Contribution ID: **57** Type: **Poster**

Solving the inverse problem of Turing patterns

Tuesday, 16 September 2025 16:16 (1 minute)

Patterns such as stripes, spots, and digit arrangements in animals emerge from multicellular organization driven by gene regulatory networks and intercellular signaling. In developmental biology, these processes can be modeled as reaction–diffusion systems, where intracellular gene interactions act as reactions and protein-mediated communication between neighboring cells is captured by diffusion. Turing's mechanism provides a powerful framework to explain how local gene activity translates into large-scale biological patterns.
However, the nonlinear nature of reaction–diffusion equations makes purely numerical simulations insufficient to address fundamental questions: Can distinct gene regulatory networks generate the same observed pattern? How robust are patterns to perturbations in initial conditions? How quickly and precisely do patterns form?
We address these questions by deriving explicit analytical formulas in the linear and weakly nonlinear regimes. First, we find a parametrization of the gene regulatory network that allows us to generate all possible Turing patterns with two morphogens. Second, we deduced an inequality showing that patterns with larger wavelengths take longer to form. Thirdly, we compute a formula for the final amplitude of the pattern, whose region of applicability is precisely where the pattern is the most robust to noise in the initial conditions. These results allow us to characterize pattern sensitivity, likelihood, and dynamics. To validate our approach, we compare the analytical predictions with numerical simulations across several well-studied models, showing strong agreement.
Taken together, our findings could help to identify regions of Turing space that provide most stable and reproducible biological patterns.

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Session Classification: Coffee and Poster Session