

An ODE Model of Root Zonation in *A. Thaliana* Mutants

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Acknowledgements

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- Dr. Geoffrey Wasteneys (Experimental Collaborator)
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Root Zonation

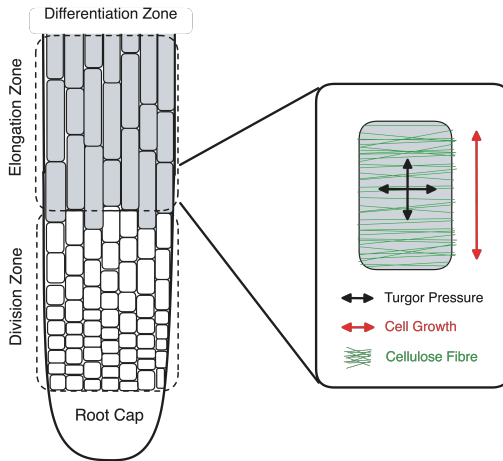


Figure: Zonation of the root apical meristem in *A. thaliana*.

Signalling Network

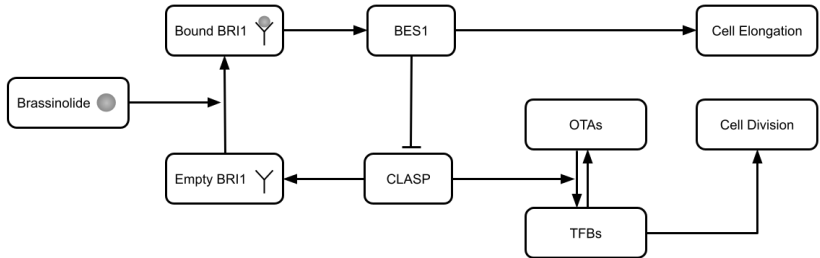
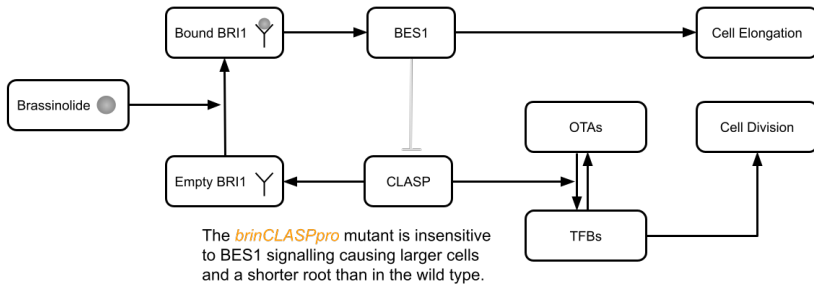
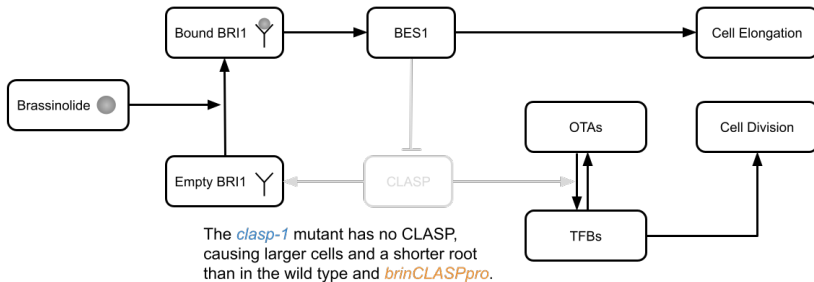


Figure: Hormone interactions observed in *A. thaliana* roots.

brinCLASPpro Mutant



clasp-1 Mutant



Abridged Signalling Network

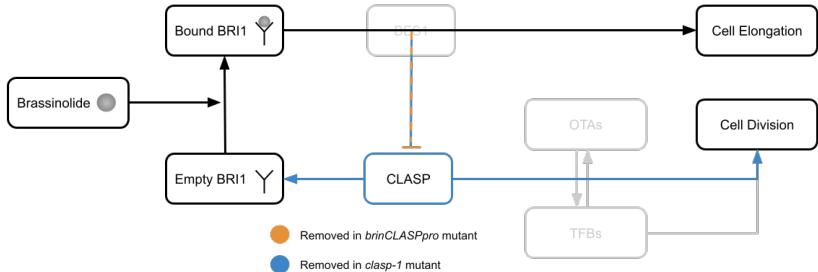


Figure: Simplified signalling network used in the model.

