A simple probabilistic model of submicroscopic diatom morphogenesis

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Unicellular algae called diatoms morph biomineral compounds into tough exoskeletons via complex intracellular processes about which there is much to be learned. These exoskeletons feature a rich variety of structures from submicroscale to milliscale, many that have not been reproduced in vitro. In order to help understand this complex miniature morphogenesis, here we introduce and analyse a simple model of biomineral kinetics, focusing on the exoskeleton's submicroscopic patterned planar structures called pore occlusions. The model reproduces most features of these pore occlusions by retuning just one parameter, thereby indicating what physio-biochemical mechanisms could sufficiently explain morphogenesis at the submicroscopic scale: it is sufficient to identify a mechanism of lateral negative feedback on the biomineral reaction kinetics. The model is nonlinear and stochastic; it is an extended version of the threshold voter model. Its mean-field equation provides a simple and, as far as the authors are aware, new way of mapping out the spatial patterns produced by lateral inhibition and variants thereof.

1. Introduction

Collect any spoonful of mud from a riverbed, any scrape of residue from a tidal rock or any potful of coastal water. Upon its examination under the light microscope, the careful eye will most likely observe a number of microscopic entities, each of a curious, well-defined shape. These are diatoms, unicellular algae that are prolific in the world's waters. Their shapes are bestowed by tough, biosilica cell walls called frustules. These frustules display a variety and an intricacy of hierarchical structures so plentiful that they have delighted and puzzled natural scientists for some 300 years, since the development of the first light microscopes [1,2]. The structures range from the curious micro-milliscale bounding cell wall shapes seen in the microscope (figure 1a), to slender spines radiating out from the cell to reach a length of up to five times the cell's diameter, to interlocking finger-like joints between diatoms that maintain filamentous colonies [3,4]. The delight and puzzlement are reinforced by the fact that some of these structures are extremely tiny, yet they are rebuilt faithfully repeatedly upon each mitotic cell division.

Documenting the range of frustule morphologies has been a bewildering task: taxonomists use frustule morphology to categorize diatoms, by which measure there are over 100 000 species. In order to understand how the diatom cell coordinates its components to bring about this morphology, there is much work to be carried out—this is a more bewildering task, and it is useful to break it into smaller, more digestible pieces. In this study, we focus on the tiniest biosilica structures of frustules, the nano-mesoscale morphologies called pore occlusions that occlude larger microscale pores.

Ultrastructural studies evidence a complicated morphogenetic sequence that is common to all diatoms ([3–5]; figure 1b). In brief, upon mitotic cell division, each daughter cell inherits one-half of its mother's frustule, frustules being always bipartite, usually comprising two large, primarily planar, components called valves connected by a series of smaller linking bands called girdle bands. During the cell cycle, each daughter cell constructs afresh one-half of a frustule

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(a new valve plus new girdle bands), which mirrors its inherited half frustule, thereby propagating the frustule's form over generations.

Construction of the valve proceeds intracellularly over about 1–2 h following cytokinesis within a large, purpose-built lipid bilayer vesicle called the *silica deposition vesicle*. Within this vesicle, firstly, biosilica precipitates to form a baselayer of microscale pores; then, subsidiary structures, such as microscale interlocking fingers, form; finally, within the microscale pores, biosilica precipitates to form the nano-mesoscale pore occlusions [4]. Each daughter cell becomes integral when the new valve is completed and putatively exocytosed through the cell membrane; then new girdle bands are added throughout the cell cycle. Silica for the frustule is derived ultimately from monosilicic acid in the diatom’s aquatic environment; it is sequestered actively by cell membrane transporters called silica uptake transporters, then processed intracellularly and delivered to the silica deposition vesicle via unknown pathways. It is likely that the cells’ turgor pressures bring about the primarily flat shape of the valves by causing sibling valves to be pressed against one another.

Inspections of bulk parts of pore occlusions, i.e. parts away from the boundaries of the microscale pores, suggest a morphological scheme: blocked, porous, hexagonally netted, convoluted netted, concentrically striped with crossbars, minutely nano-pored, or hierarchically occluded, e.g. stripes occluded by nanopores (figure 2). All pore occlusions on a valve belong to the same class—they all look the same; moreover, class is inherited from mother to daughter cell (the corresponding diatomist’s terminology is listed in figure 2).

