Mathematical principles and models of plant growth mechanics

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Mathematical principles and models of plant growth mechanics: from cell wall dynamics to tissue morphogenesis

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We explain the principles behind mathematical models of plant growth mechanics and the biological insights they provide. We suggest open questions for mathematicians and biologists to tackle in the future.

Abstract (max 200 words)

Plant growth research produces a catalogue of complex open questions. We argue that plant growth is a highly mechanical process, and that mathematics gives an underlying framework with which to probe its fundamental unrevealed mechanisms. This review serves to illustrate the biological insights afforded by mathematical modelling and demonstrate the breadth of mathematically-rich problems available within plant sciences, thereby promoting a mutual appreciation across the disciplines. On the one hand, we explain the general mathematical principles behind mechanical growth models; on the other, we describe how modelling addresses specific problems in microscale cell wall mechanics, tip growth, morphogenesis and stress feedback. We conclude by identifying possible future directions for both biologists and mathematicians, including as-yet unanswered questions within various topics, stressing that interdisciplinary collaboration is vital for tackling the challenge of understanding plant growth mechanics.

Keywords (6-8 words)

Mechanics, modelling, growth, morphogenesis, pollen tubes, shoot apical meristem, microtubules

1 Introduction

Plant growth is a highly mechanical process, incorporating both reversible (elastic) and irreversible (plastic/viscoelastic) deformations. The cell wall withstands great tension, equivalent to 100-1000 atmospheres of tensile stress (Cosgrove 2005), and consists of three main components: cellulose, hemicellulose (e.g arabinoxylan or xyloglucan) and pectin (Scheller and Ulvskov 2010, Höfte et al. 2012, Park and Cosgrove 2015, Jarvis 2009, Cosgrove 2014). A cell wall inflated under the action of turgor pressure (causing wall stress) will be stretched to mechanical equilibrium, exhibiting a constant elastic strain or deformation. For growth to occur, there must be an irreversible deformation, which begins when the mechanical load exceeds some critical value (yield). Growth is carefully mediated

via active control of the wall's mechanical properties (e.g. by enzymatic action or new material deposition), altering either the yield or the post-yield behaviour

Growth is inherently a multiscale process, from rearrangement of the cell wall microstructure to the behaviour of a whole tissue (figure 1). On the microscale, bond breakage and polymer network rearrangement (wall loosening) results in a relaxation of wall stress, allowing for viscous flow of the cell wall, whilst thinning of the wall can be balanced by deposition of wall material. Drawing water into the cell during extension allows for permanent volume increase (plastic growth) and maintains a high level of turgor. Wall loosening can be mediated by the action of proteins or enzymes, such as expansins, xyloglucan endotransglucosylase/hydrolase (XTH), pectin-modifying enzymes (PME) and/or regulated by the action of hormones, such as auxin, gibberellins, abscisic acid, and so on (Cosgrove 2005, 2016). Turgor acts in all directions simultaneously as an isotropic force. To achieve directional growth, cell walls can be mechanically anisotropic; this anisotropy is often induced by the alignment of cellulose, thus cell walls highly regulate the direction of growth (Baskin and Jensen 2013, Kierzkowski and Routier-Kierzkowska 2019). On the macroscale, plant cells are rigidly connected to one another through their cell walls (unlike animal cells); no slippage can occur. As a result, macroscale morphogenesis and growth must be a collaborative process across the whole tissue (Hamant and Haswell 2017).

Existing reviews which examine the principles of plant growth mechanics include Geitmann and Dyson (2013), Geitmann and Ortega (2009), Prusinkiewicz (2004) and Bruce (2003). Chebli and Geitmann (2007) review specifically the mechanics of pollen tube growth. Kierzkowski and Routier-Kierzkowska (2019) highlight the role of geometry in plant growth, citing studies which incorporate imaging approaches. Hamant and Haswell (2017) summarise the role of mechanical cues. Ali et al. (2014) and Chickarmane et al. (2010) both examine morphogenesis, the latter specifically looking at the use of computational modelling. Experimental procedures for quantifying mechanical behaviour have also been reviewed by Bidhendi and Geitmann (2019), including discussion of how mathematics can aid in this quantification.

There can be a lack of mutual appreciation between biologists and mathematicians about their respective disciplines. Papers in biological journals receive 28% fewer citations for each additional equation per page in the main article (Fawcett and Higginson 2012). This hinders

communication between researchers of different backgrounds. In this review we hope to tackle this issue by highlighting the crucial biological insights which are generated through mathematical models, in a way that readers who are unfamiliar with the underlying theory can appreciate. We lay out an argument that mechanics is fundamental to plant growth and morphogenesis, and that mathematical frameworks are required to describe the mechanistic processes. Such frameworks allow access to details that experiments cannot determine (Chickarmane et al. 2010), can bypass the practical challenges of experimentation on living tissue (Dupuy et al. 2007), and provide a means of testing whether proposed mechanisms are sufficient to explain observed behaviour. We begin our review by explaining in section 2 the mathematical frameworks that underlie various plant growth models. In section 3, we dissect mathematical models concerning a number of plant systems, ending each subsection with an overview of the main ideas and future outlook. We finish the review by describing in sections 4 and 5 some prospective future directions for the field in general, including both biological and mathematical questions.

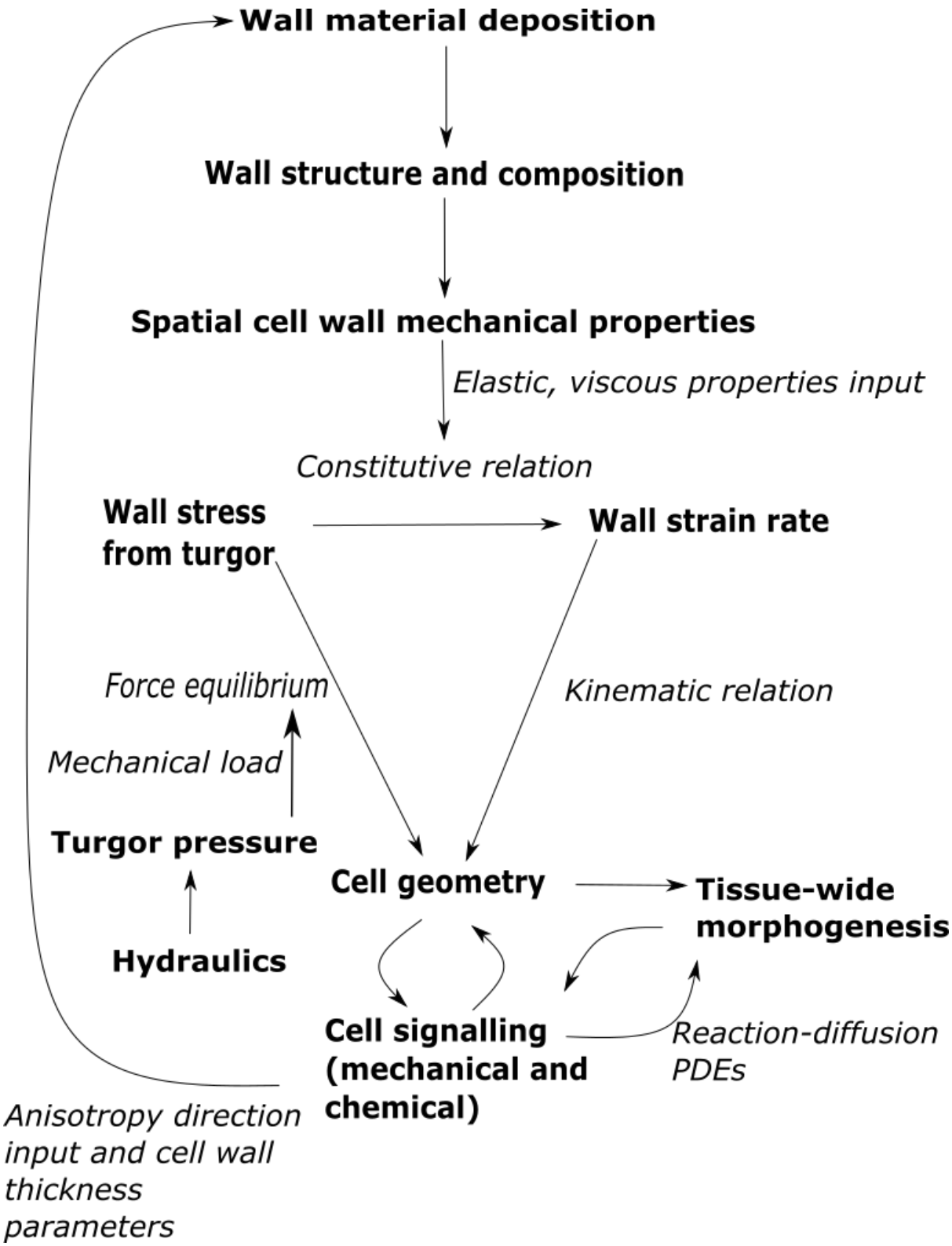

Figure 1: Various aspects of growth mechanics (bold) connected by mathematical modelling concepts (in italics), inspired by Dumais et al. (2006)

2 Mathematical Principles

Here, we introduce the mathematical principles which underlie the models described in section 3. We give brief overviews of different classes of methodology, aiming to explain the basic concepts only; references are given for more details on each technique. Key words/concepts are indicated in italics, whilst interactions between concepts are summarised in Figure 1.

