

AperTO - Archivio Istituzionale Open Access dell'Università di Torino

Intrinsic Properties of Epidemiological Dynamics on Bipartite Networks

This is the author's manuscript

Original Citation:

Availability:

This version is available <http://hdl.handle.net/2318/92988> since

Terms of use:

Open Access

Anyone can freely access the full text of works made available as "Open Access". Works made available under a Creative Commons license can be used according to the terms and conditions of said license. Use of all other works requires consent of the right holder (author or publisher) if not exempted from copyright protection by the applicable law.

(Article begins on next page)

Intrinsic Properties of Epidemiological Dynamics on Bipartite Networks

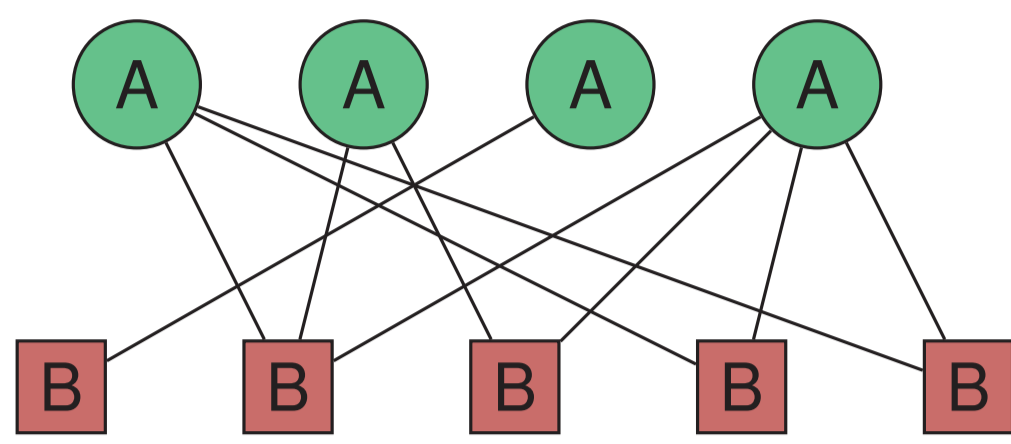
Luca Ferreri, and Mario Giacobini
 {luca.ferreri,mario.giacobini}@unito.it

Complex Systems Unit, Molecular Biotechnology Center
 Computational Epidemiology Group - Department of Animal Production, Epidemiology and Ecology, Faculty of Veterinary Medicine
 University of Torino, Italy