In recent years, two whole diatom genomes have been published [6–8], and a number of elegant studies have identified various organic molecules that are embedded within the frustule (for a review, see [9]). Despite this progress, little is established about how various molecular players interact to determine this nano-mesoscale morphology, or about how this morphology is prescribed by genetics. Experiments are often very difficult to perform on diatoms, precisely because of their tough exoskeletons.

So motivated, we introduce and analyse a model that accounts for the following three well-established facts.

1. Morphogenesis proceeds by biosilica precipitation reactions, and these reactions occur only at the solid biosilica/fluid solution interface that is being formed: there is good microscopical evidence that silica precursors nucleate a solid biosilica structure only upon initiation of valve morphogenesis [3,4].

2. Each class of pore occlusion features a characteristic wavelength of 10–100 nm, defined by regions of solid biosilica alternating with regions that are unoccluded and presumably fluid. The model must feature at least one tunable length-scale of order 10–100 nm.

3. Because pore occlusion substructures are tiny, in some cases approaching the macromolecular scale, the kinetics of their formation is likely to be subject to continual fluctuations. It is simplest to use a model that is probabilistic and discrete, because fluctuations of a rescalable size are then automatically built into the model.

The complexity of the chemical, electrostatic and mechanical interactions involved in morphogenesis, and our present
Figure 2. A morphological classification scheme and the corresponding diatomist’s descriptive terminology (Latin) for the substructures of microscale pores. These substructures are called pore occlusions. Each panel displays between one and four microscale pores; the lengths of the major axis of each microscale pore are listed at the bottom. From the left, pore occlusions in the first five panels have wavelengths 60–100 nm, whereas the nanopores/occluded stripes have wavelengths of 10/220 nm. Hexagonal nets are reproduced with permission from Round et al. [3]. Blocks are courtesy of Nelson Navarro.

2. Material and methods

2.1. Laterally dependent stochastic aggregation

The model, which we call laterally dependent stochastic aggregation (LDSA), is a continuous-time Markov chain \( U(t) \) that evolves the configuration of white and black cells over a discretized square grid lattice \( \Omega \). At any time \( t \), a configuration assigns to each discrete grid position \( x \) in \( \Omega \) (each cell) either the colour white \( U_x(t) = W \), or the colour black \( U_x(t) = B \). To model diatom morphogenesis, we adopt the convention that white cells represent precipitated biosilica, and black cells represent the fluid solution. From a given initial configuration, \( U(t) \) thereafter evolves as \( x \) change their colours according to the kinetics that is now described.

The interface of a configuration \( U(t) \) is the union of

\[
\begin{align*}
\partial W & = \{ \text{black cells in } \Omega \text{ with one or more white neighbours} \} \\
\partial B & = \{ \text{white cells in } \Omega \text{ with one or more black neighbours} \}
\end{align*}
\]

where in two dimensions the neighbours of cell \( x \) are the eight cells adjacent to it. Only cells along the interface \( \partial W \cup \partial B \) may flip their colour. This represents a kinetics of biosilica where biosilica precipitates only by aggregating onto precipitated biosilica, then may dissolve back into fluid solution. A black cell flipping to white is called precipitation, whereas a white cell flipping to black is called dissolution.

We introduce a long-range length-scale by requiring that, along this interface, colour flips occur at a rate that depends on the local density of white, or equivalently black, cells within a specific radius \( r_1 \) (in units of a cell diameter, \( r_1 \gg 1 \)). Later, we introduce a second short-range length-scale in the same way but with a different specific radius \( r_2 < r_1 \). Precisely, around each \( x \), we consider two concentric spheres \( L_x S_{r_1} \) of radii \( r_1 \gg r_2 \). Denote by \( n_{W_x} \) the excess fraction of white cells among all cells in \( L_x \) (i.e. \( n_{W_x} \) is the fraction of white cells minus the fraction of black cells), and denote by \( b_L \) the excess fraction of black cells among all cells in \( L_x \). Of course we have \( n_{W_x} = -b_L \). Likewise for \( n_{W_x}, b_L \). As a time increment \( \Delta t \) elapses from \( t \) to \( t + \Delta t \), \( U(t) \) may evolve by a colour flip according to

