

How does a developing organism find its shape?

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A developing organism does not consult a miniature drawing of its adult outline. Its shape emerges as cells divide, change their geometry, move, adhere and exert forces in a structured environment. Genetic regulation helps organize those activities, but a list of expressed genes is not yet a three-dimensional tissue. The explanation must connect molecular differences to motion, force and the boundaries against which cells act.

The word “find” is therefore a convenient question, not a claim that an embryo sees a target and steers toward it. The scientific task is to explain how local processes generate reproducible forms, which disturbances those processes tolerate, and where the explanation stops being predictive.

1. Cell identity and tissue shape are different outcomes

Knowing that cells have adopted a particular identity does not specify how they are arranged. A sheet, a tube and a cluster can contain related cell types but differ in geometry and function. Conversely, a tissue can change shape before its cells acquire visibly different mature identities.

A complete account of a particular shape change must identify both the active processes and the physical constraints. Cells generate forces, but neighboring cells and surrounding structures transmit or resist them. The same internal contraction can produce different movements when the external attachments differ. That is a mechanical consequence, not evidence that the genes have become irrelevant.

This also changes what counts as a successful model. Reproducing one final outline is weaker than reproducing the sequence of deformations and its response to a controlled perturbation. Several mechanisms can converge on a superficially similar shape.

2. A concrete case: making an epithelial sheet fold

One experimentally studied process is apical constriction: cells narrow their exposed upper surface. Coordinated constriction can contribute to tissue folding, but whether it is sufficient depends on the tissue and geometry. In early *Drosophila* embryos, researchers used light-controlled activation of Rho signaling to impose localized contractile activity and examine the resulting shape changes. [1]

The intervention is informative because it changes where and when a candidate driver acts. The investigators did not merely observe that a signaling molecule and a fold occurred together. They activated the pathway in a selected region and followed the tissue response. The experiment supports a causal role in that setting; it does not imply that every embryonic fold is generated by the same arrangement.

A key distinction is between imposing the pattern of activation and explaining how an unperturbed embryo normally establishes that pattern. An artificial intervention can reveal what a tissue is capable of doing while leaving part of its natural control system to be explained.

3. Geometry constrains the response

Imagine a row of wedge-shaped cells as a simplified geometric model. If each cell's upper edge becomes shorter while neighboring cells remain connected, the row can bend. A ring of such cells, a flat sheet and a sheet attached to a rigid boundary will not necessarily deform in the same way. The model names a plausible relation between local shape and collective curvature without specifying a real force law.

The distinction between shape and force is crucial. A cell can become narrower because it actively contracts, because its neighbors pull it, or because it is compressed by external loading. A movie of the shape alone may not distinguish these possibilities. Intervention, force-sensitive measurements and comparisons with mechanical predictions are needed to identify the contribution of each process.

Nor does stronger activity always mean a proportionally larger movement. Resistance can change as the tissue deforms, cells can rearrange, and attachments can constrain displacement. The outcome depends on the evolving system, not only on the starting strength of one molecular signal.

4. Movement can modify the next signal

Developmental control need not run in one direction from gene expression to force. A study of *Drosophila* endoderm morphogenesis distinguished local transcriptional initiation from a propagating sequence involving cell deformation and mechanical feedback. It reported that the advancing wave of Rho1-myosin II activation did not require sustained transcription in the same way as its initiation. [2]

This offers a concrete alternative to imagining that every successive cell must receive an independently drawn positional instruction. Under the studied conditions, deformation and the resulting interactions helped connect one part of the process to the next. The finding does not remove genetic control: it changes which stage of the process that control directly explains.

A chain of coupled events can be robust in some respects and vulnerable in others. Breaking the relevant mechanical connection could interrupt propagation even if the initiating signal remained. Altering the initiation could stop the process before the feedback had anything to propagate. These are different experimental predictions.

5. What a model must earn

A developmental model should say which organism, stage, tissue and boundary conditions it describes. It should also identify which quantities were fitted and which were predicted. If a parameter is adjusted separately for every observed shape, agreement alone gives limited evidence that the model has captured a general mechanism.

A stronger test holds part of the model fixed and asks whether it predicts a new perturbation. Does moving an activation region move the fold? Does changing an attachment alter the direction of deformation? Does inhibiting force generation interrupt a wave at the predicted stage? These questions make alternative accounts diverge.

Such tests also expose what a simple model leaves out. Growth, division, material turnover and three-dimensional contacts can matter on different timescales. A useful reduced model need not include everything, but its omissions should match the question rather than disappear behind the phrase "self-organization."

6. Reproducibility without a complete drawing

An organism's form can be reproducible because inherited regulatory systems and physical interactions repeatedly generate similar developmental trajectories. Reproducibility does not require a nucleotide-by-nucleotide geometric drawing, and variation does not imply the absence of regulation. The two experimental cases show particular ways in which signaling and mechanics can be connected. [1,2]

They do not establish one universal mechanism for all organs or species. The open task is to determine which interactions control each process, how they combine across scales, and how their limits change under perturbation. The common mistake is to let one successful metaphor, whether blueprint or self-organization, stand in for that causal work.

Related reading

Article 030, “How can cells with the same DNA do different jobs?”, addresses the regulation of cellular state. This article follows a different outcome: the arrangement and deformation of cells in a tissue. The link is optional and reciprocal.

References

- [1] E. Izquierdo, T. Quinkler and S. De Renzis. Guided morphogenesis through optogenetic activation of Rho signalling during early *Drosophila* embryogenesis. *Nature Communications* 9, 2366 (2018). <https://doi.org/10.1038/s41467-018-04754-z>
- [2] A. Bailles et al. Genetic induction and mechanochemical propagation of a morphogenetic wave. *Nature* 572, 467–473 (2019). <https://doi.org/10.1038/s41586-019-1492-9>