Intracellular Model

Since the intracellular signalling within the cell occurs faster than cell growth and division, we assume it is in a quasi-steady state.

$$0 = \frac{dC}{dt} = (c_0 - c_1 R_B) - c_2 C$$

$$0 = \frac{dR_T}{dt} = (r_0 + r_1 C) - r_2 R_T$$

$$R_B = f(B, R_T, K_d)$$

Extracellular Model

Shown below are the equations for cell growth (dL/dt) and division (dD/dt). When $D = 1$, the cell divides into two cells with $D = 0$.

$$\frac{dD}{dt} = (1 + \delta_0 C) \left(1 - \frac{L^n}{\delta_1^n + L^n} \right)$$

$$\frac{dL}{dt} = (\gamma_0 + \gamma_1 R_B) L$$

Initial Results

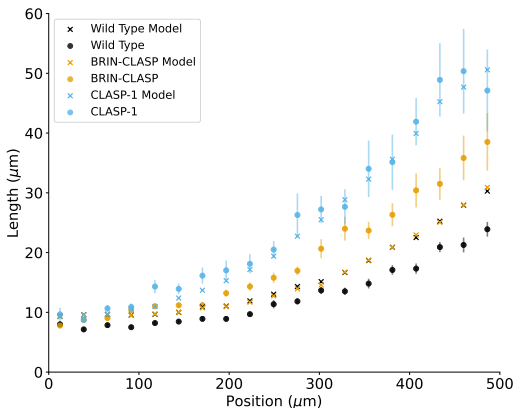


Figure: The model failed to differentiate cell lengths in the root apical meristem of the *brinCLASPpro* mutant from the wild type.

Explaining the *brinCLASPpro* Mutant

Idea: Inhibit division in the *brinCLASPpro* mutant relative to the wild type in order to increase cell length. We hypothesize this is caused by an excess of CLASP.

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To implement this change, we modify the division equation:

$$\frac{dD}{dt} = (\sigma_0 + \sigma_1 C - C^2) \left(1 - \frac{L^n}{\delta_1^n + L^n} \right)$$

Updated Results (1/2)

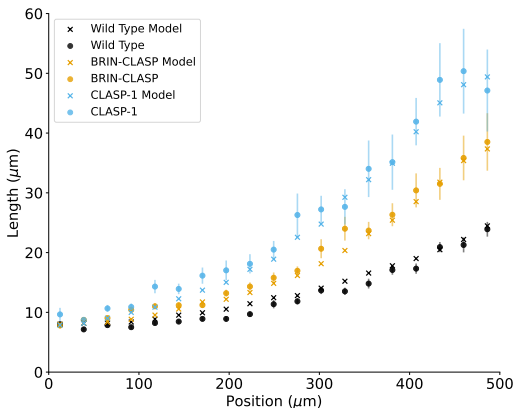


Figure: The updated model correctly differentiates cell lengths in the *brinCLASPpro* mutant from the wild type.

Updated Results (2/2)

The updated model accurately explains the mutant phenotypes:

Mutant	Length	Division Zone Size	Divisions
Wild Type	43 692 μ m	456.5 μ m	324
<i>brinCLASPpro</i>	28 352 μ m	275.0 μ m	213
<i>clasp-1</i>	19 241 μ m	234.5 μ m	142

Conclusion

Key Idea: A mechanism which causes the CLASP protein to inhibit cell division at superphysiological concentrations is sufficient to explain the *brinCLASP^{pro}* mutant.

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Next Steps:

- Integrating this work with intracellular microtubule models.
- Modelling the effects of CLASP on auxin signalling.

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Thanks for listening. Any questions?