\[
x \in \partial W : B \xrightarrow{\mathcal{U}} W \quad \text{with probability} \quad \sqrt{\kappa e^{km_x + \sigma m_y} \Delta t},
\]

and

\[
x \in \partial B : W \xrightarrow{\mathcal{U}} B \quad \text{with probability} \quad \sqrt{\kappa^{-1} e^{kb_x + \sigma b_y} \Delta t},
\]

the probabilities being specified up to of \( \Delta t \). This Arrhenius kinetics represents an unknown homogeneous and isotropic mechanism which effects a slowing down \( \lambda \) (or negative) or speeding up \( \lambda \) (or positive) of precipitation versus dissolution that depends exponentially on the nearby, lateral concentration(s) of precipitated biosilica/fluid solution within set length-scale(s). In §3 and the supplementary material, the kinetics (2.1) and (2.2) are perturbed in order to demonstrate that our conclusions apparently do not depend on the specific reciprocal or exponential form of the kinetics, but rather depend only on a few key features of the model. The reciprocal form of the kinetics was chosen in order to make our exposition more concise.

\( \lambda \) and \( \sigma \) control the degree to which the nearby, lateral densities of white/black cells feed back to alter reaction rates. When \( \lambda \) and \( \sigma \) are zero, the model is a particular version of the threshold voter model. The lateral feedback over \( r_1 \) is positive when \( \lambda \) is positive and negative when \( \lambda \) is negative. As \( |\lambda| \to 0 \), all white/black cells on the interface become equally likely to flip their colour and there is more noise in the dynamics, while as \( |\lambda| \to \infty \) then only the white/black cells on the interface with the highest \( \lambda \) positive or lowest \( \lambda \) negative lateral density of oppositely coloured cells are able to flip their colour and the model tends to a deterministic cellular automaton. Likewise for the lateral feedback over \( r_2 \) controlled by \( \sigma \). \( \kappa \) is the rate of precipitation versus dissolution when the lateral feedbacks are switched off. The number of cells on the major axis of \( \Omega \) is denoted by \( d(\Omega) \).

blocks confined pores hexagonal nets concentric stripes/nets + cross-bars convoluted nets nano-pores nano-pores (foriculae) (cribra) (cribra) (rotae) (cribra) (hymenes) (hymenes)

600–800 nm 200–300 nm 600–800 nm 500–600 nm 600–800 nm 200 nm 1500 nm
At time \( t = 0 \), the cells inside \( \Omega \) have colours that are independent and identically distributed, taking value \( W \) with probability \( p^W \) — the colour of each cell is determined by a coin toss that is biased when \( p^W \neq 1/2 \). This constitutes the initial condition, the configuration \( U(0) \), which ensures that initially the colours of different cells are uncoupled. For the cells outside \( \Omega \), their colours are prescribed at time \( t = 0 \) and thereafter remain fixed for all \( t > 0 \); these permanently coloured cells constitute the boundary condition. The colour prescriptions for different \( x \) outside of \( \Omega \) are independent and identically distributed, taking value \( W \) with probability \( p^W \).

### 2.2. Simulations

LDSA was simulated by an algorithm that requires no finite time-step approximation. This algorithm is commonly known as the Gillespie algorithm (see [13]). Simulations run until time \( T \) when the ergodic mean of the fraction of cells in \( \Omega \) that are white no longer changes beyond the admissible error \( \epsilon \leq 10^{-5} \). In all simulations, the kinetics have approximately converged to a time-invariant attractor by time \( T \); this happens in a computationally efficient and reproducible manner (see the electronic supplementary material). In all figures, cells outside of \( \Omega \) that set the boundary condition are coloured grey; white cells are coloured light grey and black cells are coloured dark grey. In an attempt to emphasize visually the distribution of stochastic activity, white cells along the interface (online version) or grey (printed version).

