Utrecht
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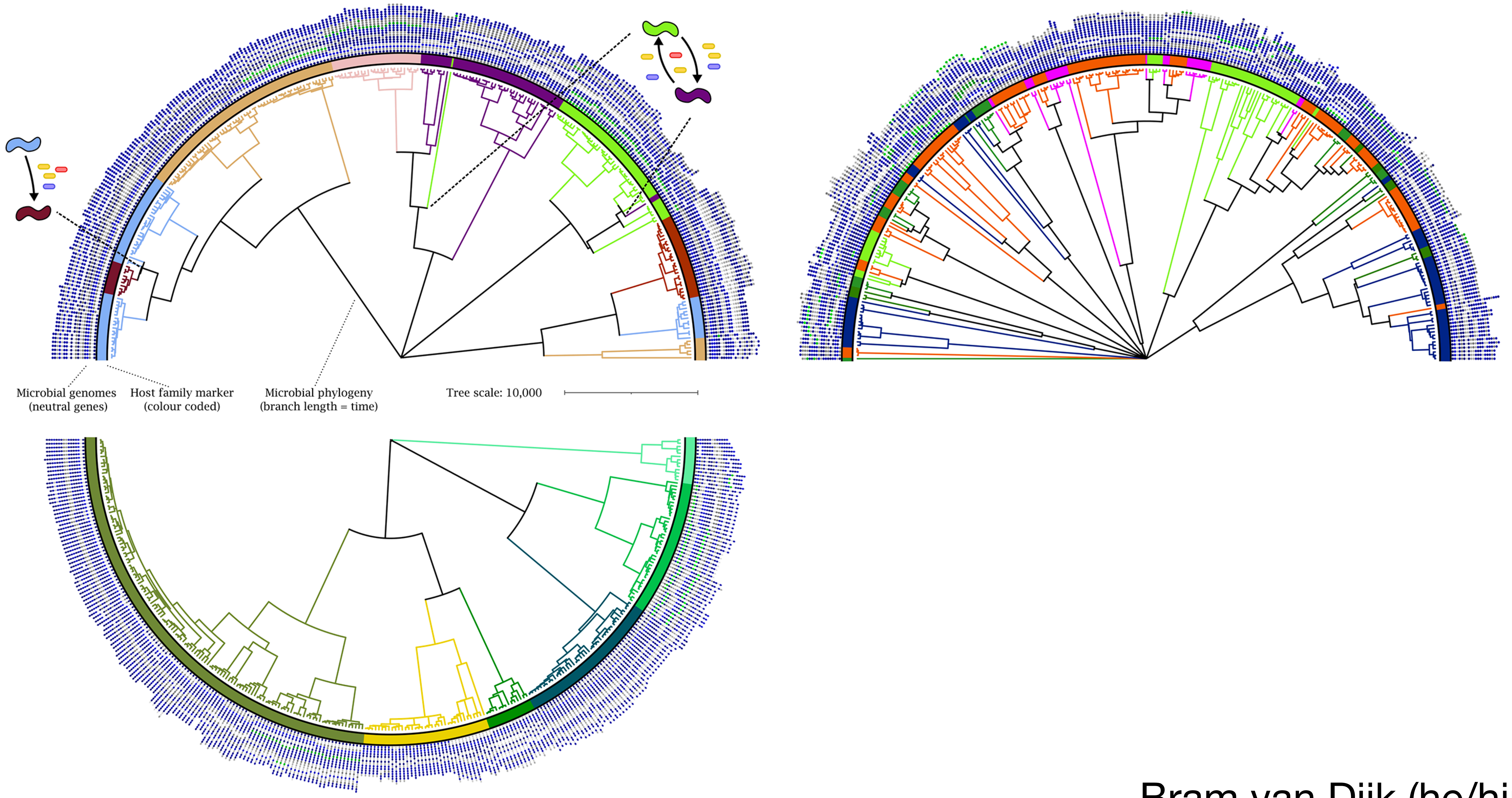
I: Horizontal transmission of genes and microbiomes

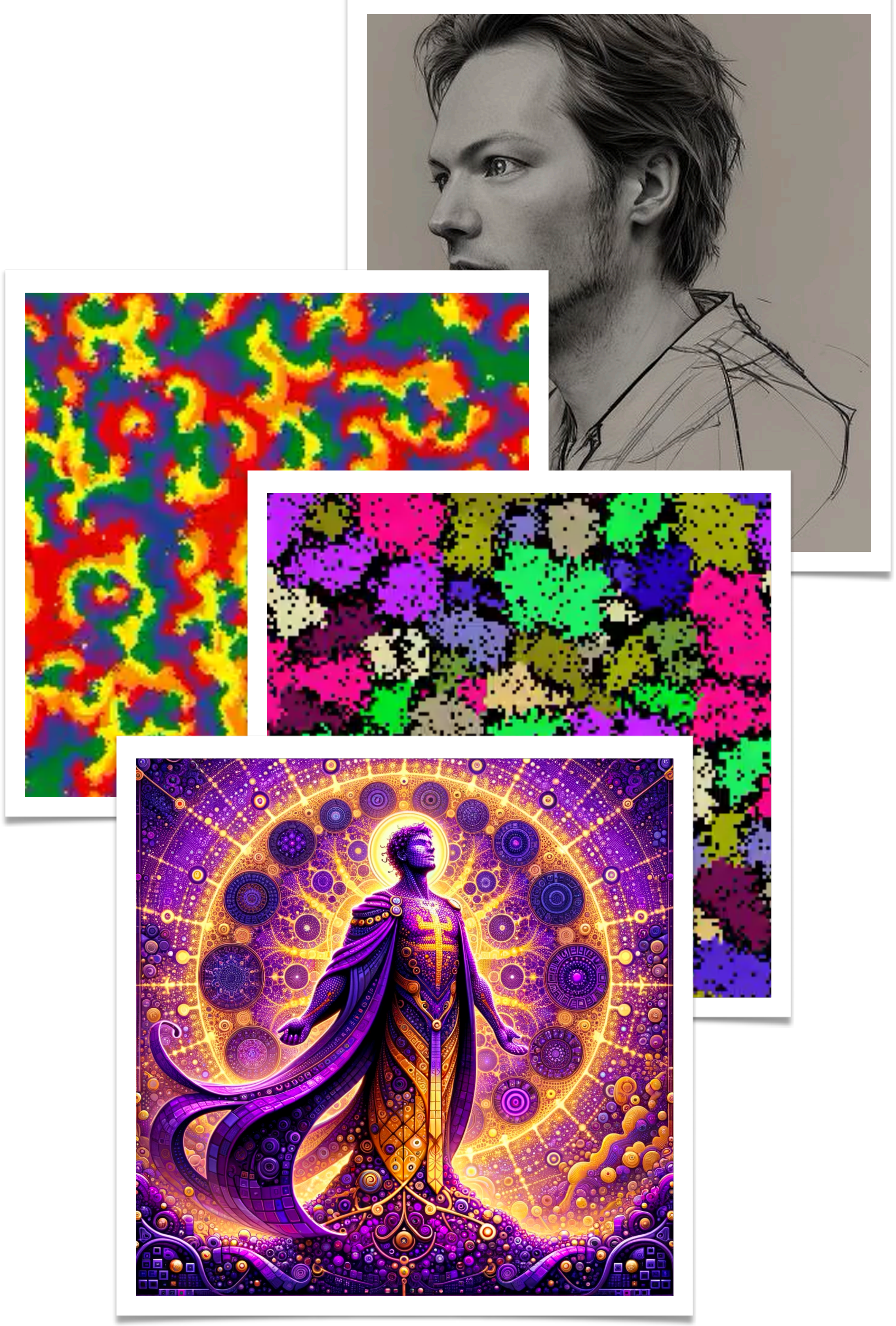


II: Black Queen dynamics and how to prevent it

Bram van Dijk (he/him)
Computational Biology - 27-02-2024

Who I am, and what I work on

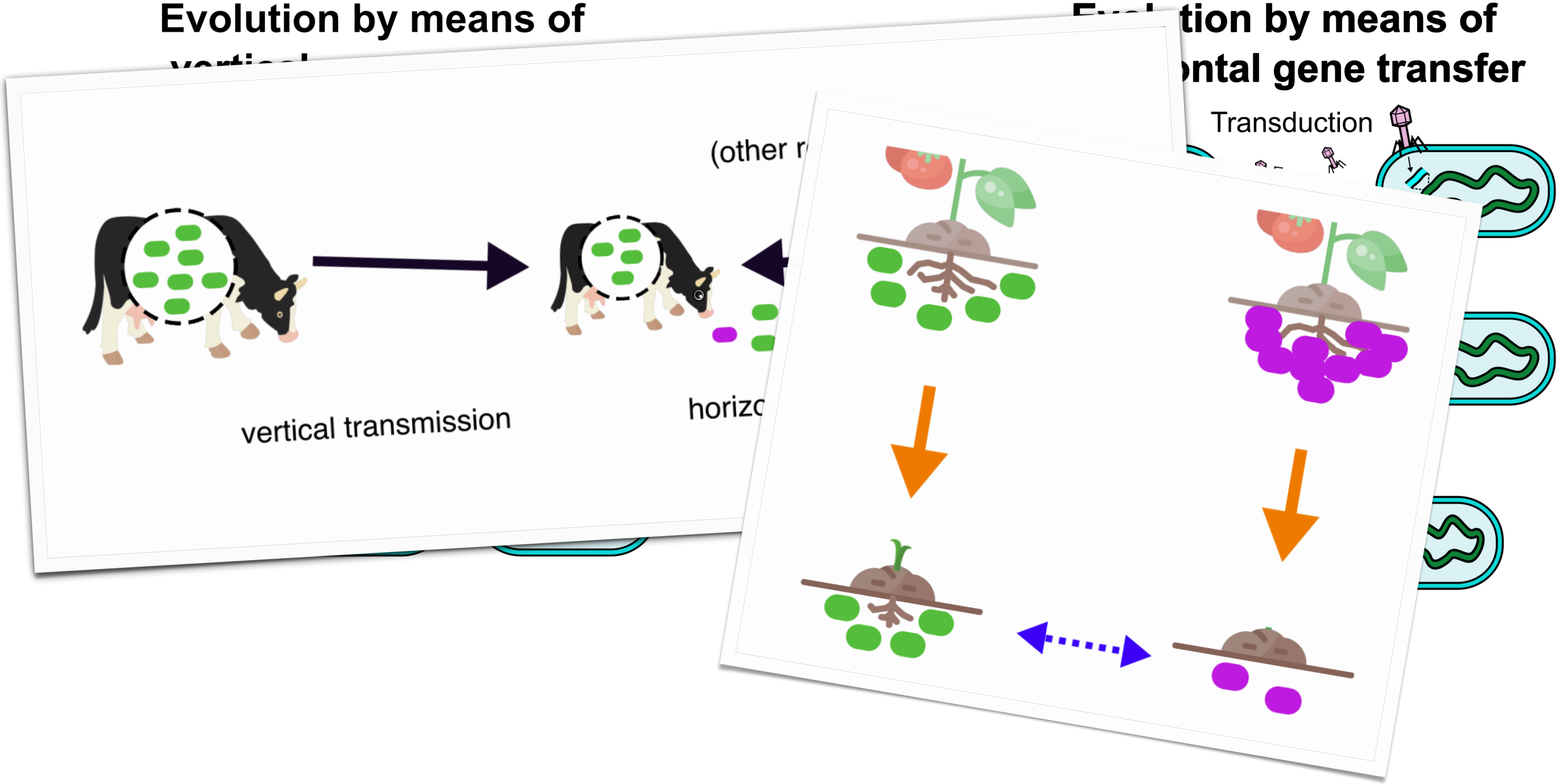
- Bram van Dijk (he/him), assistant professor at TBB (Kruyt building, N605)
- Did my PhD with Paulien (2015-2020)
- Alkmini said I shouldn't show a picture of myself
- The mother of Cacatoo 💃
- I work on **microbial ecology and evolution**
- Mostly: **spatially structured simulations** that include **more than 2 levels**
- I am looking for students! If you like this lecture: BSc (scriptie) or MSc interns very welcome!



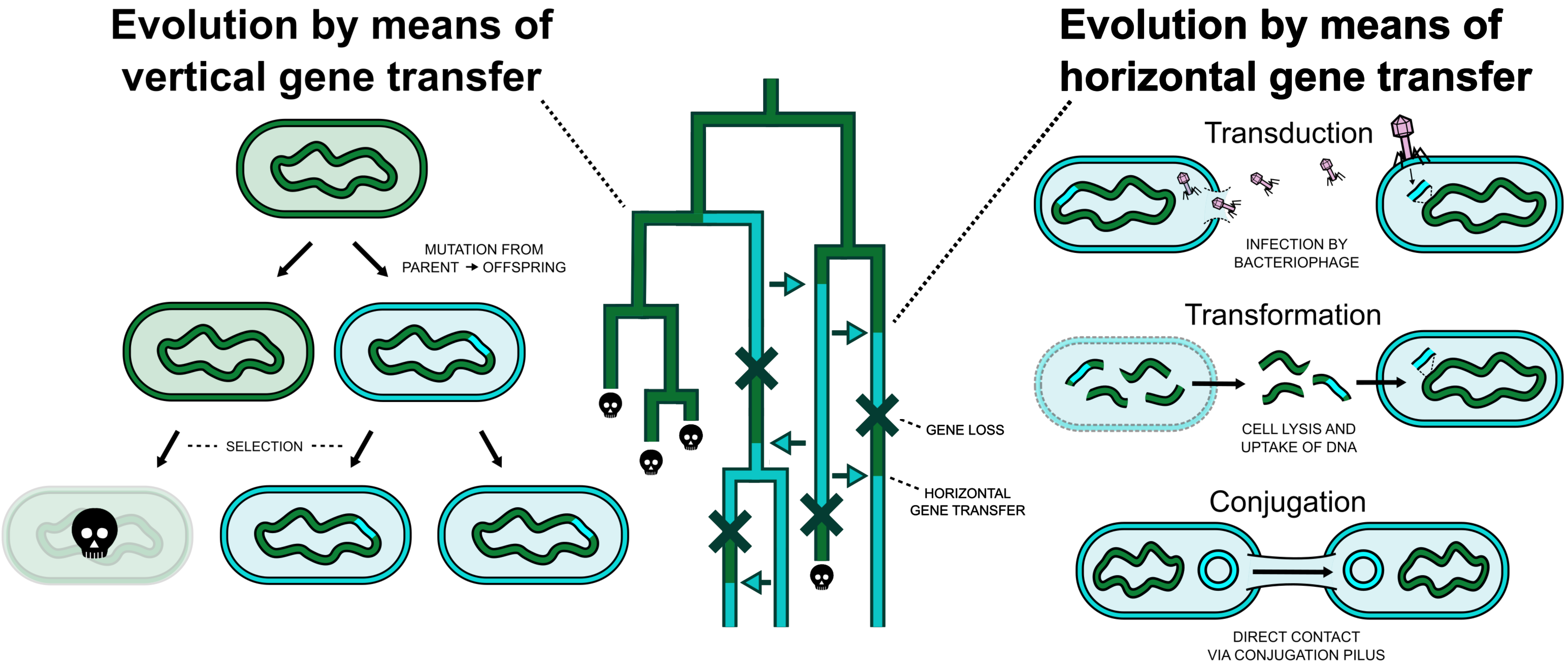
Horizontal vs. vertical transmission

Evolution by means of

Evolution by means of
ntal gene transfer



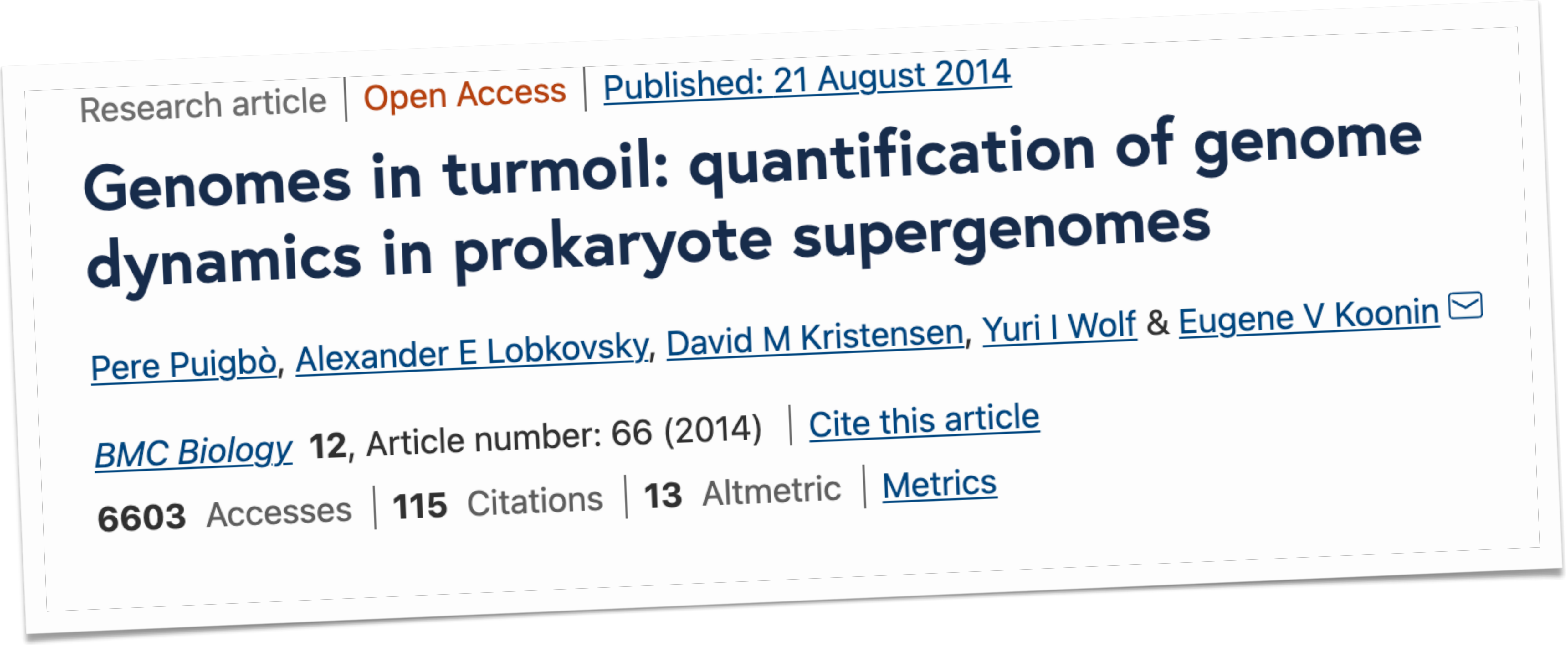
Horizontal vs. vertical **gene transfer**



“The walls that divide bacteria from one another are far from solid. Taken to extremes, the preponderance of HGT could even imply that microbiomes are better conceptualized as collections of locally adaptive genes, rather than communities of locally adapted species”

— James P.J. Hall, 2021

Taken to extremes?



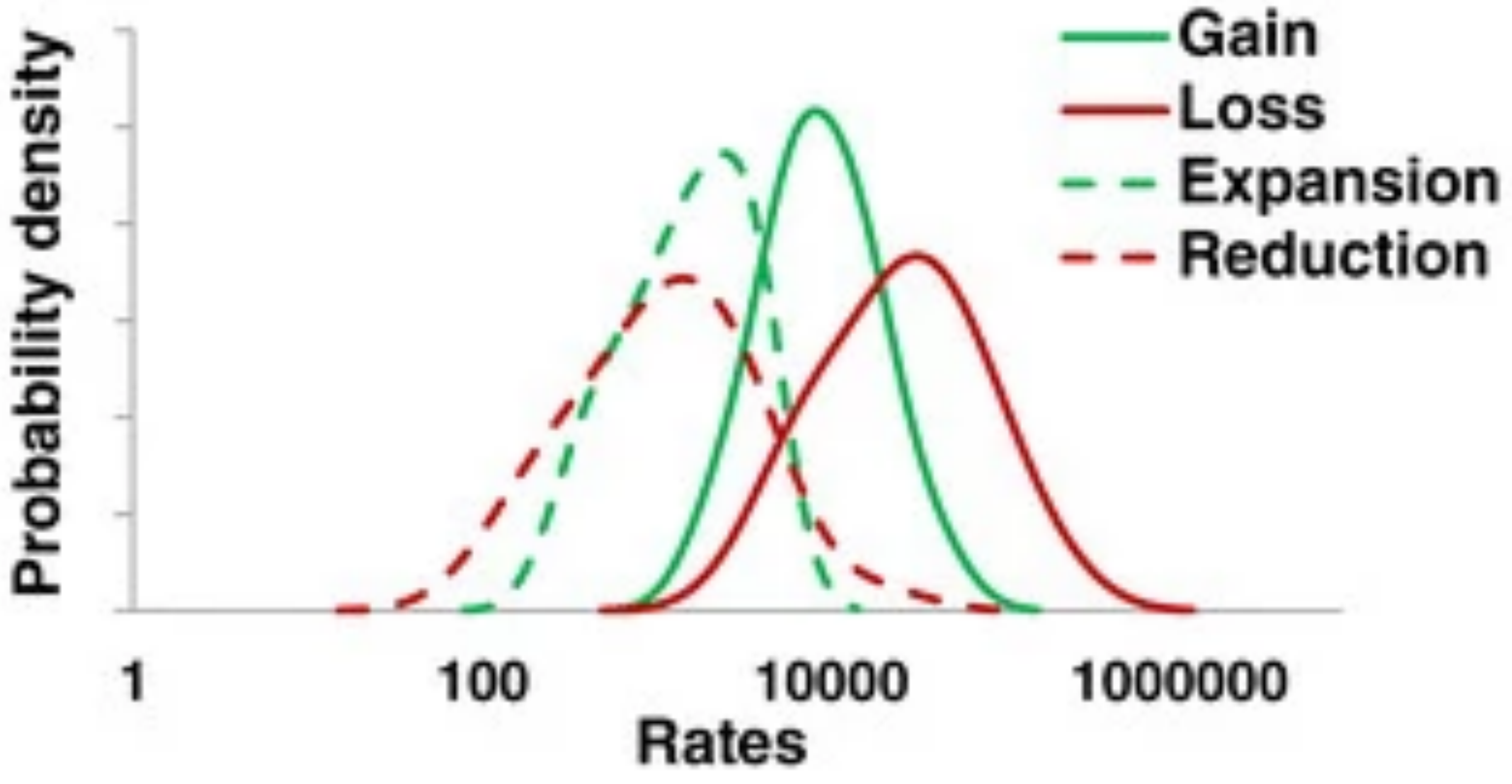
Research article | Open Access | Published: 21 August 2014

Genomes in turmoil: quantification of genome dynamics in prokaryote supergenomes

Pere Puigbò, Alexander E Lobkovsky, David M Kristensen, Yuri I Wolf & Eugene V Koonin

