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An epidermis-driven mechanism positions and scales stem cell niches in plants

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How molecular patterning scales to organ size is highly debated in developmental biology. We explore this question for the characteristic gene expression domains of the plant stem cell niche residing in the shoot apical meristem. We show that a combination of signals originating from the epidermal cell layer can correctly pattern the key gene expression domains and notably leads to adaptive scaling of these domains to the size of the tissue. Using live imaging, we experimentally confirm this prediction. The identified mechanism is also sufficient to explain de novo stem cell niches in emerging flowers. Our findings suggest that the deformation of the tissue transposes meristem geometry into an inductive scaling and positional input for the apical plant stem cell niche.

INTRODUCTION

Development requires coordination between molecular patterning and morphogenesis (

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). Several studies suggest how morphogens can

drive gene patterning, growth, and sh

ape generation in different systems

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) and how mechanical constraints might feedback on growth (

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Although adaptation to size is ofte

nseen in developmental tissues (

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how tissue geometry and size feedback on gene expression domains is

less understood. Using a combination of computational and experimental approaches, we address this question in the shoot apical meristem (SAM), the stem cell niche source of all aboveground plant organs (8).

Considered as a main orchestrator of the SAM activity (9), WUSCHEL (WUS) is expressed in a central domain, a few cell layers below the apex of the SAM. It encodes a transcription factor, moving between cells, and is involved in perpetuating stem cell activity and keeping differentiating cells at bay (11).

At the very tip of the SAM, the stem cells express the CLAVATA3 peptide (CLV3) that diffuses in the tissue and down-regulates WUS, closing the canonical CLV3/WUS feedback loop (13).

thought to be the main factor maintaining SAM homeostasis. On the flanks of the meristem, WUS also represses the differentiation program including the KANADII (KAN1) marker (12).

Spatial aspects of WUS maintenance via cytokinin signaling have recently been elucidated: LONELY GUY (LOG) proteins, which catalyze cytokinin biosynthesis, are expressed in the epidermis (L1), and ARABIDOPSIS HISTIDINE KINASE (AHK) cytokinin receptors are expressed inside the tissue, a few cell layers away from the L1 (15).

(Fig. 1A). Along with previously published data (17), this implies that the epidermis may provide a major positional cue for SAM maintenance.

RESULTS AND DISCUSSION

To investigate this hypothesis, we propose an integrated quantitative description of the processes regulating the SAM homeostasis where the L1 is central to the patterning of the stem cell regulatory genes (Fig. 1B). The spatial model integrates the SAM regulatory interactions described in a system of differential equations, using Hill formalism for transcription together with mass action and passive diffusion-like transport (Supplementary Materials) (11).

It includes the CLV3/WUS feedback loop and the repression of KAN1 by WUS. In particular, the L1 produces four signals modeled as diffusive molecules. Of those, cytokinin acts as an activator of WUS by binding the AHK receptors. Given the expression pattern of the AHKs (Fig. 1A), we hypothesized a repressive L1 signal, keeping the AHKs away from the epidermis (termed AHK-). Cytokinin and AHK- form an incoherent feed-forward motif regulating WUS

(
20
) (Fig. 1, A and B). Two additional signals produced by
the epidermis also activate
CLV3
(
11
,
12
,
19
)and
KAN1
(
12
).

Different representations of the meristematic tissue are used
throughout this study. Two abstract tissue templates, one 2D and
one 3D, are composed of overlapping spherical cells constrained in
a parabolic shape (Fig. 1C and fig. S1). A realistic tissue template
obtained from segmented confocal data (
20
) is also used and includes
cell volumes and cell contact surfaces (Fig. 1D). For all these tem-
plates, the bottom layer of cells implements a specific boundary
condition (referred to as sink), abstr
acting the diffusion of molecules
outside of the meristem and into the
tissues below (stem and vascula-
ture), where molecules are removed
from simulations based on their
diffusion rate. As such, the faster a molecule diffuses in the meristematic
tissue, the faster it is carried out of the system at the bottom boundary.
In contrast, epidermal
cells have a nonflux boundary to the outside of
the tissue.
Model parameters are optimized to obtain the wild-type expression
domains of
WUS
,
CLV3
, and
KAN1
using a dedicated strategy based
on the Covariance Matrix Adaptat
ion Evolution Strategy (CMA-ES)
algorithm (
21
) (Supplementary Materials and fig. S2). At equilibrium,
the model achieves a correct representation of the expression domains
showing that the epidermis-driv
en model is sufficient for SAM
patterning. This result is robust in the different representations of the
meristematic tissue (Fig. 1, C and D). The model is also able to qual-
itatively reproduce a large set of experimentally observed perturba-
tions including perturbations of the
CLV3/WUS
feedback loop and
perturbations of cytokinin signaling (Supplementary Materials and
fig. S3).
Several mutants of
Arabidopsis thaliana
result in meristematic
tissue defects (
12
,
22
–
24
); an example is the
clavata
phenotype,
causing plants with markedly enlarged and flattened meristems.

