The mystery of bacteria diversity

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1- Introduction

- Microorganisms are engaged in an endless arms race $\rightarrow$ a diversity of antimicrobial compounds are produced by most species.
- These substances include bacteriocins (protein antibiotics).
- Such allelopathic compounds are mediators of intra- and interspecific interactions $\rightarrow$ relevant factors in maintening microbial diversity.

- Bacteriocins actively secreted from a bacterial cell (microcins) are significantly more common than those released as a result of cell lysis (colicins).
- Bacteriocin resistance occurs when mutations eliminate or alter the cell surface receptors to which a particular bacteriocin binds.
- The main question: *can a stable bacterial community be established through allelopathic interactions?*

2- Model

- ✓ The population consists of *N* different, competing bacterial strains.
- ✓ Each strain secretes specific microcins that can kill other strains. Also, each strain is immune to its own microcin.
- ✓ Mitotic cell division $\rightarrow$ mutations: the two resulting cells can transform into one of its "nearest-neighbors" strains.

✓Mutators can evolve resistance to their competitors' microcins.

⬇

change the edges of the interspecific interaction network.

✓Population dynamics:

$$\vec{u}(t+1) = (A - B)\vec{u}(t)$$

(a_{ij})=average number of offspring of the strain *i* produced per generation by a bacterium of the strain *j*.

(b_i)=average fraction of individuals of the strain *i* dead per generation

$$\vec{m}(t+1) = (1-\gamma)\vec{m}(t) + \beta\vec{u}(t+1)$$

Microcins' decay rates

Microcins' synthesis rates

$$A = 2\begin{pmatrix} p_1(1-\sum_{j\neq 1}\nu_{1j}) & p_2\nu_{21} & \cdots & p_N\nu_{N1} \\ p_1\nu_{12} & p_2(1-\sum_{j\neq 2}\nu_{2j}) & \cdots & p_N\nu_{N2} \\ \vdots & \vdots & \ddots & \vdots \\ p_1\nu_{1N} & p_2\nu_{2N} & \cdots & p_N(1-\sum_{j\neq N}\nu_{Nj}) \end{pmatrix}$$

$$p_i = \begin{cases} p, \, se \sum_{j\neq i}\xi_{ij}m_j \leq \theta_i \\ p\exp\left\{-a_i\left[\left(\sum_{j\neq i}\xi_{ij}m_j\right)-\theta_i\right]\right\}, otherwise. \end{cases}$$

$$b_i = \begin{cases} q, \, se \sum_{j\neq i}\xi_{ij}m_j \leq \theta_i \\ 1-(1-q)\exp\left\{-d_i\left[\left(\sum_{j\neq i}\xi_{ij}m_j\right)-\theta_i\right]\right\}, otherwise. \end{cases}$$

where:

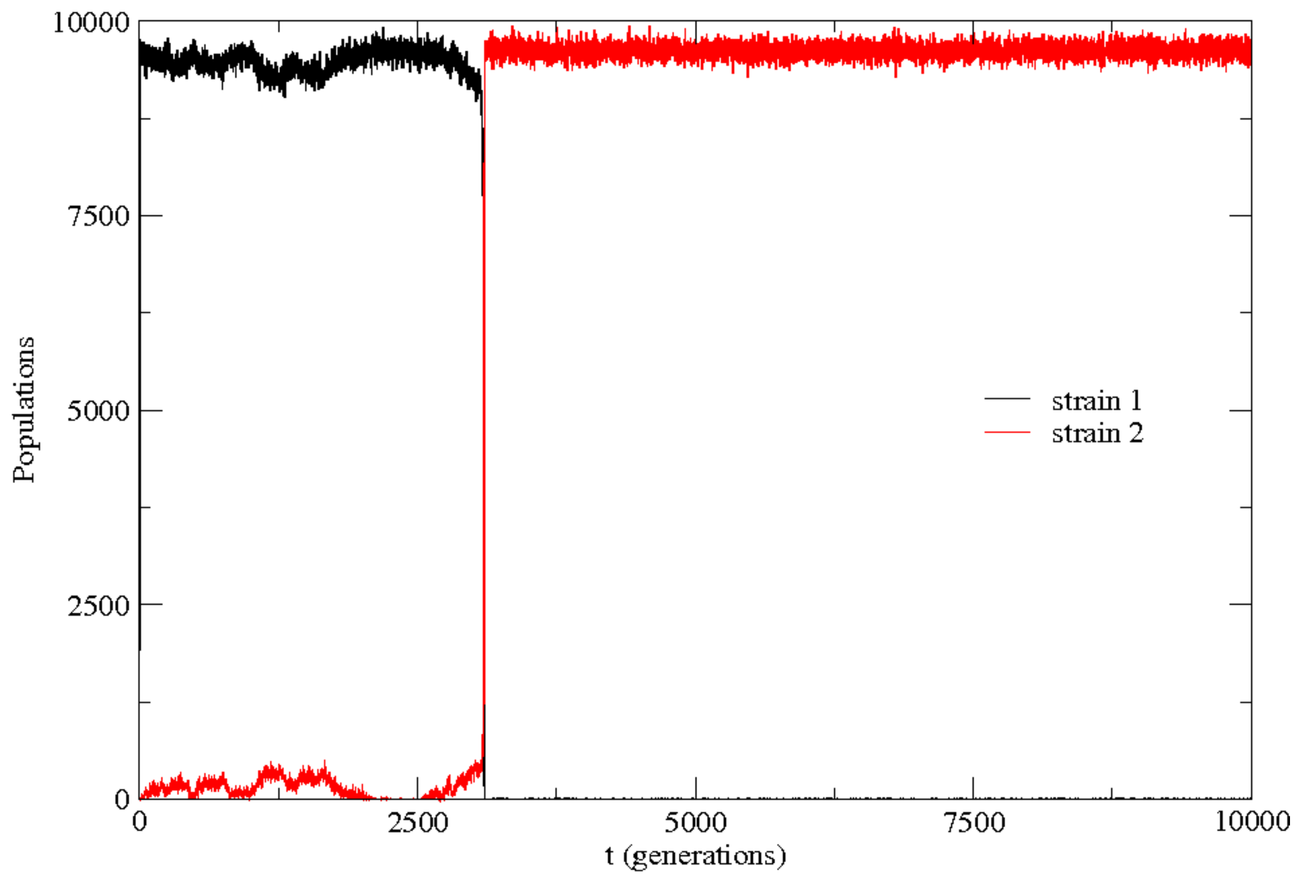
$$p = \exp\left(\frac{1}{K}\sum u_i\right); \quad q = 1 - p \quad \text{e} \quad \xi_{ij} = \begin{cases} 1, \textit{if strains } i \textit{ and } j \textit{ interact} \\ 0, \textit{otherwise.} \end{cases}$$