Any mechanical model of a material (e.g. solid or fluid) is a set of mathematical equations which relate the material's intrinsic variables (including but not limited to deformations, flow rates, potential energies and heat fluxes) to internal and external forces. Those equations contain any number of *parameters,* which describe properties of the material. A parameter can be *geometric*, meaning it involves dimensions of length only, such as an area or volume; *kinematic*, involving dimensions of length and time, such as diffusivity; *dynamic*, involving length, time and mass, such as Young's modulus; or *thermodynamic*, involving length, time, mass and temperature, such as heat capacity. The algebraic procedure of *non-dimensionalisation* creates *dimensionless* parameters from suitable combinations of the aforementioned dimensional ones, and uses scaling factors to remove dimensions (i.e. units) from variables. Examples of dimensionless parameters include aspect ratios, efficiencies, and Reynold's number which is vital in many models of fluid flow. Expressing a mechanical model in terms of dimensionless variables and parameters usually affords valuable insights into the physical system, as the absence of "sizes" in the model implies that its outputs will hold irrespective of sizes in the system. An exemplary explanation of non-dimensionalisation can be found in Edelstein-Keshet (2005).

A primary example of a mechanical model of plant growth is the Lockhart equation (Lockhart 1965):

$$\frac{1}{V}\frac{dV}{dt} = \Phi_0(P - Y)\ if\ P \geq Y \text{ and}$$

$$\frac{1}{V}\frac{dV}{dt} = 0\ if\ P < Y\ , \qquad (1)$$

where V is the volume of a growing cell, t is time, P is turgor pressure within the cell, the parameter Y is a turgor yield threshold below which no growth occurs, and Φ_0 is an

extensibility parameter. Despite its shortcomings, variations on the Lockhart equation have become a standard paradigm for plant cell and tissue growth, as we discuss in section 3.

2.1 Continuum mechanics

Mechanical modelling of a material cannot be achieved at the scale of individual particles such as electrons and atoms, for two reasons. Firstly, the vast number of particles involved would make calculations practically impossible; secondly and more fundamentally, behaviours of a material at the macroscale are emergent phenomena which, despite being caused by collective interactions between particles, cannot be predicted from those interactions (Anderson 1972). A well-developed theoretical framework, which bypasses particle interactions and models a material as an infinitely divisible medium, is that of continuum mechanics.

Every material must obey the 'four fundamental axioms of mechanics', each of which is a *balance equation*, relating the rate of change of a variable – specifically mass, momentum (mass × velocity), angular momentum (mass × orbital radius squared × angular velocity), or energy – to the internal and external influences that could cause such a change (Eringen 1980). *Reaction-diffusion* equations are an important type of balance equation, which determine how the concentrations of quantities vary in time and space. In the biological context, many cell growth models involve a reaction-diffusion component which governs the dynamics of certain chemicals (e.g. pollen tube growth, section 3.2). If a continuum model is *thermodynamic*, i.e. involving some flux of heat, then an additional, fifth axiom of mechanics must be obeyed; this is the balance of entropy (Sandler 1999). An entropy balance equation relates the rate of change of disorder in a physical system to heat transferred and/or mechanical work done by the system. In some models of cell wall mechanics, thermodynamic principles are used to determined energetically favourable wall structures (e.g. micromechanics of cell wall construction, section 3.1).

As well as the universal balance equations, a mechanical model must include *dynamical equations* which are specific to the material. A typical dynamical equation is the *constitutive law*, relating a material's *stress* (internal forces) to its *deformation* (extension) (Astarita and Marrucci 1974, Paolucci 2016). The simplest constitutive law is Hooke's law: force is proportional to extension, with the constant of proportionality denoted by Young's modulus,

also known as the spring constant (see figure 2(a)). Some models of tissue-wide growth phenomena involve variations of Hooke's Law (e.g. the shoot apical meristem, section 3.3). In general, a constitutive law describes the mechanical behaviour of the given material (is the material solid/fluid, hard/soft, does it display any directional dependence, etc.). Most balance and dynamical equations are mathematical entities known as *partial differential equations* (PDEs). For a good introduction to PDEs, and examples rooted in real-world problems, we refer the reader to Mattheij et al. (2005).

Broadly speaking, the two types of material with which we are concerned are *solids* and *fluids* (more specifically, liquid fluids, as gaseous fluids are beyond the scope of this review). The difference between them is clearly reflected in the constitutive law which, for a solid, relates stress to *strain*, i.e. the amount of deformation, via a material property known as *stiffness;* in contrast, the constitutive law for a fluid relates stress to strain-rate, i.e. the velocity at which deformation occurs, via a property of the fluid called *viscosity*. For an illustration of the various concepts we have introduced, see figure 2. However, the categorisation of materials is far from binary. For instance, *viscoelastic* materials are considered intermediate between solids and fluids, with constitutive laws relating stress to a combination of strain and strain-rate (Dill 2007). Mathematical models of plant growth require a choice of constitutive law appropriate to capture the key behaviour for a given system on the time and length scales of interest (for example treating the cell wall as a viscous fluid on a long timescale (see section 3.1).

Stress, strain and strain-rate in a material are represented mathematically by quantities known as *tensors*. A tensor is written as an array of numbers and/or functions, with each entry known as a *tensor* component. A rank-1 tensor is commonly known as a vector, whilst a rank-2 tensor can be represented by a matrix. In 3-dimensional space, a vector has 3 components, and a rank-2 tensor – the most common type in continuum mechanics – has 3^2=9 components. In a stress tensor, each of the 9 components can be interpreted as stress in a particular direction, such as normal stress (due to forces perpendicular to material cross-sections) or shear stress (due to forces parallel to material cross-sections). Of particular note in plant biomechanics are those stress tensors that display *anisotropy*, i.e. directional variations. This type of stress is related to the geometry of cells (figure 1). For instance, within spherical shapes the stress in the cell wall created by turgor tends to be isotropic (same in every directions) but in elongated cells the stress is anisotropic, which is why cells need to

have circumferential cellulose reinforcement to resist the stress. Moreover, stress distribution depends on morphology, in the sense that areas of reduced stress correspond to elongation (Kierzkowski and Routier-Kierzkowska 2019, Kierzkowski et al. 2012). A practical, physics-oriented description of generic tensors is contained in Arfken and Weber (1995), while the technically-minded reader may enjoy the rigorous treatment of tensor algebra in Renteln (2014) from the perspective of differential geometry. Specificities of stress, strain, strain-rate and viscosity tensors are excellently elucidated in Spencer (2004).