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m

m-thick transversal sections cutting through the center of the meristem were extracted and further projected following maximum intensity.

When necessary, images were rotated so that the meristem would be displayed vertically. The images were then cropped to contain only the SAMs. Finally, each image was converted to png format and analyzed using the python PIL library (www.pythonware.com/products/pil/).

In the following, we consider a reference frame with an origin at the bottom left corner of each image where discrete

x

and

y

values

correspond to pixel positions in the image. To measure the shape of a meristem, for each row of pixels, we extracted the topmost pixel registering FM4-64 fluorescence (for each

x

: maximum

y

with red

channel value >50). This results in a collection of

x

,

y

coordinates

outlying the shape of the meristem. The parameters of a second-degree polynomial (describing a parabola) were then fit to the extracted coordinates using the numpy.polyfit function. The parameter

a

of a

parabola described by

ax^2

+

bx

+

c

= 0 controls how narrow the domain contained within the parabola is (where

ax^2

+

bx

+

c

<0).

In the case of the parabolas outlying the meristem shape, the higher the value of the

a

parameter, the flatter the meristem. This value was

computed for each meristem. The parabolas and

a

parameters are

displayed on the meristem images in figs. S8 to S19.

These same images were used to quantify the shape of the expression domains of the

pWUS

>>

GFP

and

pCLV3

>>

GFP

constructs. We

chose to measure the elongation of the domains along the axis of the reference frame. To do so, we find the maximal distance between two pixels registering GFP fluorescence (green channel value >50) and having the same

y

coordinate, giving us the longest horizontal axis of

the GFP expression domain. We repeat the procedure using the same

x

coordinate and find the longest vertical axis. As a measure of the

GFP domain elongation, we use the ratio of the longest vertical axis over the longest horizontal axis. Thus, the higher the value, the more the domain is horizontally elongated. Axis and elongation values are displayed on their corresponding meristem images in figs. S8 to S19. Figure 3A and fig. S7A show a correlation between the shape of the meristematic tissue and the shape of the expression domains of both WUS and CLV3.

The flatter the SAM, the more horizontally elongated the domains of expression are. Figure S21 shows the correlation between the size of the meristems from Fig. 2 and the shape of the meristems. We observe a strong correlation between the shape of the tissue and its size, with the larger meristems also being the flatter ones. Note that the number of meristems plotted in fig. S21 may differ from the number of meristems displayed in figs. S8 to S19, because only the meristems that could also be processed for Fig. 2 are displayed. Regression analysis is performed as described in the previous section. To assess if the L1 model can capture this effect, we generated a set of new 2D templates. The optimization template was first scaled by a factor and then scaled horizontally again by the same factor. This generated a set of smaller and narrower tissue templates and a set of larger and flatter ones. The factors displayed in fig. S20 are 0.9, 0.95, 1, 1.05, 1.1, 1.15, and 1.2. As exemplified, the larger and flatter meristems show elongated domains of expression for both WUS and CLV3.

whereas the domain tends to narrow down for the smaller and narrower meristems. The data obtained for all parameter sets optimized for the 2D template are shown in Fig. 3C and fig. S7C.

Primordium growth (Fig. 4 and movie S1)

Cells are allowed to grow by increasing the cell radii and are allowed to divide into two daughter cells when a threshold size is attained. A set of spring forces between cell centers moves cells apart when overlapping and back inside the lid when pushed out of it. Cells falling below the lower boundary of the lid are removed from the simulation (12).

