Comparison of Domain Nucleation Mechanisms in a Minimal Model of Shoot Apical Meristem

Dorjsuren Battogtokh[1,2] and John J. Tyson[2]

Short Abstract — Existing mathematical models of the shoot apical meristem (SAM) explain nucleation and confinement of a stem cell domain by a Turing mechanism, assuming that the diffusion coefficients of the activator (WUSCHEL) and inhibitor (CLAVATA) are significantly different. As there is no evidence for this assumption of differential diffusivity, we recently proposed a new mechanism based on a "bistable switch" model of the SAM. Here we study the bistable-switch model in detail, demonstrating that it can be understood as localized switches of WUSHEL activity in individual cells driven by a non-uniform field of a hypothetical hormone. By comparing domain formation on a cell-network driven by Turing and bistable-switch models, we show that better domain control is possible with the new mechanism.

***Keywords* —minimal model of SAM, reaction-diffusion systems, Turing instability, bistability, fast diffusive field.**

I. Purpose

The stem cells residing in the shoot apical meristem (SAM) give rise to above ground tissues [1]. Hence, maintenance of stem cell niches is of central importance to plant growth [2,3]. Negative feedback between the proteins WUSCHEL (WUS - a homeodomain transcription factor) and CLAVATA (CLV - a receptor kinase) is at the core of the signaling pathway controlling the central domain – the reservoir of stem cells [1]. Recently, theorists have proposed reaction-diffusion models of the SAM [4-7] that explain nucleation and confinement of the central domain as a Turing instability.

The most well-known mechanism of pattern formation in dissipative systems is associated with a Turing instability [8]. In this case, a spatially uniform steady state, which is globally stable with respect to uniform perturbations, becomes unstable with respect to non-uniform perturbations, provided that the diffusion range of an inhibitor significantly exceeds the diffusion range of an activator [9]. Under these conditions, a periodic pattern emerges in a monostable system at a certain critical wavenumber [10]. For a mathematical model of SAM, a Turing mechanism requires that the diffusion coefficient of CLV (inhibitor) significantly exceeds that of WUS (activator). At present, the diffusive properties of CLV and WUS are not well established; therefore, there is no clear experimental evidence on whether the Turing condition of differential diffusivity is applicable within the WUS and CLV expression zones of the SAM.

Existing models of the molecular biology of SAM regulation have positive and negative feedback loops that can generate not only Turing patterns but also alternative stable steady states (bistability) in a certain range of parameter values [2,6,7]. Recently bistable reaction-diffusion models have been studied to simulate experimental data on cytokinin controlled domain confinement in SAM [3]. In our previous work [11], a mechanism different from Turing instability was proposed for pattern formation in a minimal, bistable model of SAM. In the present work, we study in detail the mechanism of domain nucleation reported in Ref. [11].

II. Conclusion

Here we illustrate how a spatially non-uniform field of a peptide hormone synthesized by WUS can drive domain nucleation in a SAM model exhibiting bistability. We compare central-domain formation by Turing and bistable-switch mechanisms on a polygonal cell-network and show that, in the latter case, domain nucleation at a target location is possible without the additional assumptions required by the former.

References

[1] E. Aichinger, N. Korner, T. Freidrich, T. Laux, Ann. Rev. Plant Biol. **63**, 615(2012).
[2] J. C. Fletcher, E. M. Meyerowitz Curr. Opin. Plant Biol. **3,** 23(2000).
[3] S. P. Gordon, V. S. Chickarmane, C. Ohno, E. M. Meyerowitz, PNAS USA, **106**, 16529(2009).
[4] Jonsson et. al., Bioinformatics **21**, Suppl. i232(2005).
[5] T. Hohm,E. Zitzler, and R. Simon, PLoS One **12**, e9189(2010).
[6] H. Fujita, K. Toyokura, K. Okada, M. Kawaguchi, PloS One, **6** e18243(2011).
[7] S. V. Nikolaev et. al., Russ Journ. Dev. Biol. **38**, 383(2007). S. V. Nikolaev et. al., Biofizika, **51**, 583 (2006).
[8] A. M. Turing, Philos. Trans. Royal Soc. London, Ser B, **237**, 37(1952).
[9] A. Gierer and H. Meinhardt, Kybernetik, **12,** 30(1972); H. Meinhardt, *Models of Biological Pattern Formation,* Academic Press, London 1982; A. J. Koch and H. Meinhardt, Rev. Mod. Phys. **66,** 1481(1994).
[10] J. D. Murray, *Mathematical Biology II*, Springer-Verlag, Berlin, 2003.
[11] D. Battogtokh, Phys. Rev. E. **91**, 032713(2015).

[1]The Institute of Physics and Technology, Mongolian Academy of Sciences, Ulaanbaatar 51, Mongolia. E-mail: dbattogt@vt.edu
[2]Department of Biological Sciences, Virginia Polytechnic and State University, Blacksburg, Virginia 24061, USA. E-mail: tyson@vt.edu

Nothing should be here on page 2! Please limit your abstract to a single page, and create a one-page .pdf file for submission.