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

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Acknowledgements

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- Dr. Geoffrey Wasteneys (Experimental Collaborator)
- NSERC USRA Program

Root Zonation

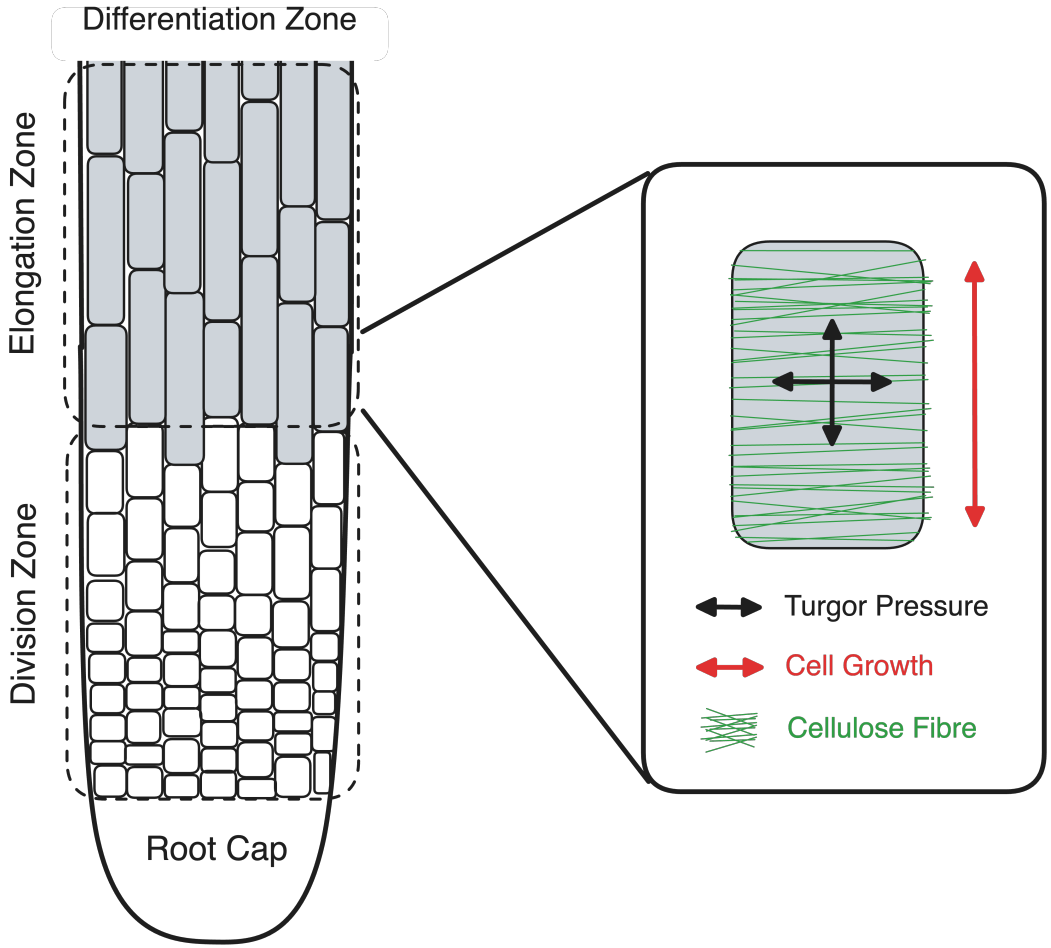


Figure: Zonation of the root apical meristem in *A. thaliana*.