BMC Biology **12**, Article number: 66 (2014) | Cite this article

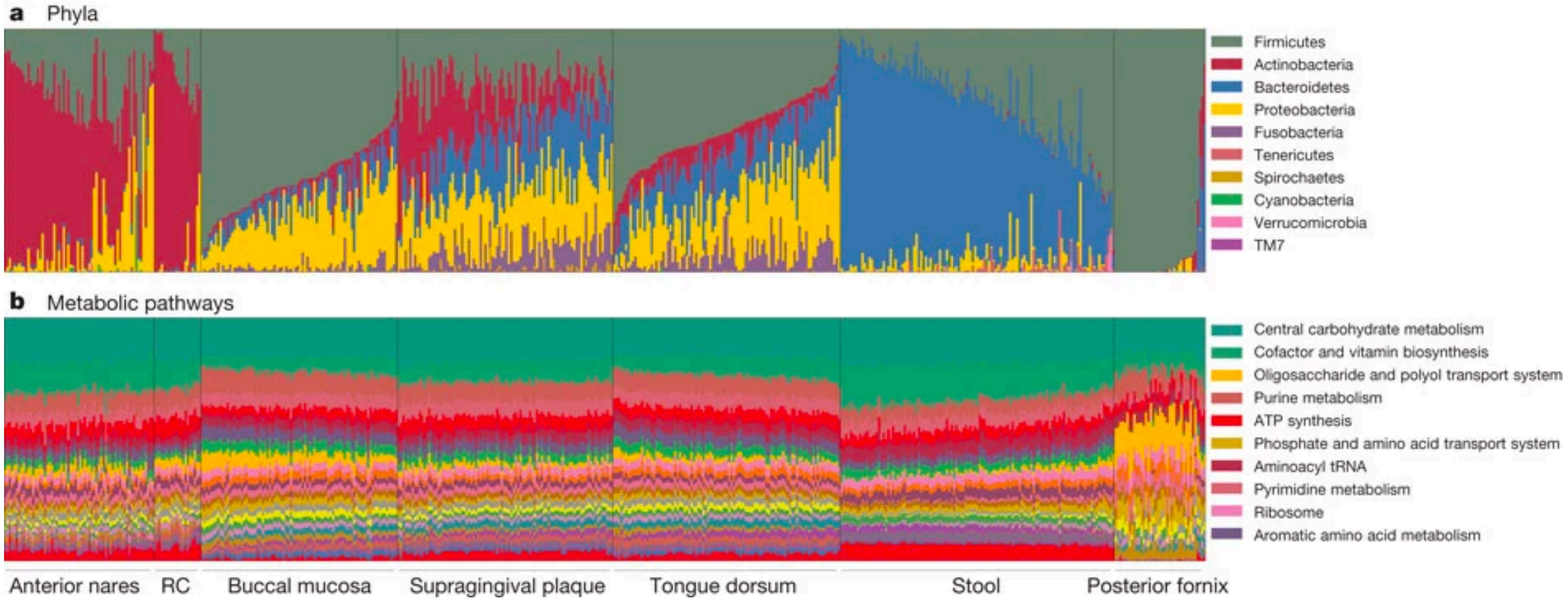
6603 Accesses | **115** Citations | **13** Altmetric | Metrics



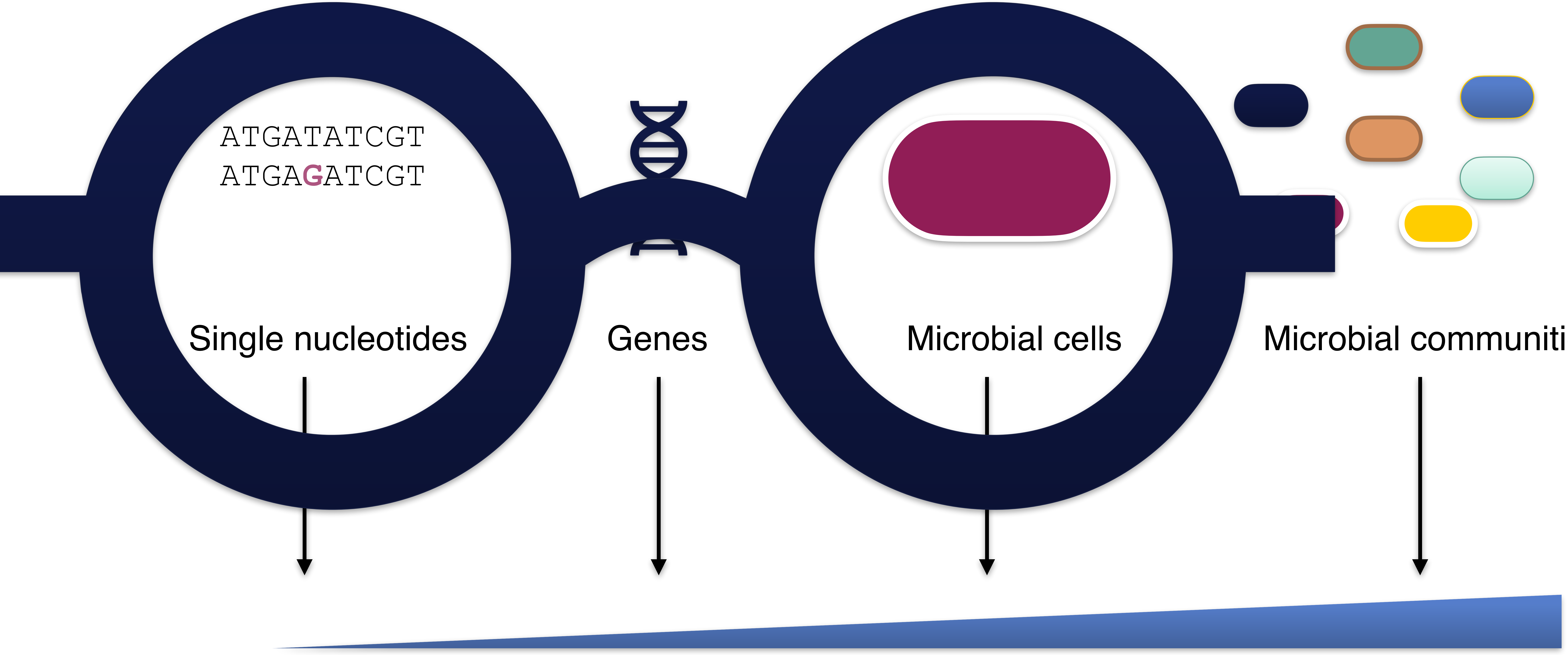
"Indeed, the estimated rates of gene family gain and loss in some groups of bacteria are such that **multiple genes appear to come and go over the time required for a single nucleotide substitution to occur** in an evolving gene. "

Conclusion: bacteria aren't "*waiting for beneficial mutations*", they are "*waiting for beneficial genes*"

Moreover: bacterial evolution is about **community gene content**, not species!



Nucleotides, genes, individuals, and communities



Single nucleotides

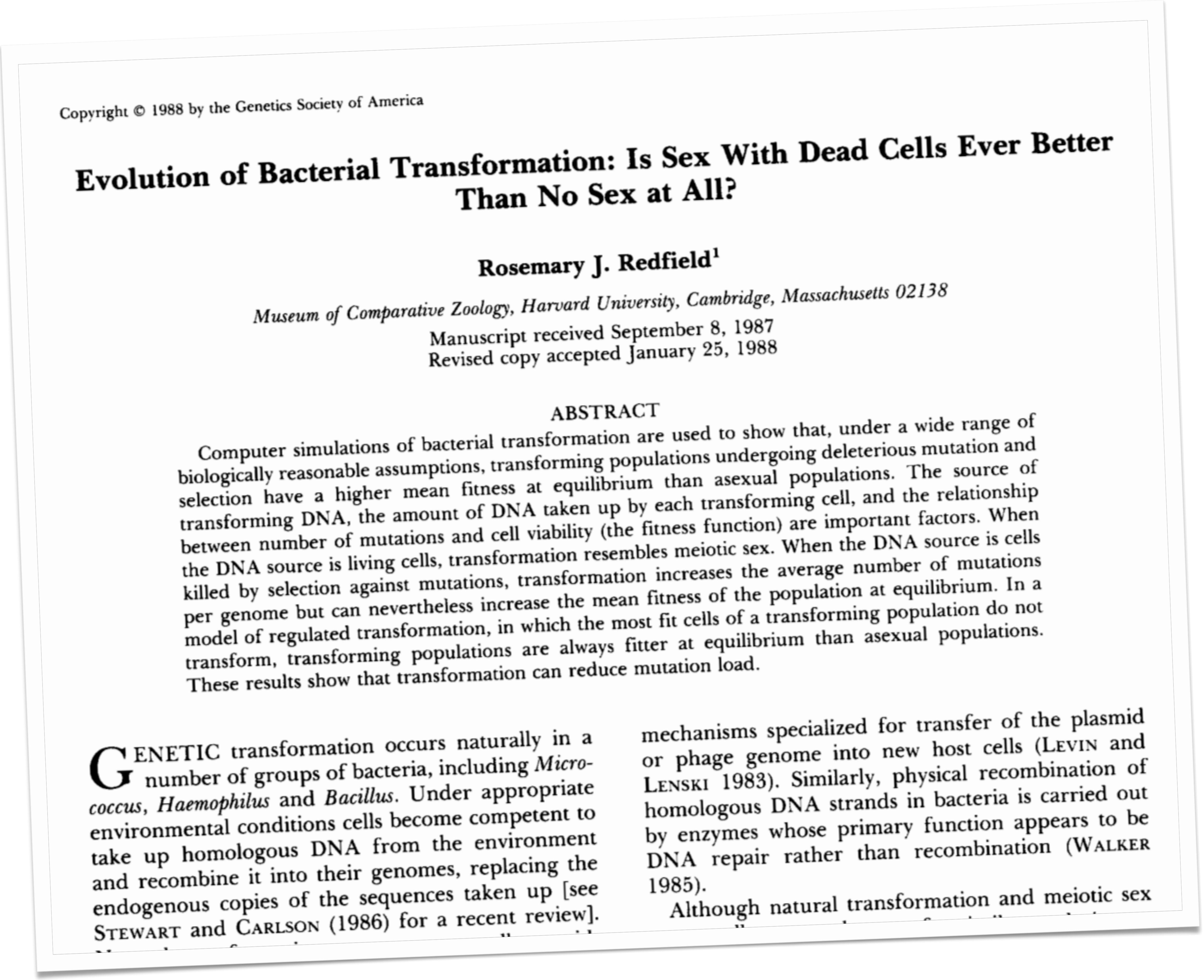
Genes

Microbial cells

Microbial communities

Increasing scale

HGT is often compared to sex, and seen as a side-effect



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Evolution of Bacterial Transformation: Is Sex With Dead Cells Ever Better Than No Sex at All?

Rosemary J. Redfield[1]

Museum of Comparative Zoology, Harvard University, Cambridge, Massachusetts 02138

Manuscript received September 8, 1987
Revised copy accepted January 25, 1988

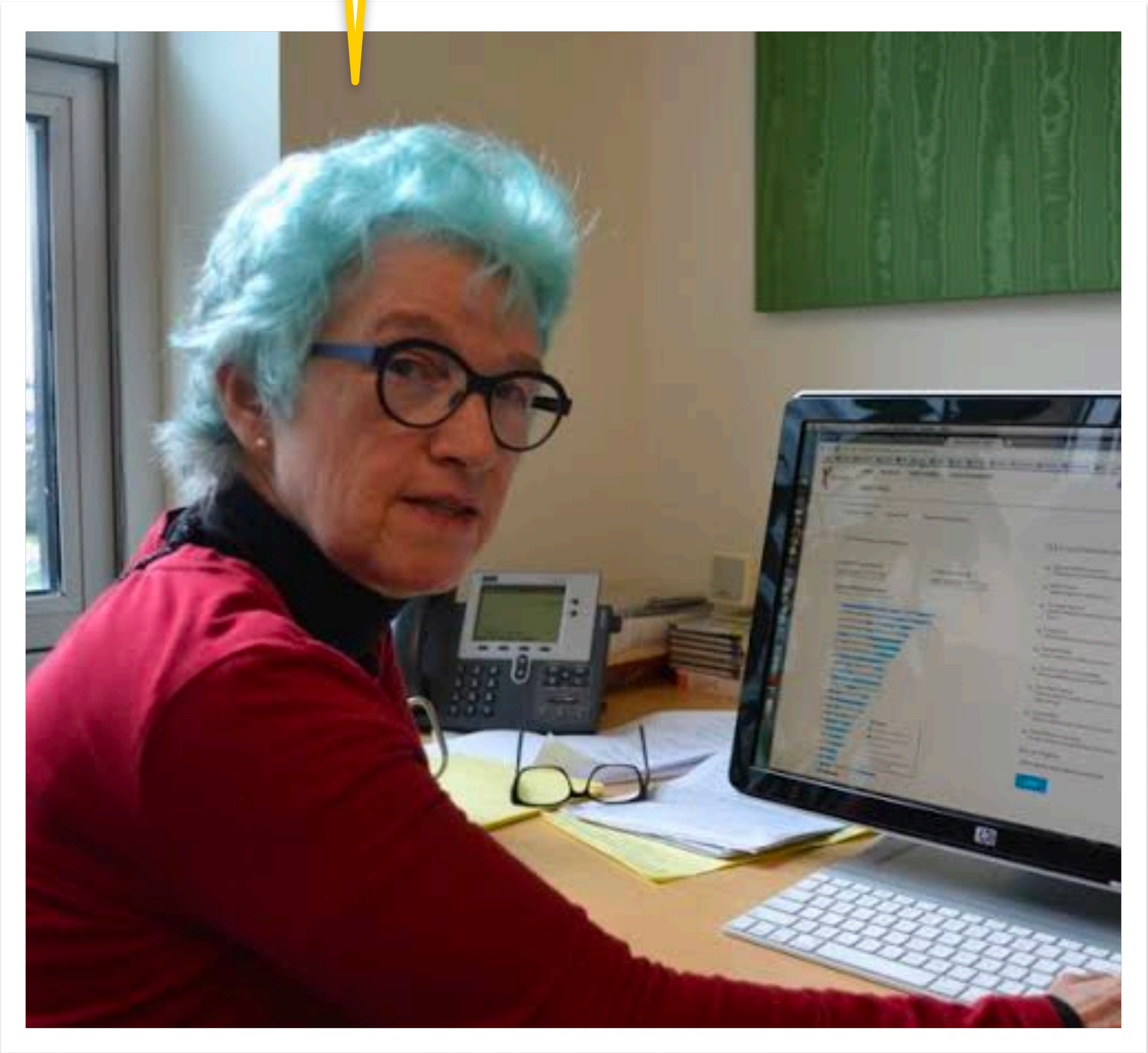
ABSTRACT

Computer simulations of bacterial transformation are used to show that, under a wide range of biologically reasonable assumptions, transforming populations undergoing deleterious mutation and selection have a higher mean fitness at equilibrium than asexual populations. The source of transforming DNA, the amount of DNA taken up by each transforming cell, and the relationship between number of mutations and cell viability (the fitness function) are important factors. When the DNA source is living cells, transformation resembles meiotic sex. When the DNA source is cells killed by selection against mutations, transformation increases the average number of mutations per genome but can nevertheless increase the mean fitness of the population at equilibrium. In a model of regulated transformation, in which the most fit cells of a transforming population do not transform, transforming populations are always fitter at equilibrium than asexual populations. These results show that transformation can reduce mutation load.

GENETIC transformation occurs naturally in a number of groups of bacteria, including *Micrococcus*, *Haemophilus* and *Bacillus*. Under appropriate environmental conditions cells become competent to take up homologous DNA from the environment and recombine it into their genomes, replacing the endogenous copies of the sequences taken up [see STEWART and CARLSON (1986) for a recent review].

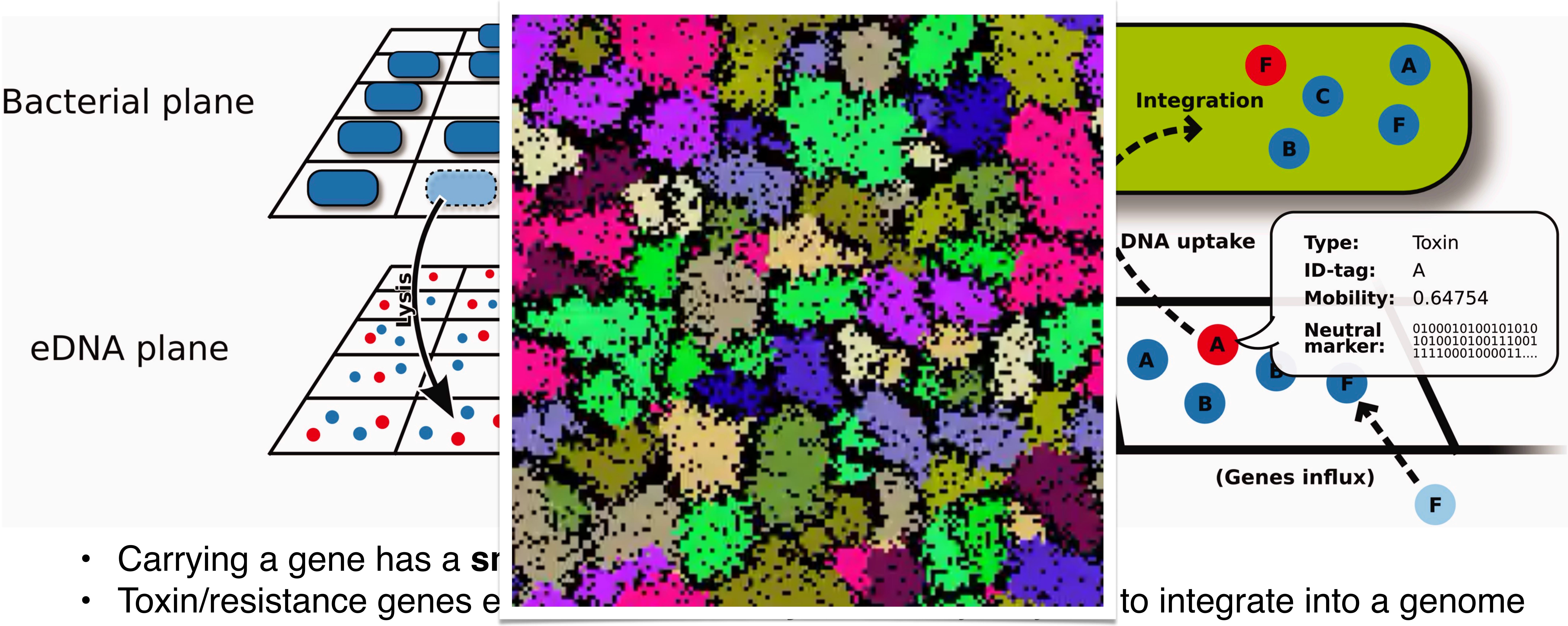
mechanisms specialized for transfer of the plasmid or phage genome into new host cells (LEVIN and LENSKI 1983). Similarly, physical recombination of homologous DNA strands in bacteria is carried out by enzymes whose primary function appears to be DNA repair rather than recombination (WALKER 1985).

Although natural transformation and meiotic sex



"HGT is just a side-effect of bacteria consuming DNA for resources. The adaptive benefits are secondary."

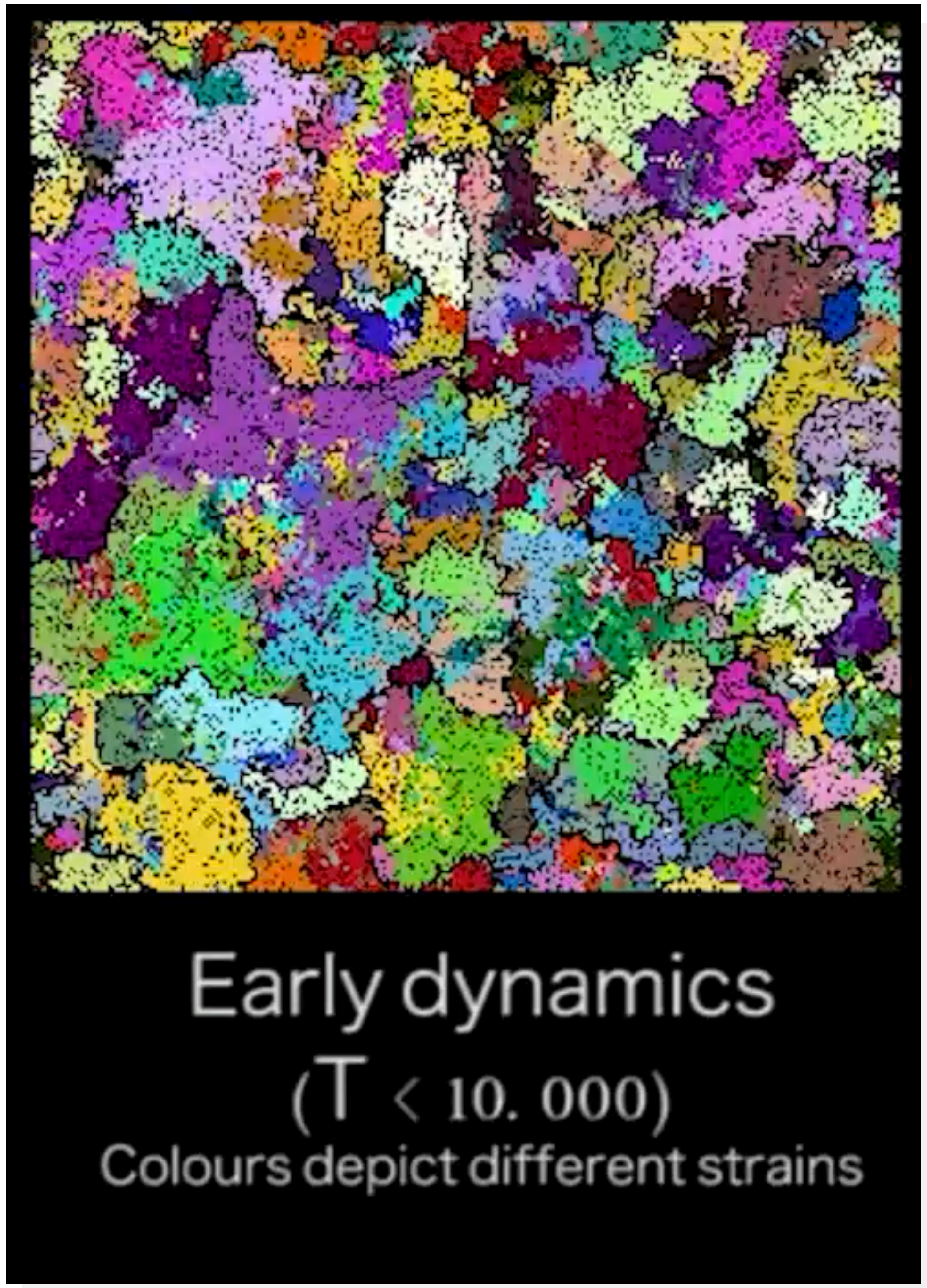
Let’s start there. Bacteria simply take up DNA.



