fluid to solid at a certain area fraction, and a rigidity transition from fluid to solid at a different (lower) area fraction. At present, we cannot determine whether these transitions are true phase transitions or crossovers.

The existence of a rigidity transition suggests that certain aspects of our model and potentially the behavior of a cell membrane can be related to the physics of glass transition in binary mixtures. In binary mixtures consisting of large

and small particles, different arrested states are observed. Depending upon the fraction of small particles, the mechanism for this arrests are (1) the depletion-induced attractions from small spheres, (2) gelation, and (3) depletion-induced bonding between large particles [47]. A characterization of these states is made by studying the MSD of the large particles. The MSD remains flat over the timescales the arrested states are observed. To investigate this possibility in our model we compute the MSD of the solid nodes for different solid fractions. Our results show that no flat region is observed in the MSD; the dynamics remain subdiffusive throughout. This suggests that the usual glassy behavior is absent in our system under the studied conditions.

We emphasize that although the motivation behind our model is the cell membrane, we do not attempt to capture all the incredible complexity of natural biological membranes in this model. A simpler experimental analog of our model is a giant unilamellar vesicle with cytoskeleton [38,48–51]—a step towards building synthetic cells. In such composite systems, also in cells, the coupling of the cytoskeleton to the membrane can be tuned. Experiments [49] show that inclusion of cytoskeleton has minimal effect on the bending modulus (measured by the fluctuation spectrum of height field), and we find the same. A different technique (hydrodynamic tube pulling) shows that the effective cortical tension increases [52]. The tension is proportional to the area modulus K_A which in our model does increase. To the best of our knowledge, the effective shear modulus of such systems have not been measured yet. Note that, while comparing our model with these experiments we expect at best a qualitative agreement because we do not take into account either the active nature of the cytoskeleton or its viscoelastic property. It is straightforward to include activity and study how it influences the mechanical properties in our model along the lines described in Ref. [29].

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DATA AVAILABILITY

The figures in this paper are plotted using the free software matplotlib [53]. We use FFTW [54] for fast Fourier transform. The version of the code used in these computations are available at [55]. The data to reproduce the results in this paper are available at [56].

[1] A. D. Dupuy and D. M. Engelman, Protein area occupancy at the center of the red blood cell membrane, *Proc. Natl. Acad. Sci. USA* **105**, 2848 (2008).

[2] S. J. Singer and G. L. Nicolson, The fluid mosaic model of the structure of cell membranes, *Science* **175**, 720 (1972).