As the simulation runs, a sphere moves from the inside of the parabola, exiting laterally, and stopping when its center reaches the parabola. As the sphere moves, the lid is updated to comprise the parabola and the part of the sphere outside of the parabola. This results in an increase of the space allocated to cells. The growth simulation is performed using the organism software (<http://dev.thep.lu.se/organism>).

For 200 time points evenly spaced over the length of the simulation, the cell positions and radii are recorded and the cells belonging to the boundaries (L1 and Sink) are found, allowing us to find the equilibrium of the L1 model as previously described. The final movie is obtained by assembling the equilibrium variables of the model for each time point.

It is worth noting that the growth and cell division algorithm used to generate the movie is noisy, likely more than what could be expected from the actual meristematic tissue. This generates large unrealistic fluctuations of the expression domains of WUS and CLV3 as the simulation runs. Keeping such an algorithm, the fluctuations could however be reduced by extending the model to add additional feedback loops known to be present in the system, such as the negative feedback of cytokinin of its signaling mediated by type B Arabidopsis response regulators. Another plausible approach is to trade some of the expression domain pattern quality for more robustness of the patterns by adding additional components to the cost functions used in the optimization of the parameters.

WUS and CLV3 live imaging (Fig. 4)

pWUS::GFP-ER and pCLV3::dsRED-N7 plants were grown under

conditions described in the Realistic template section. For the live imaging, plants were transferred immediately after bolting to square boxes containing lukewarm MS media with 1% agar and allowed to solidify. The plants were then stained with FM4-64 and imaged with a Zeiss LSM 780 confocal microscope.

1D simulations (Fig. 5)

To illustrate the scaling effect driven by the incoherent feed-forward loop controlling

WUS

expression, simulations were performed on a set of 1D tissue templates (ranging from 5 to 14 cells), each having an L1 cell at one extremity and a Sink cell at the other. The simplified model comprises the activation of

WUS

mediated by cytokinin and its repression mediated by AHK4.

The 10-cell template was chosen to manually fit the parameters of the system to obtain a central

WUS

domain. The chosen parameters are

ν

w

$\frac{1}{4}$

1

:

7

k

L_c

w

$\frac{1}{4}$

0

:

75

n

L_c

w

$\frac{1}{4}$

10

k

L_a

w

$\frac{1}{4}$

0

:

2

n

L_a

w

$\frac{1}{4}$

10

g

w

$\frac{1}{4}$

1

:

7

p

L_c

$\frac{1}{4}$

15

g

L_c

$\frac{1}{4}$

1

D

L_c

$\frac{1}{4}$

100

p

L_a

$\frac{1}{4}$

2

g

L_a

$\frac{1}{4}$

1

D

L_a

$\frac{1}{4}$

1

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Supplementary material for this article is available at <http://advances.sciencemag.org/cgi/content/full/2/1/e1500989/DC1>

Fig. S1. Templates.

Fig. S2. Optimization strategy.

Fig. S3. The model is able to represent a large collection of perturbations.

Fig. S4.

pCLV3::WUS

.

Fig. S5.

WUS

domain size variation.

Fig. S6.

CLV3

domain size variation.

Fig. S7.

pCLV3

>>

GFP

meristem shapes and expression domains.

Fig. S8. clasp1

pWUS

>>

GFP

meristems, grown in long days.

Fig. S9. clasp1

pWUS

>>

GFP

meristems, grown in long days followed by short days.

Fig. S10. Col.0

pWUS

>>

GFP

meristems, grown in long days.

Fig. S11. Col.0

pWUS

>>

GFP

meristems, grown in long days followed by short days.

Fig. S12. WS-4

pWUS

>>

GFP

meristems, grown in long days.

Fig. S13. WS-4

pWUS

>>

GFP

meristems, grown in long days followed by short days.

Fig. S14. clasp1

pCLV3

>>

GFP

meristems, grown in long days.

Fig. S15. clasp1

pCLV3

>>

GFP

meristems, grown in long days followed by short days.

Fig. S16. Col.0

pCLV3

>>

GFP

meristems, grown in long days.

Fig. S17. Col.0

pCLV3

>>

GFP

meristems, grown in long days followed by short days.

Fig. S18. WS-4

pCLV3

>>

GFP

meristems, grown in long days.

Fig. S19. WS-4

pCLV3

>>

GFP

meristems, grown in long days followed by short days.

Fig. S20. Example of expression domain variations upon tissue shape changes.

Fig. S21. Meristem size measure versus shape measure.