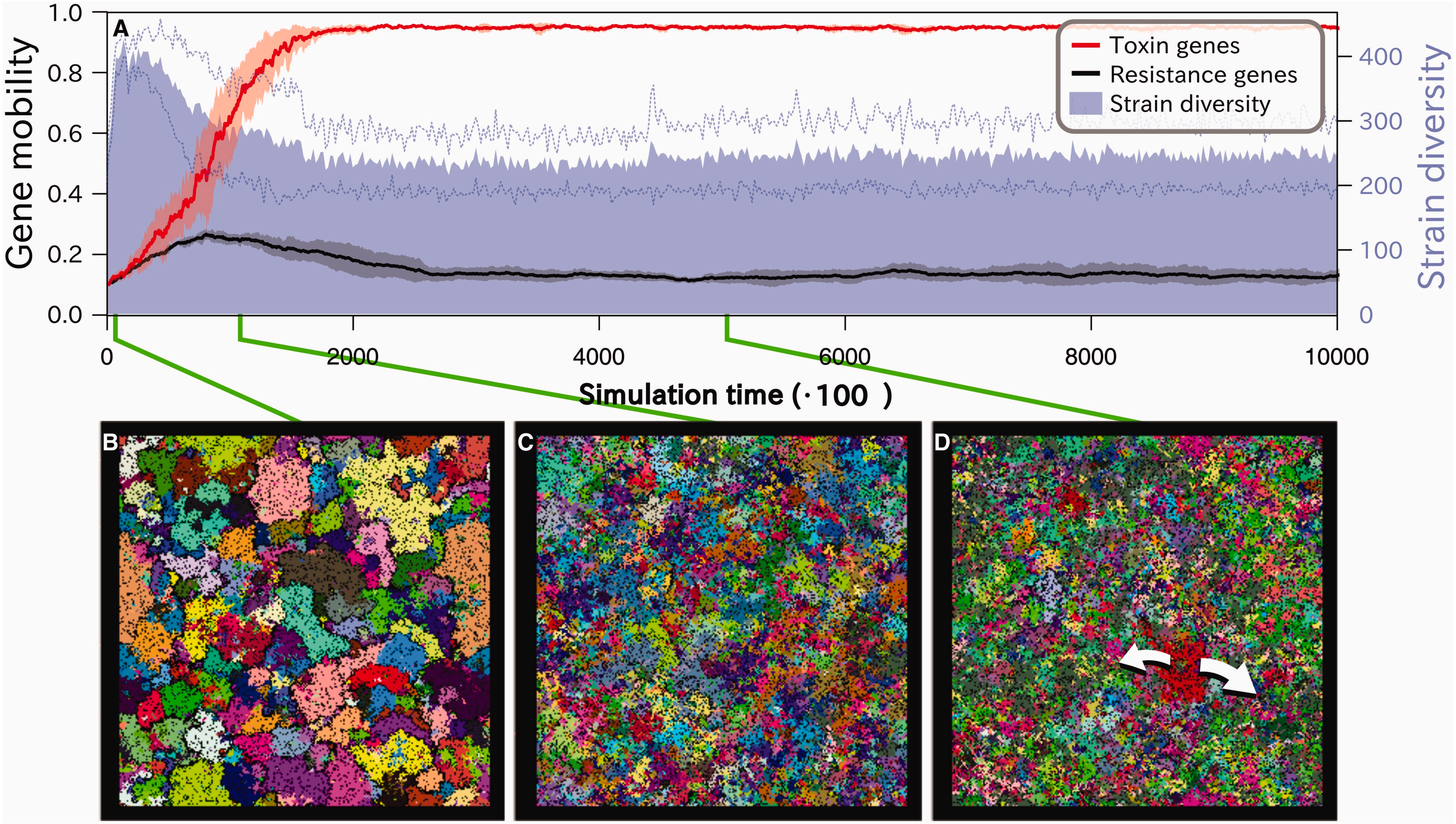
Type: Toxin
ID-tag: A
Mobility: 0.64754
Neutral marker: 0100010100101010 1010010100111001 11110001000011....

- Carrying a gene has a **s**
- Toxin/resistance genes e to integrate into a genome after uptake (whether that happens via transposon, plasmid, *etc*., we ignore for now)
- Genes can also flux into the system externally

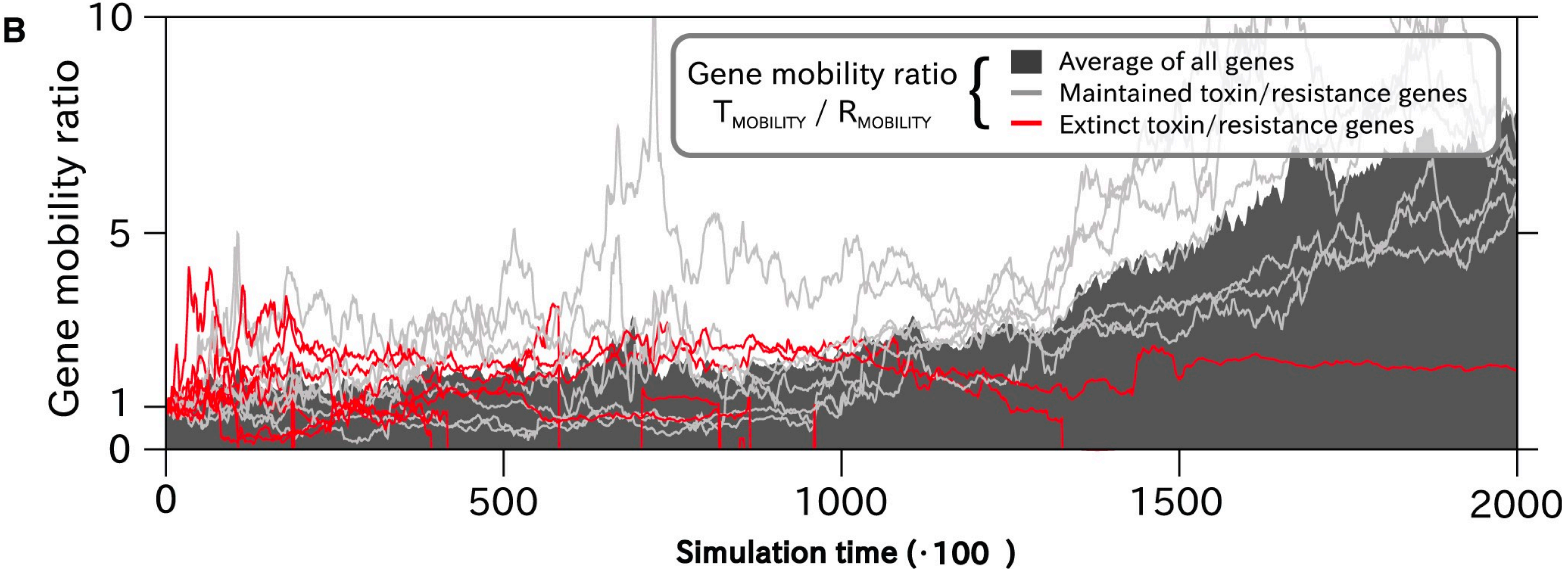
Simulating 1,000,000 time steps



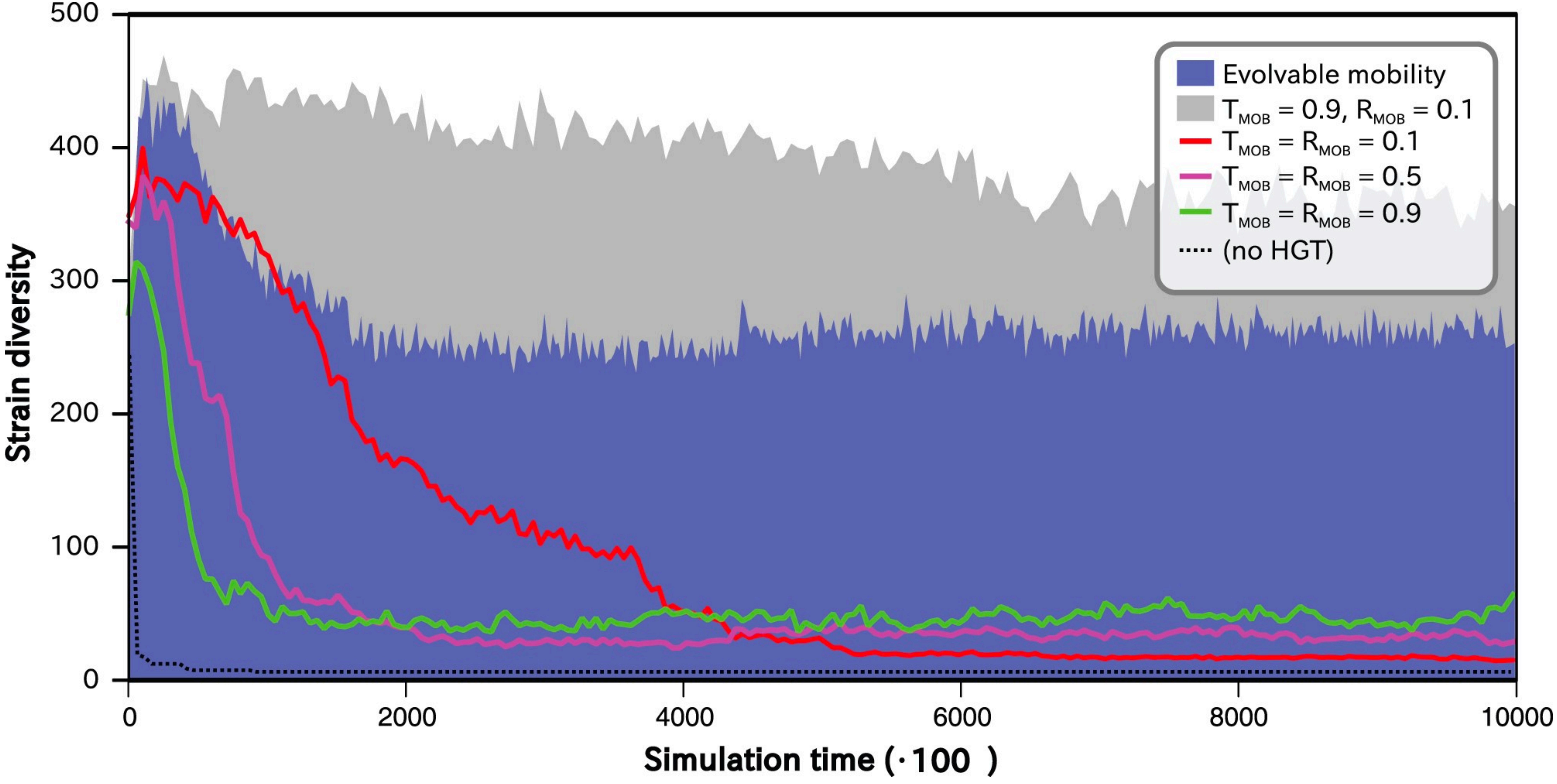
Early dynamics
(T < 10. 000)
Colours depict different strains



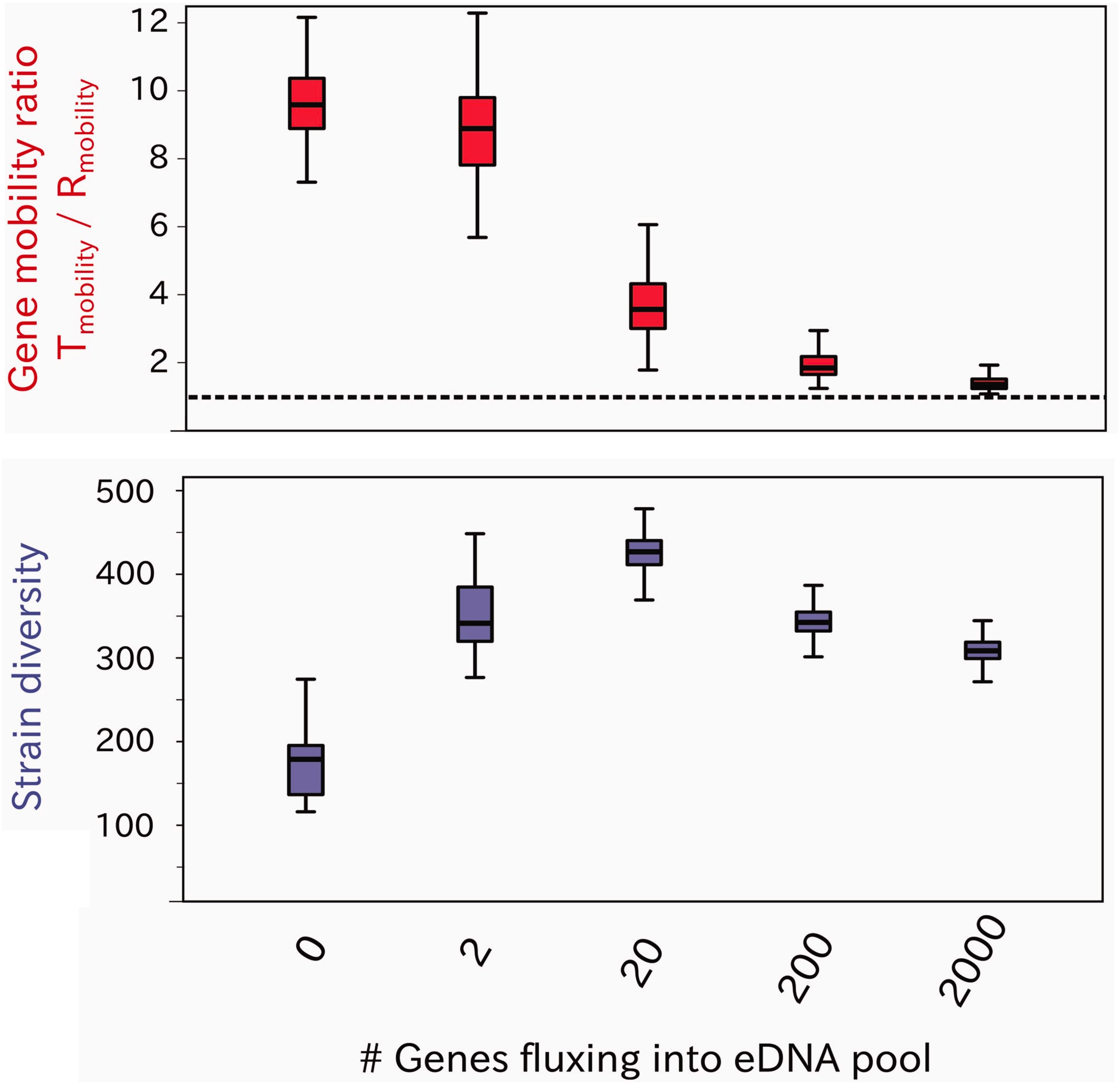
Which “sets” of toxin/antitoxin survive?

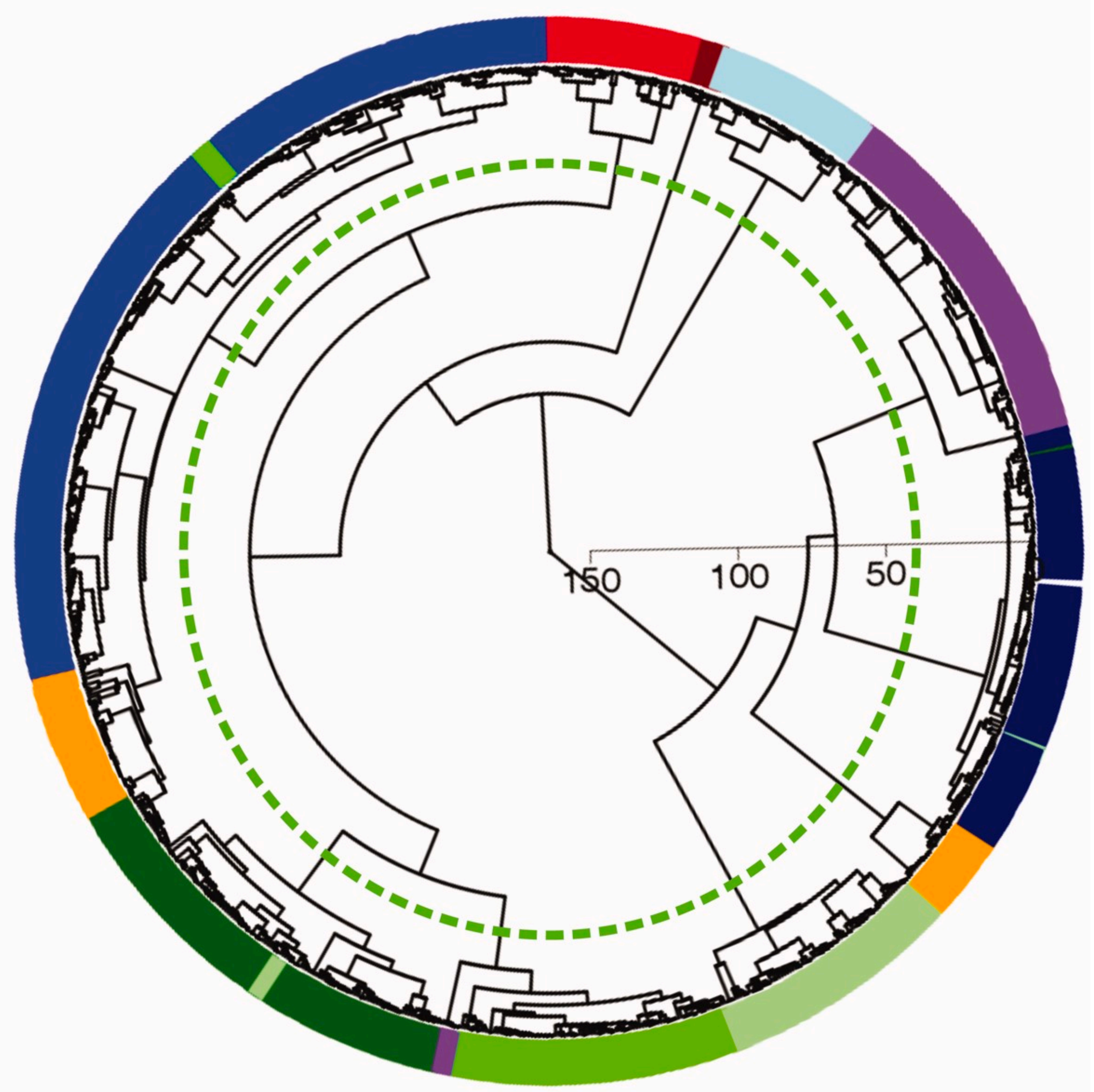


Without differential mobility, diversity crashes!

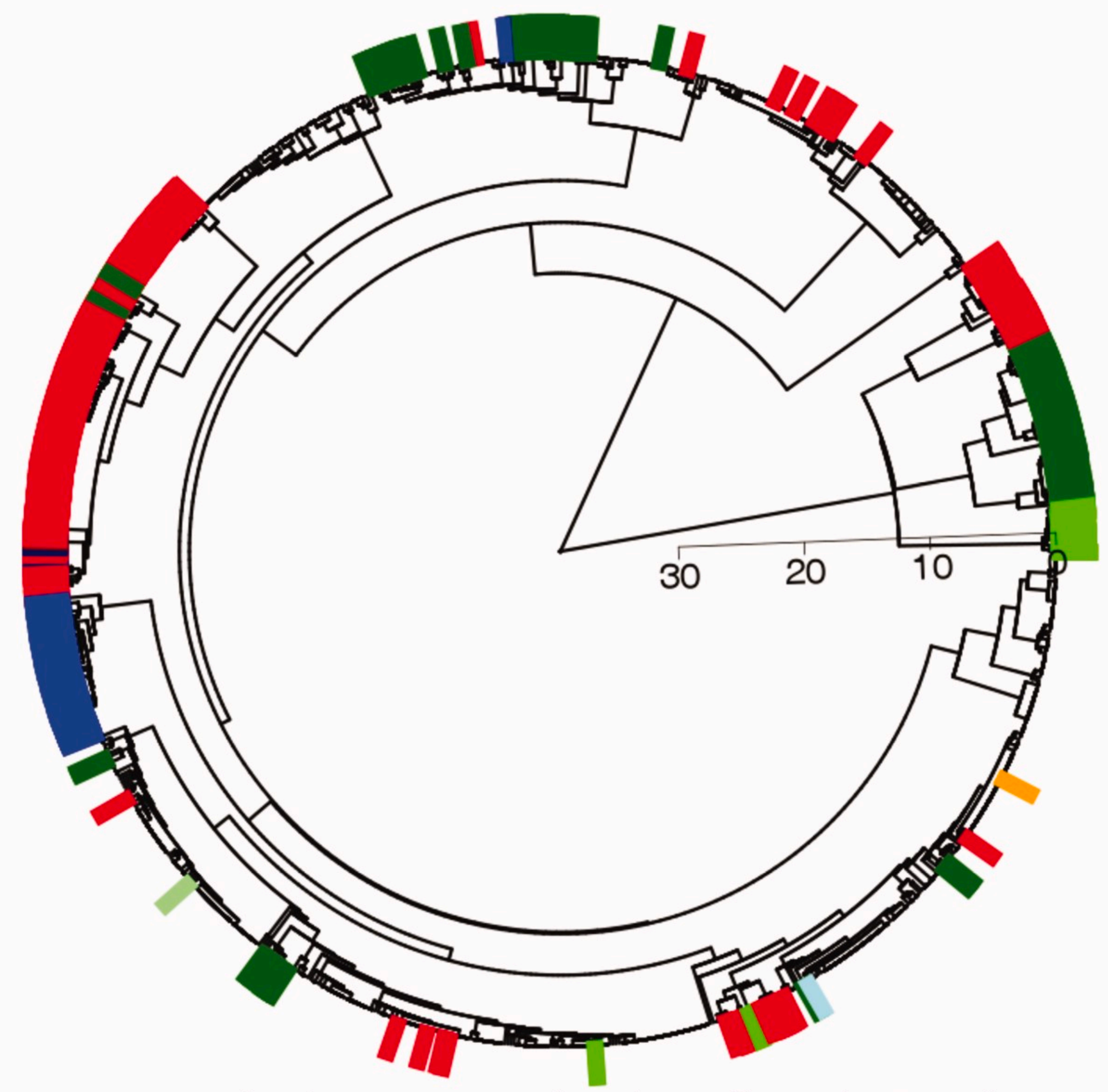


Driving up the “discovery rate” also enhanced diversity…





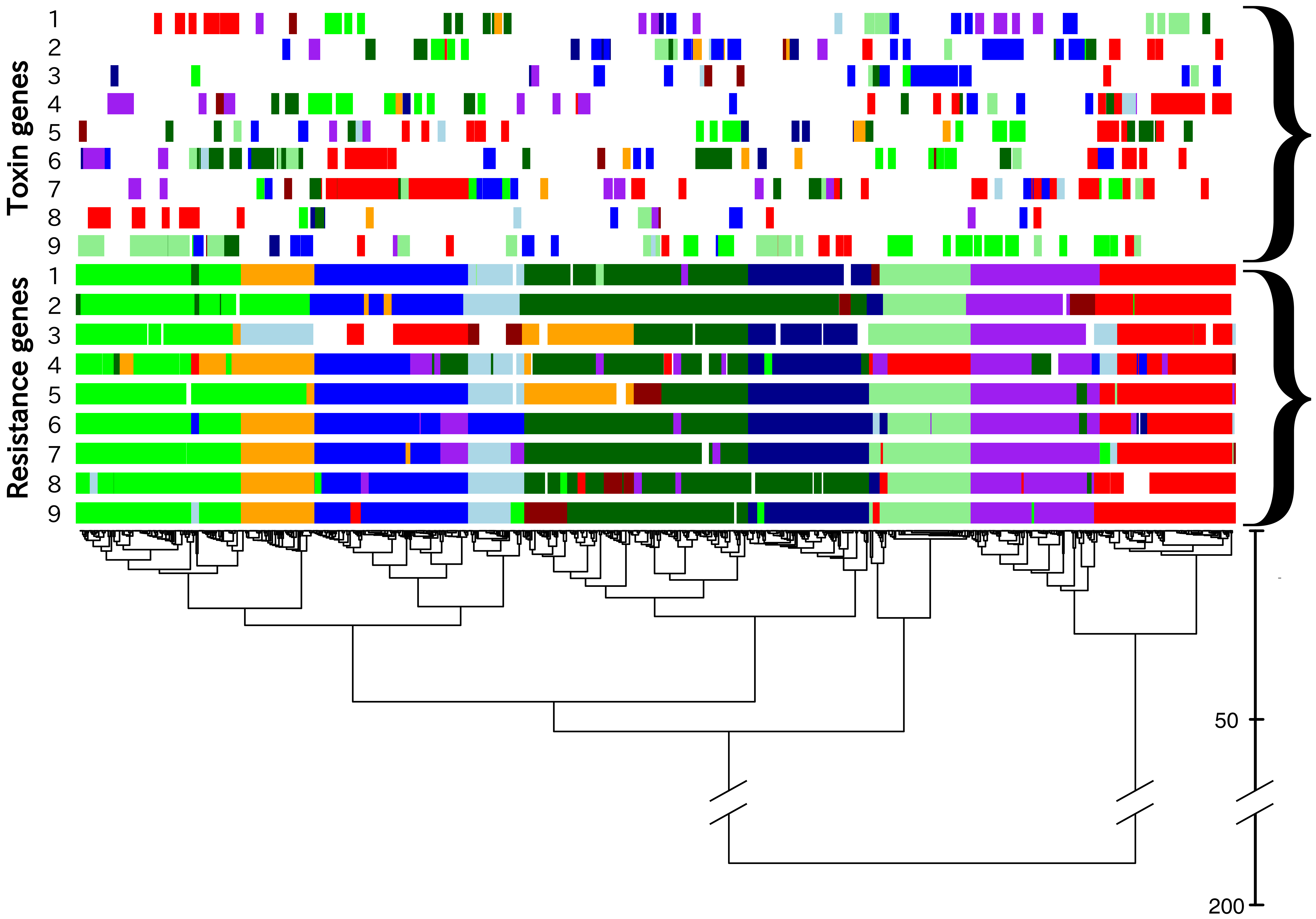
Phylogeny at low influx (2)



Phylogeny at high influx (2000)

When we keep introducing new genes,
phylogenetic diversity is lost!

