On reverse-engineering natural computation

using reaction-diffusion approaches beyond linear stability

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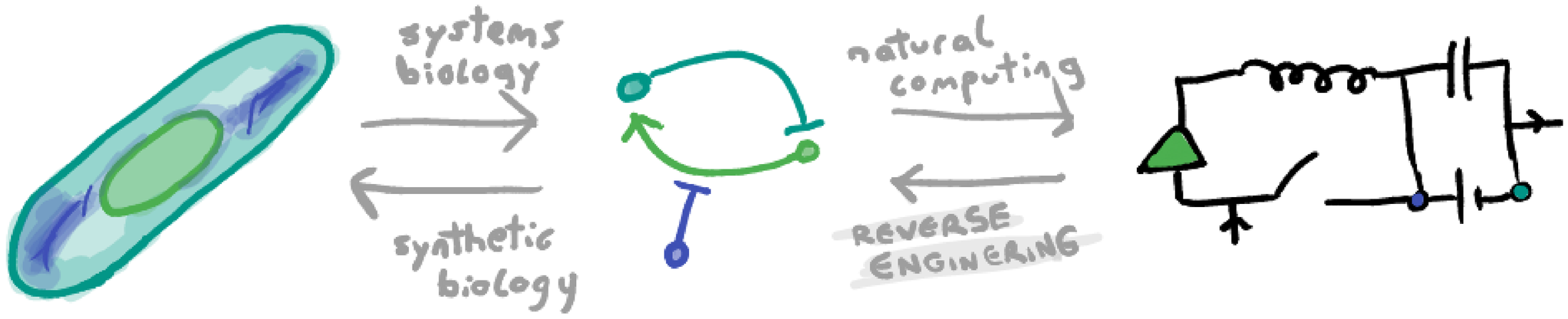
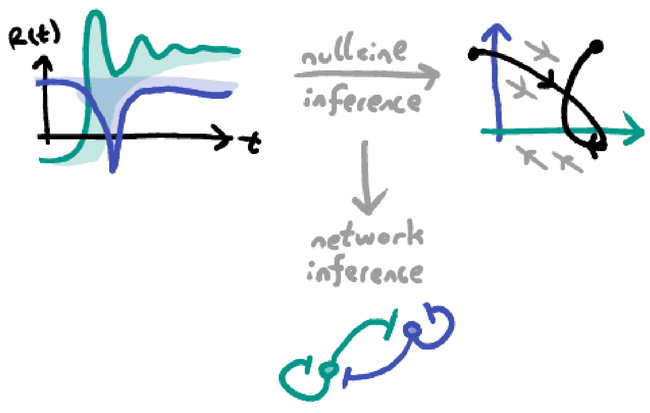


1 Evolution of response behaviour

Specific mappings have been explored between between algorithms, electrical engineering circuits and chemical reaction networks [2]. Understanding function of large biochemical networks from a computer science perspective guides experiments in synthetic biology and in-vitro reconstructive approaches [5].

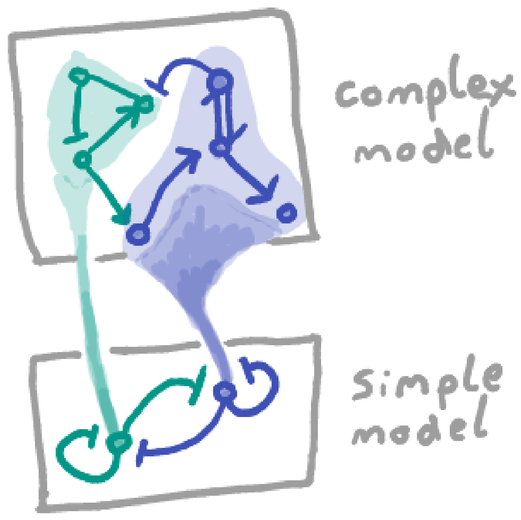
How does one design the *least complex* chemical reaction network that obeys a given *response function*?



The function of known biochemical networks such as the MAPK pathway, circadian rhythms and cell-cycles can be understood in terms of simple response functions; decomposition of large networks into switches and clocks are proposed in literature.

The networks found in nature are far from the least complicated realisations of particular response functions. The additional complexity can be explained by molecular evolutionary paths towards robust biological function [3].

Can model reduction methods [1] identify relevant components, parameters and reduce complexity in reaction networks?



Using measures of relative complexity between two given networks, can we construct evolutionary trees and understand how primitive switches and clocks evolved?