A useful check on the simulation is to set parameters \( \kappa = 1 \) and \( p^W = p^B = 1/2 \), which produces kinetics that is statistically invariant under \( W \to B \) inversion ((2.1) and (2.2) appear identical when white cells are swapped with black cells). Spot-checks where \( d(\Omega), r_L \) and \( r_S \geq 2 \) were simultaneously rescaled had no qualitative effect on the simulations, indicating that the anisotropy of the square lattice does not influence the results as long as \( r_L \) and \( r_S \) are considerably greater than 1.

### 2.3. Observational data

Pore ultrastructure in a range of diatom taxa was studied by scanning electron microscopy (Philips XL30) at the Natural History Museum, London, after preparation by standard methods [14].

### 3. Results

The kinetics of LDSA invariably converges to macroscopically time-invariant attractors. These attractors bifurcate as parameters change as described by the phase diagrams below. First, we examine the effect of the first lateral feedback alone, then the effect of both lateral feedbacks. We present qualitative descriptions of our computational findings rather than quantitative statistics. The derivation of the corresponding mean-field equation is mathematically rigorous (see the electronic supplementary material).

#### 3.1. Blocks, hexagonal nets and stripes from the first lateral feedback

Figure 3a is the phase diagram for one sphere of lateral feedback (only \( \lambda \) is switched on, while \( \sigma \) = 0). When \( \kappa = 1 \), a strong positive lateral feedback (\( \lambda > 0 \)) produces bistable attractors of a monochromatic block: with probability 1/2 all cells are white, otherwise all cells are black. A weak lateral feedback (\( |\lambda| \leq 1 \)) produces homogeneous attractors that have all cells fluctuating (at first this is surprising, because there is an apparent irreversibility about the model owing to the condition of interface movement, see the electronic supplementary material). A strong negative lateral feedback (\( \lambda \ll -1 \)) produces striped attractors; decreasing \( \lambda \) sharpens the stripes. In figure 3b a worked example using an analogous model in one-dimension illustrates this convergence to stripes (see the caption of figure 3b). As \( \kappa \) increases from 1, thereby breaking the inversion symmetry of the reactions (i.e. (2.1) and (2.2) change when white and black are swapped), stripes/bistable attractors bifurcate to hexagonal nets/white blocks. Increasing \( \kappa \) further, the black regions of hexagonal nets shrink in volume until eventually they disappear and so hexagonal nets bifurcate to white blocks. In all simulations, striped attractors have the wavelength \( 4r_L/3 \).

The simple model with just one lateral negative feedback produces kinetics that converge to persistent patterns of either uniform block colour, or hexagonal nets, or stripes, depending on the values of the parameters \( \lambda \) and \( \kappa \).

#### 3.2. A second lateral feedback introduces labyrinths

Figure 4 shows the phase diagram for two spheres of lateral feedback (both \( \lambda \) and \( \sigma \) switched on). In figure 4a, \( \kappa = 1 \). The phase diagram for one sphere is recovered along either the vertical or the horizontal axis. Along the axis \( (\lambda, 0) \approx (1, 1) \), where both feedbacks are either positive or negative, no qualitatively new attractor appears. A new attractor appears only when the two feedbacks are of opposite sign and when the long-range feedback is negative, \( (\lambda, \sigma) \approx (-1, 1) \) — labyrinths appear. Figure 4b shows \( \kappa \) increasing from 1 while \( \lambda \) and \( \sigma \) are constrained to this axis: labyrinths bifurcate to convoluted nets of pores. Figure 4c fixes \( (\sigma, \lambda) = 16 \times (-1, 1) \) to get labyrinths while \( r_S \) is increased from 0 up to \( r_L \). For \( r_S < r_L, \) the wavelength of the labyrinth is again \( 4r_L/3 \); this wavelength increases as \( r_S \to r_L \) (figure 4c).

Introducing a second lateral feedback introduces labyrinths and convoluted porous net attractors. In domains for which \( d(\Omega) \approx 4r_L/3 \), all phase diagrams are reproduced up to patterns’ orientations, as both the boundary condition \( p^W \) and the initial condition \( p^B \) vary (see the electronic supplementary material).