ξ_{ij}=1 (0) with probability λ (1-λ) → random interspecific interaction network.

3- Results

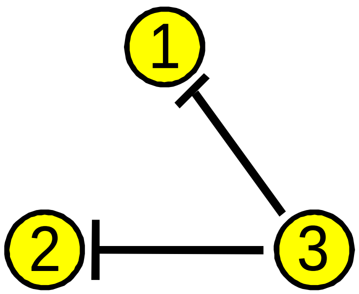
- *N*=2: the classical invasion problem

network



➢ N=3: the emergence of defensive alliances

✓ allelopathic invasion

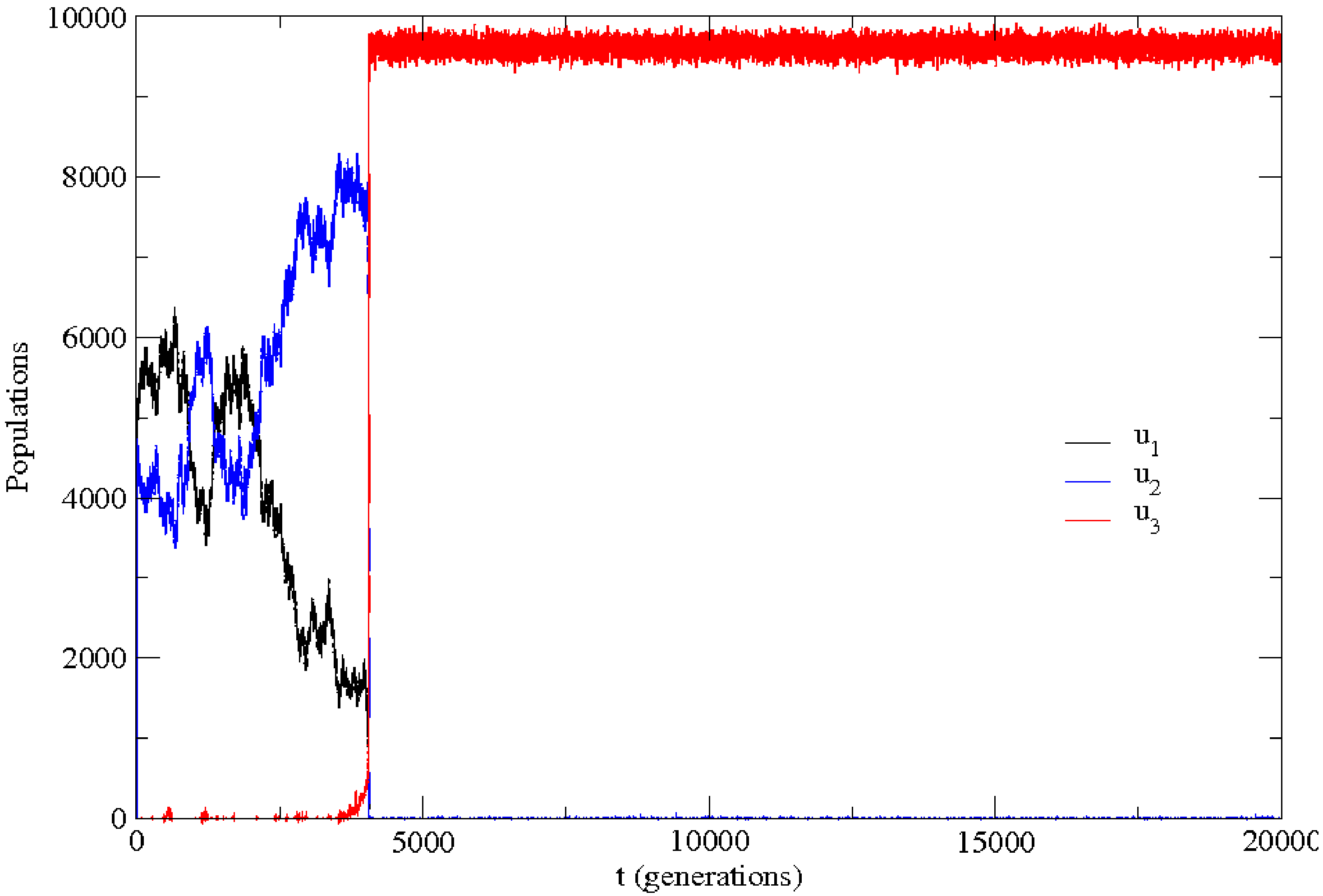