We can roughly sub-categorise solids into elastic (whose deformations are entirely reversible) and plastic (which exhibits irreversible deformation), and fluids into Newtonian ("normal" fluids such as water) and non-Newtonian ("weird" fluids such as custard). For a Newtonian fluid (which is assumed to be incompressible, i.e. with constant density), there is a simple linear relation between stress and strain-rate, with the constant of proportionality being the fluid viscosity, a measure of how much the fluid resists flow (see figure 2(b)). When a non-Newtonian fluid is considered, stress and strain-rate may be related by a viscosity tensor, giving a non-linear relationship (Brujan 2011). For a model to describe growth, irreversible deformation must be possible; when a plant cell wall is modelled as a fluid, it is typically non-Newtonian (see section 3.1); For solids, there exist numerous types of elasticity and plasticity, each requiring its own model, which Spencer (2004) outlines succinctly. Each model involves a strain energy function, which is differentiated to give the stress tensor. When deformations are small, so that a linear relationship between stress and strain is found, the solid is said to be linear elastic; for large deformations, the solid is hyperelastic. For examples of hyperelastic models with sophisticated constitutive laws, such as the neo-Hookean, Mooney-Rivlin, and Ogden models - the latter of which is particularly applicable to biological tissue - the reader is referred to the comprehensive text by Ogden (2013). There is a broad literature on the subjects of solids and fluids: Goodier and Hodge (1958) includes a rich catalogue of solid mechanics problems; Parker (2003) is a clear, elementary account of Newtonian fluids; and Brujan (2011) concisely explains the basic concept of non-Newtonian fluids, giving examples of constitutive laws from various well-known models. For an in-depth exposition of the vast number of non-Newtonian fluid models that exist, see Bird et al. (1987).

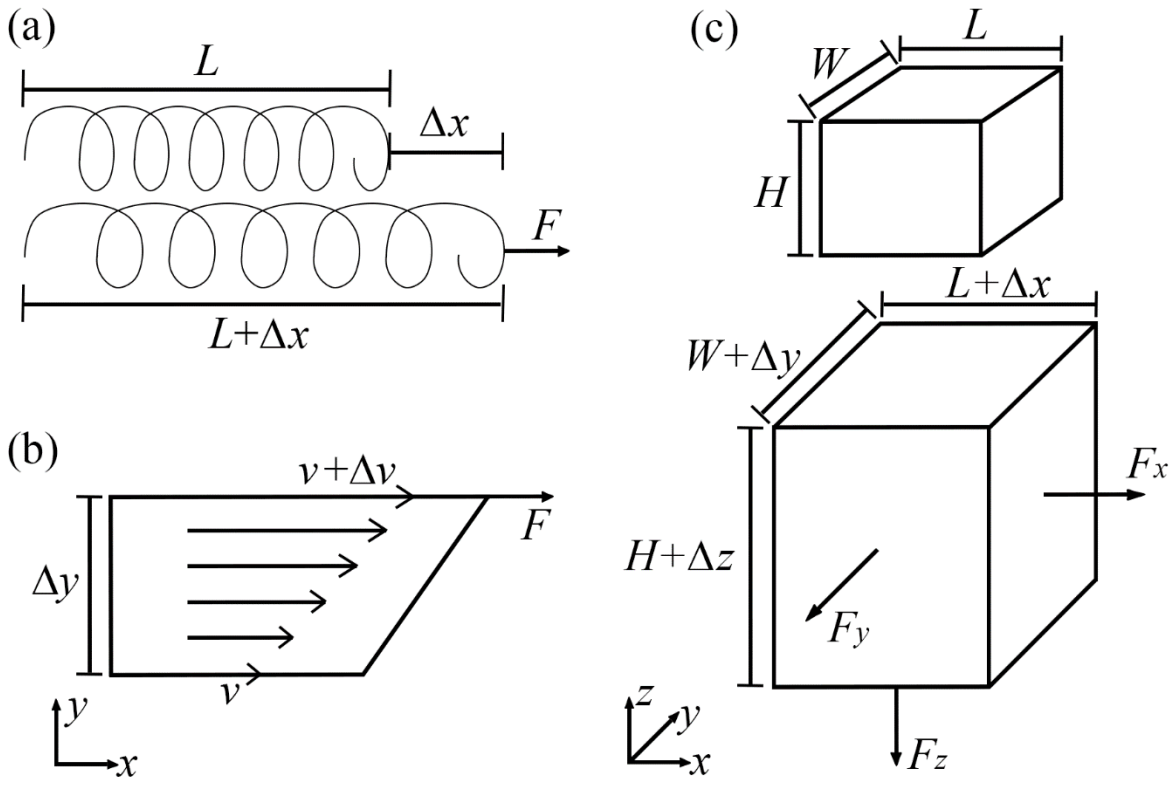

Figure 2. Stress, strain, and strain-rate. (a) The stretching and compressing deformation of a spring under an applied force is considered a one-dimensional system. Under the force F, the spring is in equilibrium (held at constant length), having extended from its natural length of L to its deformed length of $L+\Delta x$. To say the spring obeys Hooke's law means that there is some constant k, called the spring's *stiffness (*or spring constant*)*, such that $F = k\,\Delta x$. An equilibrium state with twice the force will exhibit twice the extension, halving the force halves the extension, and so on. (b) The strain-rate (velocity) of an (infinitesimal volume of) Newtonian fluid, side-view. If the fluid flow is uniform in the z-direction (out of page), then the system is two-dimensional in *(x,y)*. The bottom plate flows at velocity v while the top place, separated from the bottom by a distance Δy, flows at velocity $v+\Delta v$. The *shear stress* τ, defined as the shearing force F per unit area of the top plate, is related to Δy and Δv via a constant parameter called the fluid's *viscosity* μ: $\tau = \mu\,\Delta v/\Delta y$. (c) The strain (deformation) of an elastic solid under applied forces. The force on each surface is normal (perpendicular) to that surface, causing a strain according to a generalised Hooke's law: the vector (F_x, F_y, F_z) is related to the vector $(\Delta x, \Delta y, \Delta z)$ via some *stiffness matrix* with constant coefficients. If the force on any surface is not normal to that surface, then it will cause a shearing deformation. For example, if the force on the rightmost surface can be resolved into F_x along the x-axis and F_{xy} along the y-axis, then F_{xy} will cause a shear in the x-y plane. Components of the stress tensor are related to the (per-unit-area) forces, F_x, F_y, F_z, F_{xy}, F_{yz}, F_{zx} (at equilibrium, $F_{xy} = F_{yx}$ etc.). For further mathematical details concerning the stress tensor, as well as the analogous strain tensor and strain-rate tensor, we refer the reader to Spencer (2004).

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2.2 Asymptotics

PDEs in continuum mechanics models rarely admit exact solutions; in most situations, approximate solutions are sought. One of the most commonly used techniques for finding approximate solutions to a PDE is that of *asymptotics*. This method relies on the existence in the system of a dimensionless parameter, say ϵ, whose value is 'small'. For example, ϵ might represent the ratio of cell wall thickness to cell length, or the ratio of some cross-sectional area to surface area. One may then assume that any variable, say T, can be written as a regular asymptotic expansion in the form of $T = T_0 + \epsilon T_1 + \epsilon^2 T_2 + ..$, where T_0 is known as the 0^{th}-order solution and $T_{j>0}$ is known as the j^{th}-order correction. By substituting the asymptotic expansion into the system, one may determine $T_{j\geq 0}$ in succession; including higher-order corrections generally makes the solution more accurate. Steinrück (2010) formalises the general principles of asymptotics that we have described, and provides advanced examples. Further examples, which require the advanced method of matched asymptotics, are presented in Kevorkian (2000). For an interesting historical note on the development of asymptotic methods, we refer the reader to O'Malley (2014).

2.3 Finite elements

While *analytical* methods such as asymptotics are valuable in sufficiently simple systems, more complicated systems may require a *numerical* approach. The method of finite elements is a popular one for solving continuum mechanics models over a finite domain. The basis of the method is to partition the domain, such as the cell wall of a pollen tube, into a number of appropriately defined, usually small, regions called finite elements. One then looks for an approximate global solution which is represented within each element by a simple function. When the domain is a plant tissue, cells can be represented as vertices interconnected by edges representing cell walls; these edges are typically modelled as springs with some prescribed mechanical behaviour. This type of finite-element modelling approach is known as the *vertex element method* and will be explained further in section 3.3.

In Evans et al. (2000), the reader will find a detailed and practical introduction to finite-element methods, with worked examples that demonstrate solving some well-known physical problems. Elman et al. (2005) describes fast finite-element algorithms which are suited specifically to equations of fluid mechanics. A recent review by Bidhendia and Geitmann

(2018) offers a critical analysis of the use of FE methods in mechanical plant cell modelling, and advocates the use of FE for various plant growth problems, provided that good modelling practice is followed.