#### 3.3. Recapitulating diatom pore occlusions

The phase diagrams for one and two spheres of lateral feedbacks reveal that most pore occlusion morphologies can be recapitulated by LDSA with one lateral negative feedback plus, in some cases, a short-range positive feedback.

In figure 5a–c, all pore occlusions (bottom row) have wavelengths of between 60 and 100 nm. In the corresponding simulations (top row), the number of cells on the major axis \( d(\Omega) \) was set so that the diameter of one cell corresponds to a length equal to or exceeding 2.5 nm. The geometry and \( d(\Omega) \) were matched approximately to each pore occlusion in turn, then, while the lateral negative feedback was fixed at \( \lambda = -16 \) and \( r_S \) was fixed at 18 (14 in figure 5c) to produce wavelengths in excess of 2.5 \( \times 4 \times 18/3 \approx 60 \text{ nm} \), the precipitation versus dissolution rate (\( \kappa \)) was reduced (table 1). Figure 5b shows pore occlusions of *Achnanthes coarctata*. The diameter and the geometry of these pore occlusions can vary over the valve; concomitantly, the pores alter their configurations (insets of figure 5b, bottom row). Remarkably, the model approximately mimics these changing configurations as \( d(\Omega) \) and the geometry varied in the simulations.

Downloaded from http://rsif.royalsocietypublishing.org/ on June 28, 2017
as indicated on the axes. In all simulations, the other parameters are fixed at
most likely colour changes, and it converges from a macroscopically homogeneous attractor to a stable, striped attractor with wavelength 4
and (2.2)); because the lateral feedback is negative, the smaller the number, the quicker the change. From top to bottom, the diagram follows a succession of the
(non-interface cells are marked by ‘X’s and do not change colour), this number determines the average time until the cell changes its colour (see reactions (2.1)
the number of neighbours of opposite colour minus the number of neighbours of the same colour within the range
(since the lateral feedback per interface cell is not recapitulated.

Neither does the model reproducibly recapitulate the strands
boundaries (figure 5
features of LDSA

(A derivation from mean-field evolution equations is supplied in the electronic supplementary material.) When \( \kappa = 1 \),
\[
\sigma = 0: \quad w_{L} \approx 0, \quad \lambda = -\sigma: \quad w_{L} \approx w_{S}
\]
for all \( x \) on \( \partial W \cup \partial B \).

Straight interfaces (stripes) satisfy the approximation for one sphere (\( \sigma = 0 \)), whereas interfaces that are curved everywhere (labyrinths) satisfy the approximation for two spheres and \( \lambda = -\sigma \). For one lateral negative feedback (\( \lambda \ll -1, \sigma = 0 \)), the fraction of white cells \( w_{L} \) increases as \( \kappa \) increases or as \( \lambda \) decreases (figure 3); hexagonal nets bifurcate to white blocks when, according to (3.1), \( w_{L} \) reaches its upper bound \( w_{L} \approx 1 \), equivalently \( \kappa \approx e^{-2\lambda} \) (see the electronic supplementary material).

We see how the correspondence between parameters and macroscopic time-invariant patterns can be anticipated by inspecting this simple mean-field approximation. The derivation of (3.1) uses only the key assumptions of interface movement and of isotropic lateral feedbacks affecting reaction rates; this indicates that the reciprocal form of reaction rates (2.1) and (2.2) is an inessential restriction on the model. Indeed, further simulations indicate that when the model is perturbed, phase diagrams remain qualitatively unchanged so long as these two key assumptions are retained (see the electronic supplementary material): our results are robust against such perturbations.

3.4. Phase diagrams are determined by only a few key
features of LDSA

Time-invariant patterned attractors must have reactions (2.1)
B \( \rightarrow \) W and (2.2) W \( \rightarrow \) B happening at equal rates along interfaces \( \partial W \cup \partial B \). This gets the mean-field approximation
\[
\lambda w_{L} + \sigma w_{S} \approx -\frac{\ln \kappa}{2} \quad \text{for all } x \text{ on } \partial W \cup \partial B.
\]
4. Discussion