network

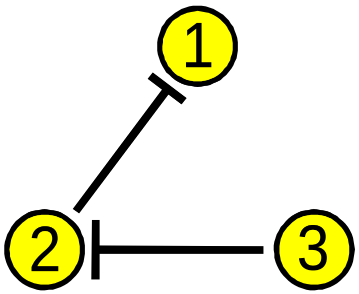
$u_1(0)=u_2(0)=10$

e $u_3(0)=10$

Strain 3 invades with P_{inv}=76%.



✓ defensive alliance



network

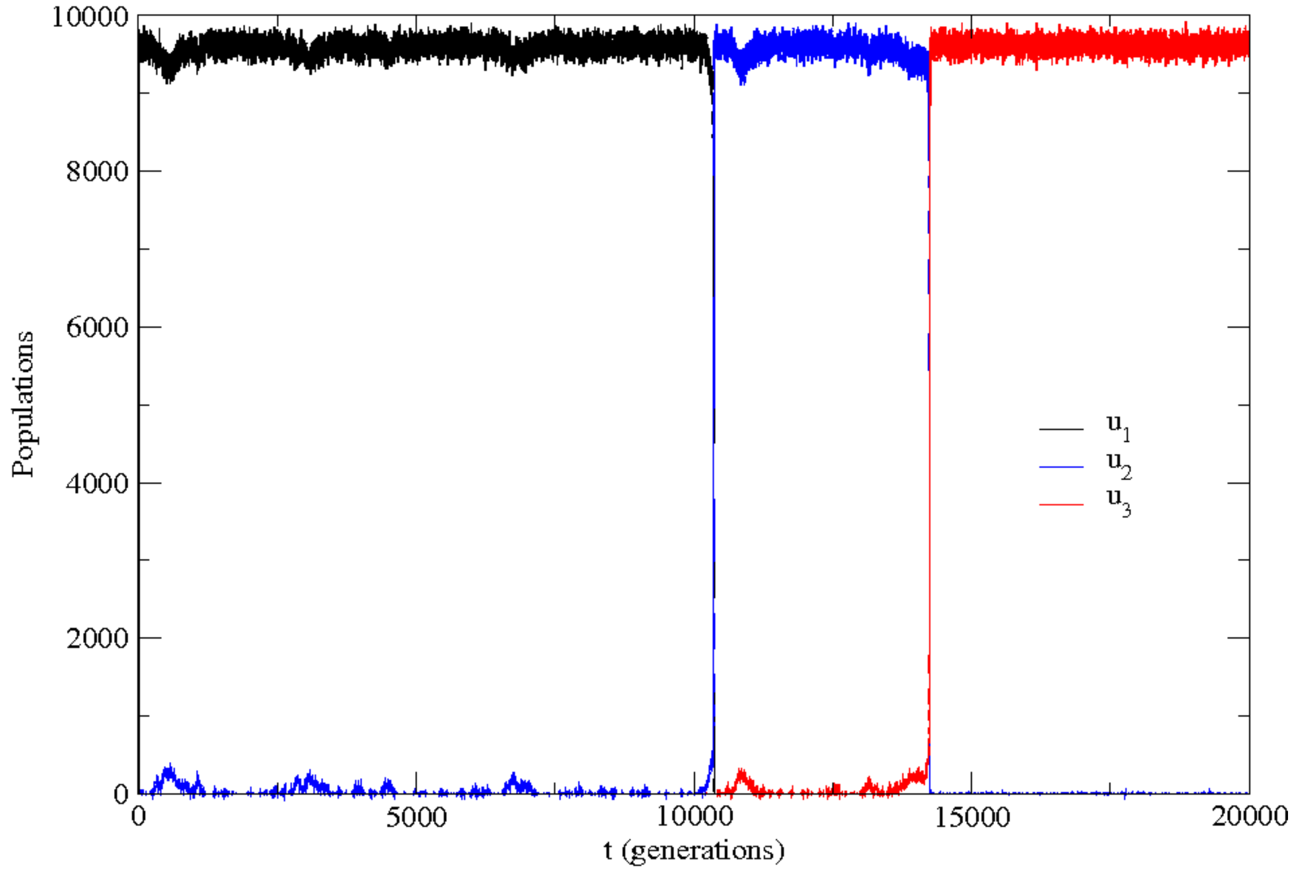
$u_1(0)=u_2(0)=u_3(0)=10$ ➡ Strain 3 invades with $P_{inv}=1$.

