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Plant morphogenesis: What drives growth?

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Studies of growing stems and leaves often emphasize the epidermis as a major restraint for organ growth. A new study of anther lobe formation shifts the spotlight from epidermal wall extensibility to the elasticity of inner cells.

For more than 150 years, biomechanical studies have pointed to the epidermis as a major constraint on stem growth[1,2]. Likewise, mechanical models of the shoot apical meristem indicated mechanical restraint by the epidermis[3,4]. Conceptually, growth or osmo-elastic stretching of internal cells is physically constrained by the epidermis with its stiffer and/or less extensible walls[5–7]. Such conflicts lead to tissue stresses that arise when turgor-generated wall tensions of inner growing cells are displaced to the restraining epidermal cell walls. Tissue stresses can also arise for non-growing tissue through differential wall stiffnesses. For growing organs, they can be generated when inner cells undergo stress relaxation[5,8], leading to higher epidermal tensions[9,10]. Tissue stresses are manifested in the classical split pea stem bioassay for auxin by an outward curvature upon splitting a growing pea stem lengthwise[11]. Stem growth may be promoted by selective loosening of epidermal walls[12,13], although internal cells may also contribute[14]. In a recent report, the concept of morphogenetic tissue conflicts has been extended to the 3D outgrowth of anther lobes in the developing flowers of *Arabidopsis thaliana*, but with a twist: Silveira, Collet *et al.*[15] propose a primary causal role for elastic 'inflation' of internal cells, with the epidermis playing second fiddle. Going further, they propose that differential elasticity of surface and internal cells causes the differential growth underlying anther lobe formation. Let's take a closer look at this study and the concepts underlying the report's conclusions.

The anther is the pollen-bearing organ of flowers. Its development starts in the floral meristem, where a primordium elongates into a fingerlike projection that swells apically, eventually forming four pollen-filled locules that in cross-section resemble a butterfly in outline[16]. Two concentric layers, the epidermis and the endothecium, surround the internal cells which ultimately develop into the nutritive tapetum and the pollen.

By meticulous time-lapse confocal microscopy combined with 3D cell segmentation and tracking, faster increases in cell volume were measured for inner tissues of the growing locule compared with the epidermis or inner cells of connective tissue. From this observation, the authors infer that anther lobation is driven by volumetric growth of the inner cells. This concept was explored by finite element models simulating elastic volume changes and stress patterns in planar layers where epidermal cells and inner cells were represented as tightly packed cubes of varying size, turgor pressure and wall stiffness. From these results, the authors formulate a quantity they name 'inflation potential', a dimensionless metric of turgor-dependent elastic change in cell volume. It is a function of cell size, turgor and wall stiffness and is closely related to the concept of cell hydraulic capacitance used in plant water relations.

To parameterize this elastic model, turgor pressures were estimated experimentally by bringing the anther cells to incipient plasmolysis with sodium chloride solutions. Cell shrinkage was then used by a novel reverse-engineering method to estimate wall stiffness, reported as E, Young's modulus. Epidermal walls were estimated to be 3X stiffer than walls of internal cells. One should expect in this situation that the epidermis would be in tension, and indeed evidence for such tension was found in the flattened shape of the epidermal cells, and their tendency to develop cracks in a mutant defective in cell adhesion. The authors conclude that anther lobe formation is driven by the