LDSA produces patterns that are familiar throughout nature. They appear in systems of chemical reactions [15–18], in directional solidification [19], in granular/fluid flows [20], hydrodynamic instabilities, animal furs, seashells and elsewhere [21–23]. The ubiquity of these patterns in nature calls for a categorization of how microscopic evolution rules
species-specific organic molecules in Kroeger changing the structure of a catalyst for precipitation (the simply by for example increasing the concentration of silica or pore occlusion morphology but a number of morphologies indicates that it may be possible to reproduce not only one solution rate (just one parameter, e.g. the original precipitation versus dissolution effects a sufficiently strong lateral negative feedback on precipitation/dissolution could be responsible for pore occlusion morphogenesis. The criteria (i) and (ii) are simple, in contrast to the apparent complexity of diatom frustule morphologies. A lateral negative feedback of precipitation/dissolution means that (a) precipitation versus dissolution slows down owing to nearby regions of solid biosilica, or (b) precipitation versus dissolution speeds up owing to nearby regions of fluid solution (equivalently non-silicified regions). It is well understood that patterns can be brought about by lateral negative feedbacks [15,24]. If a mechanism that does (i) and (ii) can be modelled in vitro, our study indicates that it may be possible to reproduce not only one pore occlusion morphology but a number of morphologies simply by for example increasing the concentration of silica or changing the structure of a catalyst for precipitation (the species-specific organic molecules in Kröger et al. [25] catalyse silica precipitation). This might provide a clue to how genetic changes alter frustule morphology, for example, if silica uptake transporters reside not only on the cell membrane but also on the frustule’s silica deposition vesicle, do these transporters vary between species in density or activity during morphogenesis, indicating that different species modulate silica concentrations differently? As already mentioned, many different mechanisms can affect the patterns that are observed in diatom pore occlusions: it is important to consider what physico-biochemical mechanisms can be plausibly acting not only at the mesoscopic length-scale, but also within the silica deposition vesicle, within the diatom cell. We describe two simple, hypothetical physico-biochemical mechanisms.

First, suppose a chemical species diffuses in solution such that, after coming into contact with precipitated silica, the species inhibits further precipitation until its inhibitory behaviour is degraded by interaction with other chemicals as it diffuses in solution. This would produce a lateral negative feedback on precipitation (see (a) above); the mesoscopic length-scale would be set by the ratio of the chemical’s diffusivity to the degradation rate. In a number of synthetic systems, microscale patterning is thought to result from the reaction and diffusion of interacting chemicals [26]. Besides catalysts for precipitation, are there organic molecules that inhibit precipitation? Second, suppose that the fluid solution contains a microemulsion (a mixture of a special surfactant and two immiscible fluids in which one phase forms thermodynamically stable mesoscale globules), and that the globules of the microemulsion tend to cluster against precipitated silica perhaps owing to electrostatic interactions. Furthermore, suppose that, while so clustering, the globules inhibit precipitation. Then in regions of more fluid solution, globules cluster less frequently, and so precipitation is quicker in these regions. This is a lateral negative feedback on precipitation (see (b) above) where the mesoscopic length-scale is set by the diameter of the globules. Species-specific organic molecules that are thought to be amphiphilic are embedded in the frustule [25,27]. Could these be forming a microemulsion that interacts with precipitating and precipitated silica?

Instead of our model, there is an alternative model. Namely that morphogenesis proceeds by silica precipitating onto a ready-made molecular template, such as an emulsion, that forms spontaneously within the silica deposition vesicle [27,28], but as yet there is no direct evidence for such a spontaneously self-organizing template. In addition, templating roles for both tiny mesoscale vesicles and larger microscopic vesicles, which were observed in the cytoplasm appressed against the frustule’s silica deposition vesicle in one set of studies [29,30], have been proposed, but these elegant

Table 1. Parameters for figure 5. In all simulations, $\rho^2 = 1/2$. 

