



Turing instabilities in a mathematical model for signaling networks

[Andreas Rätz](#), [Matthias Röger](#)

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GTPase molecules are important regulators in cells that continuously run through an activation/deactivation and membrane-attachment/membrane-detachment cycle. Activated GTPase is able to localize in parts of the membranes and to induce cell polarity. As feedback loops contribute to the GTPase cycle and as the coupling between membrane-bound and cytoplasmic processes introduces different diffusion coefficients a Turing mechanism is a natural candidate for this symmetry breaking. We formulate a mathematical model that couples a reaction-diffusion system in the inner volume to a reaction-diffusion system on the membrane via a flux condition and an attachment/detachment law at the membrane. We present a reduction to a simpler non-local reaction-diffusion model and perform a stability analysis and numerical simulations for this reduction. Our model in principle does support Turing instabilities but only if the lateral diffusion of inactivated GTPase is much faster than the diffusion of activated GTPase.

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