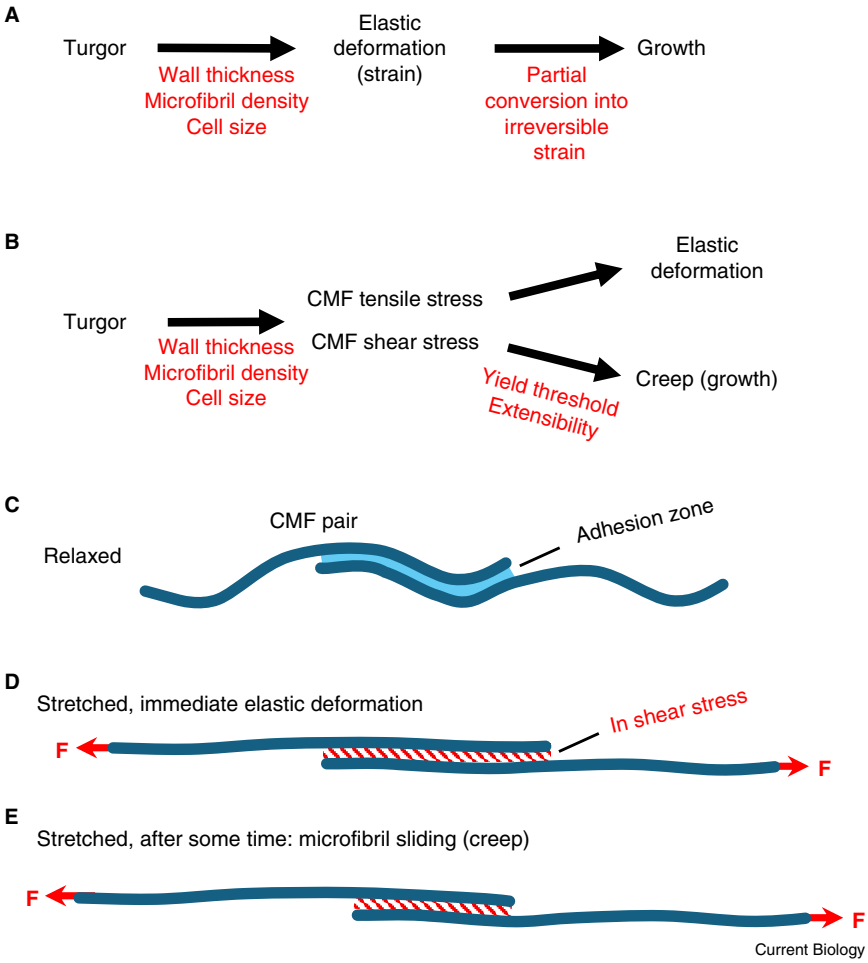

Figure 1. Alternative causal diagrams for modeling growth and underlying modes of deformation of cellulose microfibrils (CMF).
(A) Elasticity-based model of growth. Turgor pressure elastically stretches the wall, modulated by wall thickness, microfibril density and cell size. A portion of the elastic strain is then converted to an irreversible increase in wall size. (B) Creep-based model of growth. Turgor pressure elastically stretches the wall, generating two types of stresses within CMFs and shear stresses in adhesion zones between CMFs. Tensile stresses result in immediate elastic (reversible) deformations, whereas shear stresses drive irreversible creep of the wall over time, modulated by the creep yield threshold and extensibility. (C–E) Conceptual diagrams of CMF conformation and modes of deformation in a relaxed state (C), upon application of a tensile stretching force F which generates a tensile stress within CMFs and a shear stress between CMFs (D), and after some time to allow CMF sliding in the adhesion zone, leading to an irreversible increase in length of the CMF bundle (E). Elastic deformation (strain) at the CMF level entails reversible straightening, bending and stretching of CMFs[18].

greater elastic inflation potential of inner tissue, with inner cells having softer walls at early stages and becoming larger with thinner walls at later stages.

The reader comes away with the impression that anther morphogenesis is driven by elastic mechanics. However, this begs the question of how elastic deformation, which is relatively small and reversible, is transformed into growth (morphogenesis), which can span several orders of magnitude, and is irreversible. Although not answered in the main text, this question is addressed in a computational multicellular model for growth, described in the methods of the paper.

According to one framing of this model, represented in a causal diagram in Figure 1A, turgor causes elastic deformation of the wall (commonly represented as a spring), and a fraction of this elastic deformation is then converted into irreversible deformation by increasing the resting length of the spring, simulating growth. Thus, turgor causes elastic deformation which causes growth. This elastic-driven hypothesis for growth may correspond to a biophysical mechanism in which stretching of the wall is followed by crosslinking or insertion of material to increase wall length irreversibly[2]. Experimental evidence of this hypothesis is weak[2] — for instance, polymer addition to the growing wall is separable from its irreversible extension both *in vivo* and *in vitro*[17] (although the two processes may be roughly correlated in some developmental contexts).

The computational model presented in the paper is also consistent with a different interpretation (Figure 1B). The main load-bearing elements in plant cell walls are cellulose microfibrils[18]. Consider a scenario in which laterally bonded microfibrils are stretched endwise; elastic deformation occurs immediately through microfibril stretching and straightening (Figure 1C,D). This deformation is reversible upon removal of the stretching force. Irreversible deformation (growth) arises on a longer time scale through yielding to the shear stresses in microfibril adhesion zones, resulting in microfibril sliding (Figure 1E). For a given shear stress, the extent of sliding depends on the adhesive strength and length of the interface between the microfibrils. Such sliding corresponds to creep and is irreversible. The threshold tension at which sliding initiates and the rate of sliding underlie the concepts of yield threshold and extensibility, respectively. This creep-driven growth hypothesis is linked to wall loosening by expansins, and has received considerable experimental and theoretical support[17,19].

The creep-driven hypothesis can be represented with the causal diagram shown in Figure 1B. Rather than being a cause of growth, elastic deformation is a parallel outcome of microfibril stress. According to this view, elastic deformation can be used as a proxy for microfibril stress, which, together with extensibility, can allow growth to be computed using the same equations as those described in the methods section of the paper. The same equations can have different causal interpretations[20].

By portraying elastic deformation as causing growth, the paper adopts an elastic-driven hypothesis, for which there is currently little biophysical support. Moreover, by focusing on elastic deformation alone, the paper neglects the possible role of varying extensibility. For

example, the experimental data show only a 3-fold difference in stiffness between inner and outer walls, while their growth model invokes a 50-fold difference. This discrepancy is not discussed, but could be resolved by higher extensibility of inner walls. Introduction of yield thresholds would be another way to resolve the discrepancy.

Our comments are not meant to diminish the considerable achievements in imaging and quantification of anther morphogenesis presented in the paper. Moreover, this paper is not alone in favoring an elastic-driven hypothesis. Our comments are aimed at highlighting different biophysical hypotheses that may underlie growth models, and to encourage authors, particularly when making causal inferences, to make those hypotheses explicit and consider experimental evidence for or against them.

DECLARATION OF INTERESTS

The authors declare no competing interests.

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Ecology: Complexity and functionality in forests

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Forests are species-rich ecosystems and provide vital ecosystem services. A new study highlights how tree diversity, mycorrhizal fungi and soil food web structure govern forest functionality, and how tiny energy fluxes can be critical for community persistence. The findings provide new insights into how to sustainably manage forests.

Forests are among the most biologically rich ecosystems on earth. Most forests harbour a large diversity of tree and plant species. This diversity creates a large variety of habitats for organisms living above- and belowground. An important component of forest biodiversity is the soil food web, including bacteria, fungi, protists, nematodes, insects, mites, and worms. Forests provide ecosystem services that are important to human well-being. Forests produce wood and play a major role in the global cycling of matter, energy, carbon, and nutrients. Sustainable forest management is therefore recognized as 'key' in solving worldwide environmental issues such as the protection of biological diversity and the mitigation of global environmental change.

A new study from Yi *et al.*[1], published in this issue of *Current Biology*, explicitly links forest biodiversity with forest ecosystem functionality by quantifying energy fluxes. By taking a whole-ecosystem approach,