<table>
<thead>
<tr>
<th>Panel</th>
<th>$\kappa$</th>
<th>$(\lambda, r_s)$</th>
<th>$(\sigma, r_s)$</th>
<th>$d(\Omega)$</th>
<th>$\rho^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>(a) blocks</td>
<td>$2 \times 10^{14}$</td>
<td>$(-16,18)$</td>
<td>—</td>
<td>175</td>
<td>0.95</td>
</tr>
<tr>
<td>(b) confined pores</td>
<td>$10^6$</td>
<td>$(-16,18)$</td>
<td>—</td>
<td>90 (insets 70)</td>
<td>0.95</td>
</tr>
<tr>
<td>(c) hexagonal nets</td>
<td>$10^6$</td>
<td>$(-16,18)$</td>
<td>—</td>
<td>160</td>
<td>0.6</td>
</tr>
<tr>
<td>(d) concentric stripes</td>
<td>10</td>
<td>$(-16,18)$</td>
<td>—</td>
<td>130</td>
<td>0.95</td>
</tr>
<tr>
<td>(e) convoluted nets</td>
<td>$4 \times 10^2$</td>
<td>$(-16,18)$</td>
<td>$(8,5)$</td>
<td>160</td>
<td>0.95</td>
</tr>
<tr>
<td>(f) nanopores</td>
<td>$10^6$</td>
<td>$(-16,17)$</td>
<td>—</td>
<td>160</td>
<td>0.5</td>
</tr>
<tr>
<td>(g) occluded stripes</td>
<td>0.1</td>
<td>$(-16,22)$</td>
<td>—</td>
<td>160</td>
<td>0.5</td>
</tr>
</tbody>
</table>
ultrastructural studies have not yet been repeated, and there is no theory to account for how these vesicles organize into a precise pattern. Polymer networks of protein and polysaccharide are known to be embedded in the frustules at least of some species [31,32]. Because some pore occlusions look as if biosilica has precipitated onto networks of polymeric filaments (not shown), it is possible that networked filaments are important for morphogenesis in all diatoms; in particular, they may help to maintain the connectedness of the valve, a feature that our simple model does not a priori recapitulate.

In conclusion, we propose three challenging questions, answers to which could constitute major advances in understanding this diatom microscopic morphogenesis: (i) Can a mechanism of lateral negative feedback on precipitation/dissolution be identified at the length-scale of around 10–100 nm? (ii) At the length-scale of 1 μm? (iii) How does the cell control the sequence in which these mechanisms are brought into effect, producing the morphogenetic sequence of frustule formation that was outlined in §1? Perhaps another simple model that recapitulates the spatially hierarchical patterns observed in frustules, rather than only the submicroscopic patterns, would be instructive to pursue (3.1).

Thomas Duke approved the penultimate version of this manuscript, excluding the discussion, before his death in June 2012. L.W. remains deeply indebted to Tom Duke and deeply affected by his intellectual generosity. We thank Nelson Navarro for providing SEM micrographs, and the EMMA Unit at London’s Natural History Museum for technical support. We are especially grateful to Michael Cohen, Chiu Fan Lee and L. Mahadevan’s group for helpful discussions, and to Tadashi Tokieda for valuable feedback on a draft manuscript. This work was supported by an EPSRC fellowship awarded to L.W.

Endnotes

1Continuous-time Markov chains are commonly used to model network reactions where, owing to activation energies, chemicals typically interact many times before finally reacting with one another. So LDSA more ideally represents an aggregation phenomenon where precipitation and dissolution reaction rates are also (i) limited by activation energies, and (ii) affected by a deterministic field of finite range that sets up quickly compared with the times between reactions. We chose to use a continuous-time Markov chain to model diatom morphogenesis because the chemical reaction networks of precipitation and dissolution are not wholly known and because it is a simple way to incorporate fluctuations into the model. The implicit assumptions (i) and (ii) are, we believe, unimportant for our conclusions.

2For each colour configuration, the time until the next colour flip is exponentially distributed with mean 1/R, where R is the sum of all reaction rates; this exponential random variable is sampled to determine the time of the next colour flip. The probability that cell x ∈ Ω is the next cell to flip its colour is x’s reaction rate divided by R; a random variable from the corresponding distribution is sampled to determine which cell flips its colour. So, the algorithm evolves the dynamics by simulating two random variables for each successive colour flip and updating the colour configuration and the time accordingly.

3In reactions (2.1) and (2.2), reparametrizing κ as its reciprocal is equivalent to swapping white with black, so phase diagrams are shown only for κ ≥ 1.

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