Phylogenetic diversity: core- vs. accessory genomes evolved!

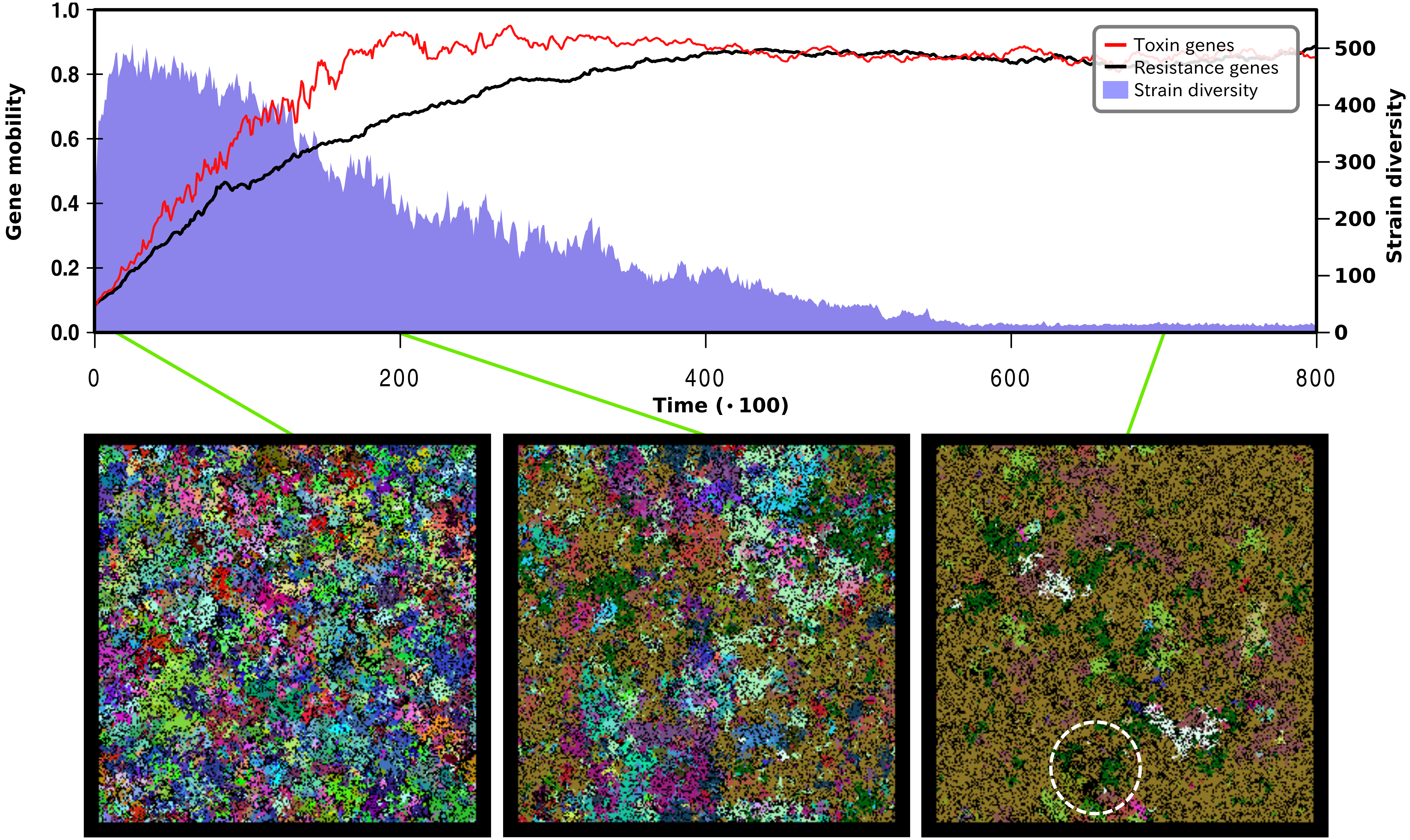


“Accessory” genome

“Core” genome

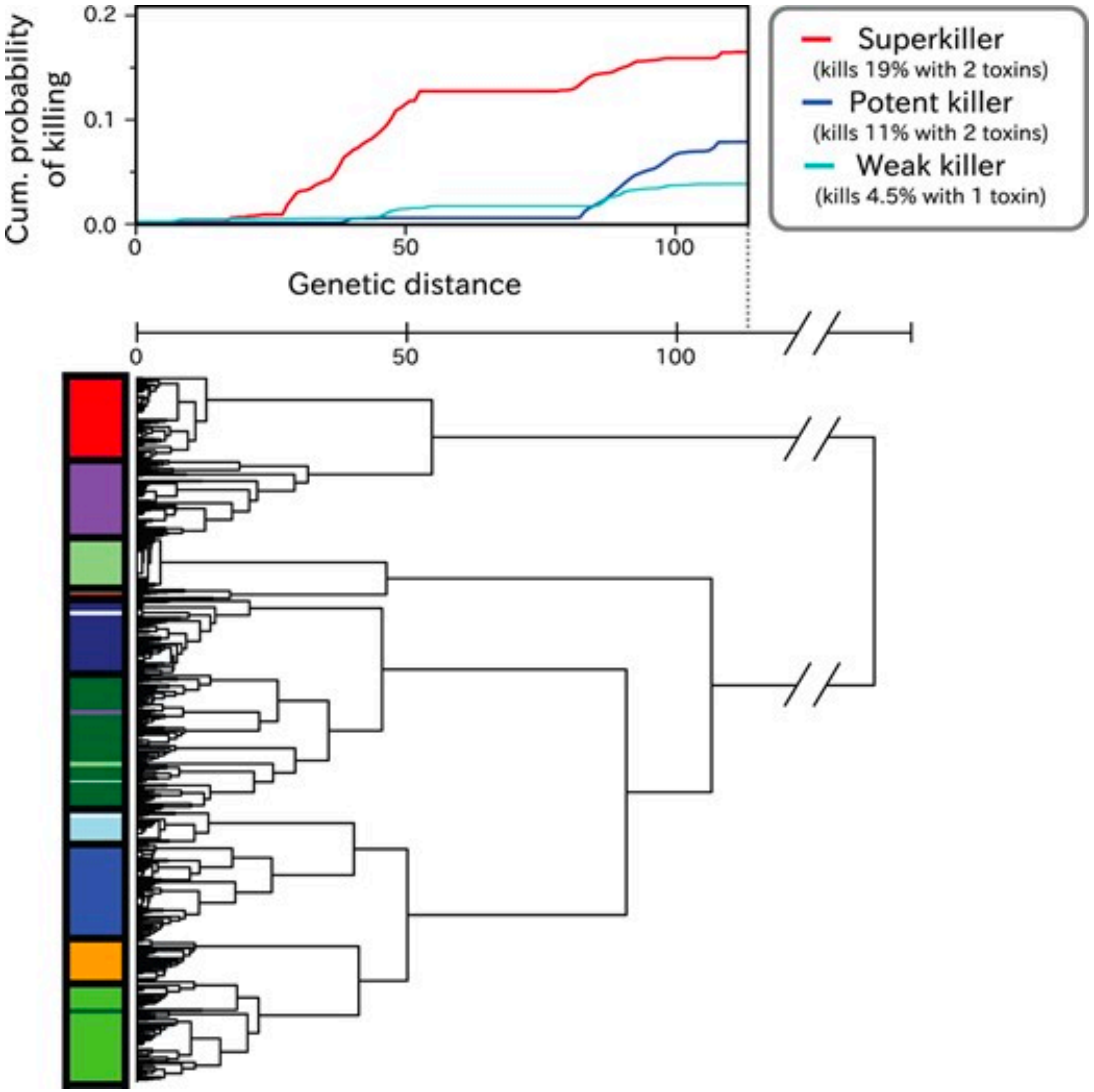
Toxins genes are best conceptualised as “sometimes-useful parasites”

Why don’t **all genes** become parasites?



Sparing close kin: without kin-recognition!

- In nature, bacteria often "spare" close kin from killing
- The model does not include kin recognition: **they don't know who they are killing**
- Through the interplay of local interactions and pattern formation, however, they end up "sparing close kin" anyway
- This system is maintained due to the toxin genes transferring more frequently than their corresponding resistance genes!



Conclusions so far

On the cell-level:

- Despite HGT driving a lot of “genetic mixing”, that doesn’t mean everyone becomes the same.
- Concepts like “individuality" and “species" still persist for bacteria, but the gene- and group-level are equally (or more!) important

On the gene-level:

- Mobile genes are Darwinian entities themselves, and **evolve towards “parasitism” if they get the chance**!
- However: a feedback with the local environment prevents this from getting out of control!
- Toxin genes can get away with being parasites in the short term, because they are **occasionally beneficial**

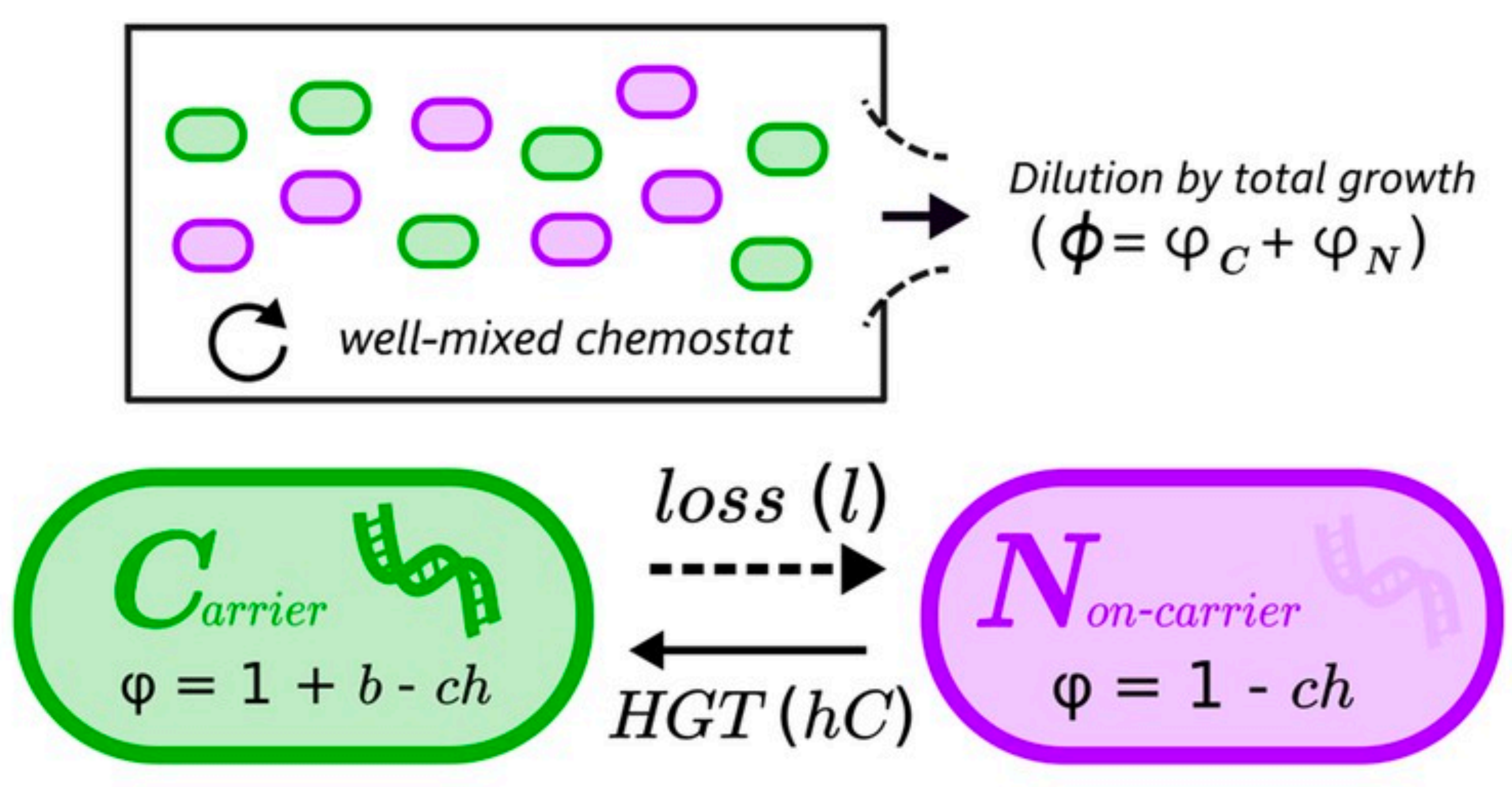
Genes evolve to play nice… or not. What do the **cells** want?

"HGT is just a side-effect of bacteria consuming DNA for resources. The adaptive benefits are secondary."

- Toxin genes give major benefits **under the right circumstances**.
- Toxin genes cost energy when not used
- On average, I measured them to be "slightly beneficial" in the model
- Do the cells then "want" to take up toxin genes?
- **No**. No they don't. But is that because:
  - A) HGT is not adaptive for the cells, or
  - B) Taking up toxin genes can be **extra** bad

Slightly beneficial genes

A) ODE model (carriers and non-carriers)



Q: How does HGT impact the population growth rate?

$$\frac{dC}{dt} = \underbrace{(1-ch+b)C}_{\text{reproduction of C}} - \underbrace{lC}_{\text{gene loss}} + \underbrace{hCN}_{\text{HGT}} - \underbrace{\phi C}_{\text{chemostat}}$$

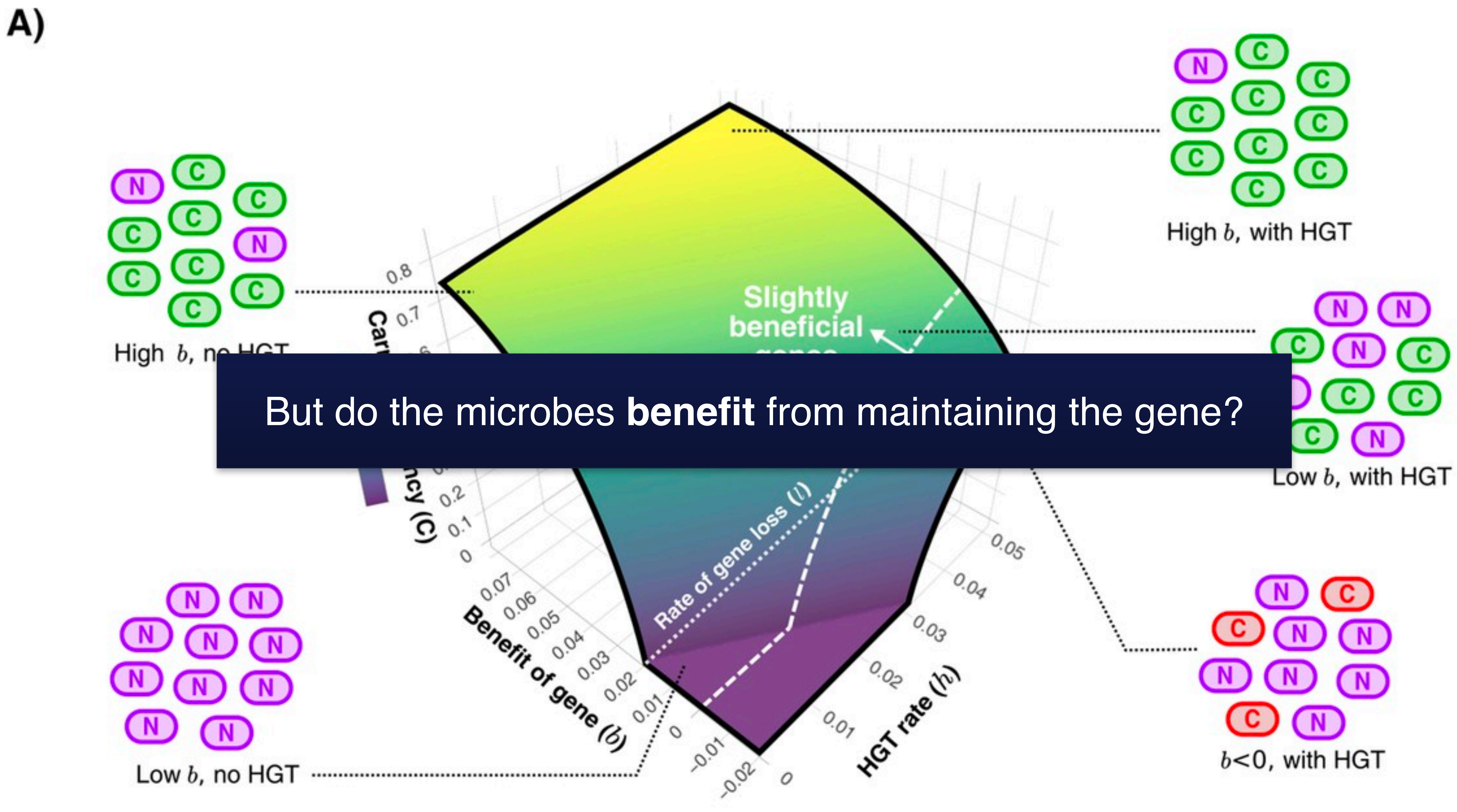
$$\frac{dN}{dt} = \underbrace{(1-ch)N}_{\text{reproduction of N}} + \underbrace{lC}_{\text{gene loss}} - \underbrace{hCN}_{\text{HGT}} - \underbrace{\phi N}_{\text{chemostat}}$$

$$\phi = \underbrace{(1-ch+b)C}_{\text{total growth of C}} + \underbrace{(1-ch)N}_{\text{total growth of N}}$$

$$C + N = 1 \text{ (constant population size, ensured by chemostat assumption.)}$$

- As a proof-of-principle, we assume **HGT has a cost** (uptake of DNA, transfer machinery, *etc.*) rather than giving it a direct benefit for cells. (A <u>hard-case</u> for adaptive benefits of HGT!)
- How does this **costly** HGT impact growth rates?

Maintaining beneficial genes depends on parameters



But do the microbes **benefit** from maintaining the gene?

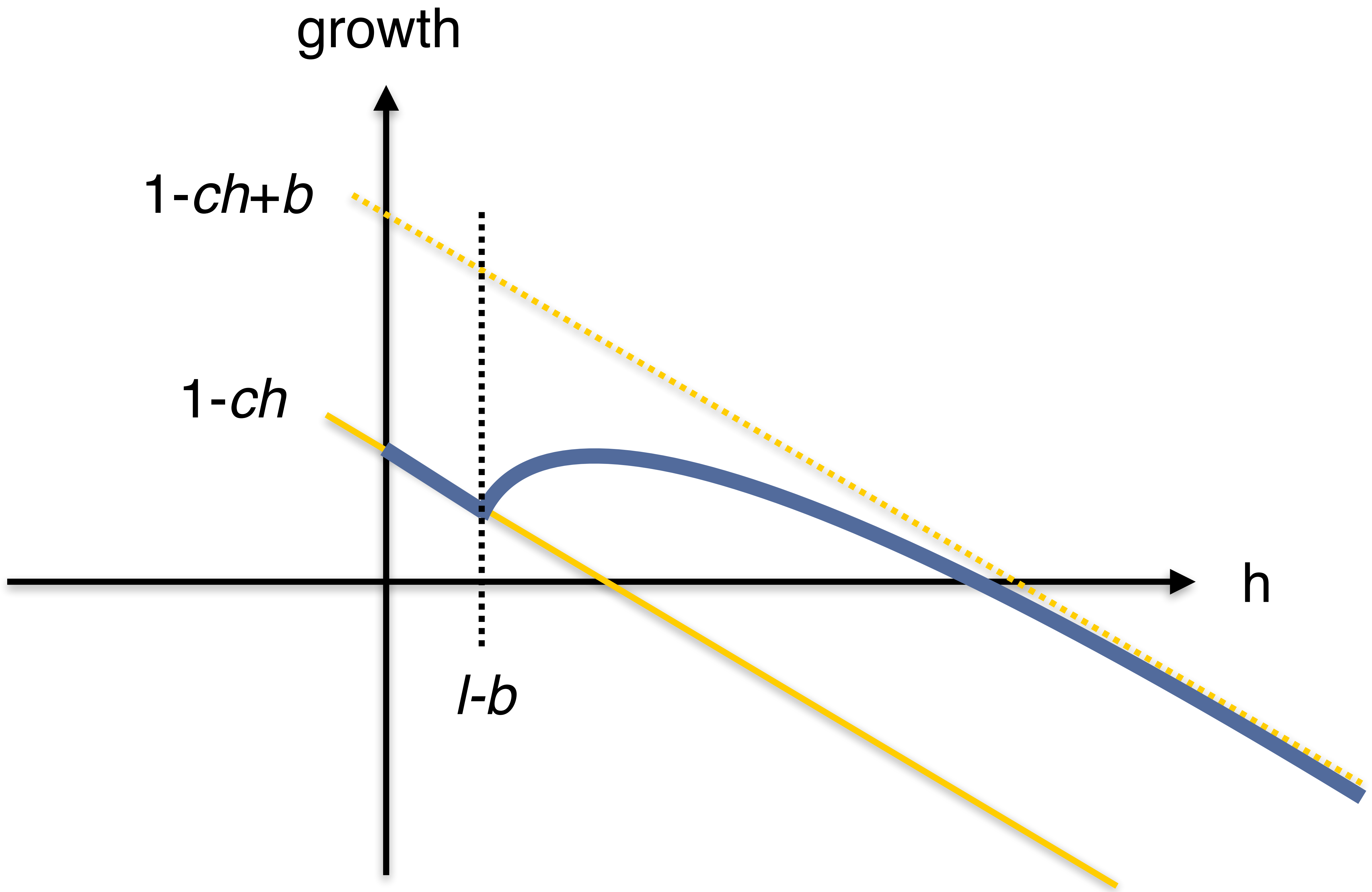
Math math math....