3 Models of Growth Mechanics

In this section, we summarise the insights into some plant growth scenarios provided by mathematical modelling techniques. Where appropriate, we explain how the models have been derived and solved. Beginning with the microscale and cellular aspects of growth in section 3.1, we review how cell wall components hold stress and how they are arranged. In section 3.2 we give an overview of models of tip-growing cells, specifically pollen tubes. This is followed by section 3.3, where we look at models on a larger scale, which deal with the mechanical process in morphogenesis, specifically in pavement cells and shoot apical meristem (SAM), and tissue signalling (thus following the flow of figure 1 by starting at the top and reading down).

We assume in this review that cellulose deposition angle is highly influenced by microtubules; even though it has been shown that this is not necessarily the case, and there is ongoing debate about the regulatory effect of microtubules on cellulose alignment and anisotropy (Baskin 2005, Cosgrove 2014, Baskin 2001). We will expand on this issue in Section 4. We will also be focusing only on the primary cell wall.

3.1 Cell wall properties and construction

Models of cellular and microscale dynamics within the cell wall may help to resolve the long-standing apparent paradox that the wall is weak enough to yield under turgor, yet strong enough for the cell to remain intact and resist bursting. The models described below incorporate elements of the cell wall microstructure to determine the emergent growth behaviour (see figure 1) and/or macroscale mechanical characteristics

It has been a matter of debate as to how cellulose fibres are connected within the cell wall. This is an important question, as the links between cellulose microfibrils by matrix polysaccharides determine most of the physical properties of the cell wall (Cosgrove 2005).

One early theory was the tethered/sticky network model, which assumed cellulose molecules were joined continuously along their lengths, and peel off as they get increasingly stretched during cell growth (Cosgrove 1993). There has been growing experimental evidence against this theory; for instance the observation that xyloglucan-digesting enzymes (xyloglucan is a hemicellulose that is said to crosslink the microfibrils) do not have a significant impact on the strength of the cell wall (Cosgrove 2014). According to a finite-element model featuring a network of cellulose molecules tethered together by hemicellulose via hydrogen bonds, a deformed network is not strong enough to withstand the strain (Yi and Puri 2012). This is evidence that the tethered network model is not a feasible explanation as to how the cell wall retains integrity. A plausible alternative theory of cell wall connectivity is the biomechanical hotspot hypothesis, which suggests that wall extensibility is controlled by a limited number of cellulose-cellulose contacts, potentially coordinated by xyloglucan (Cosgrove 2014). One hotspot model considers a network of cellulose connected by hotspots represented as linear springs (Nili et al. 2015). The model implies that a group of short xyloglucan strands is stiffer than a single long strand, and it can produce the requisite wall stiffness to oppose turgor. The hotspot hypothesis also claims that a small amount of degradation of the hotspots could lead to the load being carried by pectin, which then enables viscous flow of the cell wall, providing a possible mechanism for growth.

The micromechanics of cell wall construction allows for controlled creep and determines the ability of the cell wall to withstand turgor pressure, but the exact roles of the different wall elements in strength and in wall loosening are still unknown (Park and Cosgrove 2015, Cosgrove 2014, Braybrook et al. 2012). The fibres may play different roles at different states of the cell wall. It has been suggested that pre-yield (low strain) dynamics of the cell wall are dominated by hemicellulose fibres stretching and breaking, while post-yield behaviour (high strain) is dominated by pectins (Dyson et al. 2012). This was investigated using a fluid mechanical model of the cell wall, considering growth as a fluid flow which drives the stretching/straining of a network of hemicellulose fibres, each represented by a spring with stiffness κ, rest-length L_0, and evolving length L which is a function of time. These fibres connect cellulose molecules, with a breakage rate depending on the current strain. The stress resultant, Σ (essentially the axial tension), of the cell wall is found by summing (integrating) the effect of all bonds across the wall thickness, giving

$$\Sigma = \int_0^h n\kappa\left(L - L_0\right) \mathrm{d}y + \frac{\alpha}{\phi_M}, \qquad (2)$$

where h is the thickness of the cell wall, n the density of hemicellulose bonds, y the coordinate across the wall thickness, α the strain (growth) rate, and α/ϕ_M represents the contribution of pectin. The concentration of fibres might also affect wall strength. By re-deriving the Lockhart equation from thermodynamic principles, it can be shown that the cell wall yield is primarily determined by the concentration of xyloglucans and cellulose, and not the bonds between them (Veytsman and Cosgrove 1998). Even though this model considers a small time-scale on which material deposition is negligible, wall yield depends on xyloglucan concentration which in turn is determined by wall deposition. This implies an influential role of deposition in wall loosening and consequently in growth. The flexibility of the fibres also influences cell wall stress because hemicellulose could be trapped within the cellulose fibres. However, interactions with pectin are not incorporated within this framework.

The CMF is said to have a highly regulatory effect on extensibility and maintaining cell shape, but the detailed consequences of reorientation, distribution and crosslinking during growth are missing (Anderson et al 2010). Using linear elasticity under imposed turgor to examine the impact of CMF orientation, it has been found that it affects the radial elastic deformation at the ends of the cell, but that the presence of CMF on the cell end plates makes little difference to the cell's axial expansion (Ptashnyk and Seguin 2016). The model predicts that shifting the positions of the cells out of alignment in the tissue (i.e. lined up like bricks in a wall) allows for larger strains and increases the effect of varying microfibril configurations on axial expansion. A variety of orientations throughout the wall also reduces axial expansion and slightly increases radial growth. Another model has also found that the cell radius is maintained via the CMF (Dyson and Jensen 2010). Representing the cell wall as a fibre-reinforced thin sheet of viscous fluid, this model includes fibres (representing the CMF) which are convected by growth, and is analysed and solved using asymptotics. The model also finds that a variety of fundamental geometric and mechanical parameters related to the composite cell wall properties govern the cell wall extensibility.

Efforts have been made to understand pectins' regulatory effects on growth. Pectins are known to form hydrated gels that can force microfibrils apart, allowing for wall extensibility to increase and the microfibrils to slip (Cosgrove 2005). Deposition of pectin is also said to play a role, although its significance is still not completely understood. Using thermodynamic constitutive laws which involve turgor, temperature, volume, free and bound pectate, and the synthesis of pectates, a growth rate that principally depend on pectate crosslink synthesis can

be derived (Barbacci et al. 2013) (see figure 3). The comparison of this model's predictions with data concerning *Chara corallina* is "quite good". Meanwhile, by balancing cell wall growth rate and pectin insertion rate, it has been argued that turgor-driven deposition leads to cell wall polymerisation (see figure 3), which is a primary growth control mechanism (Ali and Traas 2016). The insertion rate in the model is derived from the thermodynamics principle of balancing the free energy difference between bound and unbound pectin states. Although the results match data qualitatively to *Chara corallina*, the authors acknowledge that other mechanisms are at play. Both of the pectin studies consider *Chara corallina* which contain a high amount of pectin, so these results may not be generalisable. It seems that hemicellulose connections do influence the yield threshold of growth, and these studies emphasise the role of pectins insertion in the viscous flow of the cell wall.

There are still remaining mysteries in cellular and microscale growth. For instance, despite growing evidence for the biomechanical hotspot hypothesis as we have described, there is no universal consensus on how the cell wall polymers are connected. Moreover, most studies heavily rely on xyloglucan being the load-bearing component in the cell wall, but it has been noted that xyloglucan possibly covers only a small portion of cellulose surface in the onion wall (Zheng et al. 2018), and that *Arabidopsis* mutants containing small amounts of xyloglucan have only minor changes in growth phenotype (Cosgrove 2016). The exact role of pectin in the cell wall structure has also not been carefully investigated. All of these are prerequisites for understanding how the cell wall expands. There has also not been substantial work on the action of enzymes. Some models have shown that in order to match experimental data, expansin must affect extensibility (Pietruszka 2011). Apart from this, not much work has been done to model how enzymes manipulate the cell wall to cause wall relaxation, strengthening, etc., and we will expand on this point in section 4.