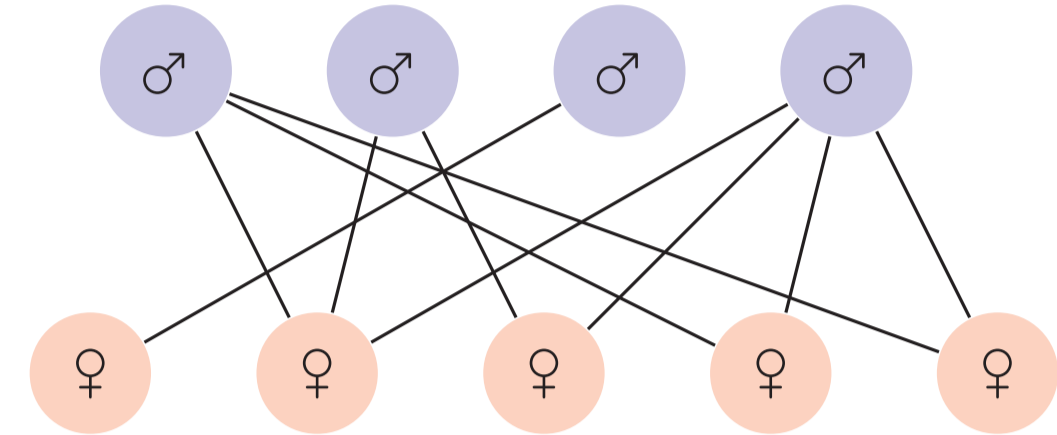


Bipartite Networks

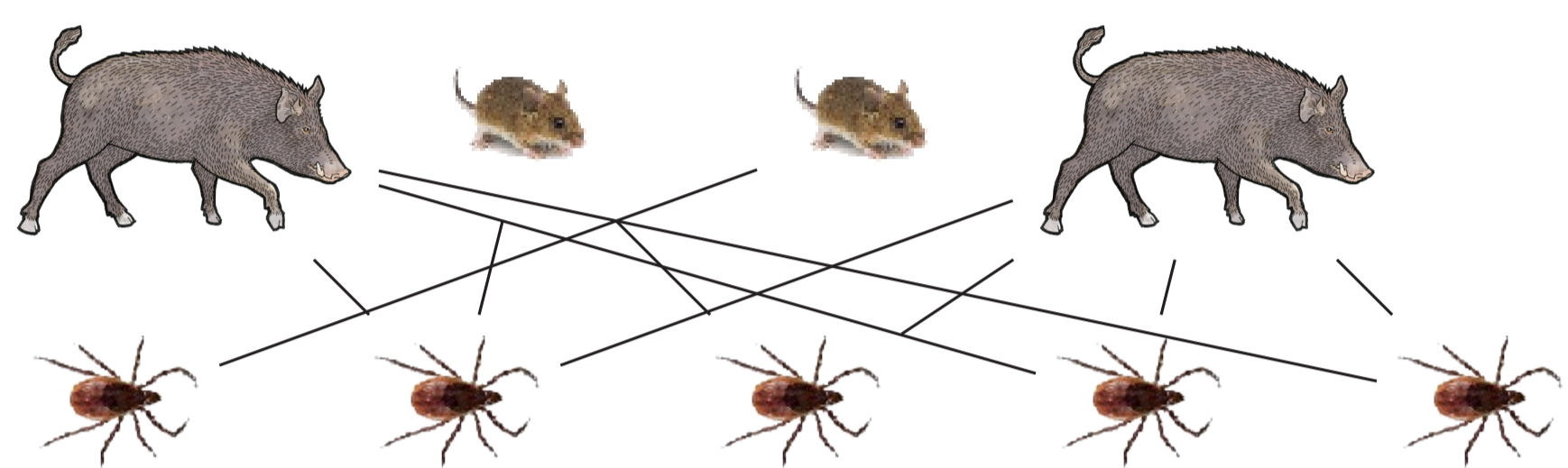
In **bipartite networks** nodes are divided in two families and edges are allowed only between individuals belonging to different families.



- Until nowadays bipartite graphs have been used to describe different structures, as:
 - ▶ co-actor movie networks, having actor and movie nodes;
 - ▶ academic collaboration networks, where researchers bind with their articles;
 - ▶ heterosexual contact networks



- ▶ vector-host contact networks



Vector-Borne Diseases

A disease is called **vector-borne** when the pathogen is transmitted from an infected individual to another individual by a blood-sucking arthropod, called **vector**.

Many vector-borne diseases are a particular kind of **zoonosis**, infectious diseases that could be transmitted from animals to humans, being of big importance for public health reasons.

