



Simplicially driven simple contagion

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Motivation

SPREADING PROCESSES CAN AFFECT EACH OTHER

- HIV increases susceptibility to other diseases.
- Unsafe behaviours boost pathogen spread.

HIGHER-ORDER STRUCTURES FOR SOCIAL CONTAGION

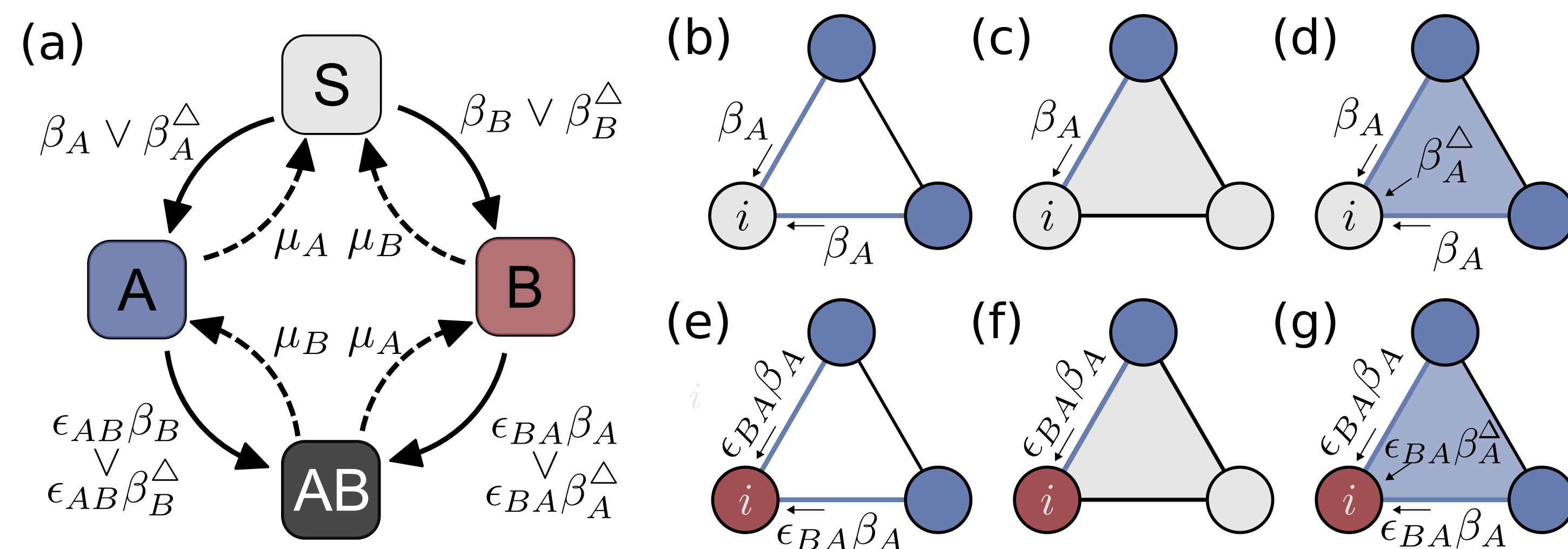
- Abrupt transition to the endemic state.
- Presence of a bi-stable region where endemic and disease free state coexist.

SO FAR:

- Modeling of processes limited to symmetric interaction and simple contagion.

Model

We consider a **model for two interacting spreading pathogens** A and B , which also include simplicial contagion [1]. Individuals can be in one of four compartments, following the standard SIS framework.



Key features of the model:

- Contagion events take place on top of a contact structure with **higher-order (non-pairwise) interactions** [2].
- For each pathogen we have **two contagion routes**: **simple** controlled by parameter $\beta_{x,1} \equiv \beta_x$ and **simplicial** controlled by parameter $\beta_{x,2} \equiv \beta_x^\Delta$. All other nodes in the simplex need to be infectious for the simplex to be considered so.
- The **interaction between the two contagion processes** is controlled via two non-negative parameters ϵ_{AB} and ϵ_{BA} . The two processes **cooperate** if $\epsilon_{xx'} > 1$ and **compete** if $\epsilon_{xx'} < 1$.