Fig. S22. Long-range and short-range signals.

Fig. S23.

WUS

sensitivity analysis.

Fig. S24.

CLV3

sensitivity analysis.

Table S1. Example parameter sets.

Movie S1. Primordium growth.

REFERENCES AND NOTES

1. M. Nahmad, A. D. Lander, Spatiotemporal mechanisms of morphogen gradient interpretation.

Curr. Opin. Genet. Dev.

21

, 726

–

731 (2011).

2. J. Jaeger, D. Irons, N. Monk, Regulative feedback in pattern formation: Towards a general

relativistic theory of positional information.

Development

135

, 3175

–

3183 (2008).

3. E. Coen, A.-G. Rolland-Lagan, M. Matthews, J. A. Bangham, P. Prusinkiewicz, The genetics of

geometry.

Proc. Natl. Acad. Sci. U.S.A.

101

, 4728

–

4735 (2004).

4. S. Sauret-Güeto, K. Schiessl, A. Bangham, R. Sablowski, E. Coen,

JAGGED

controls

Arabidopsis

petal growth and shape by interacting with a divergent polarity field.

PLoS Biol.

11

, e1001550

(2013).
5. B. I. Shraiman, Mechanical feedback as a possible regulator of tissue growth.
Proc. Natl. Acad. Sci. U.S.A.
102
, 3318
—
3323 (2005).
6. O. Hamant, M. G. Heisler, H. Jönsson, P. Krupinski, M. Uyttewaal, P. Bokov, F. Corson, P. Sahlín, A. Boudaoud, E. M. Meyerowitz, Y. Couder, J. Traas, Developmental patterning by mechanical signals in Arabidopsis
Science
322
, 1650
—
1655 (2008).
7. D. Ben-Zvi, N. Barkai, Scaling of morphogen gradients by an expansion-repression integral feedback control.
Proc. Natl. Acad. Sci. U.S.A.
107
, 6924
—
6929 (2010).
8. T. A. Steeves, I. M. Sussex, Patterns in Plant Development (Cambridge Univ. Press, New York, 1989).
9. K. F. X. Mayer, H. Schoof, A. Haecker, M. Lenhard, G. Jürgens, T. Laux, Role of WUSCHEL in regulating stem cell fate in the Arabidopsis shoot meristem.
Cell
95
, 805
—
815 (1998).
10. H. Schoof, M. Lenhard, A. Haecker, K. F. X. Mayer, G. Jürgens, T. Laux, The stem cell population of Arabidopsis shoot meristems is maintained by a regulatory loop between the CLAVATA and WUSCHEL genes.
Cell
100
, 635
—
644 (2000).
11. R. K. Yadav, M. Perales, J. Gruel, T. Girke, H. Jönsson, G. V. Reddy, WUSCHEL protein movement mediates stem cell homeostasis in the Arabidopsis shoot apex.
Genes Dev.
25
, 2025
—
2030 (2011).
12. R. K. Yadav, M. Perales, J. Gruel, C. Ohno, M. Heisler, T. Girke, H. Jönsson, G. V. Reddy, Plant stem cell maintenance involves direct transcriptional repression of differentiation program.
Mol. Syst. Biol.
9
, 654 (2013).
13. S. E. Clark, R. W. Williams, E. M. Meyerowitz, The CLAVATA1 gene encodes a putative receptor kinase that controls shoot and floral meristem size in Arabidopsis.
Cell
89
, 575
—
585 (1997).
14. M. Ogawa, H. Shinohara, Y. Sakagami, Y. Matsubayashi, Arabidopsis CLV3 peptide directly binds CLV1 ectodomain.
Science
319
, 294 (2008).
15. S. P. Gordon, V. S. Chickarmane, C. Ohno, E. M. Meyerowitz, Multiple feedback loops through cytokinin signaling control stem cell number within the Arabidopsis shoot meristem.
Proc. Natl. Acad. Sci. U.S.A.
106
, 16529
—
16534 (2009).
16. V. S. Chickarmane, S. P. Gordon, P. T. Tarr, M. G. Heisler, E. M. Meyerowitz, Cytokinin signaling as a positional cue for patterning the apical basal axis of the growing Arabidopsis shoot meristem.
Proc. Natl. Acad. Sci. U.S.A.
109
, 4002
—
4007 (2012).
17. D. Reinhardt, M. Frenz, T. Mandel, C. Kuhlemeier, Microsurgical and laser ablation analysis of interactions between the zones and layers of the tomato shoot apical meristem.
Development
130
, 4073
—
4083 (2003).
18. S. Knauer, A. L. Holt, I. Rubio-Somoza, E. J. Tucker, A. Hinze, M. Pisch, M. Javelle, M. C. Timmermans, M. R. Tucker, T. Laux, A protodermal miR394 signal defines a region of stem cell competence in the Arabidopsis shoot meristem.
Dev. Cell
24
, 125
—