$u_1(0)=u_2(0)=10$ e $u_3(0)=10$

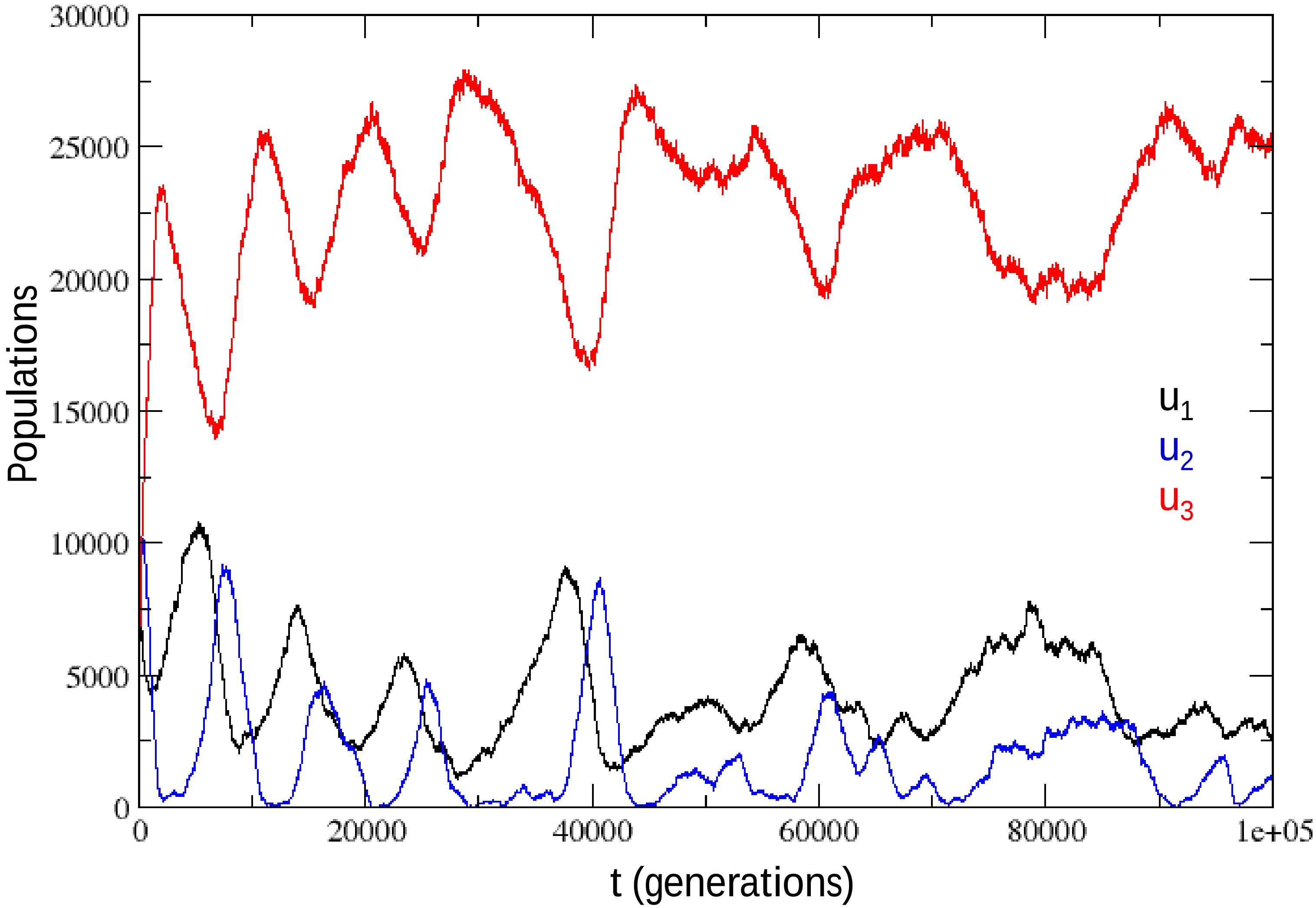
The alliance fails.