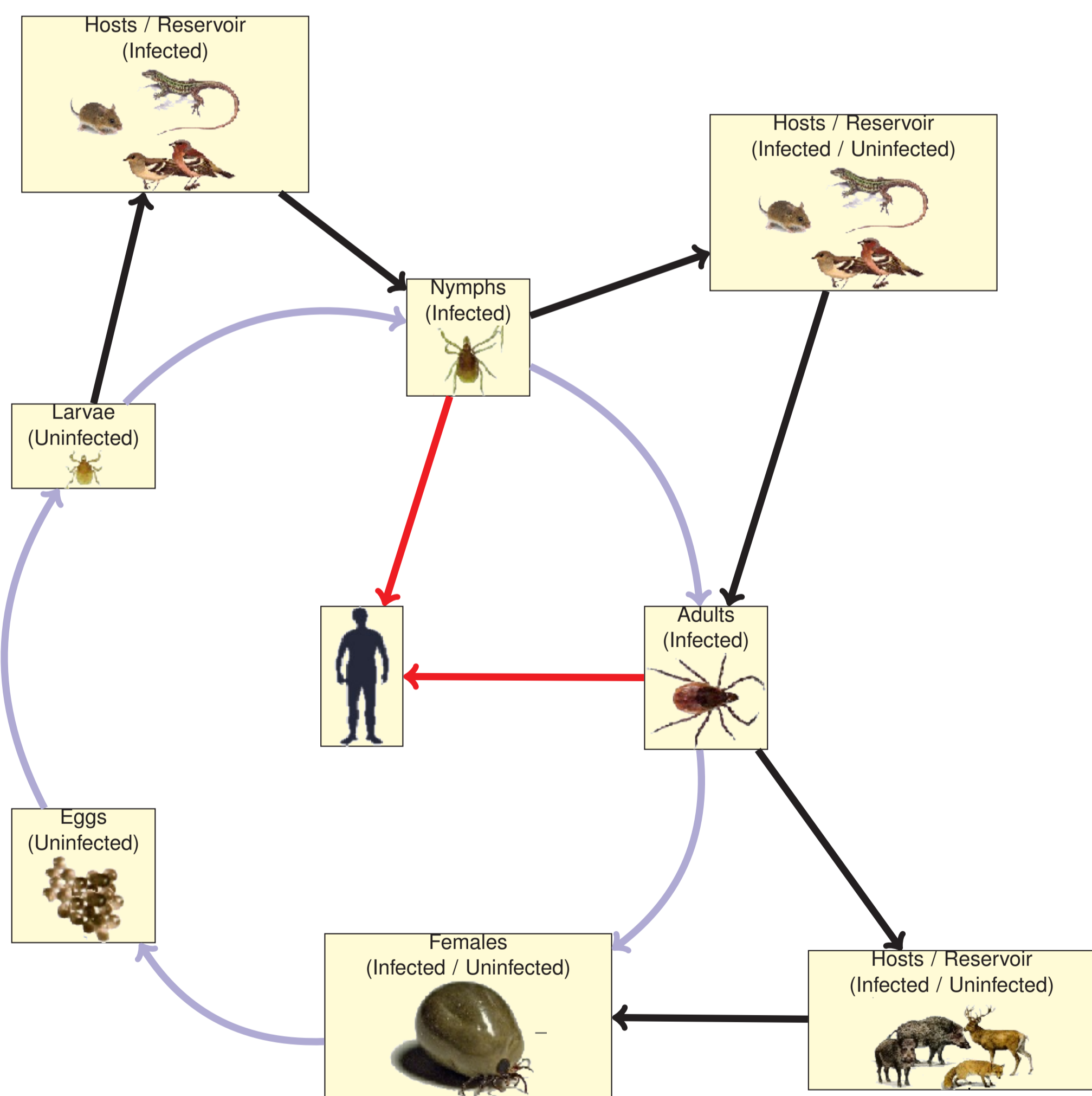
Vector-borne diseases depend upon three different classes of actors:

- ▶ the **pathogens**, either viruses, bacteria, protozoa, or helminths (worms);
- ▶ the **vectors**, commonly arthropods such as ticks or mosquitoes;
- ▶ the **hosts**, usually vertebrate, which could be infected by vectors, thus becoming source of infection for vectors successively feeding on them.

An example: Lyme-Borreliosis

Lyme Borreliosis (LB) is the most common vector-borne disease of humans in temperate climates. It can affect at different threat levels several tissues in the human body: skin, heart, nervous system, eyes, kidneys, and liver.

LB's pathogenic agent are spirochetal bacteria belonging to the *Borrelia* genus. Vectors are hard ticks of the genus *Ixodes* (in Europe *I. ricinus*), while hosts are small-medium vertebrate such as reptiles, birds and mammals depending on the host-seeking tick stage.



Infections Spreading on Bipartite Networks

The transmission of pathogens over a bipartite networks was firstly analyzed by Gómez-Gardeñes and colleagues for **heterosexually transmitted diseases**.

Those results were generalized by Bisanzio and coauthors and applied to **vector-borne diseases**. In that paper an important result was proven: if until then the aggregative behavior of vectors on hosts was usually described by **negative-binomial** distributions, they showed on different datasets that the best fitting was **power law** with exponents around 2.5.

This implies an important consequence: the epidemic threshold vanishes in the limit of large network sizes. In other terms, the condition for an endemic behavior, observed in several countries around the world, seems to be well supported by field data and by the network models. Moreover,

- ▶ on dynamical processes occurring on bipartite networks we observe two exciting phenomena pathogens become endemic for lower probability transmission than those need to spread over unipartite networks structure sharing the same topological proprieties;
- ▶ pathogens show an oscillating behavior which has an attractive biological interpretation.