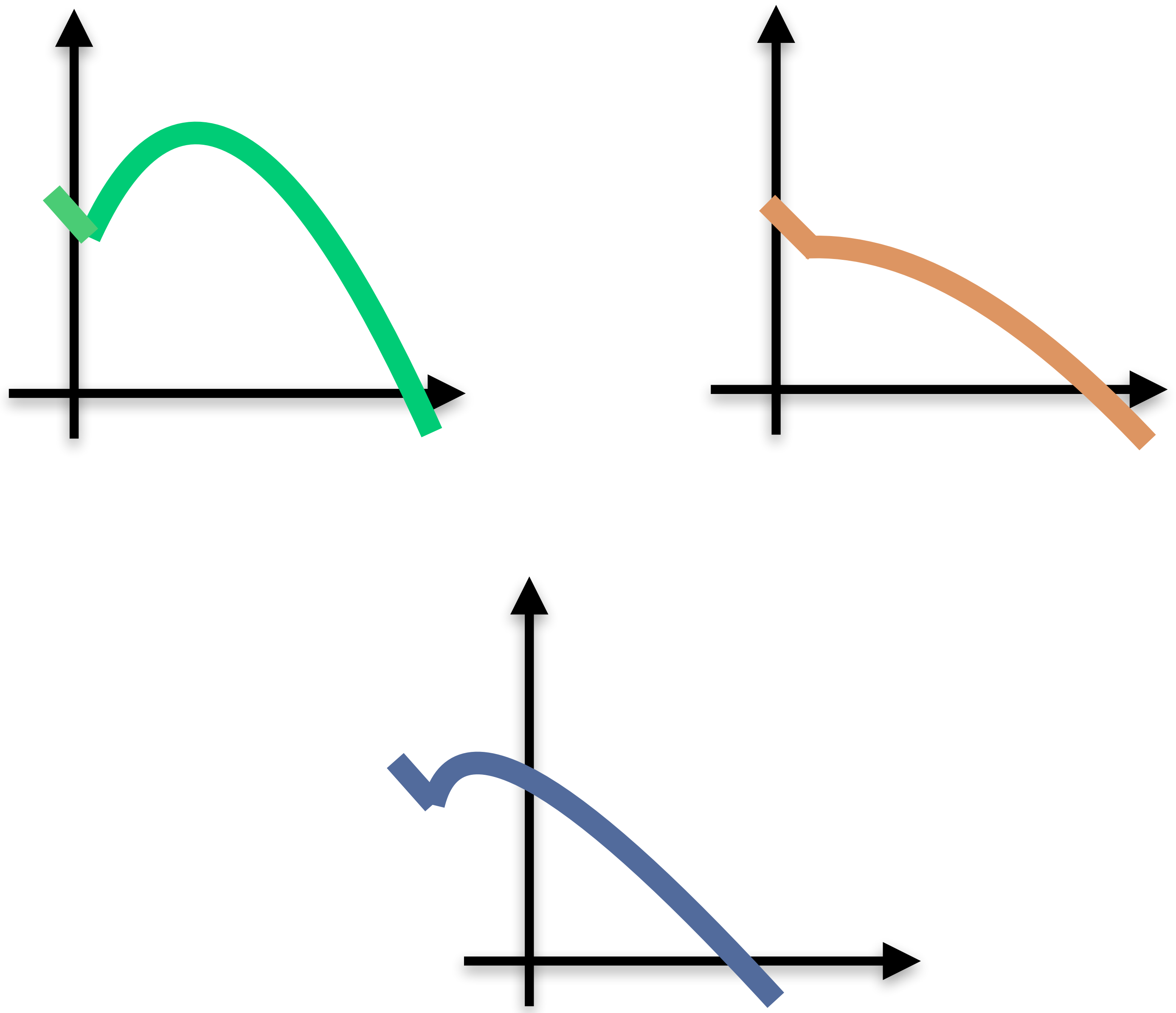
$$\begin{aligned}
\frac{\mathrm{d}C}{\mathrm{d}t} &= \underbrace{(1-ch+b)C}_{\text{reproduction of C}} - \underbrace{lC}_{\text{gene loss}} + \underbrace{hCN}_{\text{HGT}} - \underbrace{\phi C}_{\text{chemostat}} \\
\frac{\mathrm{d}N}{\mathrm{d}t} &= \underbrace{(1-ch)N}_{\text{reproduction of N}} + \underbrace{lC}_{\text{gene loss}} - \underbrace{hCN}_{\text{HGT}} - \underbrace{\phi N}_{\text{chemostat}} \\
\phi &= \underbrace{(1-ch+b)C}_{\text{total growth of C}} + \underbrace{(1-ch)N}_{\text{total growth of N}} \\
C+N = {}& 1 \;\; (\text{constant population size, ensured by chemostat}
\end{aligned}$$

$$\phi^*(h) = \begin{cases} 1-ch & \text{if } h \leq (l-b) \text{ (gene cannot persist)} \\ 1-ch+b-\frac{bl}{b+h} & \text{if } h > (l-b) \text{ (gene persists)}\,. \end{cases}$$

The growth rate of the population in steady state

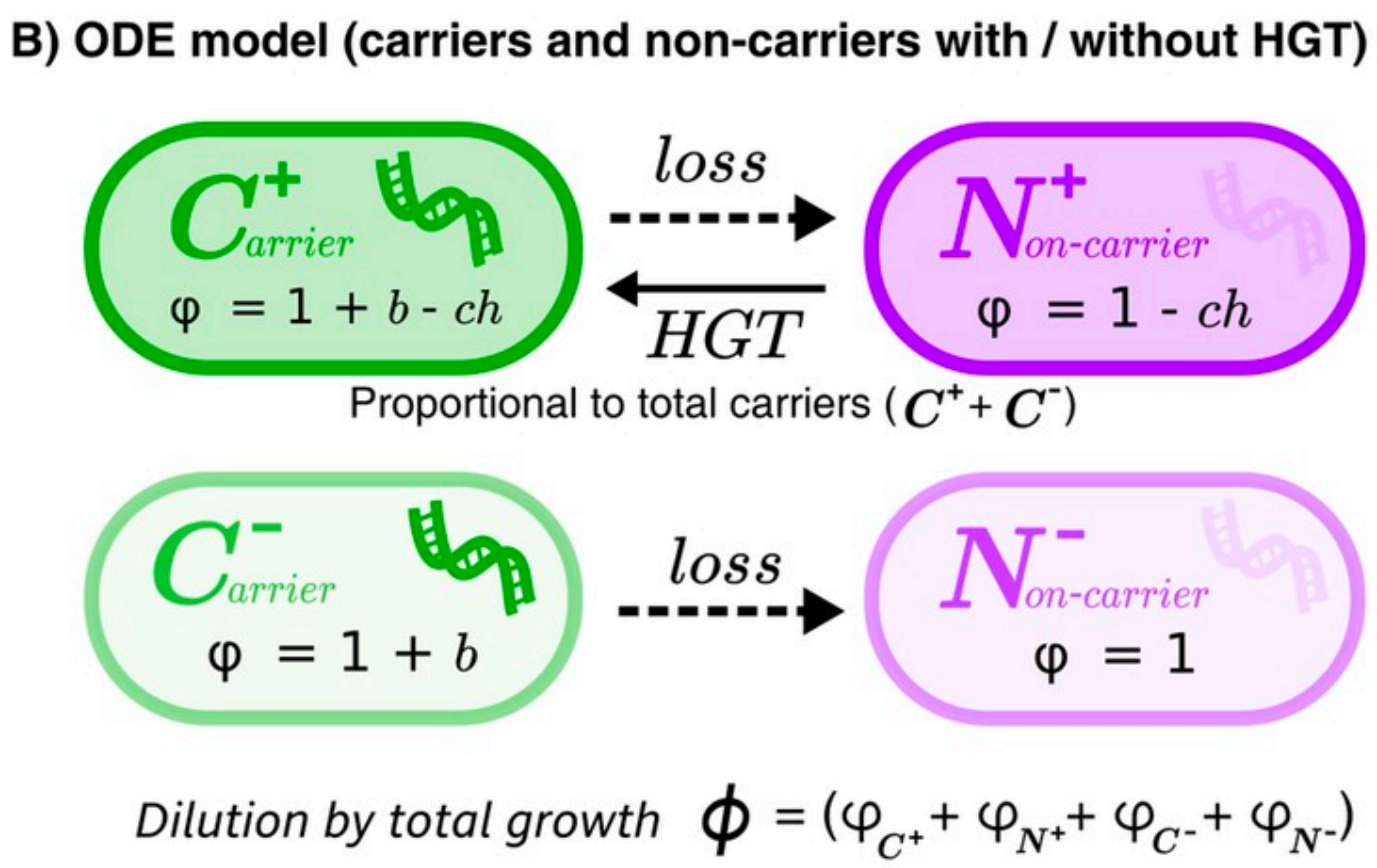
What does $\Phi^*(h)$ look like?



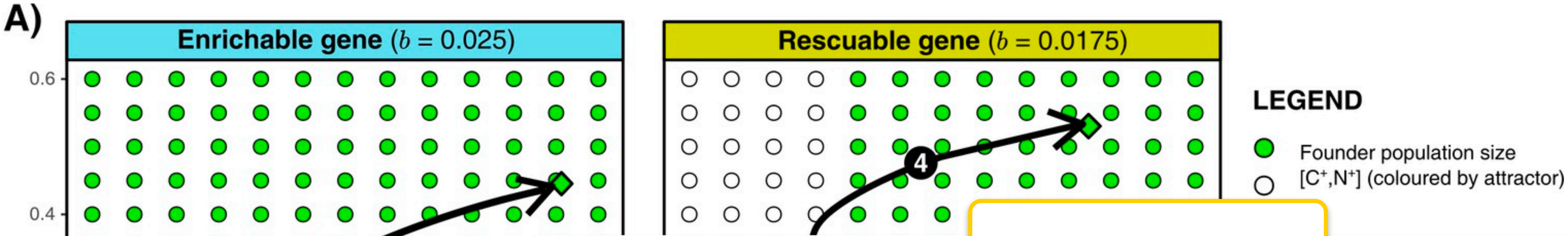


$$\phi^*(h) = \begin{cases} 1 - ch & \text{if } h \leq (l - b) \text{ (gene cannot persist)} \\ 1 - ch + b - \frac{bl}{b+h} & \text{if } h > (l - b) \text{ (gene persists)}. \end{cases}$$

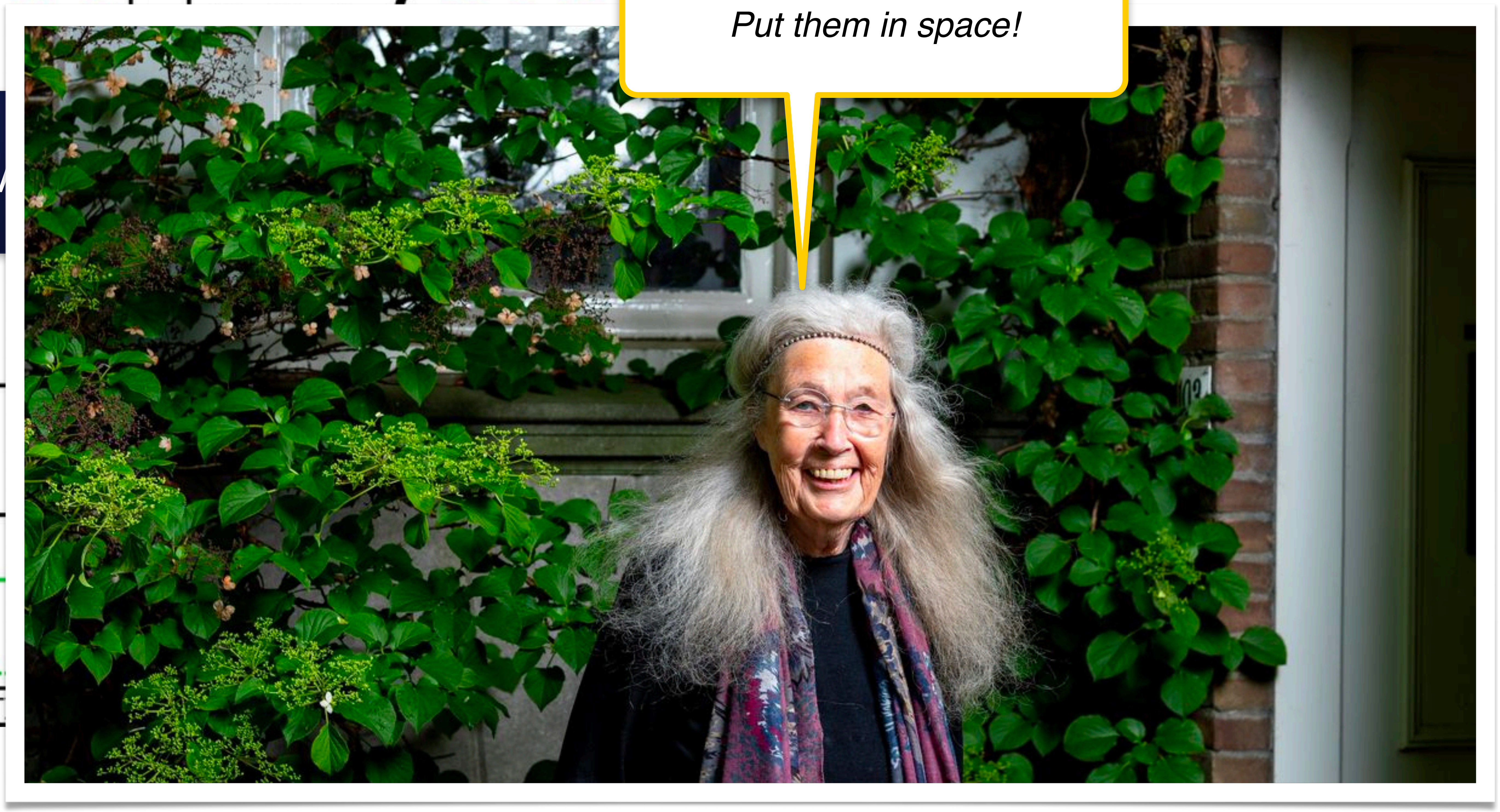
Can microbes evolve **costly HGT** to “rescue” a gene?



Evolve costly HGT to “rescue” a gene?

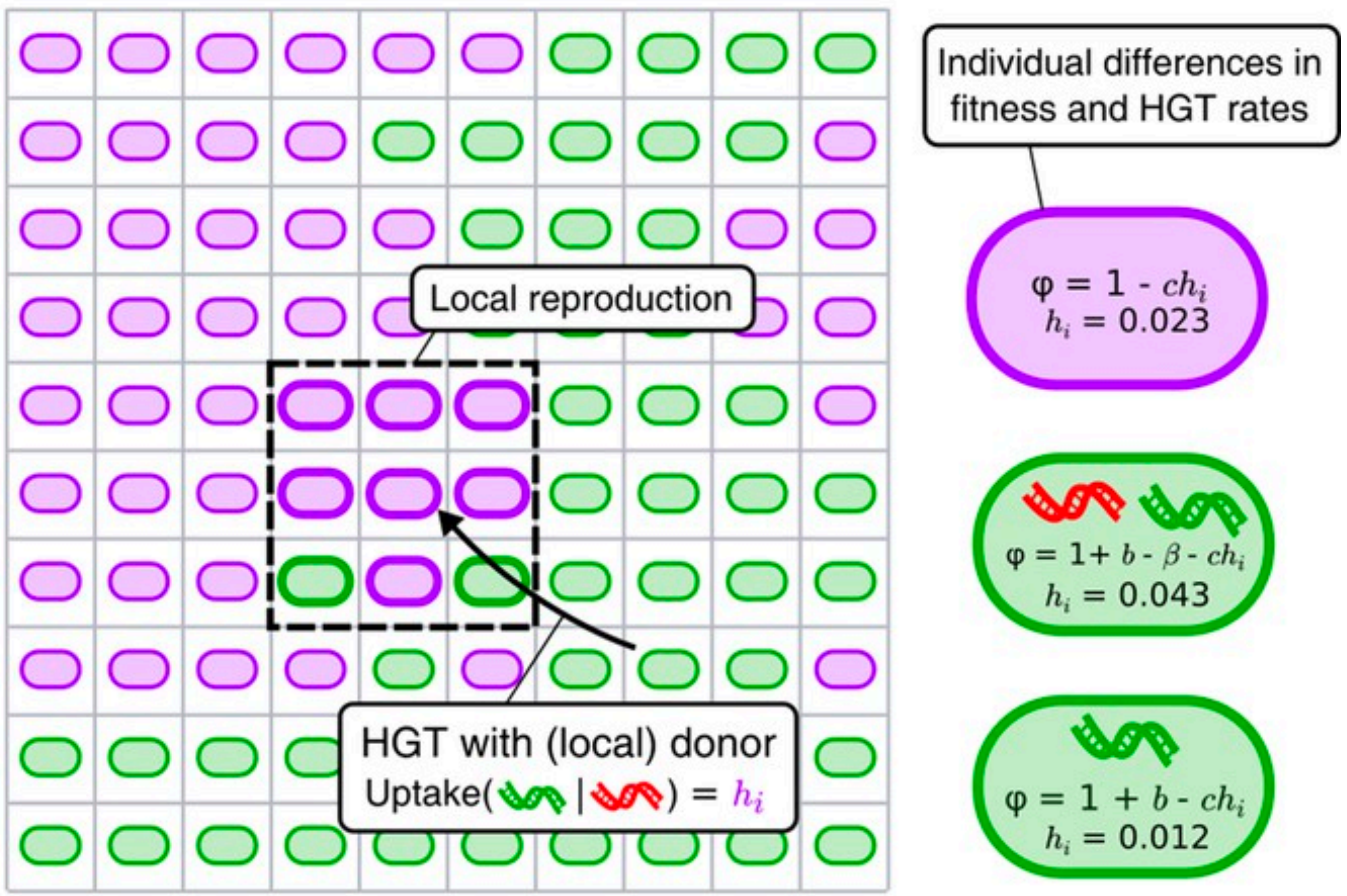


Maybe I shouldn’t hav



Put them in space!

Almost identical model. In space.



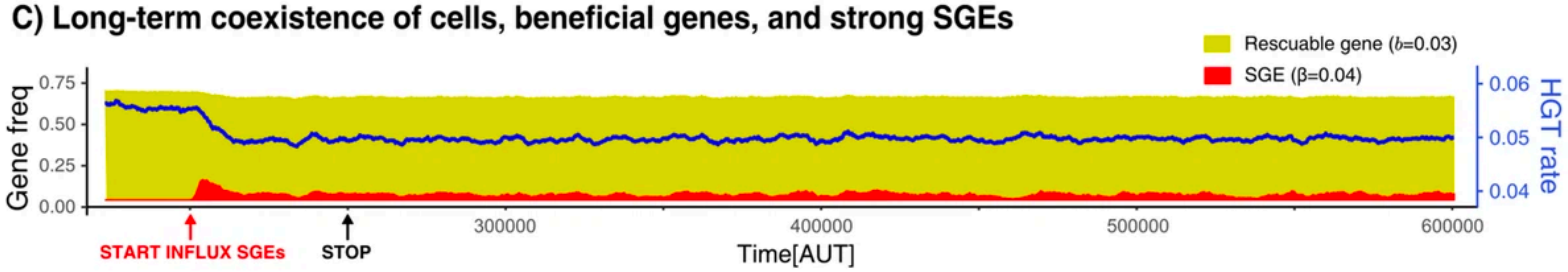
C) Individual-based, eco-evolutionary model

Q: What is the impact of spatial structure?