132 (2013).
19. H. Jönsson, B. E. Shapiro, E. Meyerowitz, E. Mjolsness, in
On Growth, Form, and Computers
. S. Kumar, P. Bentley, Eds. (Academic Press, London, 2003), pp. 156
-
161.
20. R. Fernandez, P. Das, V. Mirabet, E. Moscardi, J. Traas, J.-L. Verdeil, G. Malandain, C. Godin,
Imaging plant growth in 4D: Robust tissue reconstruction and lineaging at cell resolution.
Nat. Methods
7
. 547
-
553 (2010).
21. N. Hansen. The CMA evolution strategy: A comparing review, in
Towards a New Evolutionary
Computation. Advances on Estimation of Distribution Algorithms
. J. A. Lozano, P. Larranaga,
I. Inza, E. Bengoetxea, Eds. (Springer-Verlag, Heidelberg, Germany, 2006), pp. 75
-
102.
22. U. Brand, M. Grünewald, M. Hobe, R. Simon, Regulation of
CLV3
expression by two homeobox
genes in Arabidopsis.
Plant Physiol.
129
. 565
-
575 (2002).
23. U. Brand, J. C. Fletcher, M. Hobe, E. M. Meyerowitz, R. Simon, Dependence of stem cell
fate in
Arabidopsis
on a feedback loop regulated by
CLV3
activity.
Science
289
. 617
-
619
(2000).
24. V. Wahl, L. H. Brand, Y.-L. Guo, M. Schmid, The FANTASTIC FOUR proteins influence shoot
meristem size in
Arabidopsis thaliana
. BMC Plant Biol.
10
. 285 (2010).
25. G. V. Reddy, E. M. Meyerowitz, Stem-cell homeostasis and growth dynamics can be uncoupled
in the
Arabidopsis
shoot apex.
Science
310
. 663
-
667 (2005).
26. J. J. Kieber, G. E. Schaller, Cytokinins.
Arabidopsis Book
12
. e0168 (2014).
27. I. Antoniadi, L. Pla
č
ková, B. Simonovik, K. Dole
ž
al, C. Turnbull, K. Ljunga, O. Novák, Cell-
type-specific cytokinin distribution within the Arabidopsis primary root apex.
Plant Cell
27
. 1955
-
1967 (2015).
28. T. Kuroha, H. Tokunaga, M. Kojima, N. Ueda, T. Ishida, S. Nagawa, H. Fukuda, K. Sugimoto,
H. Sakakibara, Functional analyses of
LONELY GUY
cytokinin-activating enzymes reveal
the importance of the direct activation pathway in
Arabidopsis
. Plant Cell
21
. 3152
-
3169
(2009).
29. T. Kurakawa, N. Ueda, M. Maekawa, K. Kobayashi, M. Kojima, Y. Nagato, H. Sakakibara,
J. Kyoizuka, Direct control of shoot meristem activity by a cytokinin-activating enzyme.
Nature
445
. 652
-
655 (2007).
30. D. L. Lindsay, V. K. Sawhney, P. C. Bonham-Smith, Cytokinin-induced changes in
CLAVATA1
and
WUSCHEL
expression temporally coincide with altered floral development in
Arabidopsis
. Plant Sci.
170
. 1111
-
1117 (2006).
31. I. Bartrina, E. Otto, M. Strnad, T. Werner, T. Schmülling, Cytokinin regulates the activity of
reproductive meristems, flower organ size, ovule formation, and thus seed yield in
Arabidopsis
thaliana
. Plant Cell
23
. 69
-
80 (2011).
32. A. Bhargava, I. Clabaugh, J. P. To, B. B. Maxwell, Y.-H. Chiang, G. E. Schaller, A. Loraine,
J. J. Kieber, Identification of cytokinin-responsive genes using microarray meta-analysis and
RNA-Seq in Arabidopsis.
Plant Physiol.
162

, 272

–

294 (2013).