3.2 Tip-growing cells

There are a number of cells such as pollen tubes and root hairs which extend via tip growth, where growth is highly localised to one area of the cell wall. Growth rates are typically very high, despite turgor pressures similar to other cell types (Beauzamy et al. 2014). Tight control of both mechanical properties and new material deposition is therefore required, and modelling can help understand these coupled processes (Geitmann and Emons 2000). Here we review tip growth in pollen tubes.

Pollen tubes are of particular interest due to their high growth rate (1cm/h) (Bove et al. 2008) and their oscillatory growth patterns (Kroeger and Geitmann 2012), but their growth is as yet not fully understood. To model pollen tube growth, one needs to couple cell mechanics to biochemical processes (Cameron and Geitmann 2018). Measurements of turgor do not show significant oscillations, implying that it is likely not a driving mechanism behind growth oscillations (Beauzamy et al. 2014). One possible explanation for oscillatory growth is a vesicle recycling mechanism dependant on calcium concentration, where the fusion of vesicles at the apex is stimulated by calcium ions (Kroeger et al. 2008). In this framework, the pollen tube invading the external media is modelled as one viscous fluid being injected into another, surrounded by a viscoelastic membrane representing the wall. The overall growth velocity is calculated via Darcy's law of pressure-driven fluid flow

$$\mathbf{u} = \frac{K}{\mu}\nabla p \,, \tag{3}$$

where $\boldsymbol{u}$ is the tip apex velocity, K denotes the permeability of the external medium, μ the viscosity of the cell, p is the pressure, and ∇p gives the magnitude and direction of the pressure gradient. The idea behind this model is that the rate of flow/growth is proportional to the difference in pressure between two different regions (there is higher pressure in the cell due to turgor which drives the flow outwards), similar to diffusion. An effective elastic constant is incorporated into the fluid flow/growth rate, which in turn is dependent on calcium concentration at the cell walls (see figure 3). The calcium exocytosis rate on the cell wall is determined from a reaction-diffusion equation. These results agree qualitatively with experimental observations, with some discrepancies due to calcium absorption by the cell wall being neglected; this suggests that the assumption of calcium dynamics driving vesicle recycling could be robust. The pollen tube growth phenomenon of pearled morphology (oscillations in diameter to form a wavy boundary) is also not understood. A possible explanation is that it occurs as a result of the extension and deposition rates being out of phase (Rojas et al. 2011). Employing a principle that deposition causes crosslink turnover, this model considers a computational lattice network of nodes which are connected if there is a crosslink (modelled as linear springs), incorporating both esterified and de-esterified pectin (see figure 3). The model predicts both steady and oscillatory growth, agreeing with experimental data. Alternative morphologies have been observed in pollen tubes consisting of swelling or tapering of the pollen tube head; unlike the causes above which both relate to deposition, other mechanisms could be at play here. One model claims that the swelling

arises due to an insufficiently steep decrease in Young's modulus along the growth direction (Fayant et al. 2010). This claim is validated by the distribution of de-esterified (stiffer) pectin. The model also suggests that cellulose are important in resisting radial expansion, despite the randomness of their orientations. In conclusion, the model posits that the features affecting growth are spatially-varying components of the cell wall.

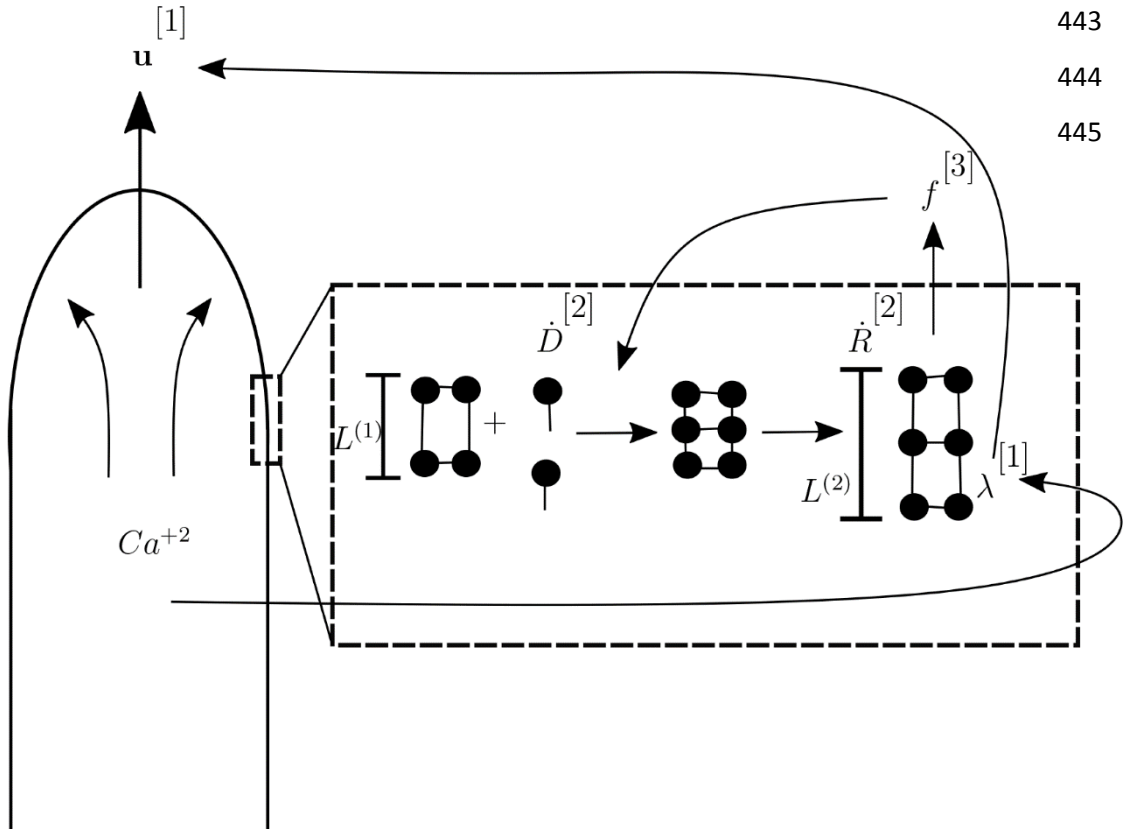

Figure 3: Pollen tube and pectin driven growth model principles. Kroeger et al. (2008) predict that the pressure gradient and calcium ion concentration both effect growth, **u** (equation 3) with the former determining the direction and latter affecting the elasticity constant, λ which in turn affects the extension rate (labelled [1]). Rojas et al. (2011) assume the deposition of pectin with rate $\dot{D}$ causes crosslink turnover, leading to extension in length from $L^{(1)}$ to $L^{(2)}$ with rate $\dot{R}$ (labelled [2]). Note diagram depicts only demethylesterified pectin. This pectin deposition driven growth framework also demonstrates the modelling ideas behind Ali and Traas (2016) and Barbacci et al. (2013) in *Chara corallina* with the former model using turgor driven extension of the wall (denoted as *f*), driving further polymerisation (labelled [3]).

There remain unexplored territories within pollen tube modelling. Most models assume axisymmetric growth with a straight centreline (in order to reduce complexity and computational time), and therefore are unsuited for investigating complex mechanisms such as steering, which to our knowledge has not been explicitly modelled. Regarding oscillatory growth, there is evidence that calcium-ion oscillations occur in non-growing pollen tubes, showing it could be independent of growth (Cameron and Geitmann 2018), motivating

452 further work in this area.

453

454 3.3 Models of tissue growth

455 We turn our attention to larger-scale models which are evaluated across tissues, where
456 mechanics influences both shape and growth (see figure 1). Plant cells are tightly fixed to
457 each other, therefore growth and morphogenesis arise due to collaborative expansion, cell
458 division and communication between cells (Cosgrove 2005, Hamant and Haswell 2017,
459 Mirabet et al. 2011). For example, careful control of organ growth is observed in leaves with
460 a reduced cell number, which still reach normal size by increasing the rate or duration of their
461 cell expansion, demonstrating shape-sensing mechanisms (Hervieux et al. 2016). In this
462 section, we review mathematical insights into problems posed by pavement cells, shoot apical
463 meristems (SAM) and stress signalling.

