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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 instructive scaling and positional input for the apical plant stem cell niche.

INTRODUCTION

Development requires coordination between molecular patterning and morphogenesis ([1], [2]). Several studies suggest how morphogens can drive genepatterning, growth, and shape generation in different systems ([3], [4]) and how mechanical constraints might feedback on growth ([5], [6]). Although adaptation to size is often seen in developmental tissues ([1], [7]), how tissue geometry and size feedback on gene expression domains is

less understood. Using a combinati
on of computational and experimen-
tal 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
,
10
),
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
,
12
).AttheverytipoftheSAM,
the stem cells express the CLAVATA3 peptide (CLV3) that diffuses in
thetissueanddown-regulates
WUS
, closing the canonical CLV3/WUS
feedback loop (
13
,
14
), thought to be the main factor maintaining SAM
homeostasis. On the flanks of the meristem, WUS also represses the dif-
ferentiation program including the
KANADI1
(
KAN1
)marker(
12
). Spa-
tial 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
,
16
) (Fig. 1A). Along
with previously published data (
17
,
18
), this implies that the epidermis
may provide a major position
al 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 differenti
al equations, using Hill formalism
for transcription together with mass action and passive diffusion-like trans-
port (Supplementary Materials) (
11
,
12
,
19
). 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
AHK
s (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 regulat-
ing
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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5-
m
m-thick transversal sections cutting
through the center of the meristem
were extracted and further project
ed 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,eachimagewascon
verted to png format and analyzed
using the python PIL library (www.py
thonware.com/p
roducts/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.
Thesameimageswereusedtoquantifytheshapeoftheexpression
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 ra
tio of the longest vertical axis
overthelongesthorizontalaxis.Thus,thehigherthevalue,themore
the domain is horizontally elongat
ed. 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 gen-
erated 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
,
whereasthedomainstendtonarrowd
own for the smaller and narrower
meristems. The data obtained for al
l 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 pa-
rabola 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 per-
formed using the organism software (h
ttp://dev.thep.lu.se/organism).
For 200 time points evenly spaced over the length of the simu-
lation, 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 ex-
pected from the actual meristematic tissue. This generates large un-
realistic 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 t
he model to add additional feed-
back 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 comp
onents 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 tr
ansferred immediately after bolting to square
boxes containing lukewarm MS media with 1% agar and allowed to solidify. The plants were then stai
ned 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
$V_W = 1$
$k_{L_c W} = 0.75$
$n_{L_c W} = 10$
$k_{L_a W} = 0.2$
$n_{L_a W} = 10$
$g_W = 1$
$p_{Lc} = 15$
$g_{Lc} = 1$
$D_{Lc} = 100$
$p_{La} = 2$
$g_{La} = 1$
$D_{La} = 1$

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SUPPLEMENTARY MATERIALS

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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.

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