Mean-field description

We assume identical recovery rates $\mu_A = \mu_B = \mu$ and introduce the **rescaled infectivity parameters** $\lambda_x = \beta_x \langle k \rangle / \mu$ and $\lambda_x^\Delta = \beta_x^\Delta \langle k_\Delta \rangle / \mu$. The general mean-field equations describing the evolution of the densities are:

$$\dot{\rho}_A = -\rho_A + \lambda_{AS}(\rho_A + \rho_{AB}) + \lambda_A^\Delta \rho_S(\rho_A + \rho_{AB})^2 + \rho_{AB} - \epsilon_{AB} \lambda_B \rho_A(\rho_B + \rho_{AB}) - \epsilon_{AB} \lambda_B^\Delta \rho_A(\rho_B + \rho_{AB})^2 \quad (1a)$$

$$\dot{\rho}_B = -\rho_B + \lambda_{BS}(\rho_B + \rho_{AB}) + \lambda_B^\Delta \rho_S(\rho_B + \rho_{AB})^2 + \rho_{AB} - \epsilon_{BA} \lambda_A \rho_B(\rho_A + \rho_{AB}) - \epsilon_{BA} \lambda_A^\Delta \rho_B(\rho_A + \rho_{AB})^2 \quad (1b)$$

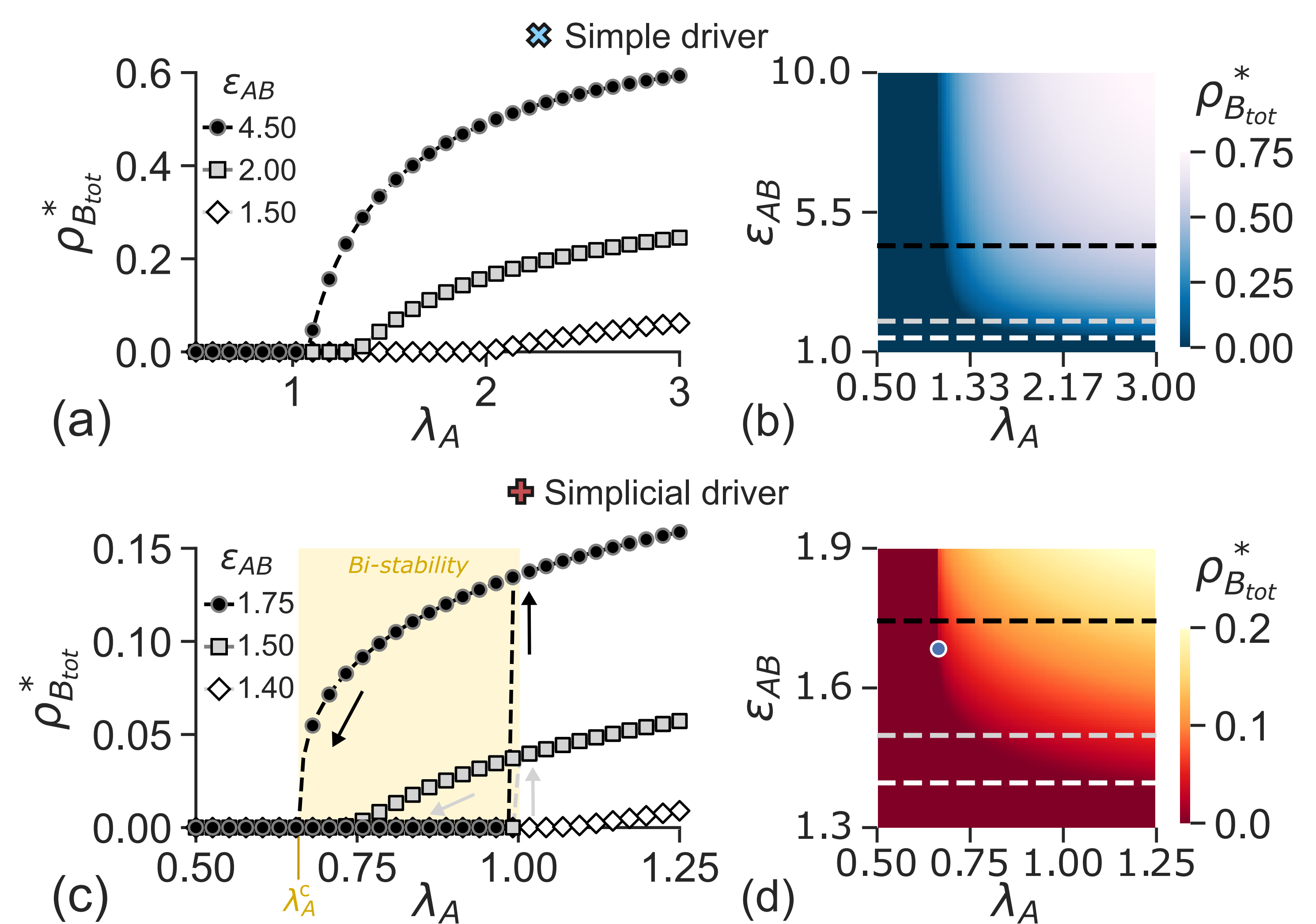
$$\dot{\rho}_{AB} = -2\rho_{AB} + \epsilon_{AB} \lambda_B \rho_A(\rho_B + \rho_{AB}) + \epsilon_{AB} \lambda_B^\Delta \rho_A(\rho_B + \rho_{AB})^2 + \epsilon_{BA} \lambda_A \rho_B(\rho_A + \rho_{AB}) + \epsilon_{BA} \lambda_A^\Delta \rho_B(\rho_A + \rho_{AB})^2 \quad (1c)$$