- [3] W. Helfrich, Elastic properties of lipid bilayers: Theory and possible experiments, *Z. Naturforsch. C J. Biosci.* **28**, 693 (1973).
- [4] M. Müller, K. Katsov, and M. Schick, Biological and synthetic membranes: What can be learned from a coarse-grained description? *Phys. Rep.* **434**, 113 (2006).
- [5] R. Phillips, J. Kondev, J. Theriot, H. G. Garcia, and N. Orme, *Physical Biology of the Cell*, 2nd ed. (Garland Science, New York, 2013).
- [6] H. J. Deuling and W. Helfrich, Red blood cell shapes as explained on the basis of curvature elasticity, *Biophys. J.* **16**, 861 (1976).
- [7] H. Deuling and W. Helfrich, The curvature elasticity of fluid membranes: A catalogue of vesicle shapes, *J. Phys. France* **37**, 1335 (1976).
- [8] C. Pozrikidis, Resting shape and spontaneous membrane curvature of red blood cells, *Math. Med. Biol.* **22**, 34 (2005).
- [9] G. H. W. Lim, M. Wortis, and R. Mukhopadhyay, Stomatocyte–discocyte–echinocyte sequence of the human red blood cell: Evidence for the bilayer–couple hypothesis from membrane mechanics, *Proc. Natl. Acad. Sci. USA* **99**, 16766 (2002).
- [10] J. B. Freund, Numerical simulation of flowing blood cells, *Annu. Rev. Fluid Mech.* **46**, 67 (2014).
- [11] S. Henon, G. Lenormand, A. Richert, and F. Gallet, A new determination of the shear modulus of the human erythrocyte membrane using optical tweezers, *Biophys. J.* **76**, 1145 (1999).
- [12] M. Dao, C. T. Lim, and S. Suresh, Mechanics of the human red blood cell deformed by optical tweezers, *J. Mech. Phys. Solids* **51**, 2259 (2003).
- [13] S. Suresh, Mechanical response of human red blood cells in health and disease: Some structure-property-function relationships, *J. Mater. Res.* **21**, 1871 (2006).
- [14] G. Bao and S. Suresh, Cell and molecular mechanics of biological materials, *Nat. Mater.* **2**, 715 (2003).
- [15] A. Ishihara, Y. Hou, and K. Jacobson, The Thy-1 antigen exhibits rapid lateral diffusion in the plasma membrane of rodent lymphoid cells and fibroblasts, *Proc. Natl. Acad. Sci. USA* **84**, 1290 (1987).
- [16] K. Ritchie, R. Iino, T. Fujiwara, K. Murase, and A. Kusumi, The fence and picket structure of the plasma membrane of live cells as revealed by single molecule techniques, *Mol. Membr. Biol.* **20**, 13 (2003).
- [17] P. S. James, C. Hennessy, T. Berge, and R. Jones, Compartmentalisation of the sperm plasma membrane: A FRAP, FLIP and SPFI analysis of putative diffusion barriers on the sperm head, *J. Cell Sci.* **117**, 6485 (2004).
- [18] K. Murase, T. Fujiwara, Y. Umemura, K. Suzuki, R. Iino, H. Yamashita, M. Saito, H. Murakoshi, K. Ritchie, and A. Kusumi, Ultrafine membrane compartments for molecular diffusion as revealed by single molecule techniques, *Biophys. J.* **86**, 4075 (2004).
- [19] A. Kusumi, C. Nakada, K. Ritchie, K. Murase, K. Suzuki, H. Murakoshi, R. S. Kasai, J. Kondo, and T. Fujiwara, Paradigm shift of the plasma membrane concept from the two-dimensional continuum fluid to the partitioned fluid: High-speed single-molecule tracking of membrane molecules, *Annu. Rev. Biophys. Biomol. Struct.* **34**, 351 (2005).
- [20] S. Manley, J. M. Gillette, G. H. Patterson, H. Shroff, H. F. Hess, E. Betzig, and J. Lippincott-Schwartz, High-density mapping of single-molecule trajectories with photoactivated localization microscopy, *Nat. Methods* **5**, 155 (2008).
- [21] A. V. Weigel, B. Simon, M. M. Tamkun, and D. Krapf, Ergodic and nonergodic processes coexist in the plasma membrane as observed by single-molecule tracking, *Proc. Natl. Acad. Sci. USA* **108**, 6438 (2011).
- [22] D. Krapf, Mechanisms underlying anomalous diffusion in the plasma membrane, *Curr. Top. Membr.* **75**, 167 (2015).
- [23] R. P. Feynman, R. B. Leighton, and M. Sands, *The Feynman Lectures on Physics: Mainly Electromagnetism and Matter* (Addison-Wesley, Reading, 1977), Vol. 2.
- [24] L. Landau and E. Lifshitz, *Theory of Elasticity*, Course of Theoretical Physics, Vol. 7 (Pergamon Press, Oxford, 1970).
- [25] G. Gompper and D. Kroll, in *Statistical Mechanics of Membranes and Surfaces*, edited by D. Nelson, T. Piran, and S. Weinberg (World Scientific, 2004), pp. 359–426.
- [26] J. Paulose, G. A. Vliegenthart, G. Gompper, and D. R. Nelson, Fluctuating shells under pressure, *Proc. Natl. Acad. Sci. USA* **109**, 19551 (2012).
- [27] T. Auth, D. A. Fedosov, and G. Gompper, in *The Giant Vesicle Book*, edited by R. Dimova and C. M. Marques (CRC Press, Boca Raton, London, New York, 2019), pp. 169–193.
- [28] V. Agrawal, V. Pandey, H. Kylhammar, A. Dev, and D. Mitra, MeMC: A package for Monte Carlo simulations of spherical shells, *J. Open Source Softw.* **7**, 4305 (2022).
- [29] V. Agrawal, V. Pandey, and D. Mitra, Active buckling of pressurized spherical shells: Monte Carlo simulation, *Phys. Rev. E* **108**, L032601 (2023).
- [30] H. S. Seung and D. R. Nelson, Defects in flexible membranes with crystalline order, *Phys. Rev. A* **38**, 1005 (1988).
- [31] C. Itzykson, in *Proceedings of the GIFT Seminar, Jaca 85*, edited by J. t. Abad (World Scientific, Singapore, 1986), pp. 130–188.
- [32] D. M. Kroll and G. Gompper, The conformation of fluid membranes: Monte Carlo simulations, *Science* **255**, 968 (1992).
- [33] P. Mehta, M. Bukov, C.-H. Wang, A. G. Day, C. Richardson, C. K. Fisher, and D. J. Schwab, A high-bias, low-variance introduction to machine learning for physicists, *Phys. Rep.* **810**, 1 (2019).
- [34] See Supplemental Material at <http://link.aps.org/supplemental/10.1103/ljw-j37w> for videos of crease formation in the sheared solid membrane ($\Phi = 1$) and the absence of creases in the fluid membrane ($\Phi = 0$).
- [35] S. Busch, C. Smuda, L. C. Pardo, and T. Unruh, Molecular mechanism of long-range diffusion in phospholipid membranes studied by quasielastic neutron scattering, *J. Am. Chem. Soc.* **132**, 3232 (2010).
- [36] N. L. Andrews, K. A. Lidke, J. R. Pfeiffer, A. R. Burns, B. S. Wilson, J. M. Oliver, and D. S. Lidke, Actin restricts FcεRI diffusion and facilitates antigen-induced receptor immobilization, *Nat. Cell Biol.* **10**, 955 (2008).
- [37] F. Heinemann, S. K. Vogel, and P. Schwille, Lateral membrane diffusion modulated by a minimal actin cortex, *Biophys. J.* **104**, 1465 (2013).
- [38] T. Härtel and P. Schwille, in *The Giant Vesicle Book*, edited by R. Dimova and C. M. Marques (CRC Press, Boca Raton, London, New York, 2019), pp. 53–70.
- [39] E. Falck, T. Róg, M. Karttunen, and I. Vattulainen, Lateral diffusion in lipid membranes through collective flows, *J. Am. Chem. Soc.* **130**, 44 (2008).