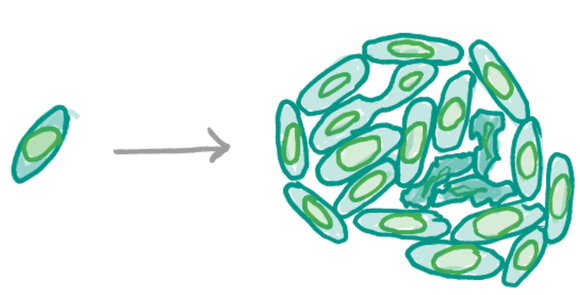
2 Patterns in dynamic populations

Ever since Turing formulated the differential diffusion condition [] for pattern formation, whether a biological pattern is truly driven by a diffusion instability or not has been a matter of debate and speculation.

It is conceivable that the differential diffusion condition is satisfied by the time-scale separation between cytosolic, membrane and inter-cellular reactions.

Finite element reaction-diffusion simulations can take these effects into account explicity at an enourmous computational cost, which leads researches to resort to more abstract Kuramoto-type models.

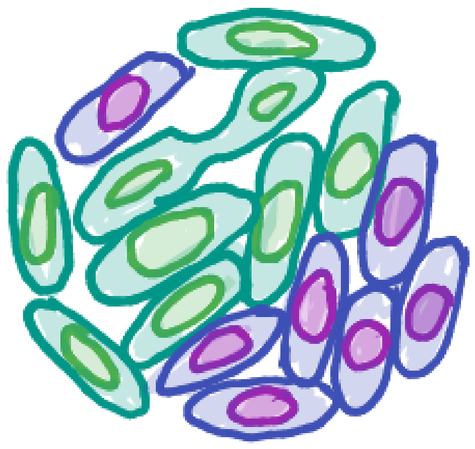
Can we construct a computationally tractable reaction-diffusion model that takes cell division and death into account?



$$\partial_t \rho = \partial_x^2 (D\rho) + R(\rho) \qquad \partial_t D = ?$$

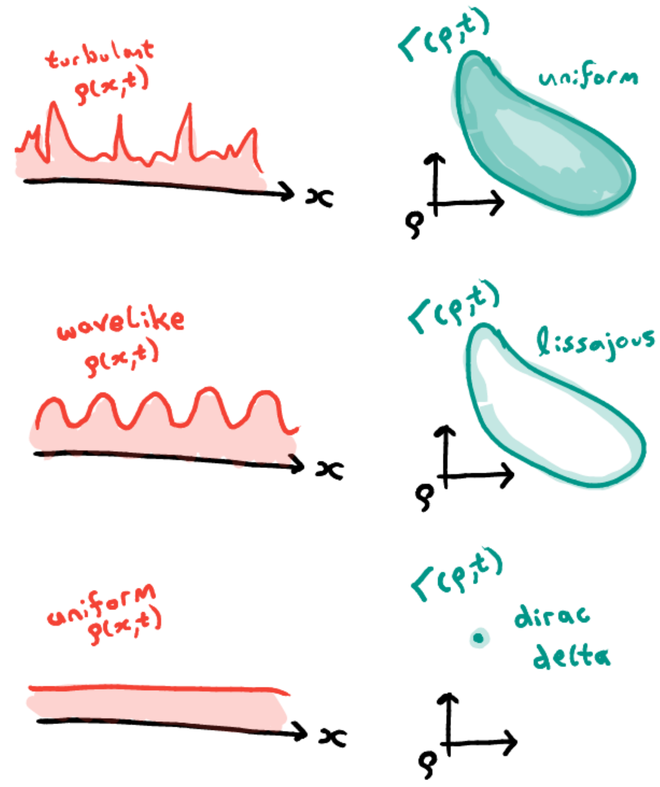
Evolution of biochemical chemical networks is not just driven by random mutation, but are subject to environmental pressures and competition. Snythetic approaches to bacterial competition experiments [] allow us to probe the fitness of different chemical networks.

How to we model competition between cell types that contain different reaction networks?



3 Geometrisation approach

Recent advances in the theory of pattern formation [4] suggest that the dynamics of *local equilibria* provide general insight into pattern formation beyond linear stability analysis.



Such a theory requires a descritption of the system in phase space, moving according to the balance of reactive and diffusive forces. Below I present preliminary equations of motion:

$$\partial_k \left(R(k)\Gamma(k) \right) + \partial_k^2 V(k) = 0$$

$$\Gamma(k,t) := \int_{\mathbb{R}^M} \delta(k - \rho(x,t))\, \mathrm{d}x$$

$$V(k,t) := D \int_{\mathbb{R}^M} (\partial_x \rho(x,t))^2\, \delta(k - \rho(x,t))\, \mathrm{d}x$$

References

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