Phew minor differences:

- Individual-based model: individual lineages can evolve their rate of eDNA uptake!
- Both “**good**” and “**bad**” genes are possible in 1 system (*b and β- parameter)*!
- Mobile vs. Selfish genetic elements

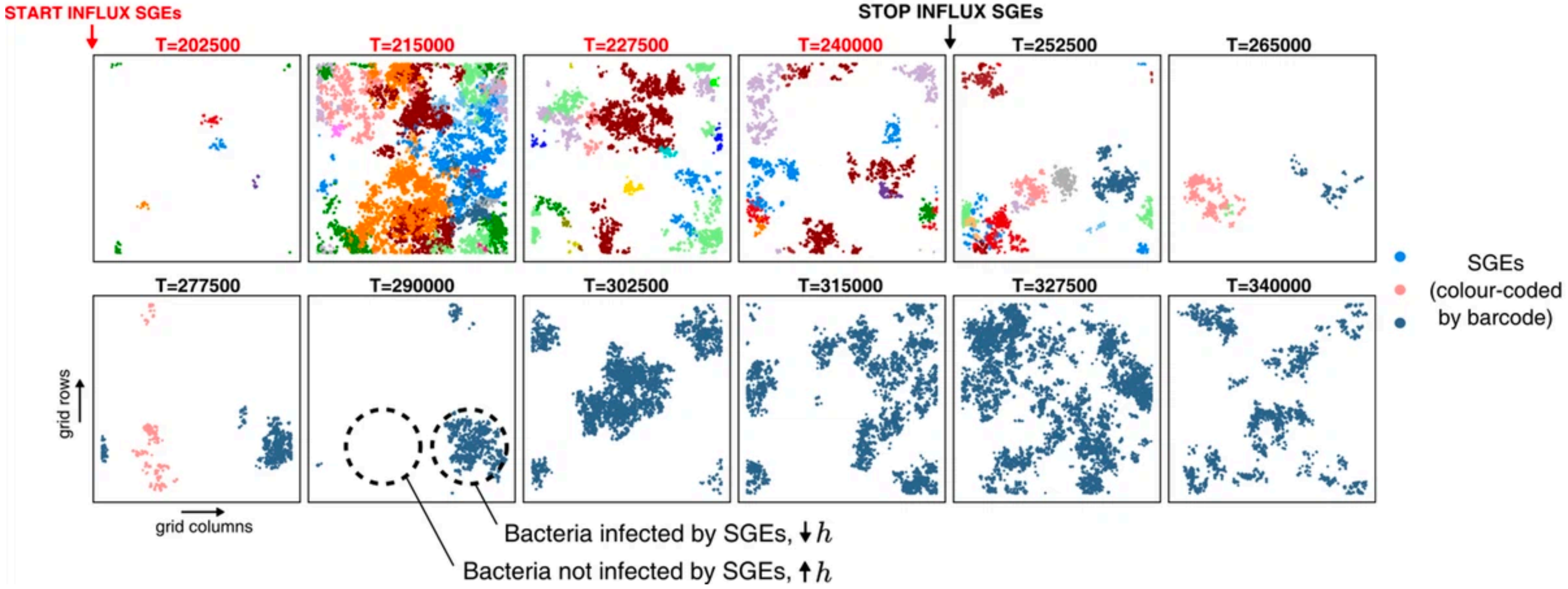
Cells maintain **costly HGT**, in the presence of **costly SGEs**



- Proof-of-principle: **HGT can be adaptive for microbes**, even under genuinely terrible circumstances

Conclusions Part Ia

- While the toxin genes were “occasionally” beneficial, similar observations for constantly (“slightly”) beneficial genes
- Spatial structure can overcome apparent “paradoxes” caused by positive frequency dependence (“Allee effects”)
- Microbes engage in HGT under terrible circumstances. It can still benefit them!
- To understand this, spatial heterogeneity is relevant:



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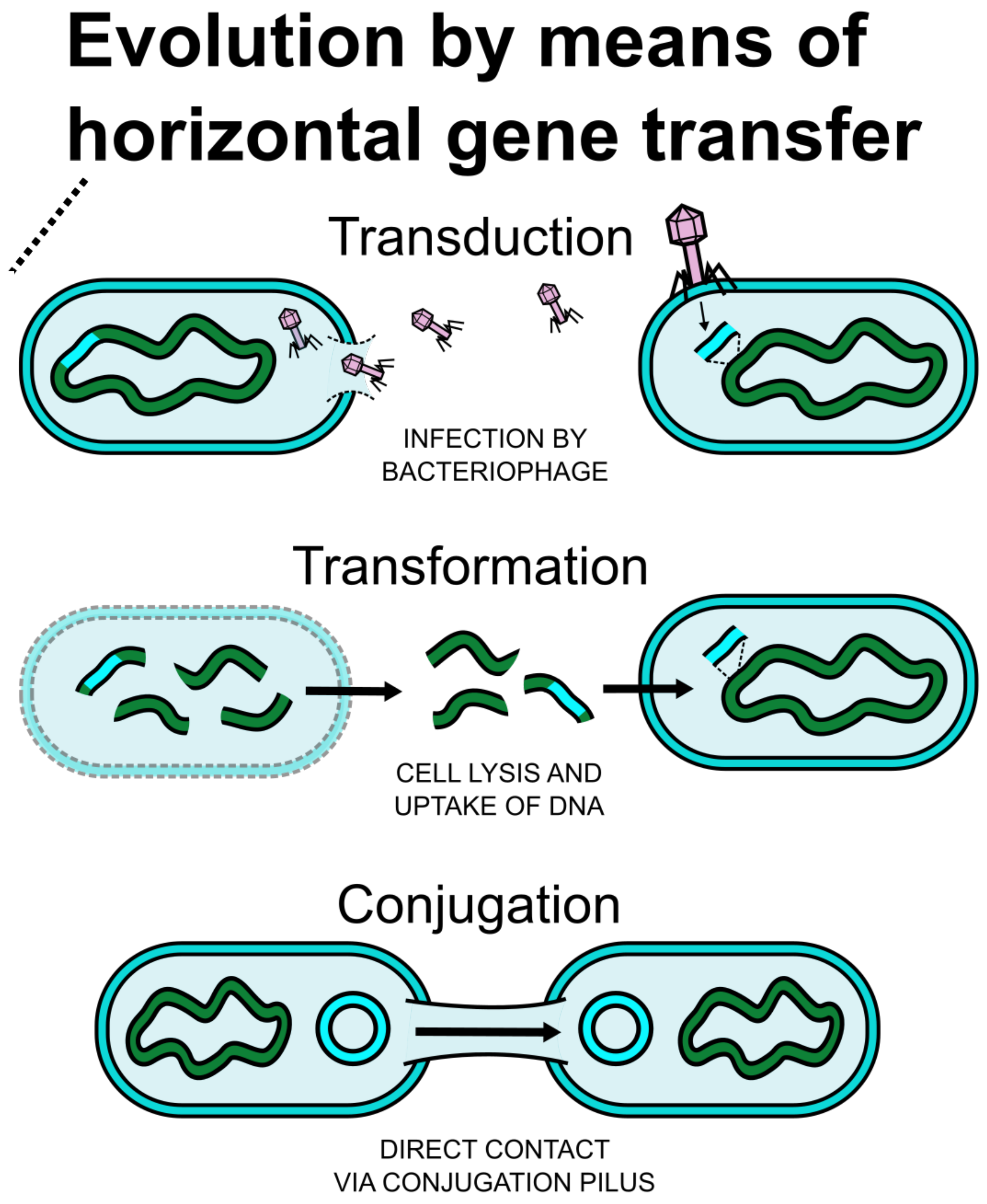
What about the vehicles of HGT?



These are the “text-book” examples of HGT, but there are **more** mechanisms and vehicles

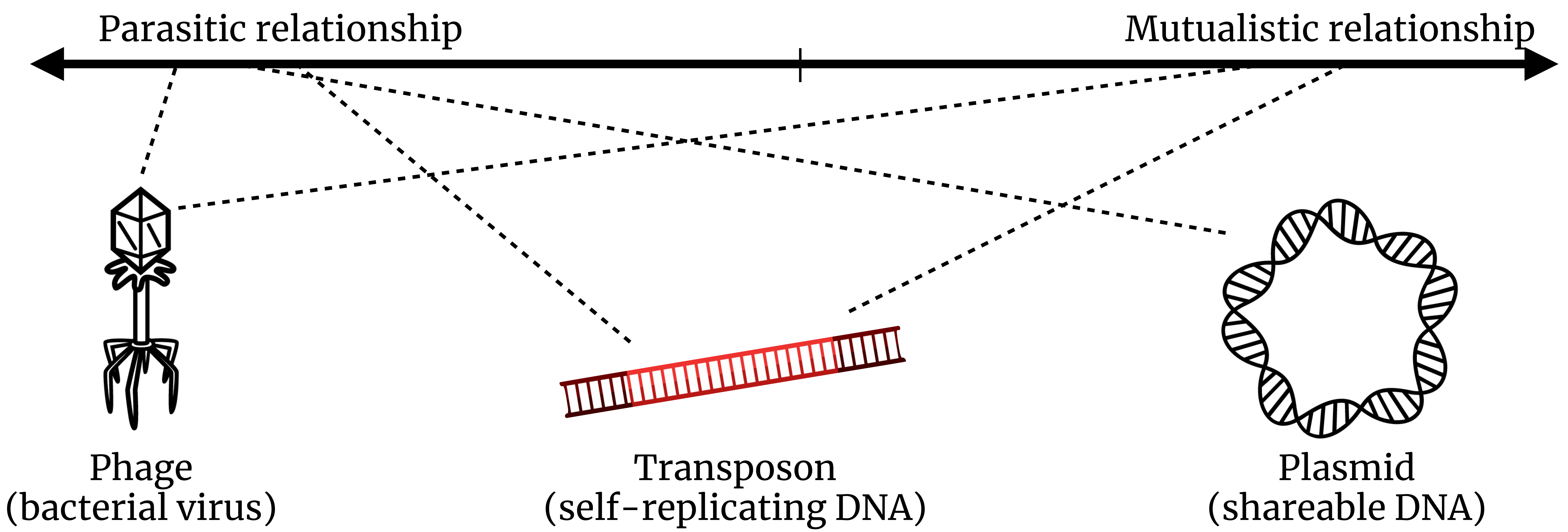
Membrane vesicles, gene transfer agents, transposons, integrative and conjugative elements (ICEs), integrons, BORGs, starships and voyagers, even mobile chromosomes…

The list is endless…

“Mobile elements are entities that evolved to persist and replicate through adaptations that move DNA.”

— James P.J. Hall, 2021; “The secret lives of Mobile Genetic Elements”

Friends or foes?



Mobile elements are diverse and evolve on a **parasitism-mutualism continuum**

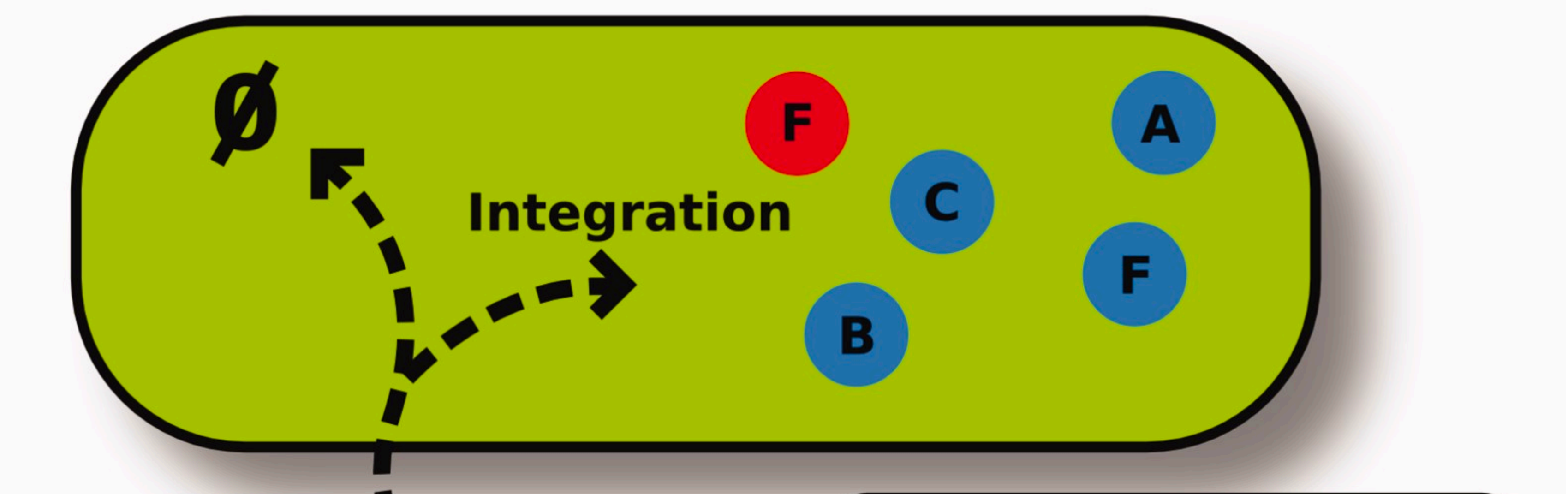
Friends or foes?

- Transposons are the simplest "nested replicator", they replicate inside chromosomes
- They can also jump from cell to cell, after uptake from the environment

- Occasionally, they carry useful genes in nature, such as antimicrobial resistance genes
- So what drives parasitism vs mutualism for these very simple entities?

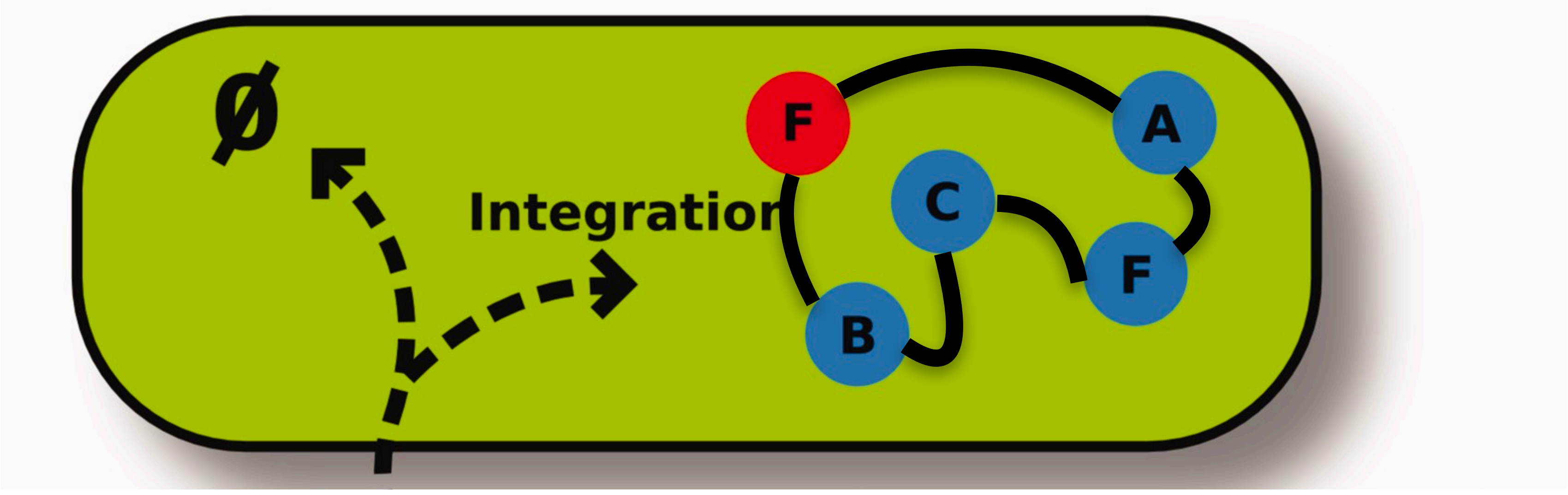
Genome evolution 101

- More models of **genome evolution** will follow later in the course, but here's a quick intro

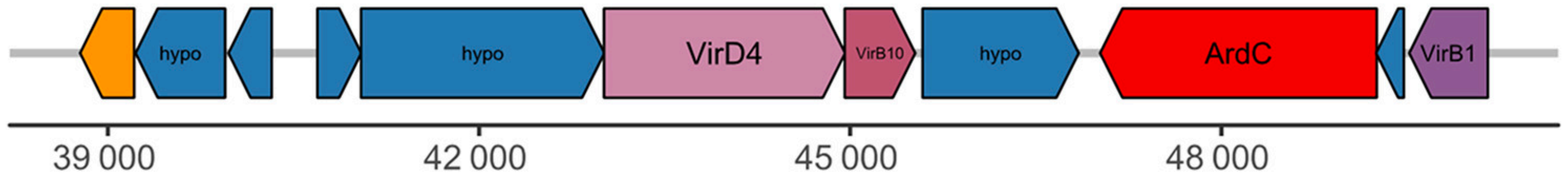


In earlier models, bacteria are "bags of genes"…

→

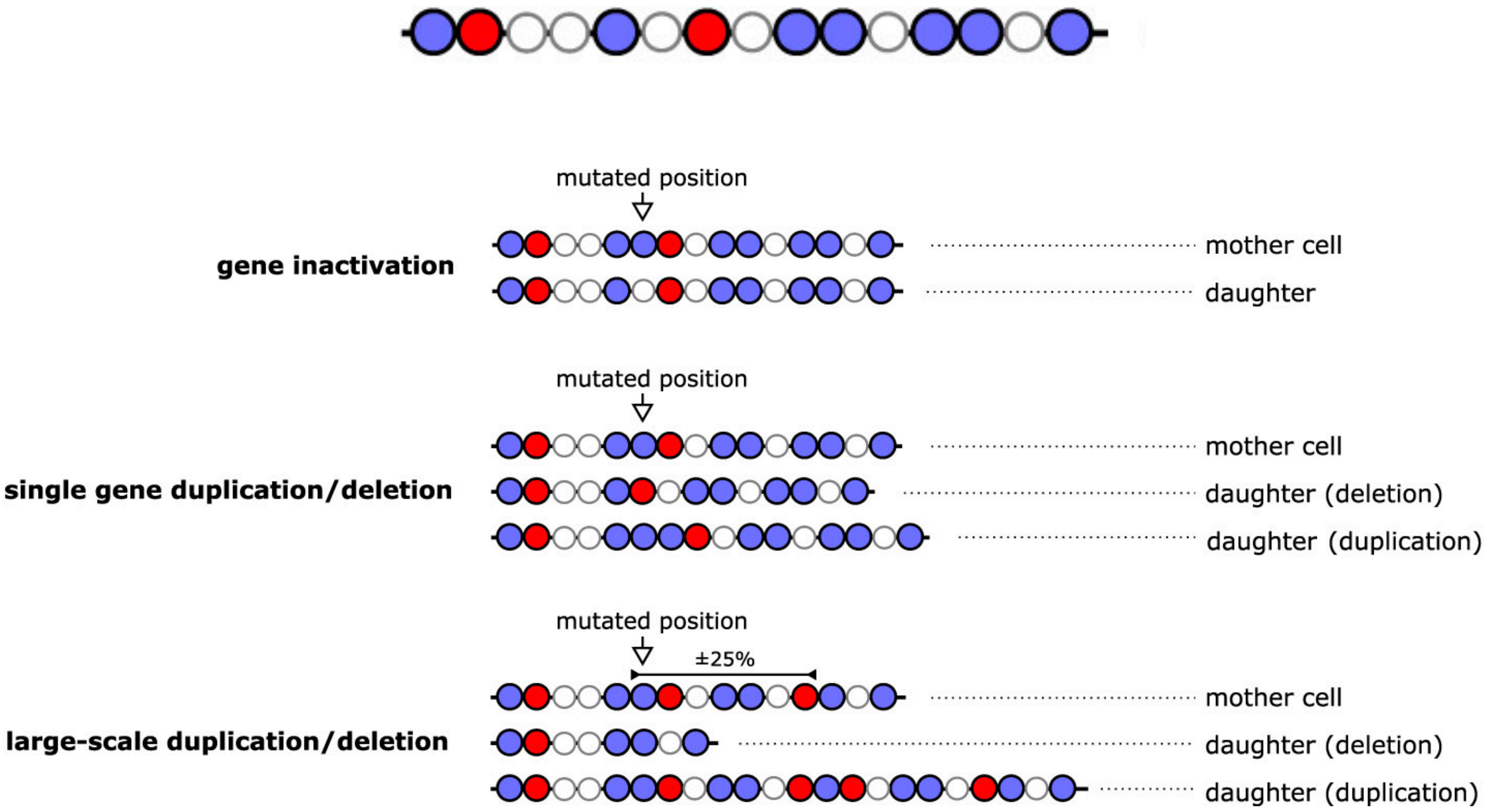


But real genes are on a chromosome!

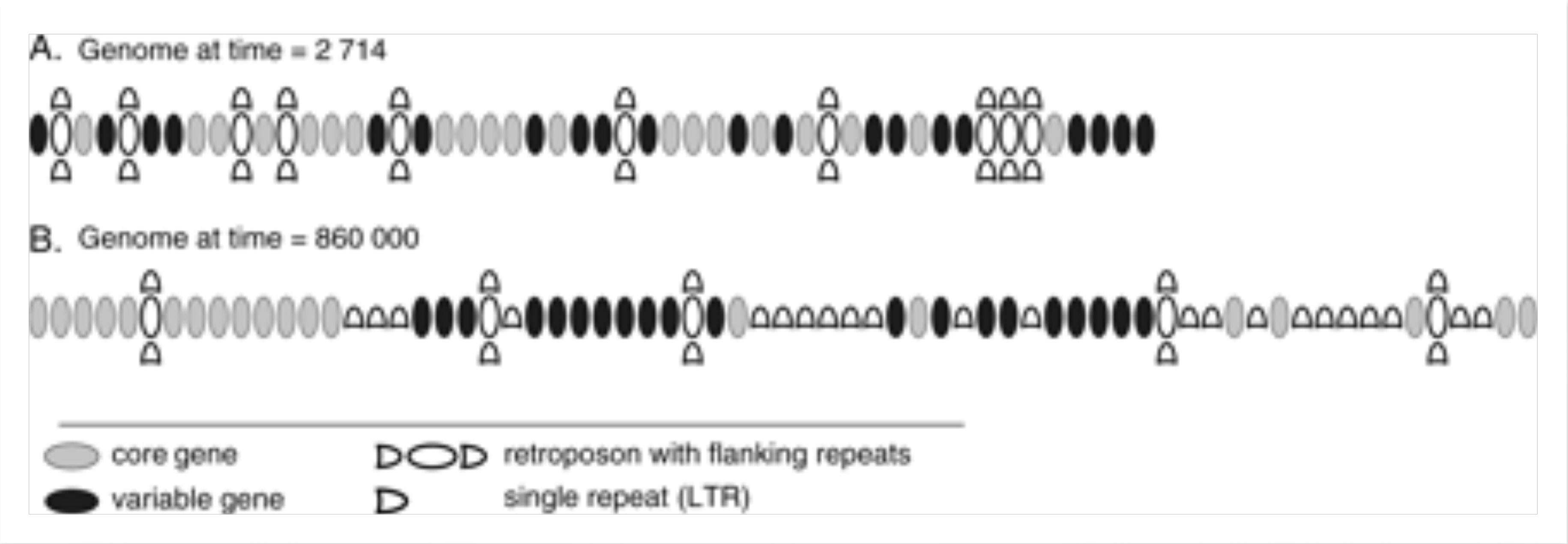


- Suddenly, the word "integration" means something else…
- Genes have to **insert somewhere** (more on this later!)