Microtubules

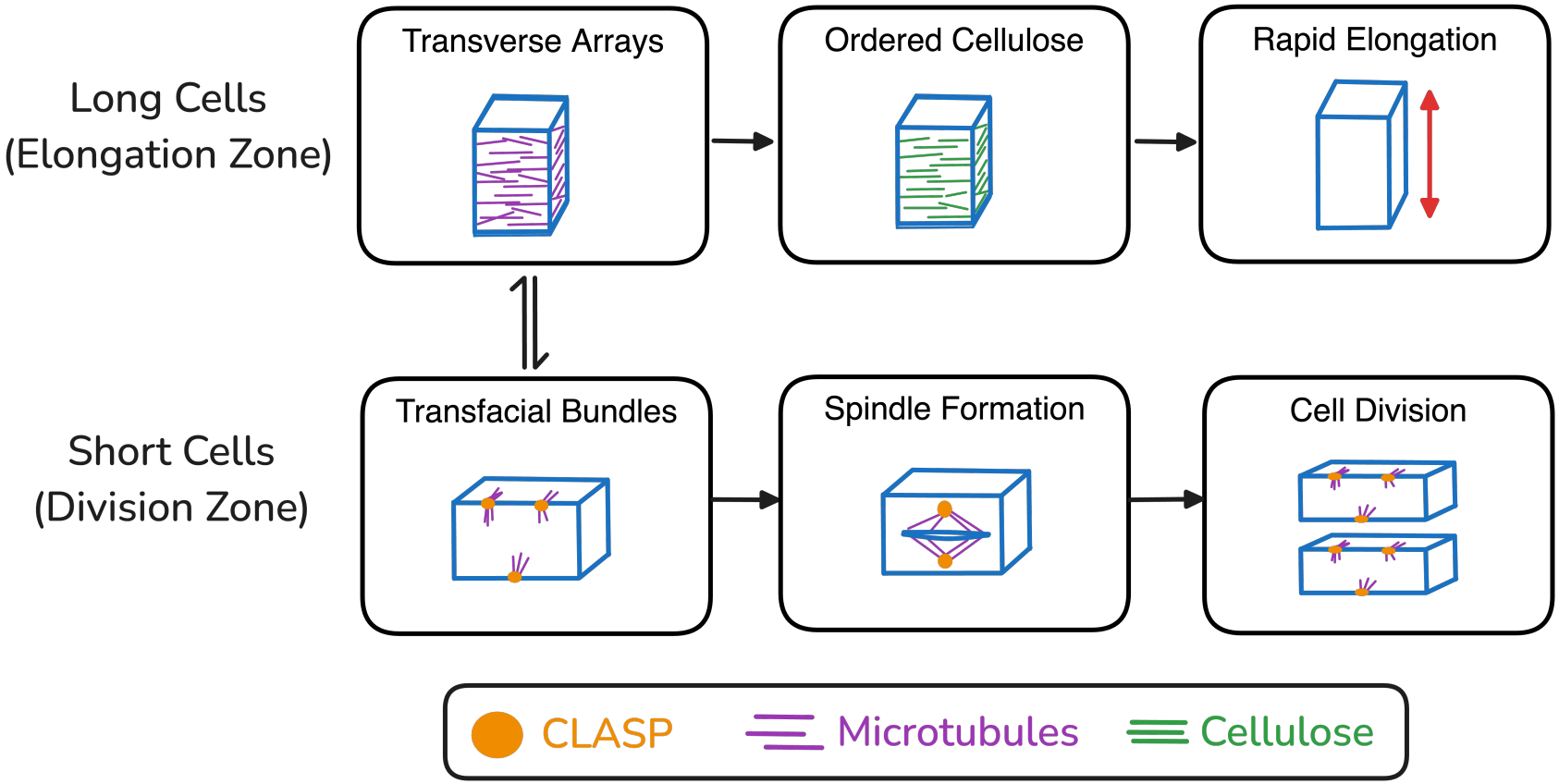


Figure: The arrangement of microtubules is linked with cell behaviour.