33. A. Schmidt, Histologische Studien an phanerogamen Vegetationspunkten.

Bot. Arch.

9

,

345

,

404 (1924).

34. B. Bozorg, P. Krupinski, H. Jönsson, Stress and strain provide positional and directional

cues in development.

PLOS Comput. Biol.

10

, e1003410 (2014).

35. A. Sampathkumar, P. Krupinski, R. Wightman, P. Milani, A. Berquand, A. Boudaoud,

O. Hamant, H. Jönsson, E. M. Meyerowitz, Subcellular and supracellular mechanical stress

prescribes cytoskeleton behavior in

Arabidopsis

cotyledon pavement cells.

eLife

3

,

e01967 (2014).

36. S. Savaldi-Goldstein, C. Peto, J. Chory, The epidermis both drives and restricts plant shoot

growth.

Nature

446

, 199

–

202 (2007).

37. M. A. Savageau, Parameter sensitivity as a criterion for evaluating and comparing the

performance of biochemical systems.

Nature

229

, 542

–

544 (1971).

38. J. I. Medford, F. J. Behringer, J. D. Callos, K. A. Feldmann, Normal and abnormal development in

the *Arabidopsis* vegetative shoot apex.

Plant Cell

4

, 631

–

643 (1992).

39. D. Weigel, J. Glazebrook,

Arabidopsis: A Laboratory Manual

(Cold Spring Harbor Laboratory

Press, New York, 2002).

40. J. C. Ambrose, T. Shoji, A. M. Kotzer, J. A. Pighin, G. O. Wasteneys, The

Arabidopsis CLASP

gene encodes a microtubule-associated protein involved in cell expansion and division.

Plant Cell

19

, 2763

–

2775 (2007).

41. O. Grandjean, T. Vernoux, P. Laufs, K. Belcram, Y. Mizukami, J. Traas, In vivo analysis of cell

division, cell growth, and differentiation at the shoot apical meristem in *Arabidopsis*.

Plant

Cell

16

, 74

–

87 (2004).

42. Y. Deveaux, A. Peaucelle, G. R. Roberts, E. Coen, R. Simon, Y. Mizukami, J. Traas, J. A. H. Murray,

J. H. Doonan, P. Laufs, The ethanol switch: A tool for tissue-specific gene induction during

plant development.

Plant J.

36

, 918

–

930 (2003).

RESEARCH ARTICLE

Gruel

et al

, Sci. Adv. 2016;2:e1500989 29 January 2016

12 of 13

on September 22, 2016

<http://advances.sciencemag.org/>

Downloaded from

43. D. R. Smyth, J. L. Bowman, E. M. Meyerowitz, Early flower development in

Arabidopsis

,

Plant

Cell

2

, 755

–

767 (1990).

44. B. Landrein, Y. Refahi, F. Besnard, N. Hervieux, V. Mirabet, A. Boudaoud, T. Vernoux, O. Hamant,

Meristem size contributes to the robustness of phyllotaxis in

Arabidopsis

,

J

,

Exp

,

Bot

,

66

,

1317

–

1324 (2014).

45. J. Schindelin, I. Arganda-Carreras, E. Frise, V. Kaynig, M. Longair, T. Pietzsch, S. Preibisch, C. Rueden,

S. Saalfeld, B. Schmid, J.-Y. Tinevez, D. J. White, V. Hartenstein, K. Eliceiri, P. Tomancak, A. Cardona,

Fiji: An open-source platform for biological-image analysis.

Nat. Methods

9

, 676

–

682 (2012).

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J.G. designed the study, performed modeling, collected and analyzed data, and prepared the manuscript. B.L. and P.T. equally contributed in collecting and analyzing data. C.S., Y.R., and A.S. collected and analyzed data. C.S. commented on the manuscript. O.H. and E.M.M. contributed in designing the study and commented on the manuscript. H.J. designed the study and prepared the manuscript.

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The authors declare that they have no competing interests.

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All data needed to evaluate the conclusions in the paper are present in the paper and/or the Supplementary Materials. Additional data related to this paper may be requested from the corresponding author.

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Sci. Adv.

2,

e1500989 (2016).

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