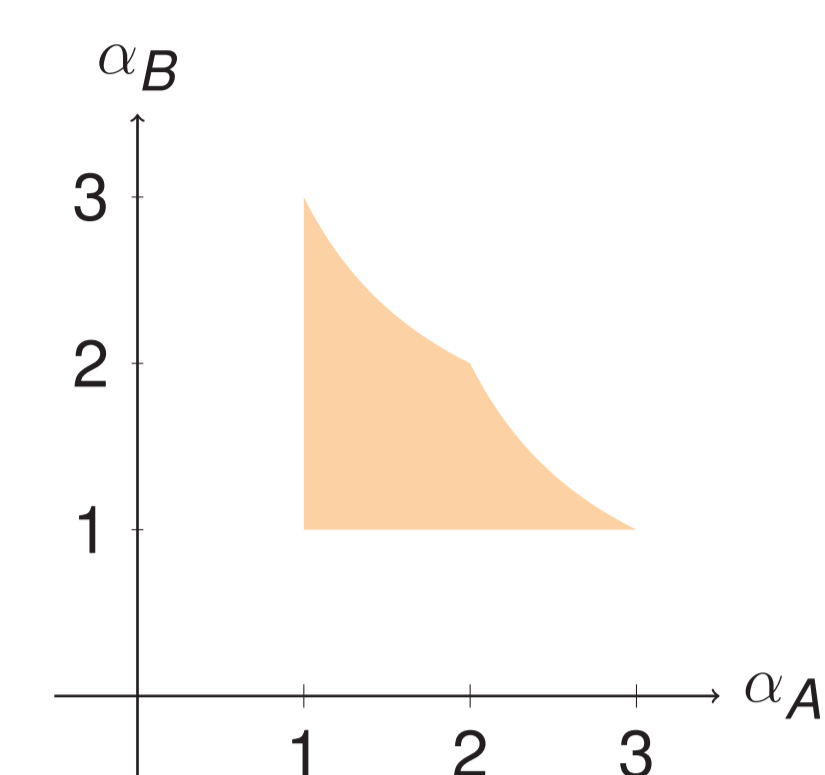
Essential bibliography

- ▶ Pastor-Satorras, R., Vespignani, A. - **Epidemic Spreading in Scale-Free Networks** - Phys. Rev. Let., **86**,14, 3200–3203 (2001)
- ▶ Gómez-Gardeñes, J., Latora, V., Moreno, Y., Profumo, E.: - **Spreading of sexually transmitted disease in heterosexually populations** - PNAS, **105**, 5, 1399–1404 (2008)
- ▶ Bisanzio, Bertolotti, Tommasone, Amore, Ragagli, Mannelli, Giacobini, Provero: - **Modeling the Spread of Vector-Borne Diseases on Bipartite Networks** - PLoS ONE, **5**, 10, 2010
- ▶ Ferreri L., Venturino E., Giacobini M. - **Do Disease Spreading on Bipartite Networks Have Some Evolutionary Advantages?** - LNCS 6623 - 2011
- ▶ Gray J., Kahl O., Lane R.S., and G. Stanek - **Lyme Borreliosis. Biology, Epidemiology and Control** - CABI 2002

Acknowledgement: this project has been supported by the Lagrange Project on Complex Systems, CRT Foundation.

Evolutionary Advantage?

We compare the epidemic threshold derived on bipartite networks with those derived on same but unipartite networks. In fact we compare the asymptotic behavior for enlarging population having degree distributed with two power-laws of exponents α_A and α_B . Analytically we demonstrate that there exist couples of exponents (α_A, α_B) for which the epidemic threshold tends to zero for bipartite but not for unipartite networks.