Signalling Network

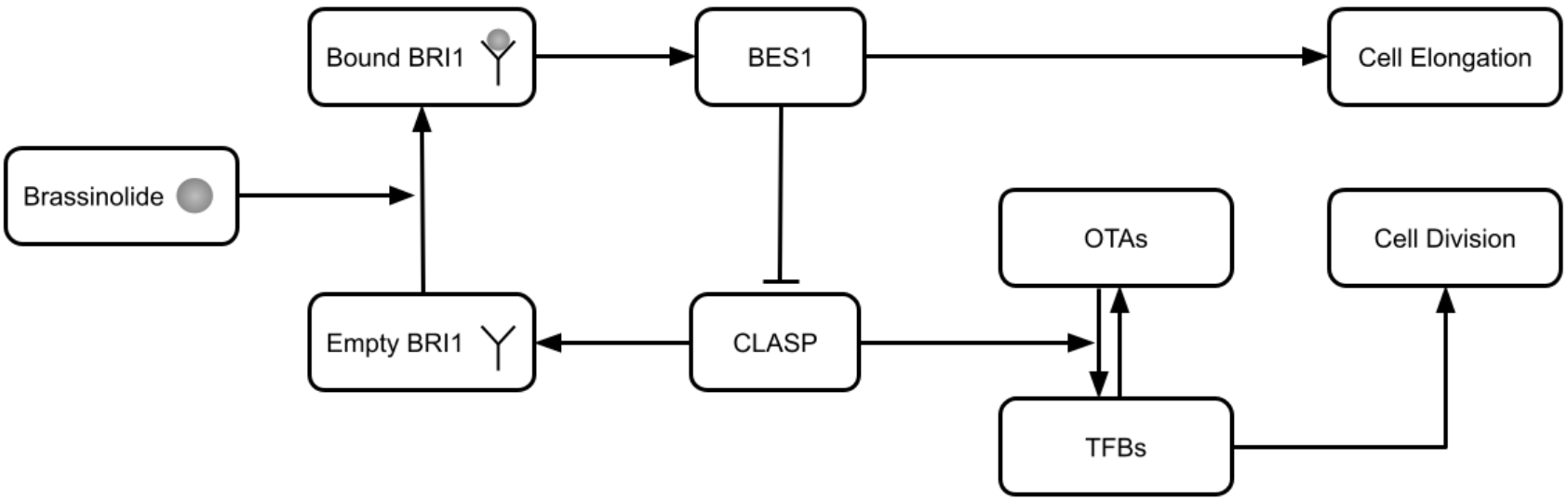
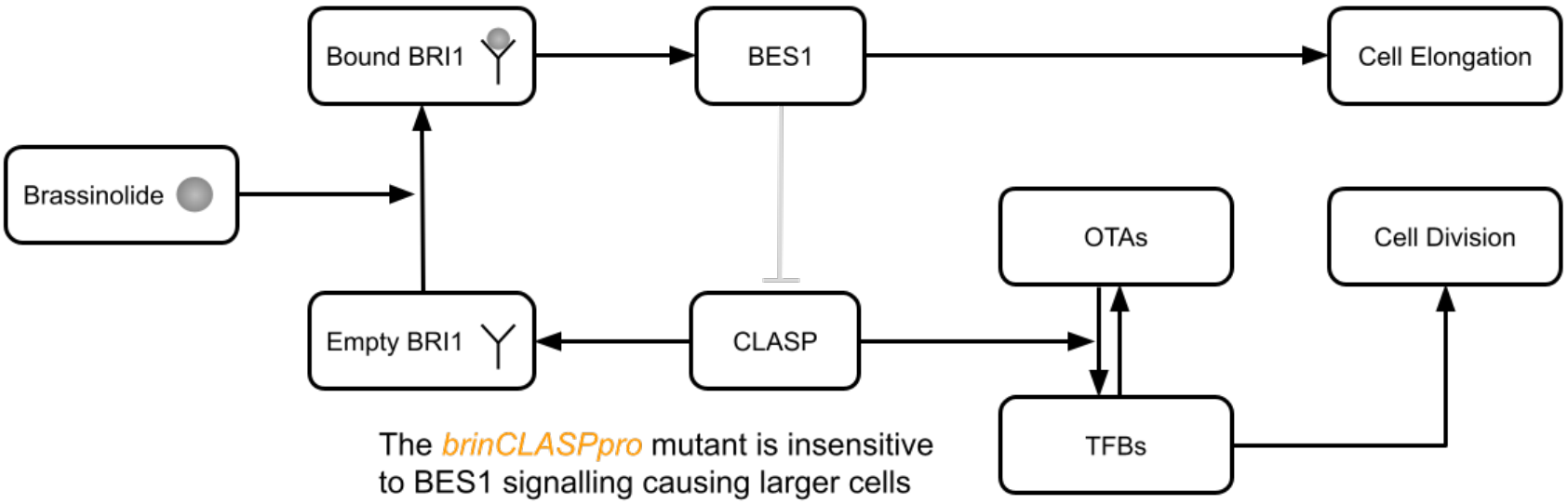