- [40] M. Javanainen, H. Hammaren, L. Monticelli, J.-H. Jeon, M. S. Miettinen, H. Martinez-Seara, R. Metzler, and I. Vattulainen, Anomalous and normal diffusion of proteins and lipids in crowded lipid membranes, *Faraday Discuss.* **161**, 397 (2013).
- [41] M. Saxton, Anomalous diffusion due to obstacles: A Monte Carlo study, *Biophys. J.* **66**, 394 (1994).
- [42] T. Bauer, F. Höfling, T. Munk, E. Frey, and T. Franosch, The localization transition of the two-dimensional Lorentz model, *Eur. Phys. J. Spec. Top.* **189**, 103 (2010).
- [43] B. J. Sung and A. Yethiraj, Lateral diffusion and percolation in membranes, *Phys. Rev. Lett.* **96**, 228103 (2006).
- [44] F. Höfling and T. Franosch, Anomalous transport in the crowded world of biological cells, *Rep. Prog. Phys.* **76**, 046602 (2013).
- [45] L. Van Hove, Correlations in space and time and Born approximation scattering in systems of interacting particles, *Phys. Rev.* **95**, 249 (1954).
- [46] D. Bi, X. Yang, M. C. Marchetti, and M. L. Manning, Motility-driven glass and jamming transitions in biological tissues, *Phys. Rev. X* **6**, 021011 (2016).
- [47] J. Hendricks, R. Capellmann, A. B. Schofield, S. U. Egelhaaf, and M. Laurati, Different mechanisms for dynamical arrest in largely asymmetric binary mixtures, *Phys. Rev. E* **91**, 032308 (2015).
- [48] M. Murrell, L.-L. Pontani, K. Guevorkian, D. Cuvelier, P. Nassoy, and C. Sykes, Spreading dynamics of biomimetic actin cortices, *Biophys. J.* **100**, 1400 (2011).
- [49] F.-C. Tsai, B. Stuhmann, and G. H. Koenderink, Encapsulation of active cytoskeletal protein networks in cell-sized liposomes, *Langmuir* **27**, 10061 (2011).
- [50] S. K. Vogel, F. Heinemann, G. Chwastek, and P. Schwille, The design of MACs (minimal actin cortices), *Cytoskeleton* **70**, 706 (2013).
- [51] A. P. Liu and D. A. Fletcher, Biology under construction: *In vitro* reconstitution of cellular function, *Nat. Rev. Mol. Cell Biol.* **10**, 644 (2009).
- [52] K. Guevorkian, J. Manzi, L.-L. Pontani, F. Brochard-Wyart, and C. Sykes, Mechanics of biomimetic liposomes encapsulating an actin shell, *Biophys. J.* **109**, 2471 (2015).
- [53] J. D. Hunter, Matplotlib: A 2D graphics environment, *Comput. Sci. Eng.* **9**, 90 (2007).
- [54] M. Frigo and S. G. Johnson, The design and implementation of FFTW3, *Proc. IEEE* **93**, 216 (2005).
- [55] <https://github.com/vipinagrawal25/MeMC/tree/semisolid>.
- [56] <https://github.com/thecalculon/ssmplots>.