$u_1(0)=10$ e $u_2(0)=u_3(0)=0$

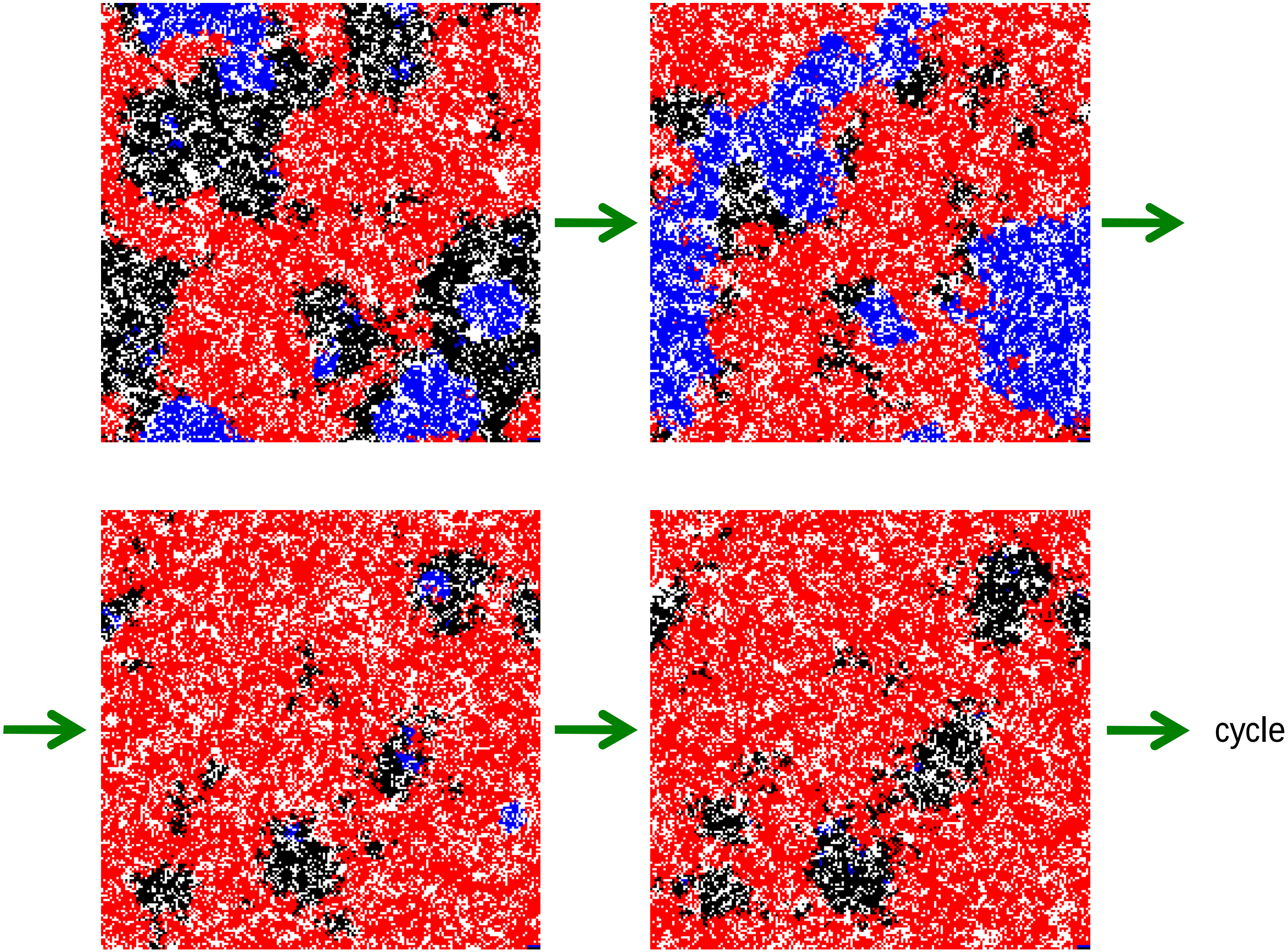
$P_{inv3}=80\%$; $P_{inv2}=15\%$
Two-strains coexistence:
P=5%.



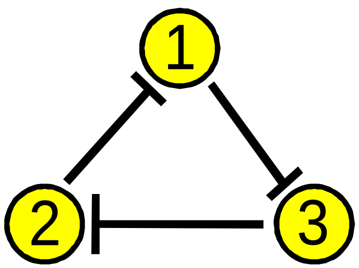
✓ the effect of space:



The deffensive alliance is successful


cycle

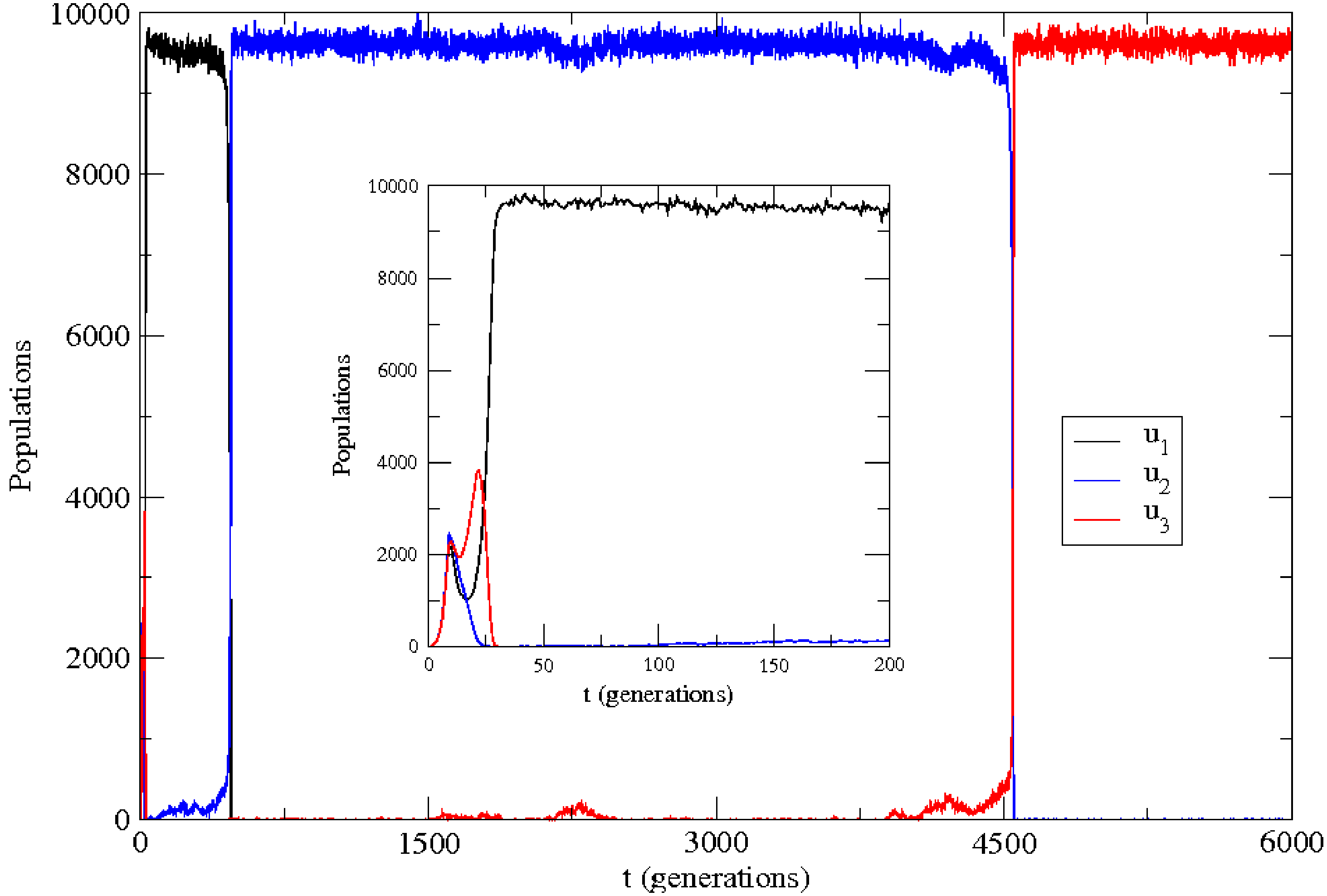
✓ Rock-Paper-Scissor



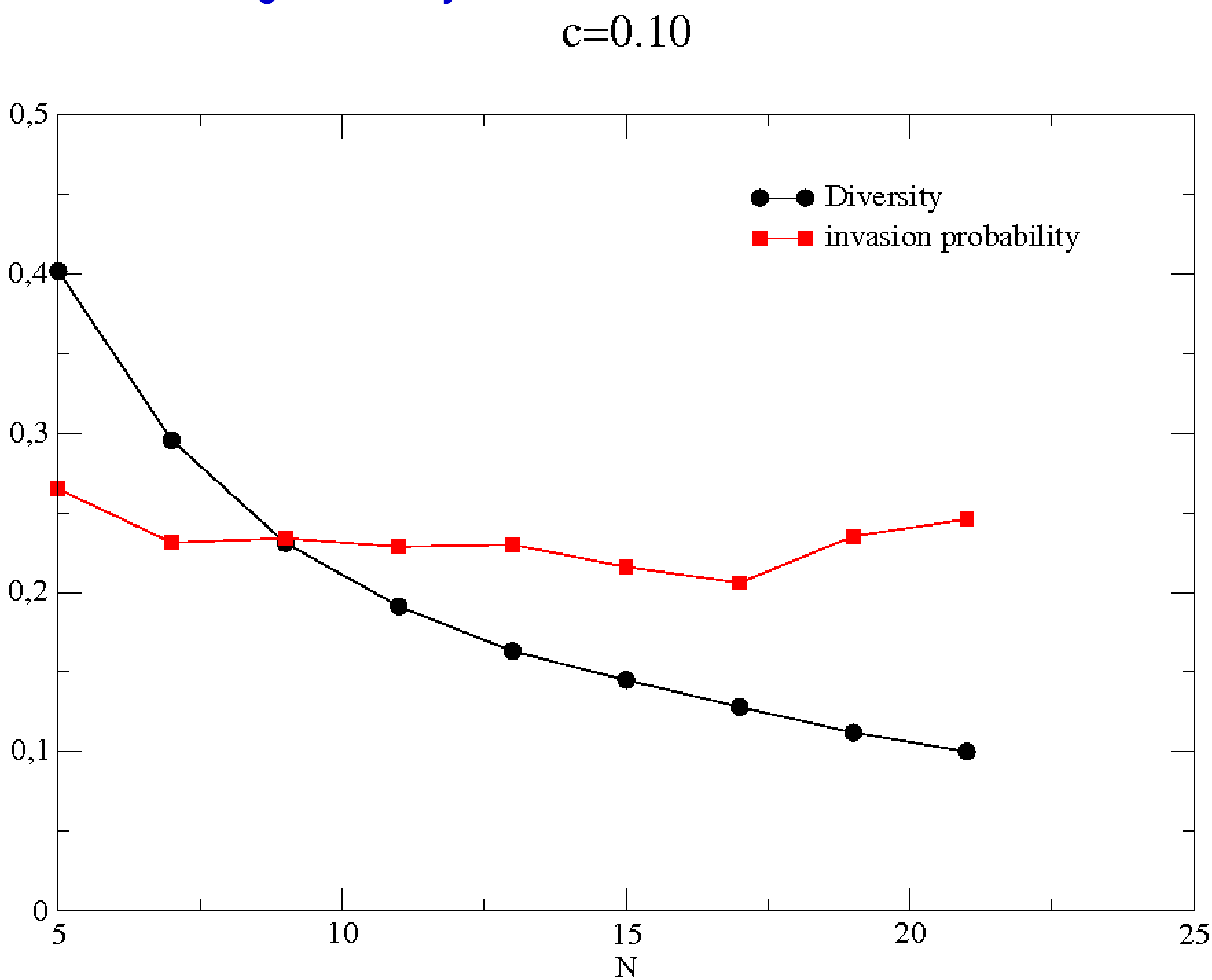
network

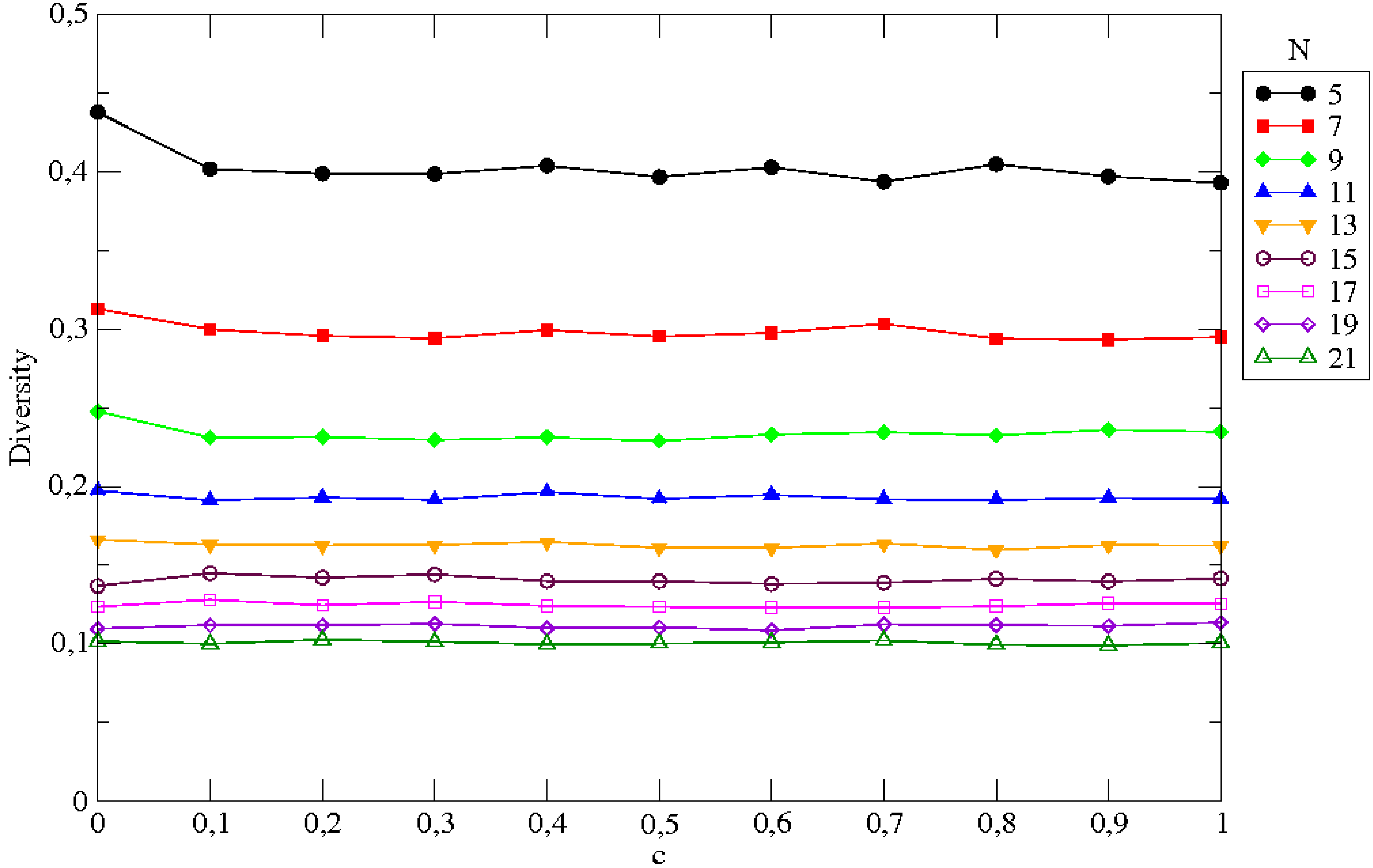
$u_1(0)=u_2(0)=u_3(0)=10$

$P_{inv3}=95\%;$
$P_{inv2}=5\%$



➢ *N*>3: decreasing diversity





0,5