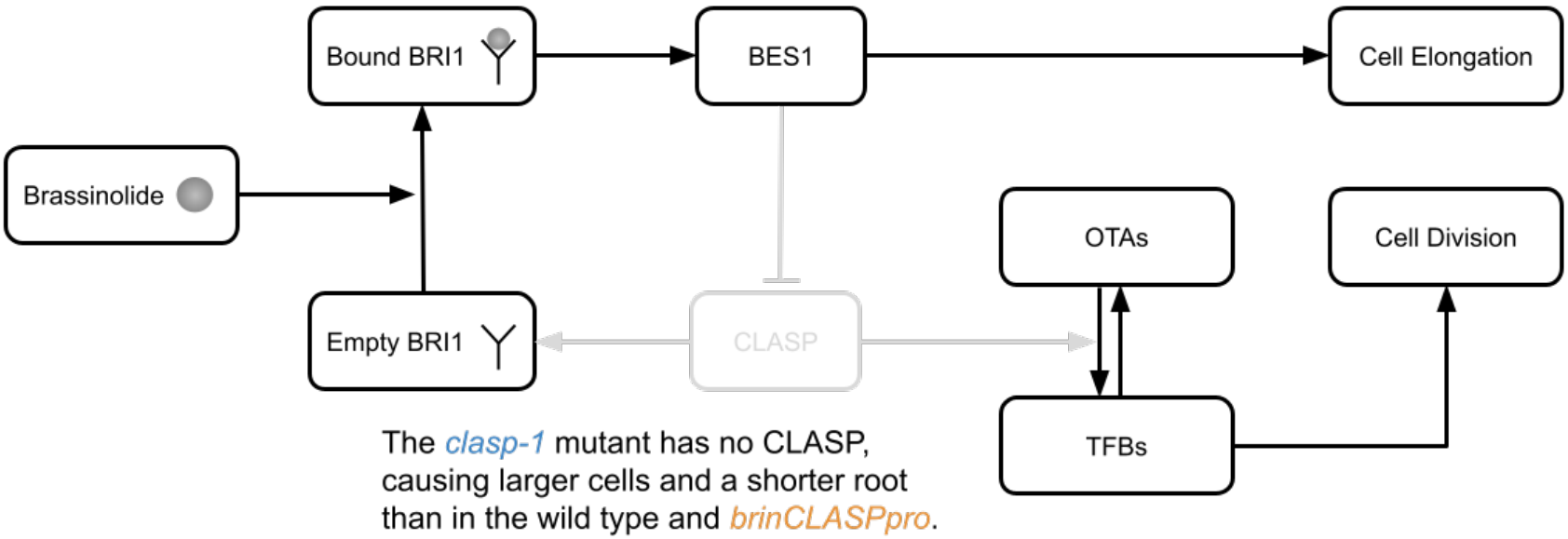
Figure: Hormone interactions observed in *A. thaliana* roots.

brinCLASPpro (BRIN-CLASP) Mutant



The *brinCLASPpro* mutant is insensitive to BES1 signalling causing larger cells and a shorter root than in the wild type.

clasp-1 Mutant



The *clasp-1* mutant has no CLASP, causing larger cells and a shorter root than in the wild type and *brinCLASPpro*.