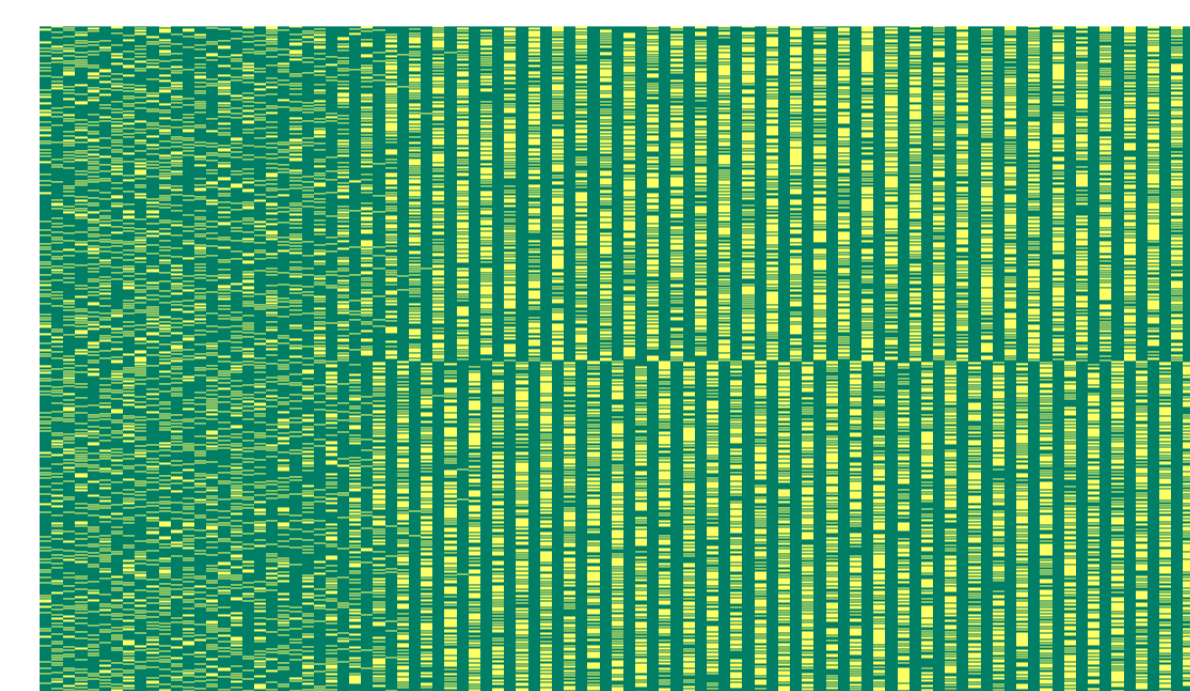


Oscillation Behavior

We generate a number of bipartite random networks with different sizes and connection probabilities p . Over these we let a disease spreads observing the following **rules**:

- ▶ a sound individual at time t connected to at least one infected nodes will be infectious at time $t + 1$
- ▶ an infectious node at time t will be susceptible at time $t + 1$, no matter if it is connect or not with infective individuals!

What we observe is that after few iterations is that the system reaches an oscillating equilibrium. In fact we found a 2 periodic solution and even more amazingly we have a (anti-)synchronization phenomena.



Analytical results

Let's consider random bipartite networks, where A and B are the two families of nodes, and two nodes $n \in A$ and $m \in B$ are connected with probability p .

For a node belonging to the A partition the degree distribution is nothing but the binomial:

$$\mathbb{P}_A(k) = \binom{N_B}{k} p^k (1-p)^{N_B-k} \quad (1)$$

and analogous for nodes belonging to B partition. Calling $x(t)$ the prevalence of infected A -nodes at time t , and respectively $y(t)$ the prevalence of infected B -nodes, and using eq. (1) we could detect the probability for a A -node to have k neighbors of which h infected is nothing but:

$$\binom{N_B}{k} p^k (1-p)^{N_B-k} \binom{k}{h} y(t)^h (1-y(t))^{k-h}$$

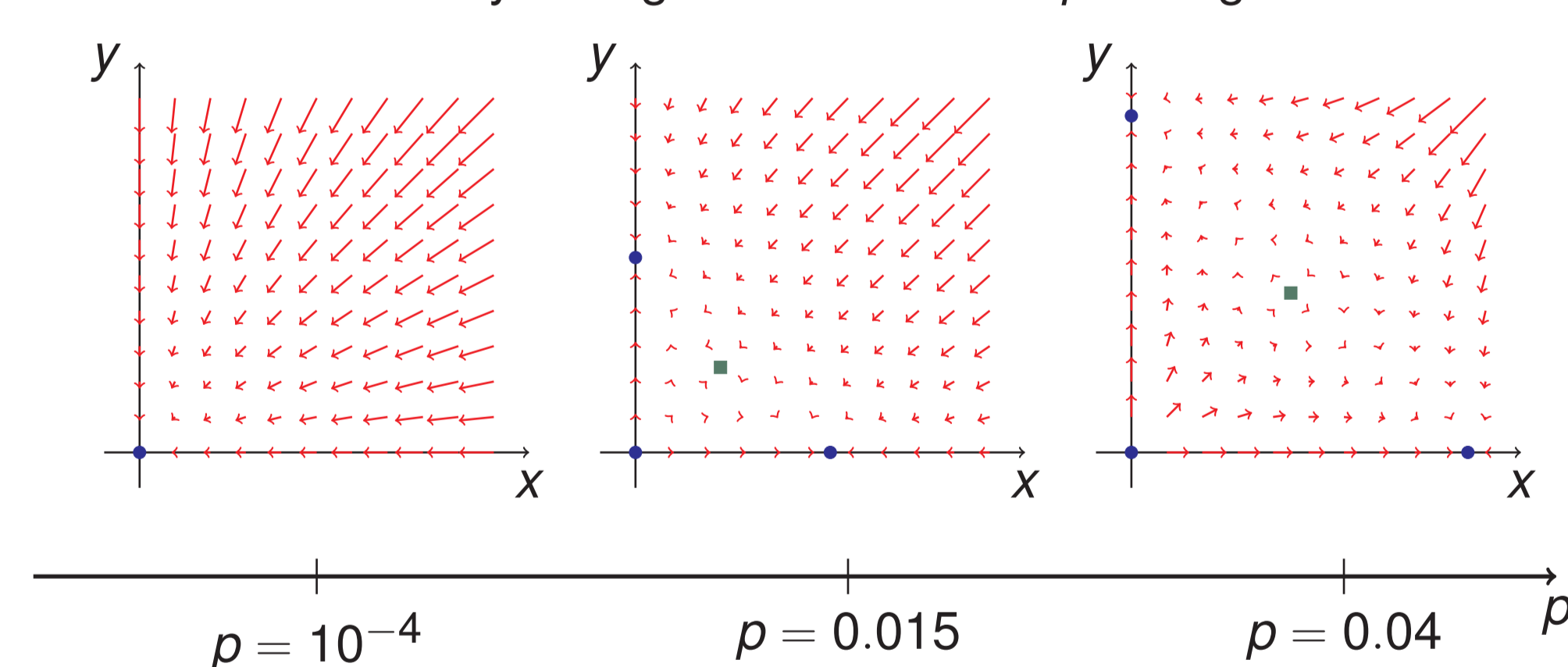
However, a susceptible node get the infection if at least one neighbor is infectious.

Therefore, we sum previous for $h = 1, \dots, k$. Thus, at time $t + 1$ the prevalence in family A is nothing but

$$x(t+1) = (1-x(t)) - (1-x(t))(1-py(t))^{N_B} \quad (2)$$

and similarly for the prevalence in family B .

Now, in order to check the 2-periodic solutions, which mean oscillations of period 2, we impose that concurrently $x_{t+1} = x_{t-1}$ and $y_{t+1} = y_{t-1}$. We numerically find the solutions using the Newton's method. We observe they change as the value of p changes.



Discussions

Results presented here are of some interests from a **biological** point of view.

- ▶ The fact that there exist scale free random bipartite networks on which the transmission probability needed for a pathogen to became endemic tends to zero, in the limit of large population size, while this does not happens on same but unipartite networks could mean that pathogen could take advantage of the peculiar transmission route described by bipartite network. That could result in an **evolutionary advantage** for these pathogens as their transmission probabilities is less subject to **selection pressure**.
- ▶ Moreover the oscillating behavior observed in infection spreading over synthetic bipartite networks is observed in real world: e.g. tick nymphs are active in Spring while larvae are active during the Summer of the same year. Hence, in order for the pathogen to be passed to a next generation of larvae, hosts are required to keep the pathogen from Spring to Summer, having been first infected by nymphs and then transmitting to larvae.