464

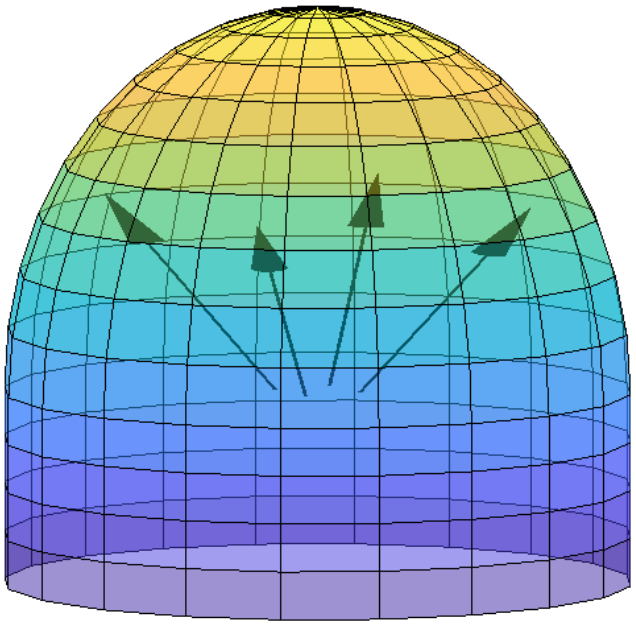

Figure 4: The concept of a shell model approximating a 3D tissue as a thin shell of epidermis inflated by pressure from the inner tissue. This significantly reduces computational time. The square objects on the shell represent a simple example mesh and do not correspond to cell walls.

465 We first explain the concept of a shell model. This is a 2D representation, a simplification of
466 the 3D tissue whereby only the outer epidermal layer is explicitly modelled, and tension from
467 the inner layers is imposed (see figure 4). The epidermis bears higher resistance to the
468 tension, as demonstrated experimentally. For example, a peeled, isolated epidermis will
469 contract, showing that it is under high stress (Savaldi-Goldstein et al. 2007, Hamant and

Haswell 2017, Beauzamy et al. 2015). Since the outer epidermis heavily restricts plant growth, the 2D shell can be a realistic assumption.

The diversity of shapes of epidermal cells in leaves (pavement cells) is truly spectacular. Some form highly undulating anticlinal walls, as in *Arabidopsis*, which lead to jigsaw-like patterns of cells (Vőfély et al. 2018). Mechanical stress is said to play an important role in determining these cells' geometry, but why and how the jigsaw patterns form has not been elucidated (Sapala et al. 2019). Here we shall refer to the indents and protrusions of a pavement cell as necks and lobes, respectively. Stress is found to be higher in the neck regions, by extracting 3D cell shapes using MorphoGraphX (de Reuille et al. 2015) and meshing the resulting surface, then incorporating fibre directions in a hyperelastic model (Sampathkumar et al. 2014). Moreover, by considering idealised ellipsoidal cells with or without protrusions, stress is found to transfer from the centres of cells to the neck regions, consequently reducing the overall stress (Sapala et al. 2018). Since the spongy mesophyll layer, which lies underneath the epidermis, contains air holes, it might not be able to provide strength to the tissue, therefore the epidermis must withstand most of the total stress. Thus, the stress reduction provided by the jigsaw morphology could be advantageous, as it could help reduce the resources needed to strengthen the tissue and/or reduce the chance of the tissue rupturing. As for *how* the jigsaw patterns come about, it has been shown that microtubules align with the direction of maximal stress, which can then reinforce the necks through the deposition of cellulose (Sampathkumar et al. 2014). The cellulose can then restrict expansion at the necks, which in turn increases the stress, creating a feedback loop where microtubules continue to align with the stress. This is a possible mechanism for how the lobes become more prominent and enlarge, but it still does not answer how the lobes *initially* form. To that end, a positive relationship between isotropic growth and 'lobeyness' has been proposed (Sapala et al. 2018). The model is validated by a 2D simulation of cells, where the walls are represented as nodes connected by linear springs (figure 6 – see description later in this section), with additional intra-cell springs to represent stiffening components such as cellulose.

It has also been suggested that lobe formation is a result of wall heterogeneities which cause buckling when the leaf epidermis is under tension from variations in growth rates across different cell layers (Majda et al. 2017). However, when this 2D modelling approach of only including the anticlinal walls (wall perpendicular to leaf surface) was recreated, the lobe

amplitude was not as significant; including the effect of the periclinal walls (wall parallel to leaf surface) the lobes are found to disappear (Bidhendi and Geitmann 2019). An alternative model including the periclinal wall was developed using finite element methods assuming the cell wall is neo-Hookean hyperelastic (Bidhendi et al. 2019). Upon analysis it was found that varying periclinal wall stiffness between neighbouring cells can induce lobe formation; a potential buckling mechanism is proposed due to cell geometry and internal pressure. Reduction of stress is therefore most likely a factor in why pavement cells form these interesting geometries but the origins of how they form could be connected with periclinal wall mechanics and possible buckling.

The shoot apical meristem (SAM) has often been of interest to plant biologists, due to its complex morphology of forming organ primordia, and because of its importance in leaf and floral meristems and stem development (Soyars et al. 2016). The SAM must maintain a source of undifferentiated cells, and differentiate cells in order to initiate organogenesis. This requires strict co-ordination and structure, which are not yet understood (Truskina and Vernoux 2018). The SAM's elastic and plastic properties which enable bulge initiation are also a mystery. There surely must be varying properties in the region to allow for the different growth rates in the shoot apex central zone and periphery. It has been proposed that the slow-growing area at the shoot tip is significantly strain-stiffened, and this may control the expansion process of the tip (Kierzkowski et al. 2012) (see figure 5). This model concludes that the difference between tip and peripheral growth rates is not due to variations in stress, but instead due to variations in other parameters such as yield threshold. In terms of methodology, the model approximates the surface as a shell of Ogden hyperelastic material (cf. section 2 and figure 4) with two regions of differing elastic properties: the shell tip where stiffness increases with strain, and the periphery where stiffness is constant. Validation of the model is provided by reproducing material behaviour from osmotic treatment experiments. Differing regions of elastic properties are also echoed by another model, which tests different mechanisms of organ emergence (Boudon et al. 2015). It finds that a bulge (similarly to the above) could be produced by changing the stiffness of the outer cell layers near the bulge tip, but not through variations in turgor or wall stiffness from interior layers. By creating a highly rigid ring around the bulge of cells, to promote further growth, the model produces a more distinct bump (see figure 5). This is a 3D model of tissue growth which includes gene regulation, and a generalised Lockhart equation relating the plastic deformation tensor to the elastic strain tensor. Both of the SAM models we have discussed illustrate the necessary

mechanical features to allow bulge initiation and maintain SAM morphogenesis, namely: in order to adapt the stiffness properties, cell wall properties of the surrounding cells vary with the distance from the tip of the initiation site.

Further on the topic of SAM, it has been noted that isotropic walls grow slower than anisotropic walls (Armezzani et al. 2018). This confused the understanding of emerging primordia, as their microtubules are in an isotropic setup while still growing faster than the surrounding meristem (Sassi et al. 2014). There also seem to be no change in cellulose deposition to alter the strength of the wall. Therefore there is likely to be some kind of signalling to promote wall loosening (Kierzkowski and Routier-Kierzkowska 2019). Through modelling and experimentation, evidence has been presented that microtubule re-organisation to an isotropic distribution can activate wall loosening genes (and vice versa), allowing organ emergence independently of auxin (Armezzani et al. 2018) (see figure 5). The model makes use of a 2D shell based on Hooke's law, which incorporates fibre orientation and plastic spring growth (figure 4 and 6). The signalling to genes is an essential part, without which isotropic walls in an anisotropic environment are unable to increase growth rates in organ outgrowth. These models all inform us that organ emergence requires not only differential mechanical properties from the tip to the periphery, but also a coupling between genes and the degree of anisotropy.