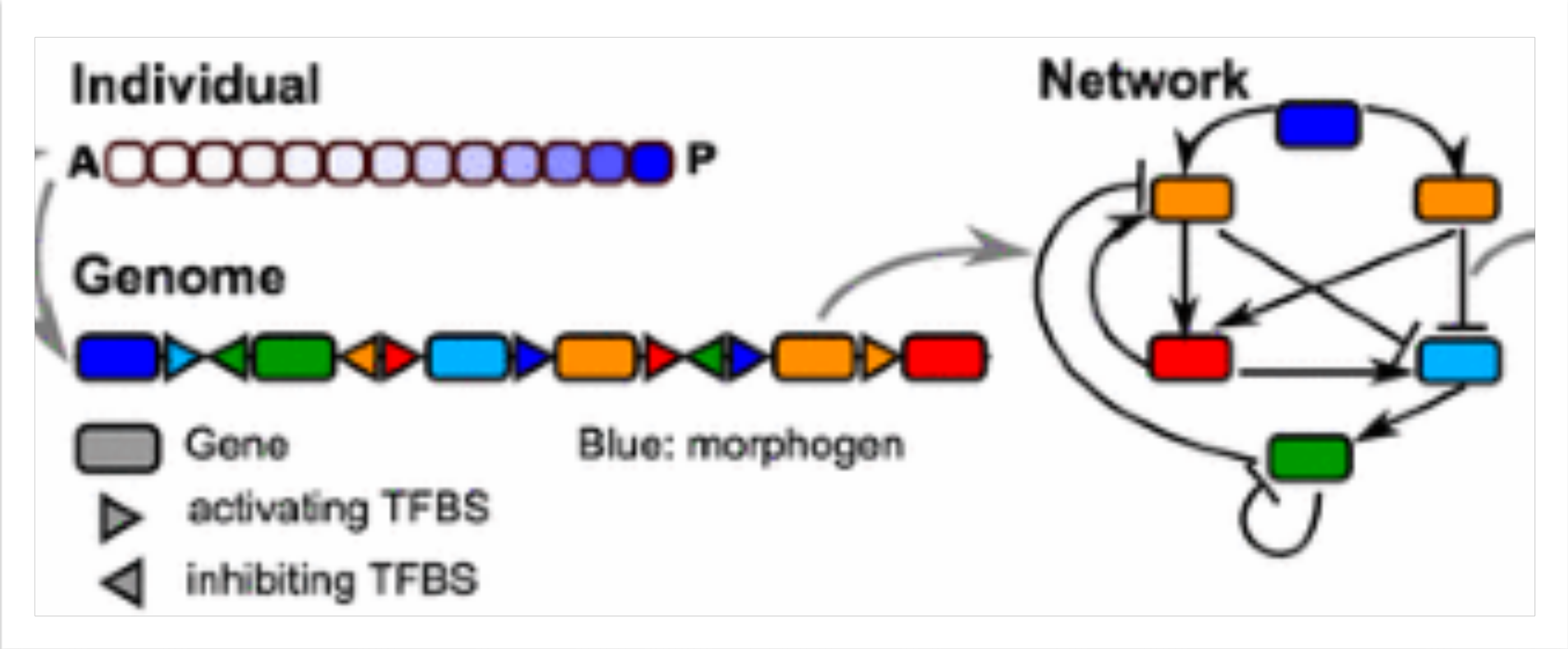
The pearls-on-strings (PoS) model of genome evolution



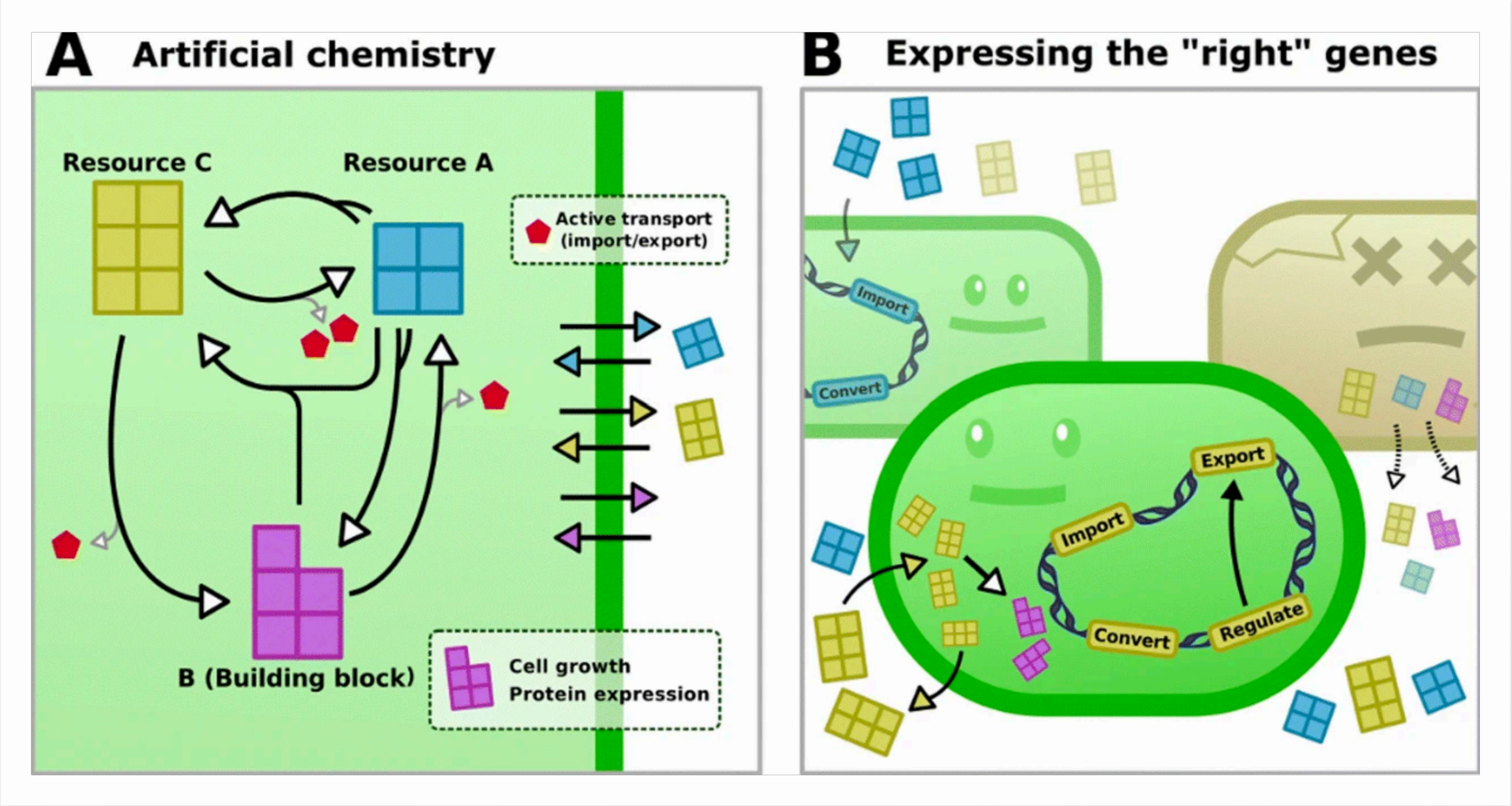
A versatile model



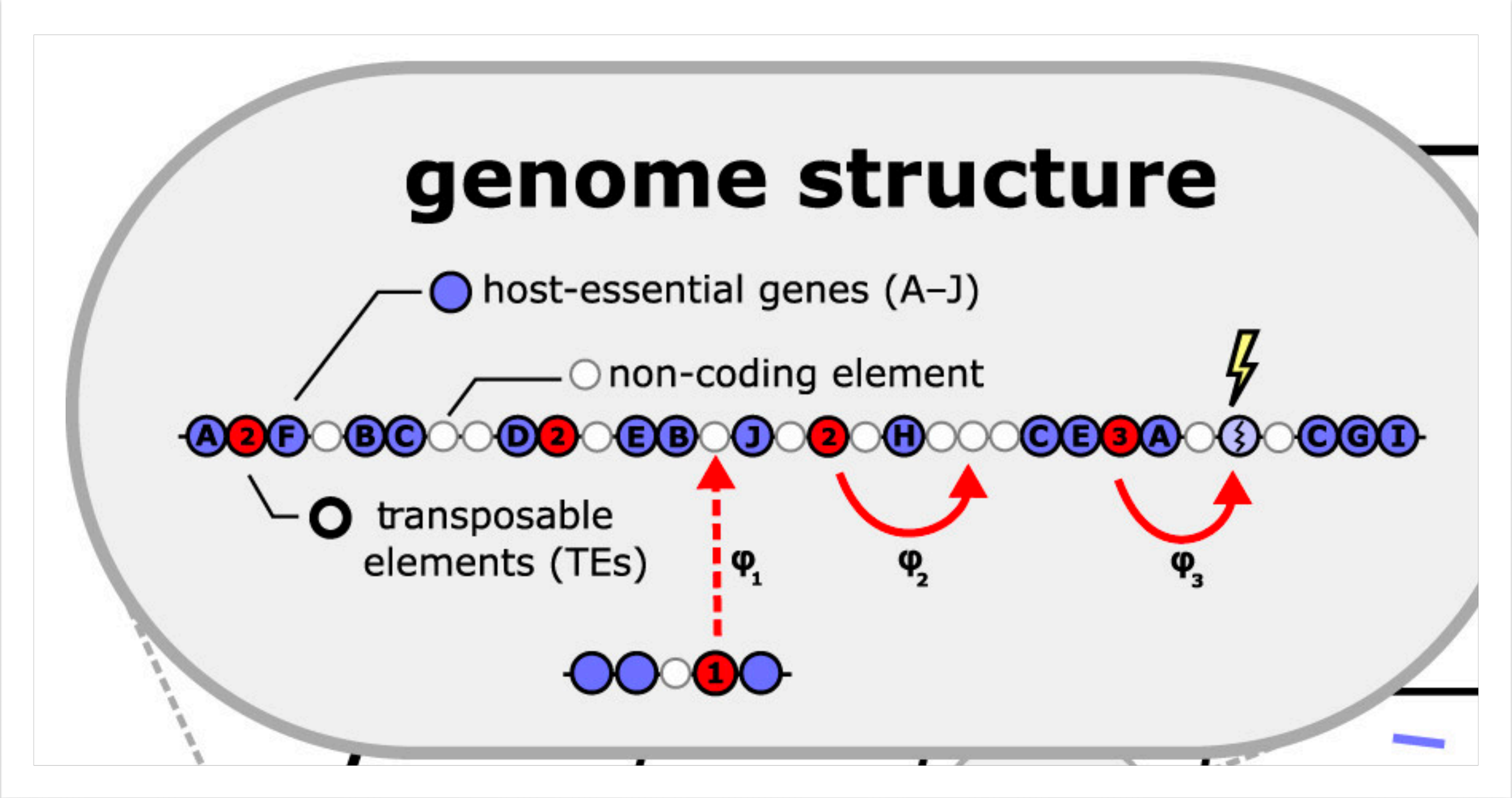
Gene clustering (Crombach *et al.*, 2007)



Gene regulatory network evolution (Vroomans *et al.*, 2017)



Combine with artificial chemistry (van Dijk *et al.* 2019)

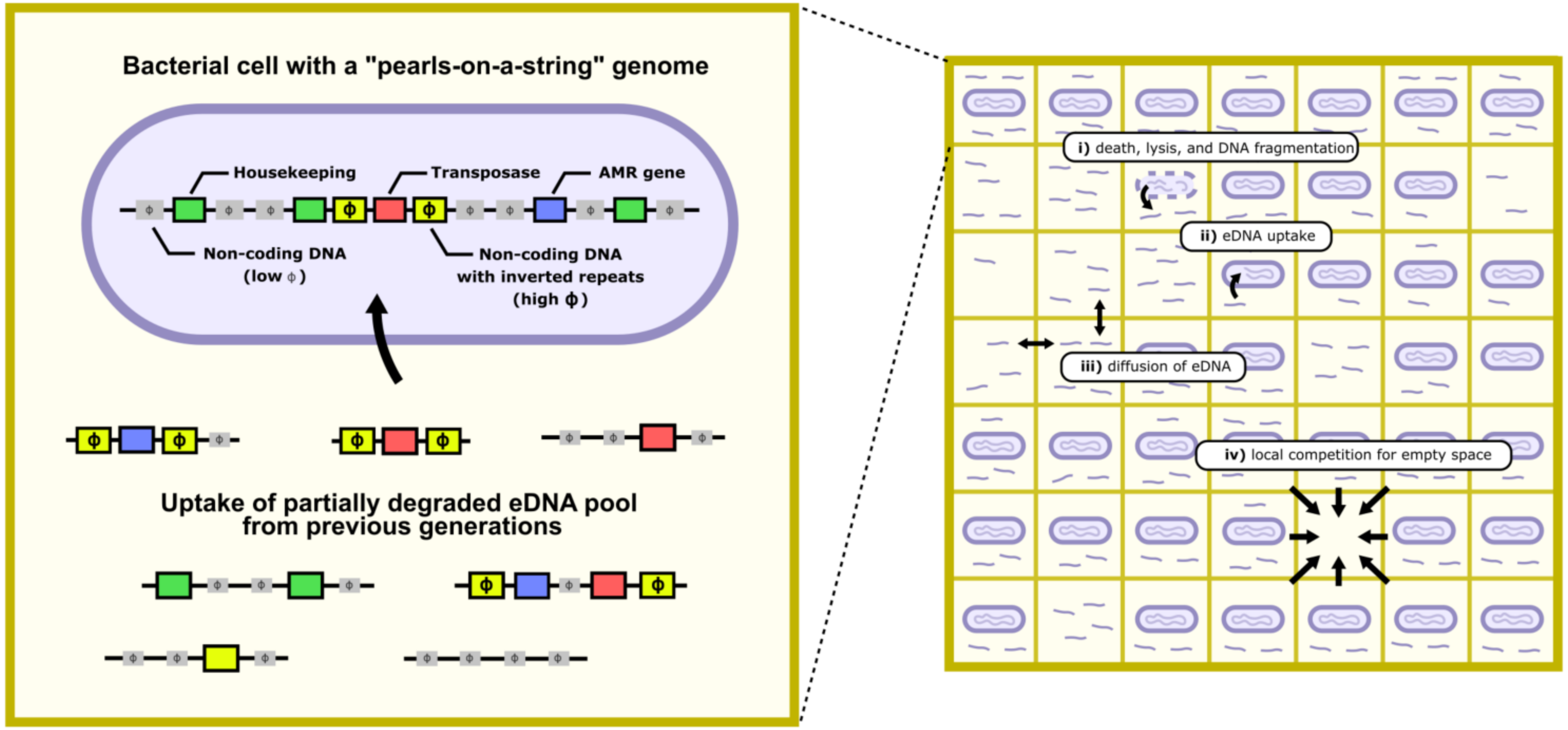


Add self-replicating pearls —> mobile genetic elements! (van Dijk *et al.*, 2021, 2024 wip)

PoS gives “linkage” of genes

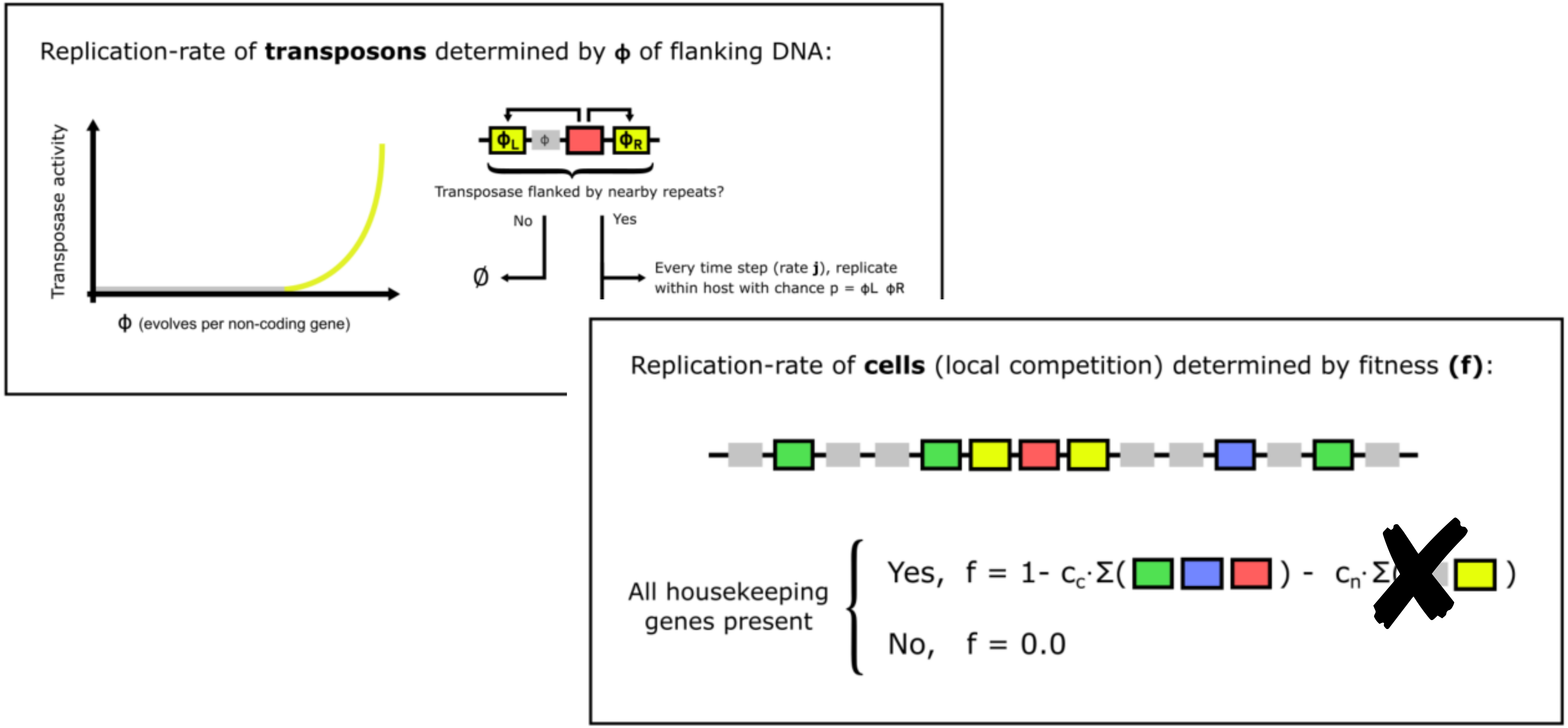
Can a **selfish genetic element** get “linked” to a good gene?
(transposons carry AMR genes, phages carry virulence genes, …)

Toy model of co-evolving genomes and transposons



Warning: the figures on the next slides never made it to publication, and are not super pretty :)

TEs are not predefined, they have to emerge!



Replication-rate of **transposons** determined by ϕ of flanking DNA:

Every time step (rate **j**), replicate within host with chance p = ϕL ϕR

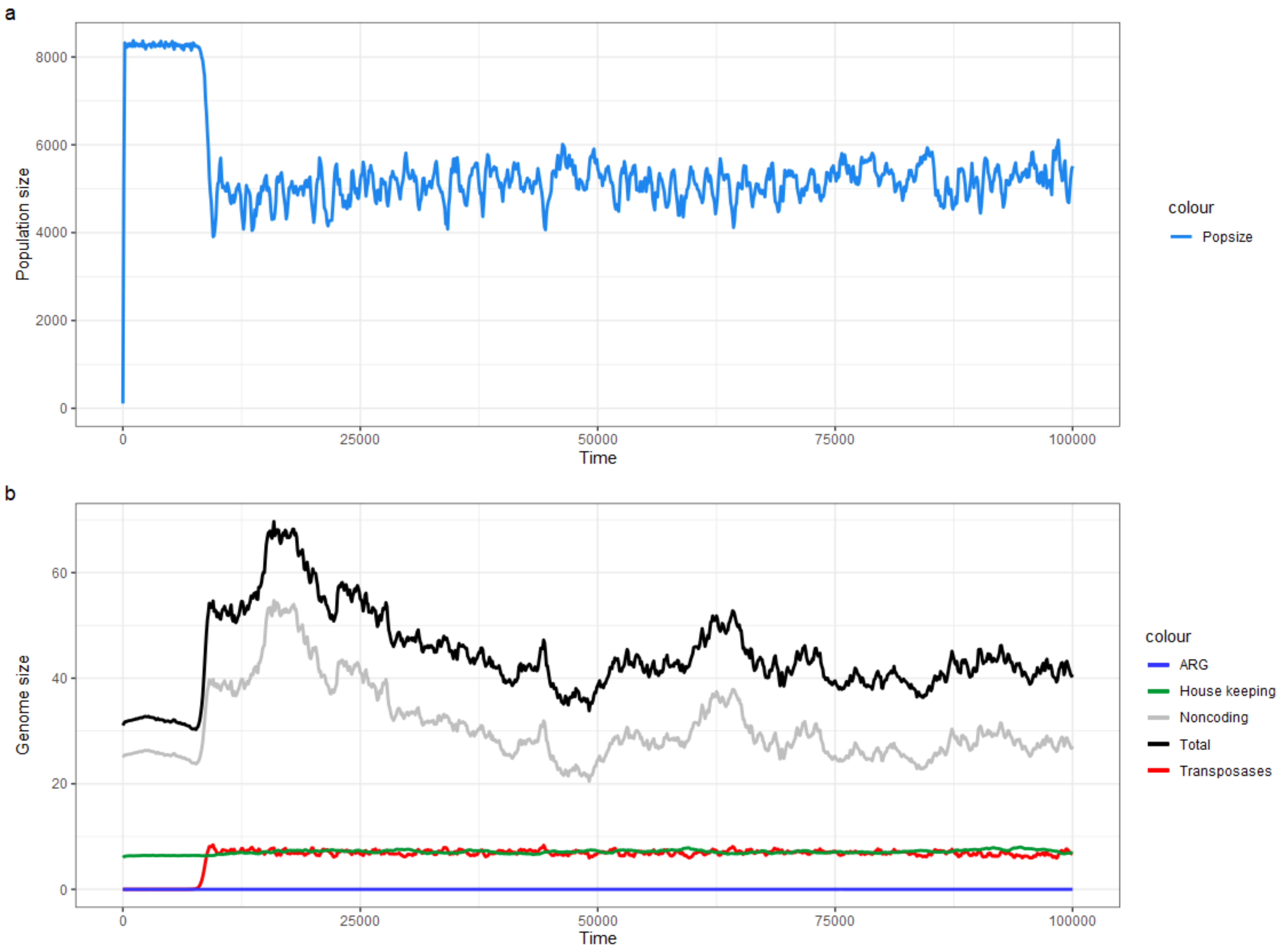
Replication-rate of **cells** (local competition) determined by fitness **(f)**:

All housekeeping genes present

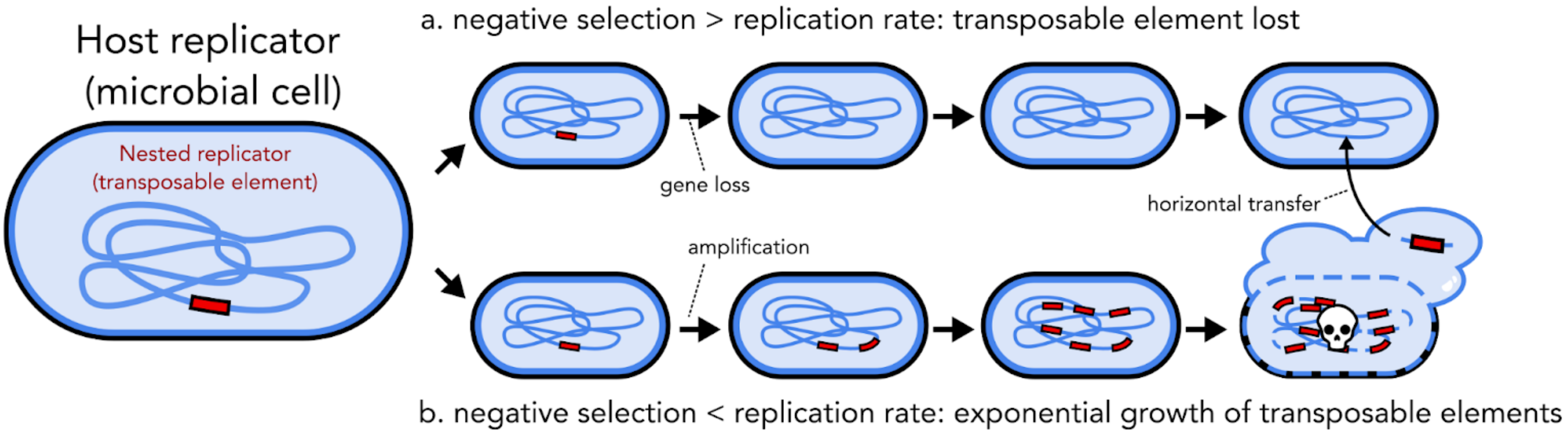
Yes, $f = 1 - c_c \cdot \Sigma($🟩🟦🟥$) - c_n \cdot \Sigma($🟨$)$