0,4
0,3
0,2
0,1
0
Diversity
0
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0,5
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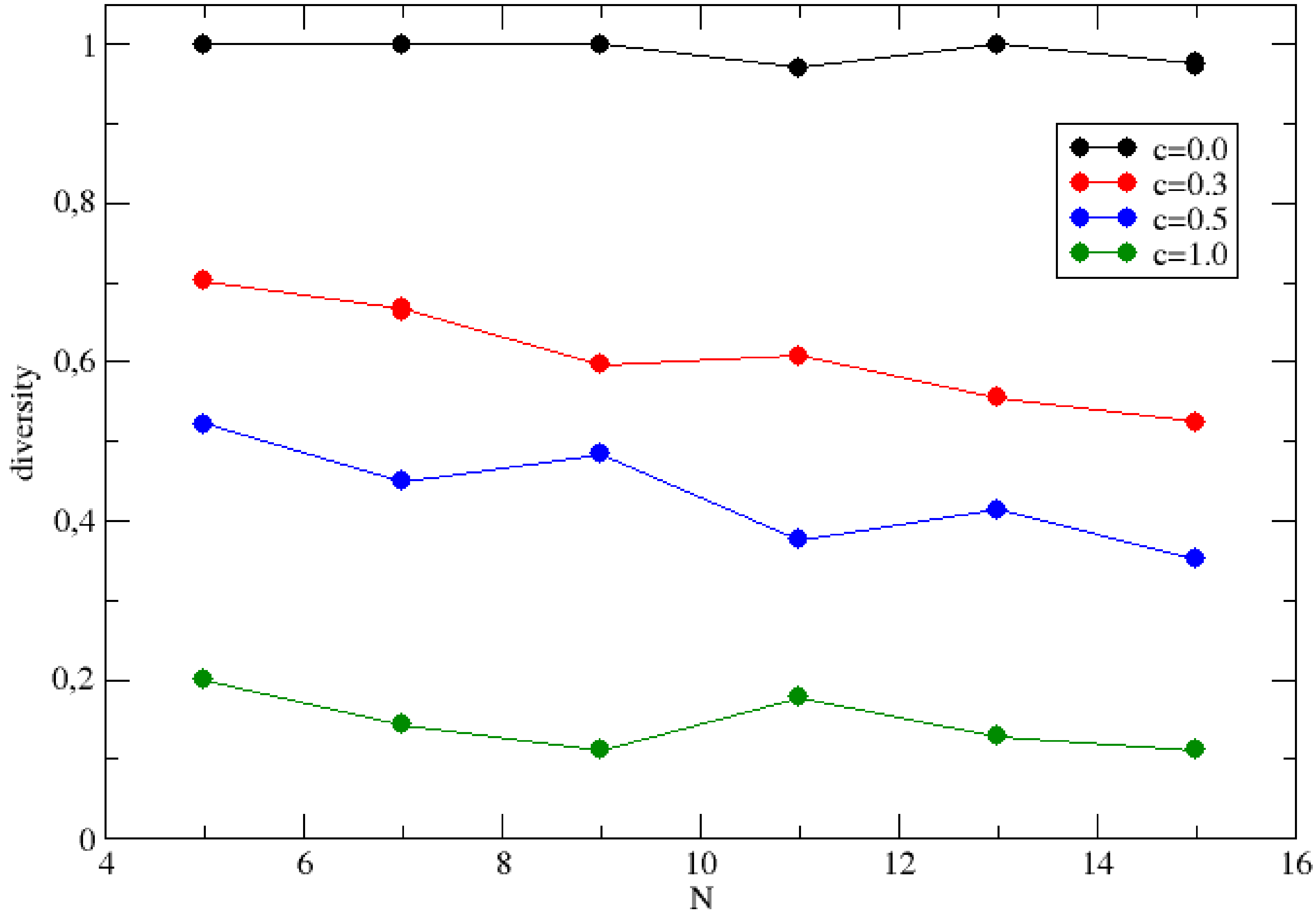
19
21

✓ Diversity in the spatially explicit model



4- Conclusions

- In random allelopathic networks, the diversity of bacteria decreases with the size of the pool of strains.
- Spatial dispersion of bacterial strains contributes to increase community diversity, but it is not sufficient.
- A self-assembly mechanism driven by correlated mutations is currently under investigation on the quest for stability and diversity.