The thick, relatively stiff cell walls of the epidermis have been postulated to regulate stem growth via intricate interactions between cells and whole tissues (Baskin and Jensen 2013). It was found that the tissue structure is stabilised by the outer layer's strain stiffening behaviour, without which the tissue could buckle (Vandiver and Goriely 2008). This model was developed by creating a cylindrical model of the stem, consisting of two material layers with different properties. They defined a deformation gradient as the product of a growth tensor, which governs the unrestricted growth of both layers, and an elastic deformation tensor, which couples the layers together. To determine the growth and bending rate of the composite *Arabidopsis* root, a model assigns a yield threshold, wall viscosity and thickness to each individual cell wall segment, varying across cell files (Dyson et al. 2014). This was integrated over the cross section to obtain a tissue-wide Lockhart equation (c.f. eq. (1)). Parametrising with experimental data such as wall thickness and turgor values, they found that the epidermis plays a dominant (6-fold influence) role in regulating extension and described the effectiveness of targeting this layer to cause root bending. Both papers therefore

demonstrate the absolute mechanical importance of the epidermis in regulating and stabilising tissues.

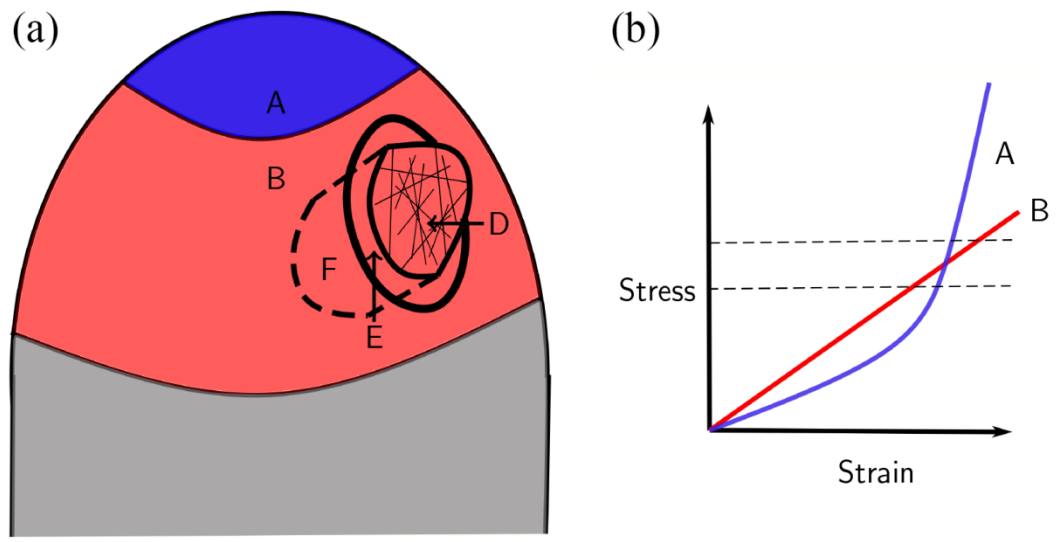

Figure 5: The requisite shoot apical meristem features for growth and organ emergence. Kierzkowski et al. (2012) demonstrated that in the slow growing apex, region A, there is strain stiffening behaviour and the faster growing periphery, region B, displayed linear behaviour (see (a)). This is depicted in (b) where the dotted line shows the strain at typical levels of turgor. The blue/red line depicts behaviour from region A/B showing the stress increasing rapidly/linearly demonstrating strain hardening in the former. The conditions for organ emergence was modelled by Boudon et al. (2015) who showed reducing the rigidity of the cells in region D allowed for organ emergence site and the creation of a rigid ring of cells in region E around the site can create a distinct bulge emergence. They also note that increased pressure from the bottom layers (region F) does not aid in organ emergence. Armezzani et al. (2018) also predict the necessary isotropic setup of microtubules which signals appropriate wall loosening genes in order for bulge initiation (region D).

Stress patterning can be crucial in tissue-wide signalling, as there is increasing evidence that cells can sense these mechanical forces, and that microtubules play a fundamental role in this phenomenon (Hamant and Haswell 2017). Evidence for this claim comes from observations that microtubules align with the stress direction, which allows for the deposition of cellulose to reinforce the cell in the principle direction of stress (Bozorg et al. 2014). Indeed this paper also shows that the microfibril direction is aligned with maximal stress direction and perpendicular to maximal strain direction in the SAM. Using mathematical modelling, one can directly approximate the distribution of stress and then compare it with the organisation of the microtubules (Mirabet et al. 2011). Moreover, by examining stress-feedback and the microtubule patterning at root hair initiation sites, it has been found that circumferential principal stresses around the loosened region lead to radial star configurations of

microtubules (Krupinski et al. 2016). More evidence is found in sepals where microtubules have been shown to align with maximal tension in sepals (Hervieux et al. 2016). At the sepal tip where growth slows down, microtubules are orientated in a setup that corresponds to fast anisotropic growth. The model includes a generalised Hooke's law, with a Young's modulus that increases from the base to the tip, and is solved by finite-element methods. Comparing this model with experiments leads to the hypothesis that microtubule stress feedback operates as a shape sensor at the tip and resists further radial expansion.

This microtubule function has also been found in the *Arabidopsis* shoot apex, whose shape is theorised to depend on the microtubule cytoskeleton, which is regulated in turn by the mechanical stress in a feedback loop (Hamant et al. 2008). The study combines experimental work, including fluorescent marking of microtubules and tissue imaging, with a shell model (see figure 4) that includes growing elastic walls elements, proliferation and anisotropy. The model defines the potential energy in the shell as

$$U = \sum_{w \in walls} \frac{k_w}{2}\left(\frac{l_w - l_w^0}{l_w}\right)^2 - \sum_{c \in cells} P_c A_c - \sum_{c \in cells} P_{c,int} V_{c,int}\,, \qquad (4)$$

where Σ denotes summation of the wall elements, $w \in walls$ (e.g. segment AC in figure 6) or cells, $c \in cells$ (e.g. the square ABCD in figure 6). The first term is the contribution of the wall element elastically stretching, where anisotropy is included in the stiffness k_w of the w^{th} wall element (this term increases when the w^{th} wall element aligns more closely with a defined direction of microtubules), which also changes due to stress feedback (microtubule directions are updated). Plastic spring growth is incorporated by increasing l_w^0. The second term in eq. (4) is the force from the internal pressure between cells in the simulated 2D shell layer (see figure 4), and the third is pressure emerging from the inner tissues including dependence on both area, A_c and volume, $V_{(c,int)}$. Models are often written in terms of the energy, because to stretch fibres (elastic deformation), break bonds and/or allow fibres to slip past each other (viscous/plastic deformation), we require energy. The energy comes from the action of turgor pressure pushing against the walls in the normal direction. There must then be a balance between turgor and wall stretching/yielding (figure 6). This is essentially what is