Results

PATHOGEN $A \rightarrow$ UNSAFE BEHAVIOUR PATHOGEN $B \rightarrow$ DISEASE

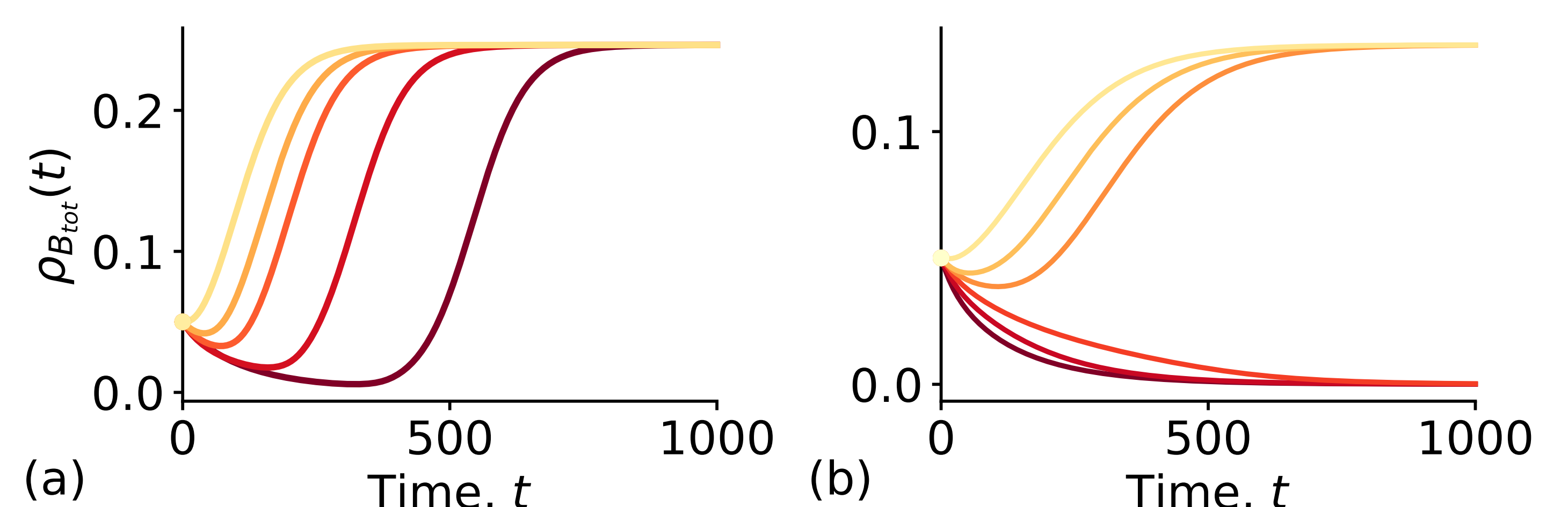
Pathogen A drives pathogen B ($\epsilon_{AB} > 1$)
Pathogen B does not affect pathogen A ($\epsilon_{BA} = 1$)

Abrupt phase transition induced by a simplicial driver



A simplicial driver with $\lambda_A^\Delta = 2.5$ can induce a **discontinuous transition** in pathogen B , contrary to a simple driver with $\lambda_A^\Delta = 0$. The transition of the simple contagion B becomes discontinuous above a critical value of cooperation ϵ_{AB}^c .

Temporal properties



The temporal properties of the driven process depends on the **initial conditions of the driver**. Observing only the driven pathogen B we see a bistability in B by changing the initial conditions of the simplicial driver A .

References

- [1] I. Iacopini *et al.*, Simplicial models of social contagion, *Nat. Comm.*
- [2] F. Battiston *et al.*, Networks beyond pairwise interactions: Structure and dynamics, *Phys. Rep.*