Mutant Roots

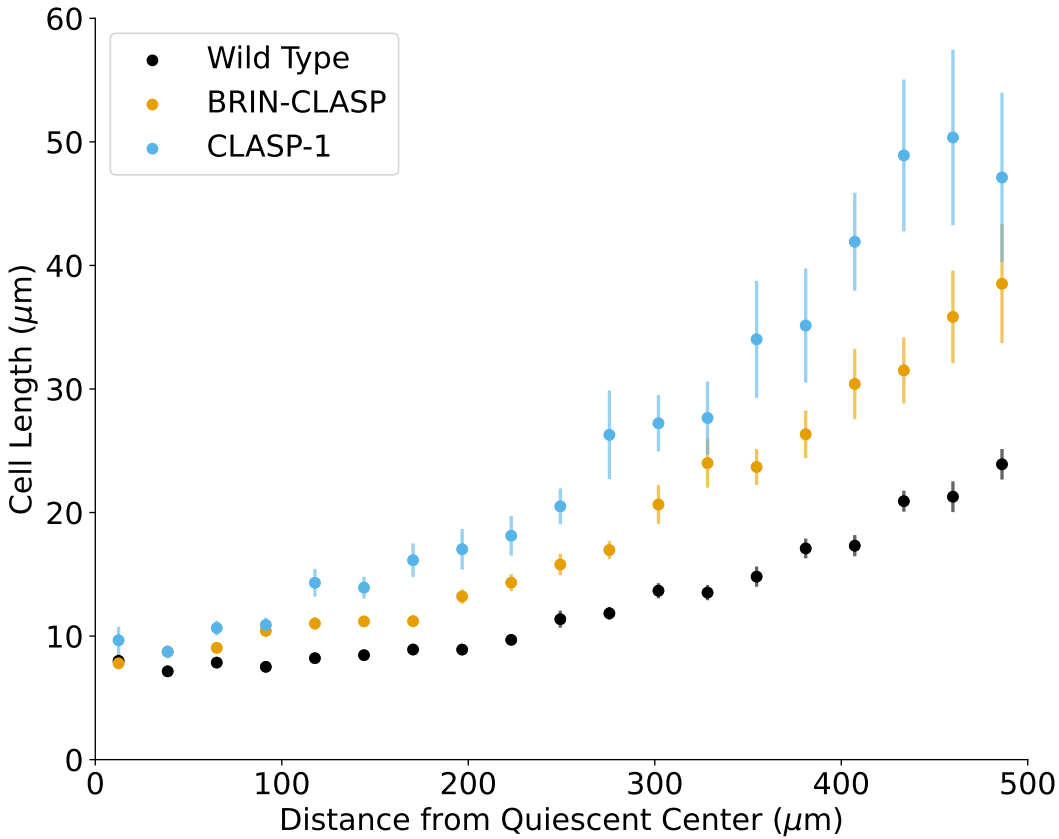


Figure: Experimental data from the wild type and mutants.