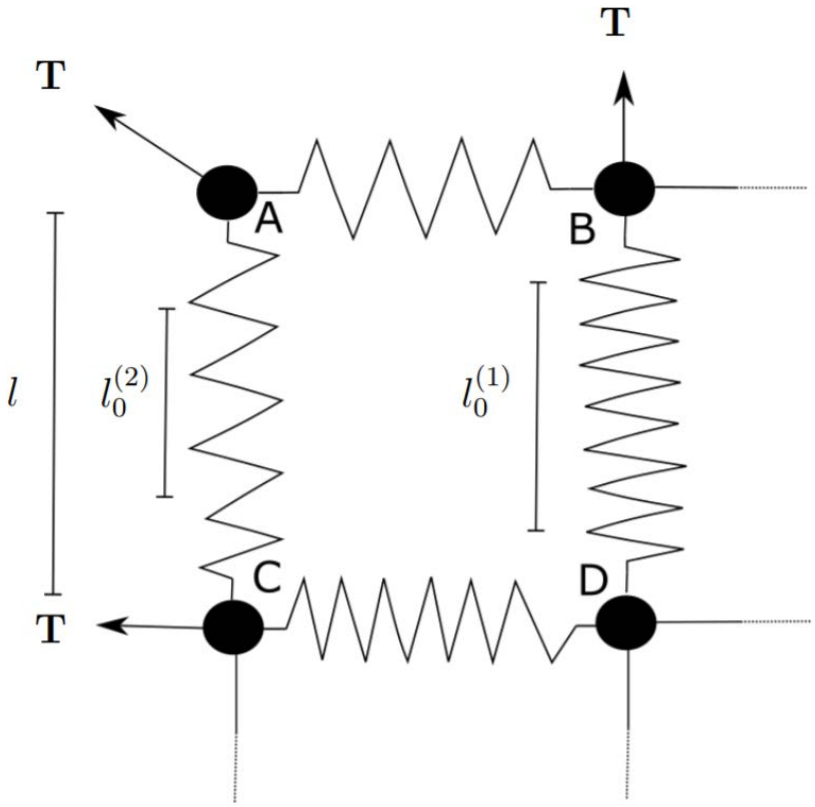

Figure 6: Principles of the vertex element method, plastic spring growth and mechanical energy balance/minimisation. A network of vertices (A,B,C,D) are attached via elements (AB,BD,CD,CA) which are represented as springs. In particular, A, B, C are outside elements with D on the inside. This simplistic system demonstrates the modelling of cell wall extension via plastic spring growth, with dynamically increasing resting lengths $l_0^{(1)}$ and $l_0^{(2)}$ (thus permanently extending the spring). Here the elements AC and BD have the same length l but AC has higher stress because its resting length $l_0^{(2)}$ is shorter than the BD resting length $l_0^{(1)}$ (so AC has undergone larger strain i.e. the difference between the resting length $l_0^{(2)}$ and the actual length l). In other words, given the same stress, BD can stretch more than AC. Plastic spring growth allows the spring to stretch further under the same stress (in practice, models usually impose constant turgor T and hence constant stress, rather than equal l; the element BD would consequently stretch further than depicted). Energy balance is also at play, as turgor T pushing the outside elements causes the springs to strain (the inside elements, e.g. D, are pushed from all sides equally). The stretch produces an elastic force which opposes the turgor. The position of the vertices are ones that balance the turgor forces and elastic forces.

written in eq. (4), where the pressure is balanced with the effect of stretching and plastic growth. The energy should then be minimised, as physical systems will always favour a state that requires the least energy (e.g. a ball in a bowl prefers to stay in the bottom where its gravitational potential energy is minimised). This model is able to replicate observed microtubule orientations in different experimental scenarios, for example primordium growth. These studies show that, firstly, microtubules are highly important in stress sensing, and secondly, they have regulatory effects on the geometry of a tissue.

The models we have described in this section suggest a fundamental role of mechanical cues in tissue mechanics, provide evidence that the interplay between mechanics and signalling is key to determining observed behaviour, and support the view that genetic regulation alone cannot account for observed phenotypes (Bassel et al. 2014). Questions remain, for example the effect of spatial variations of mechanical properties on cell behaviour, specifically in the SAM, is not yet understood (Truskina and Vernoux 2018). It has also been noted that the feedback loop between microtubules and stress cannot always completely explain how cells develop such a complex geometry in the first place (Kierzkowski and Routier-Kierzkowska 2019). Tissue stress origins are also still elusive (Baskin and Jensen 2013). Moreover, the complex relationship between anisotropies and microtubules has not been fully investigated; we will discuss further in section 4.

4 Outlook

Whilst there have been many success stories where mechanical models have had significant impact on our understanding of plant growth, there are still many exciting future directions for mathematicians and biologists alike to explore.

The roles and mechanisms of enzyme and protein action in wall loosening have not been fully understood (Cosgrove 2016). Expansins are an important group of non-enzymatic proteins that cause wall stress relaxation, enabling cell wall creep. How they interact with the linkages between microfibrils is unknown. For example, α-expansins have the apparently contradicting effects of inducing creep while maintaining wall strength (Yuan et al. 2001, Wang et al. 2008). Xyloglucan endotransglucosylase/hydrolase (XTH) also affects the cell wall but its action of cutting and rejoining xyloglucans does not necessarily cause an increase in extensibility of the wall (Cosgrove 2016). It also does not seem to affect growth in plants where XTH expression is suppressed (Cosgrove 2005). It could be interesting to explore how expansin/XTH function relates to cell wall structure, as there is the possibility that expansin can cut biomechanical hotspots, that XTH may be ineffective due to a possible inability to access the xyloglucan, or that XTH could control elongation or strengthening by affecting xyloglucan length (Cosgrove 2005, 2014). Endoglucanase expression is also said to have potential to cause wall loosening (Cosgrove 2014). The interactions between different pectin

methylesterases (PMEs) are still elusive and it has been proposed that unlocking PME action could help examine pectin's role in the cell wall (Levesque-Tremblay et al. 2015). Models similar to those in section 3.1 might be helpful, where one could examine not just which molecules hold the stress but test possible wall-loosening mechanisms (e.g. expansins targeting hotspots) against experiments.

Pectins in the cell wall have not been thoroughly investigated, despite their making up over 30% of the primary cell walls in most higher plants (Levesque-Tremblay et al. 2015). Although their role has been considered in pollen tubes (Rojas et al. 2011, Fayant et al. 2010), in other models their effect has been included as a generic isotropic term. This approach could give an accurate description of pectin's effect (Huang et al. 2015), however it neglects the potential influence of pectin-cellulose interactions, or that de-methylesterified pectin could affect the porosity of cellulose-xyloglucan networks, thereby influencing enzyme action (Cosgrove 2016). Inhibition of PME activity is known to prevent the formation of primordia at the meristem, showing that pectins influence wall extensibility, and that their spatial regulation of methylated and demethylated aids morphogenesis (Höfte et al. 2012, Braybrook et al. 2012, Braybrook and Peaucelle 2013) with pectin asymmetry in the hypocotyl epidermis shown to aid anisotropy (Bou Daher et al. 2018). Moreover, even though de-esterified pectin is found in vitro to be stiffer than methyl-esterified pectin, regions of de-esterified pectin can give rise to softer cell walls in the meristem, which is an as-yet unexplained phenomenon (Cosgrove 2016).

There is evidence that microtubules are highly influential in anisotropy/morphogenesis (see section 3.3), although this relationship is in no way straightforward. It has been found that cellulose fibres in the outer epidermal wall of most stems are orientated axially (Baskin and Jensen 2013), and that anisotropic tissues are not necessarily made up of anisotropic cells (Bou Daher et al. 2018). A related question is how cellulose orientation is decided, because the influence of microtubules on cellulose direction is questionable in some situations (Cosgrove 2014, Bou Daher et al. 2018). Cellulose can also passively re-orientate as the cell grows (Anderson et al. 2010), and although cells may have a net orientation of cellulose fibres, they are deposited in a variety of orientations between lamella layers in the cell walls (Zhang et al. 2016). These features are not often included in models. By incorporating the differences between cells into tissue expansion models, we might be able to identify the

possible origins of tissue stress. In addition, models could test different possible mechanisms for cellulose alignment and tissue anisotropy.

Models which exploit the geometry of growing plant tissues, for example using shell theory, lead to significant reductions in complexity, giving more tractable models and interpretable results. However, some features are often neglected. For example, shell models often assume a straight centreline and thus cannot be used to model steering, twisting or bending. Similarly, emergent anisotropy arising as a tissue-wide phenomenon is often neglected. Indeed, internal layers do contribute to morphogenesis such as the vasculature (Hamant and Haswell 2017). There is therefore a growing need to progress from 2D to 3D models (Kroeger et al. 2008, Fozard et al. 2013).

Finite-element models can become unstable and less accurate when simulated cell growth causes the elements to increase in length (Fozard et al. 2013). This requires the system to be re-meshed, which can be computationally expensive (Chickarmane et al. 2010). It has also been pointed out that the implementation of finite-element methods in iterations implies that growth occurs in small discrete steps (Fayant et al. 2010). Depending on the number of nodes, this could lead to subtle shape changes, even though a fine mesh density could possibly ensure the errors are small.

5 Summary

In this review we have demonstrated that mathematical models can help unravel mysteries of how the cell wall structure allows controlled growth, what causes intricate tissue morphologies and how stress feedback works across whole tissues. For mathematicians, plant biomechanics is a truly exciting field in which great opportunities beckon, with numerous fascinating questions and opportunities for impactful work.

Some of the best models have resulted from multidisciplinary collaborations between mathematicians and biologists, which allow modelling and experimentation to be adapted to each other in real time. It is therefore vital for the progress of the field that mathematicians and biologists work together to produce models which are experimentally verifiable, can

explain biological phenomena mechanistically, and can raise important new questions about the elusive nature of plant biology.

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