Hypothesis

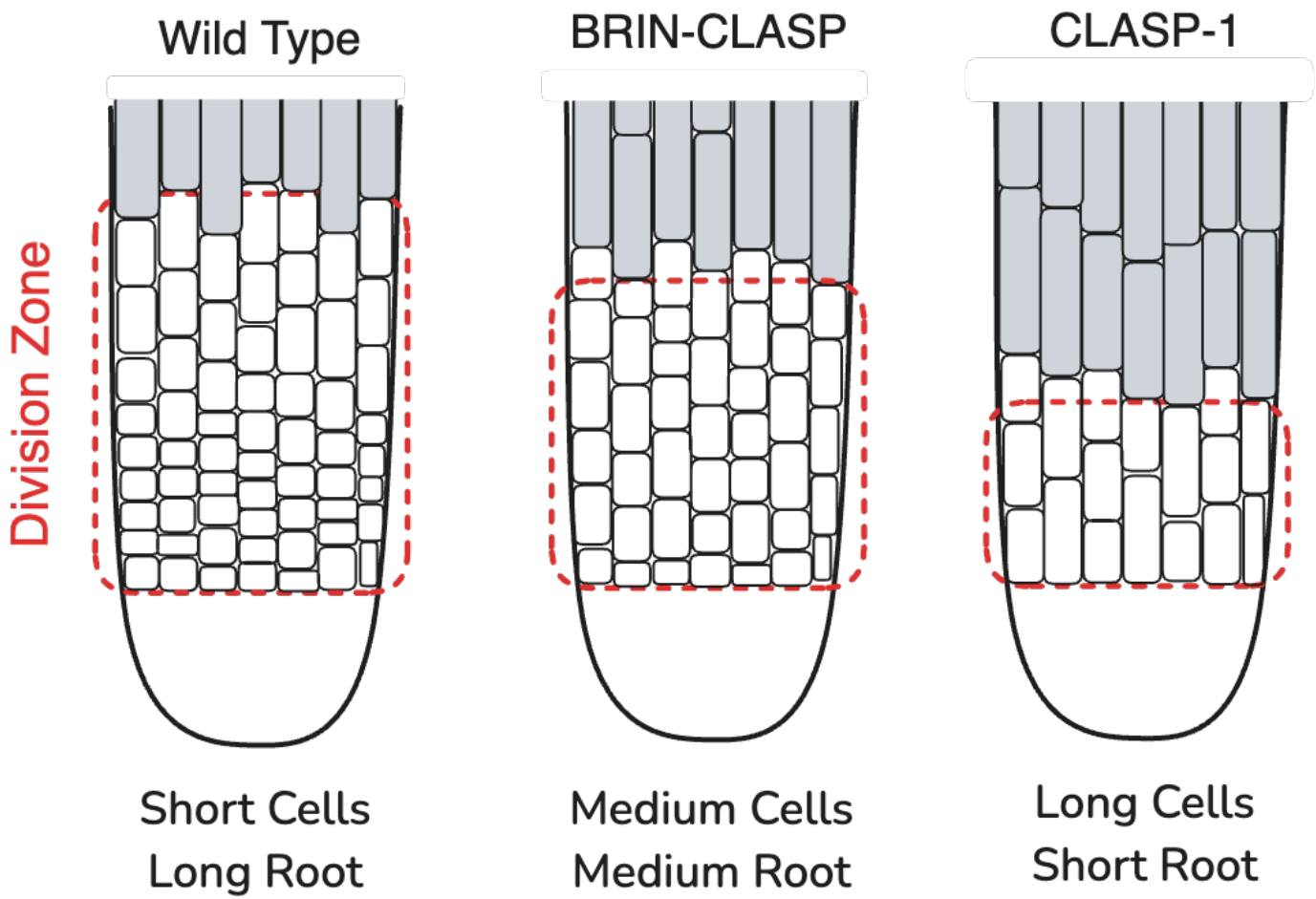


Figure: A "sizer" mechanism for division zone exit produces the different phenotypes in the wild type, BRIN-CLASP, and CLASP-1 roots!

Growth Model Assumptions

- We model a *single column* of cells over time.
- Our data has no time dependence so Δt is arbitrary.
- Cells grow at a basal rate $\gamma_0 L$.
- Cell growth is increased by BES1 at a rate γ_1. The exact model for BES1 signalling is discussed later.

Division Model Assumptions

- Cells complete a cell cycle and divide when $D = 1$.
- Cells also must be at least $m\,\mu$m long to divide.
- Cell division creates two cells with length $L/2$ and $D = 0$.
- Progress in the cell cyle proceeds at a basal rate d_0.
- Progress in the cell cyle is inhibited by *length*.

Abridged Signalling Network

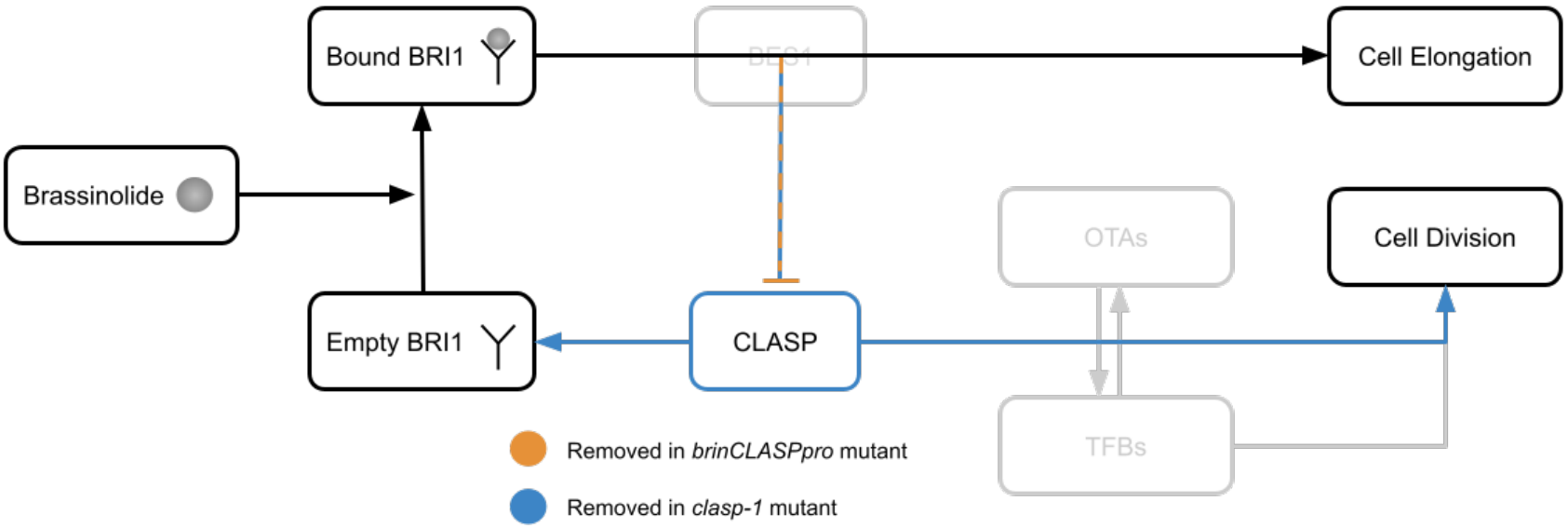


Figure: Simplified signalling network used in the model.

Equations

The intracellular equations are assumed to be in QSS:

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

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

$$0 = \frac{dR_B}{dt} = k_{\text{on}}(R_T - R_B)B_{\text{free}} - k_{\text{off}} R_B$$

Growth and division take place on a much longer time scale:

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

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

Initial Results

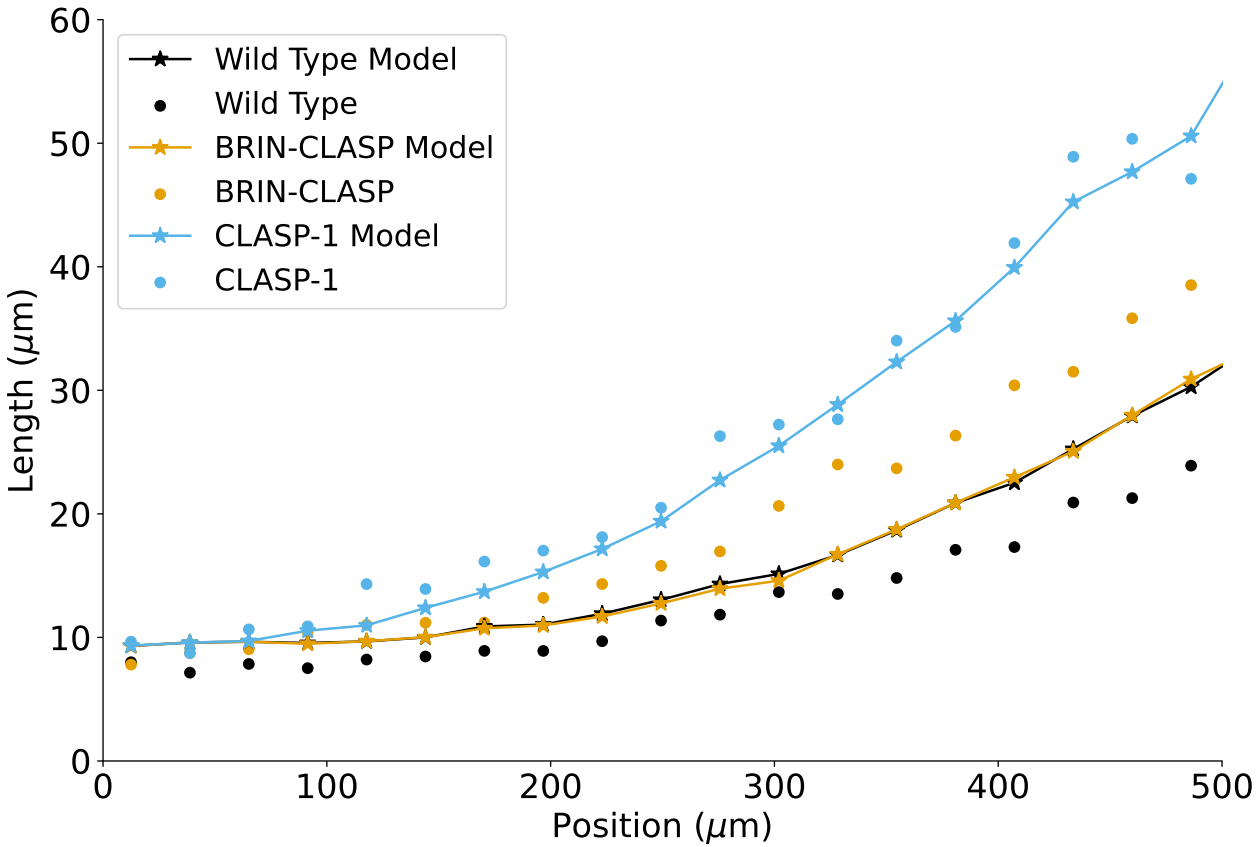


Figure: The model failed to differentiate cell lengths in the BRIN-CLASP mutant from the wild type.

Troubleshooting the Model

The BRIN-CLASP mutant is behaving almost identically to the wild type. There are two possible ways to rescue the mutant:

1. Make cells in the BRIN-CLASP mutant **grow faster**.
2. Make cells in the BRIN-CLASP mutant **divide slower** relative to the wild type, which makes them larger on average.

Solution 1: Promoting Growth in BRIN-CLASP

Why? The BRIN-CLASP mutant has more CLASP and thus more BRI1 receptors. These additional receptors could be binding to brassinosteroid molecules that weren't included in our model, increasing BES1 signalling and growth.

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How? Increase the level of extracellular BL to account for other brassinsteroids. This (as well as some other changes) ultimately *did not* rescue the BRIN-CLASP mutant.

Solution 2: Inhibiting Division in BRIN-CLASP

Why? The higher concentration of CLASP in the BRIN-CLASP mutant increases the amount of transfacial microtubule bundles. An excess of these bundles could prevent tubulin from forming the mitotic spindle.

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How? To implement this change, we modify the division equation to lower the division rate for low *and* high CLASP concentrations.

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

Updated Results (1)

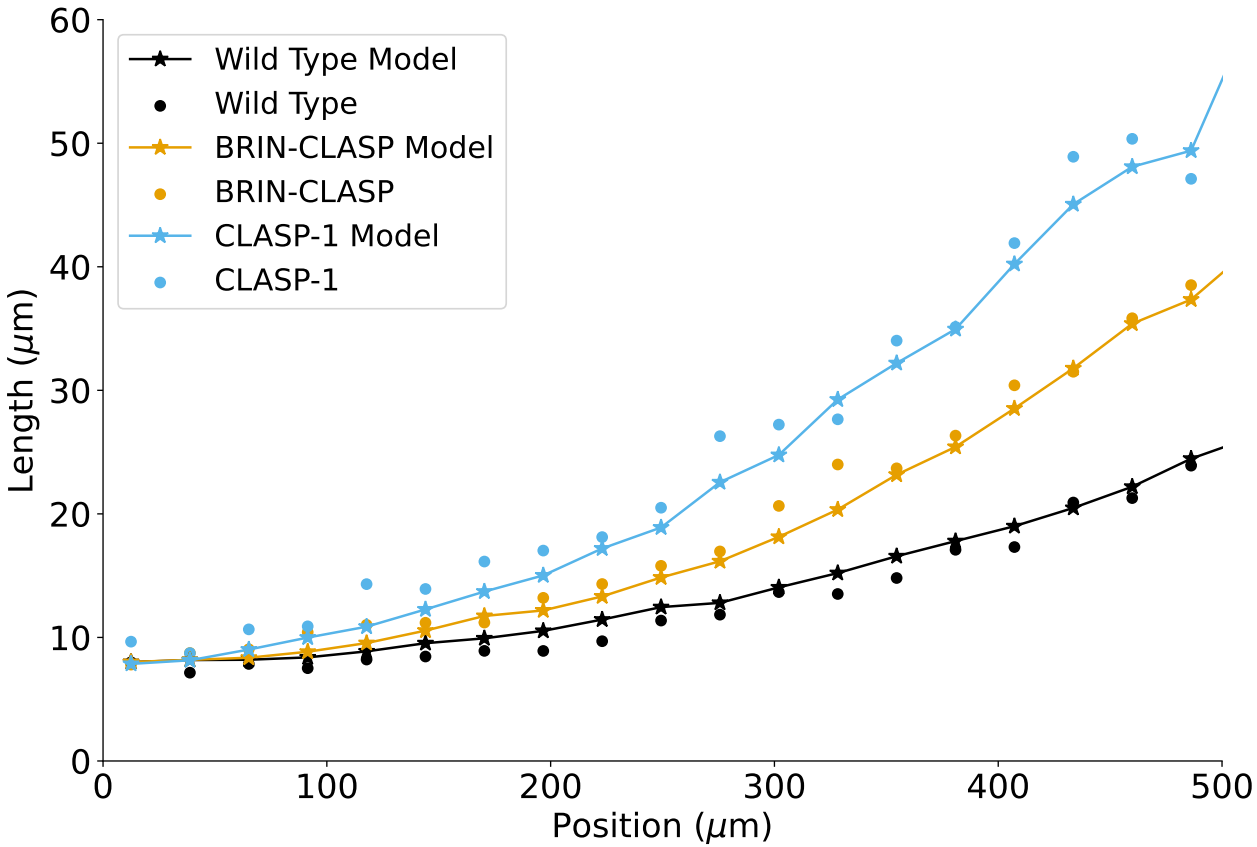

Figure: The updated model correctly differentiates cell lengths in the BRIN-CLASP mutant from the wild type.

Updated Results (2)

The updated model accurately explains the mutant phenotypes:

Mutant	Length	Division Zone Size	Divisions
Wild Type	43 692μm	456.5μm	324
BRIN-CLASP	28 352μm	275.0μm	213
clasp-1	19 241μm	234.5μm	142

Conclusion

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- Exploring other mechanisms for zonation (i.e. "timer").
- Integrating this work with intracellular microtubule models.
- Modelling the effects of CLASP on auxin signalling.

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Thanks for listening. Any questions?