No, $f = 0.0$

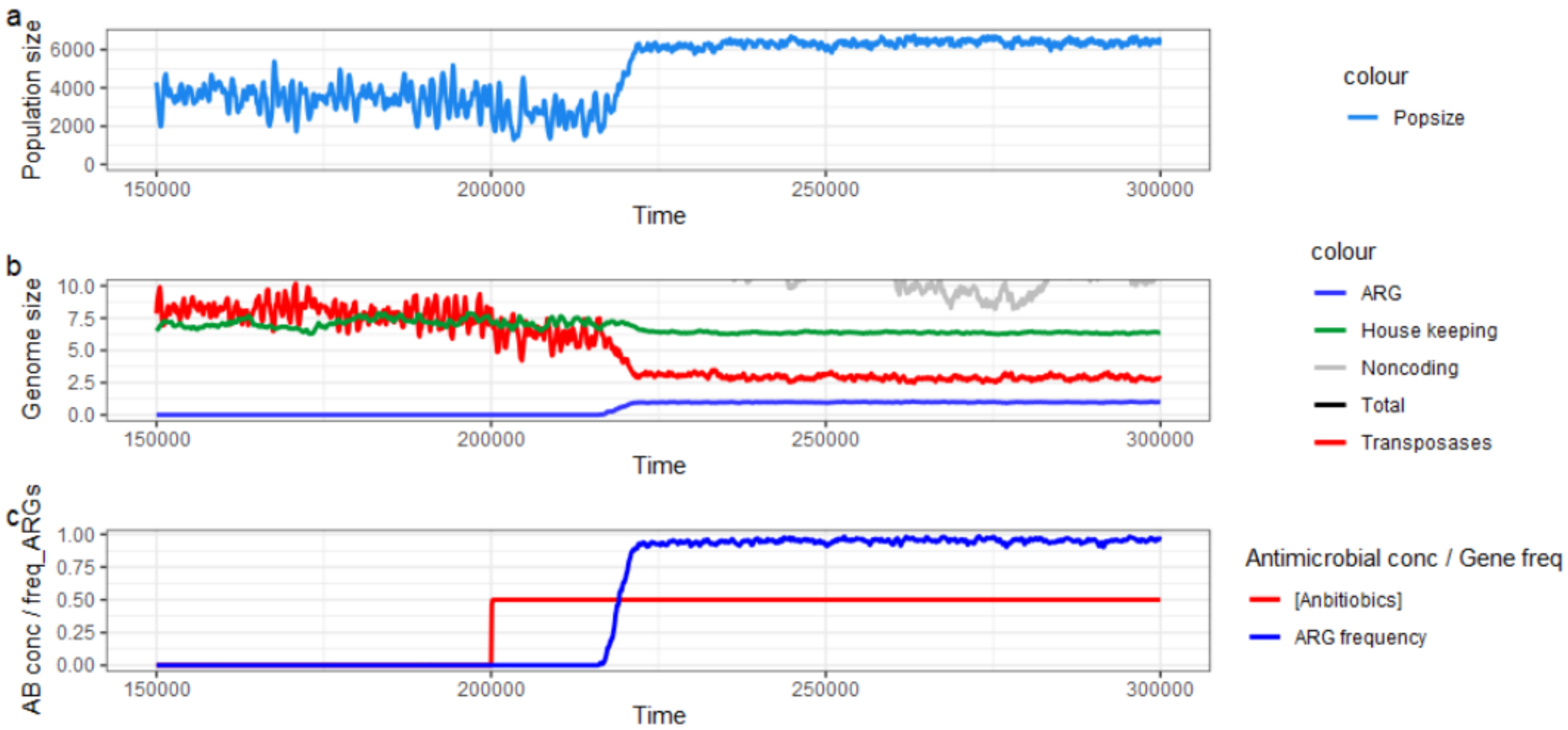
TEs emerge after some time (no AMR selection)



System persists because of spatial structure



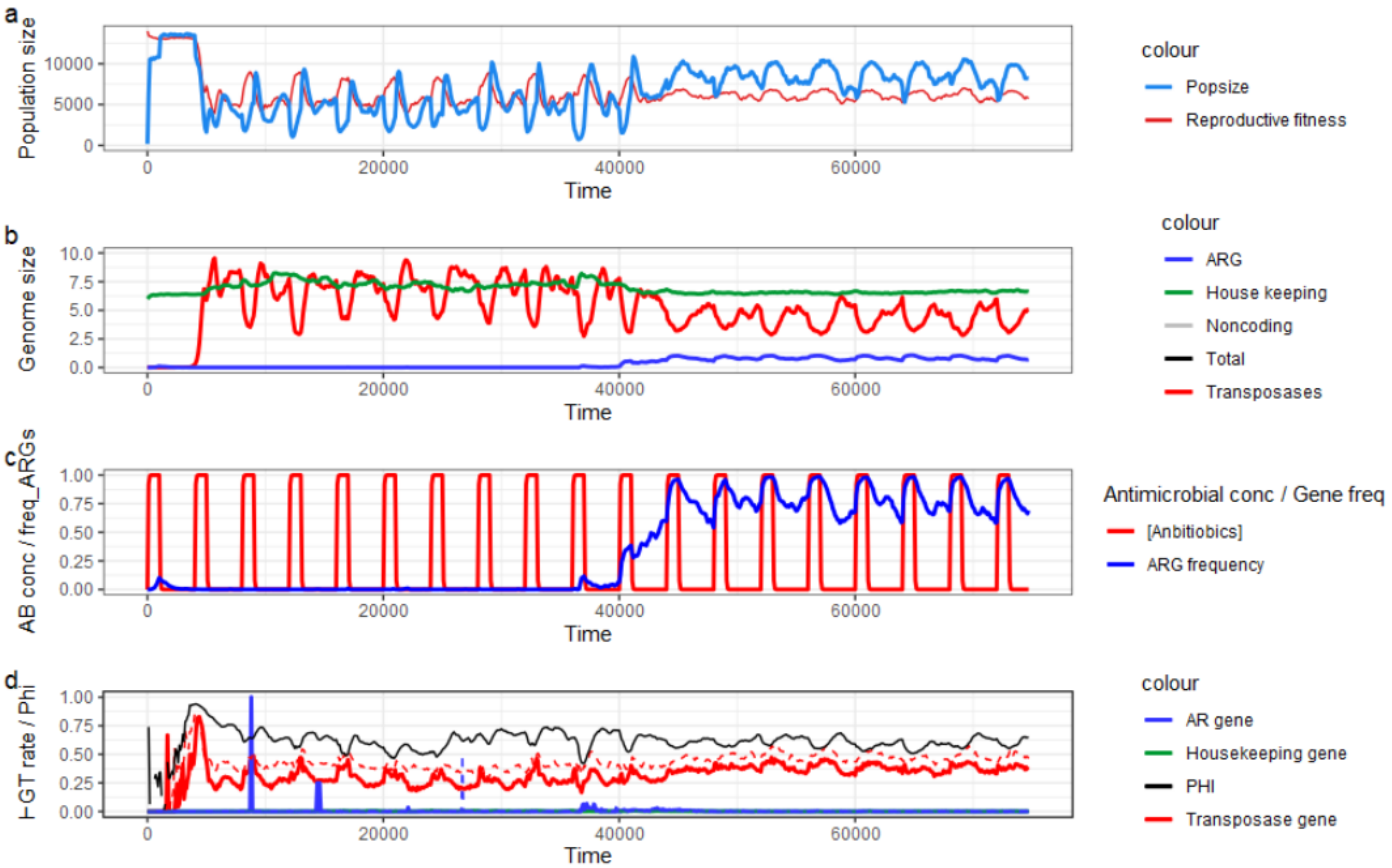
Adding selection for AMR genes (constant)



- AMR did emerge (blue line), but it is not linked to a transposon at all… (not shown)

Adding selection for AMR genes (pulsing)

./R0_SpreadAB_2/

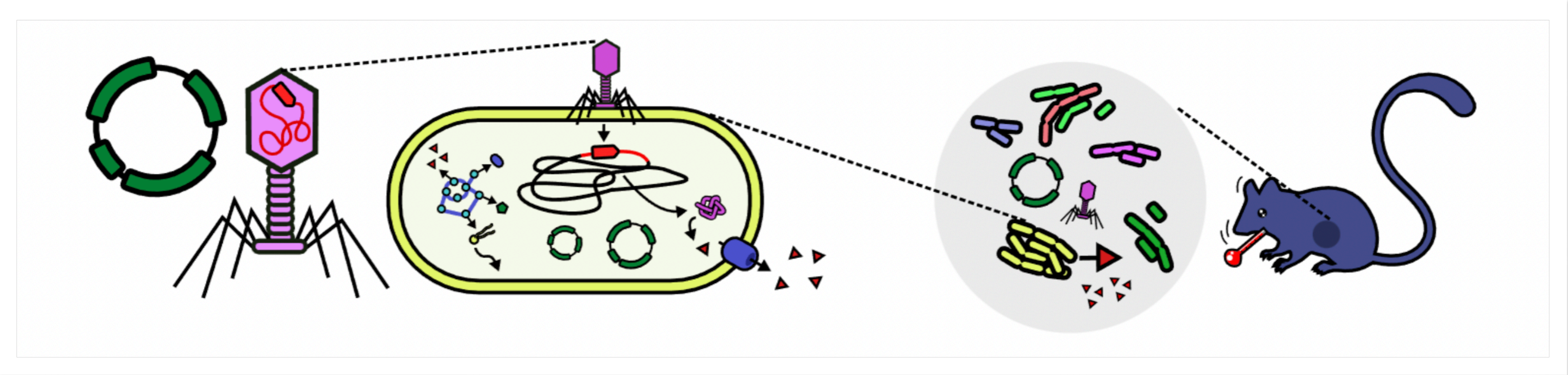


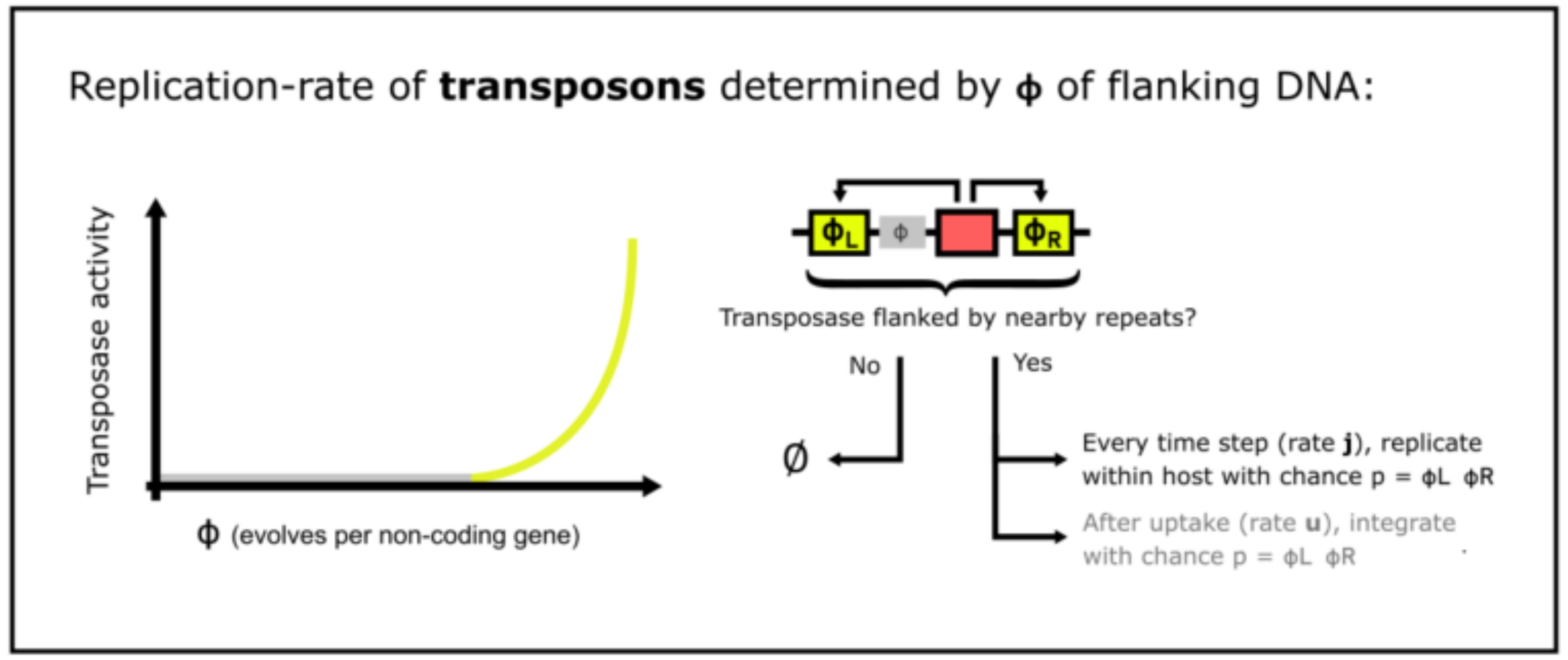
- Pulsing for antibiotic resistance: still TEs and AMR do not get linked...

Okay, so getting SGEs “linked”
to beneficial genes is really hard…

Then why do so many SGEs (transposons, phages)
carry ecologically relevant genes?

Part 1 - Horizontal- vs vertical transmission of genes and microbiomes





Replication-rate of **transposons** determined by **ϕ** of flanking DNA:

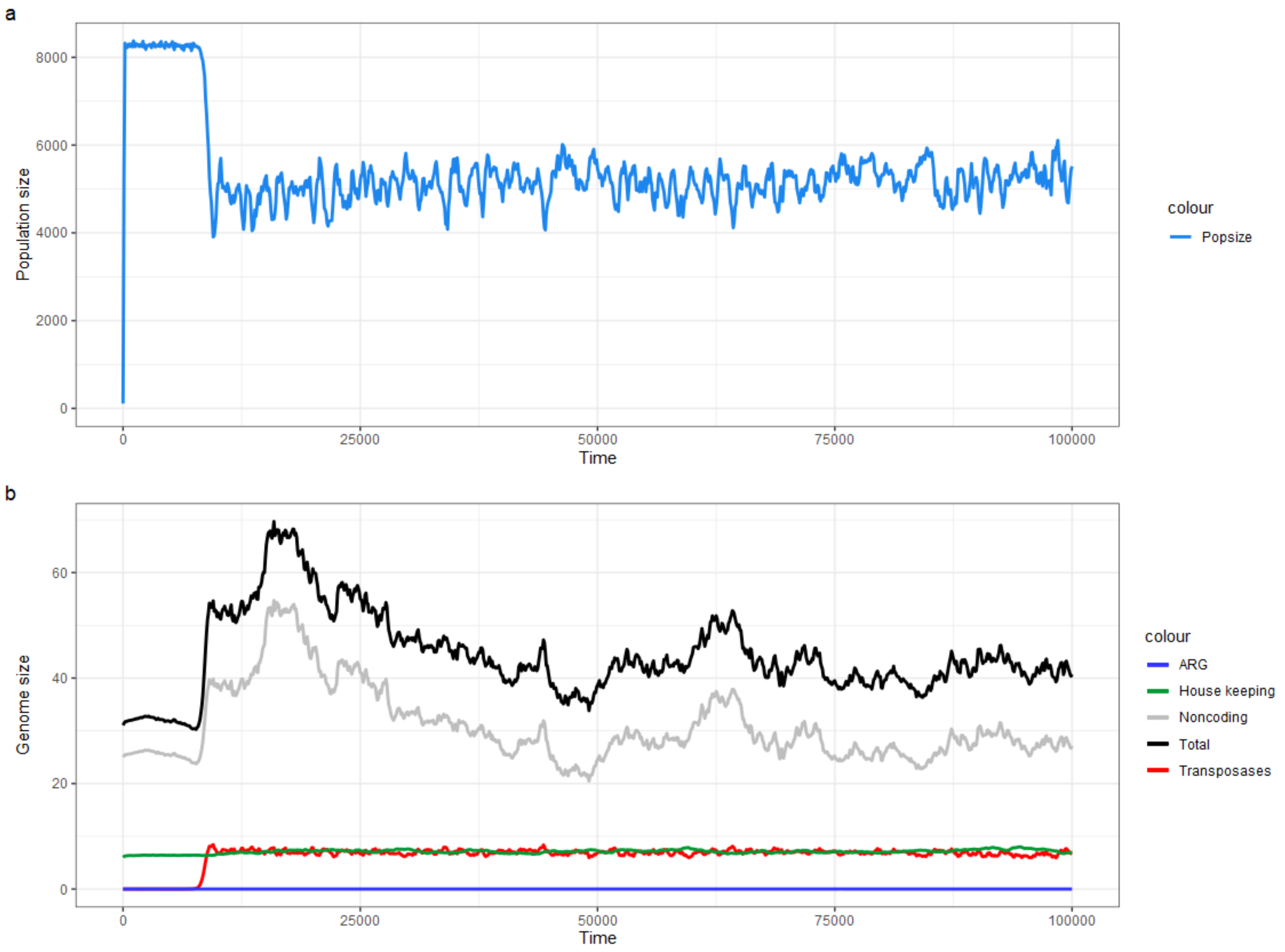
Every time step (rate **j**), replicate within host with chance p = ϕL ϕR

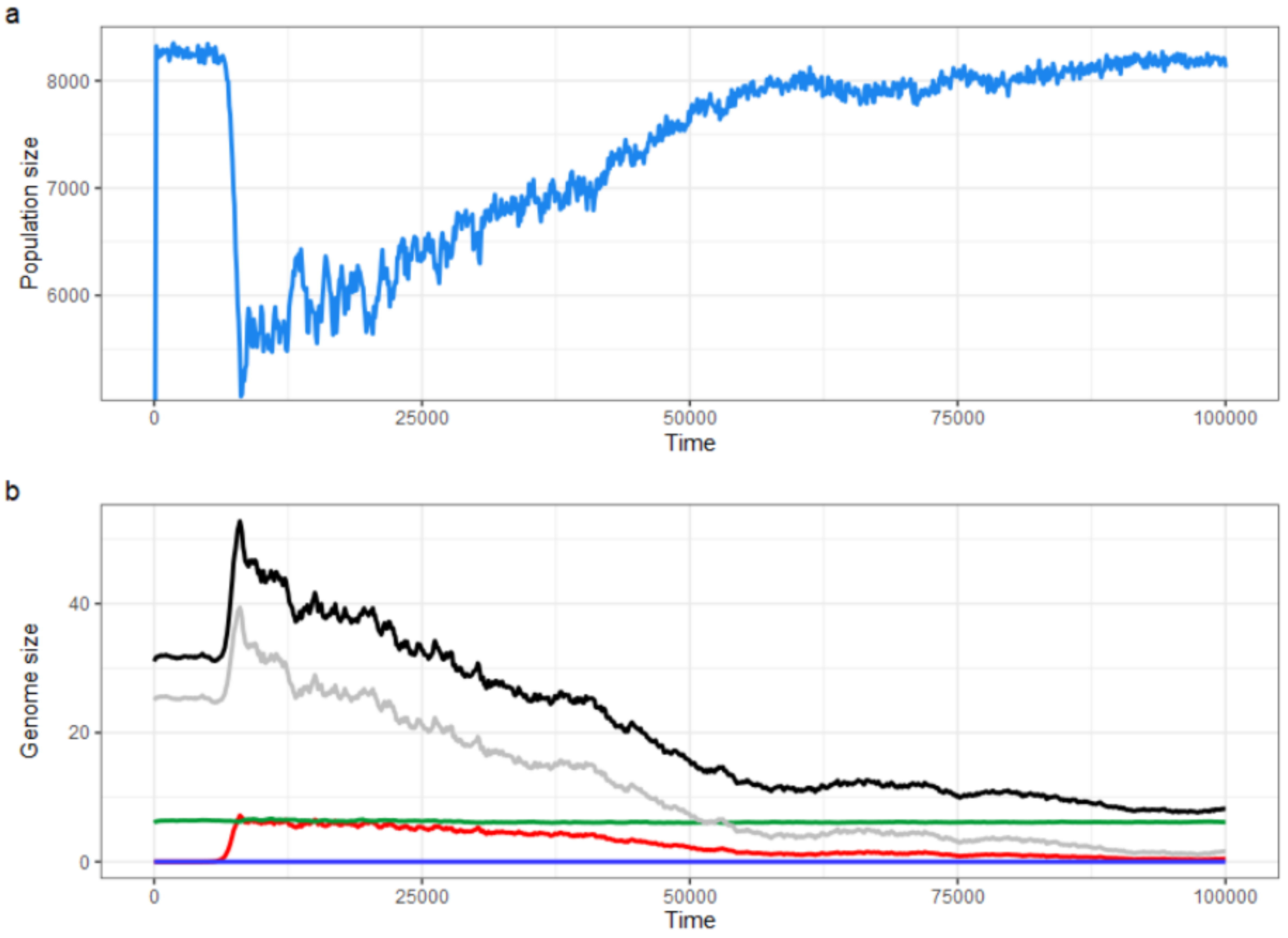
After uptake (rate **u**), integrate with chance p = ϕL ϕR

→ **You?**

What if jumping causes damage?

What if jumping causes damage?

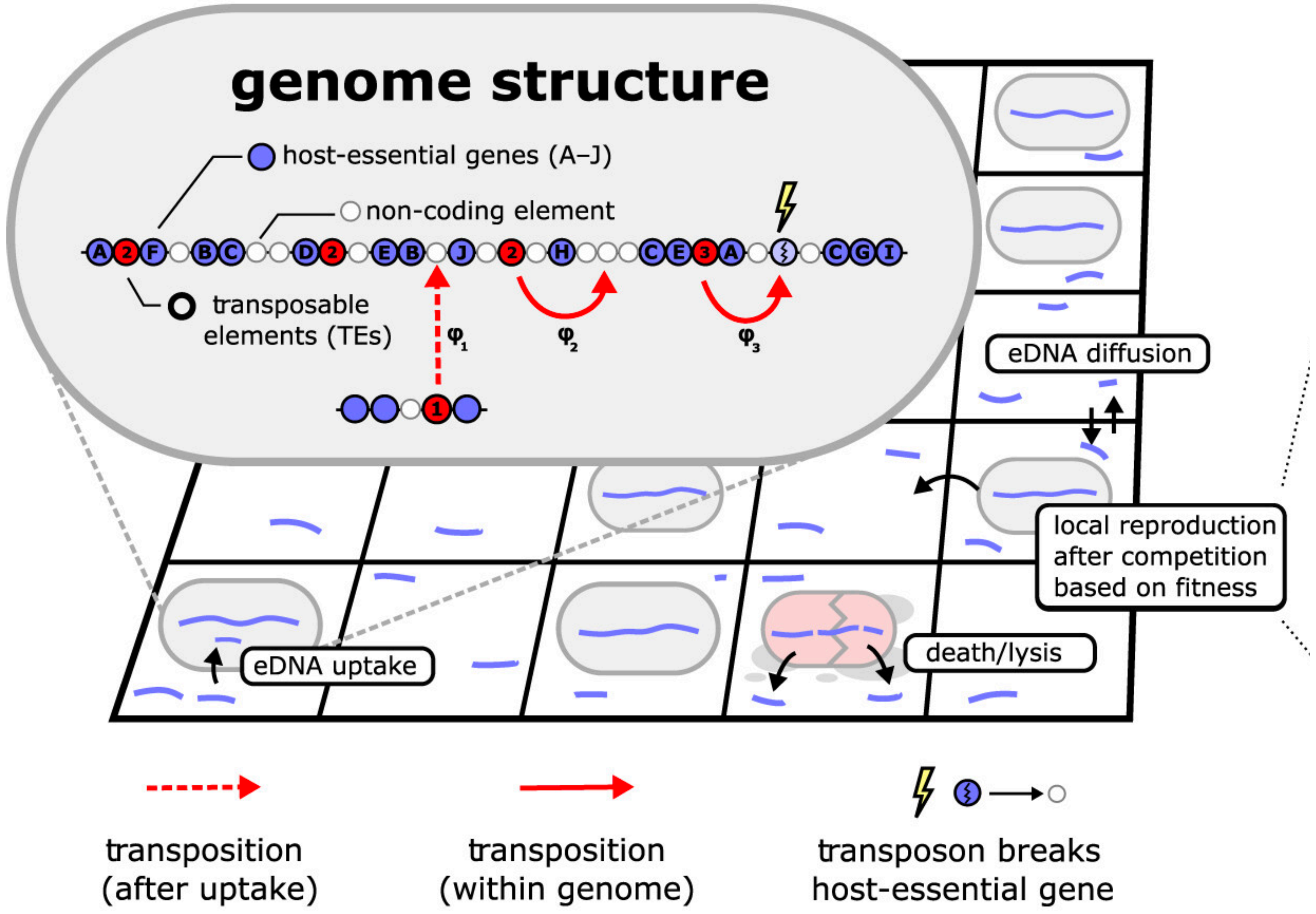




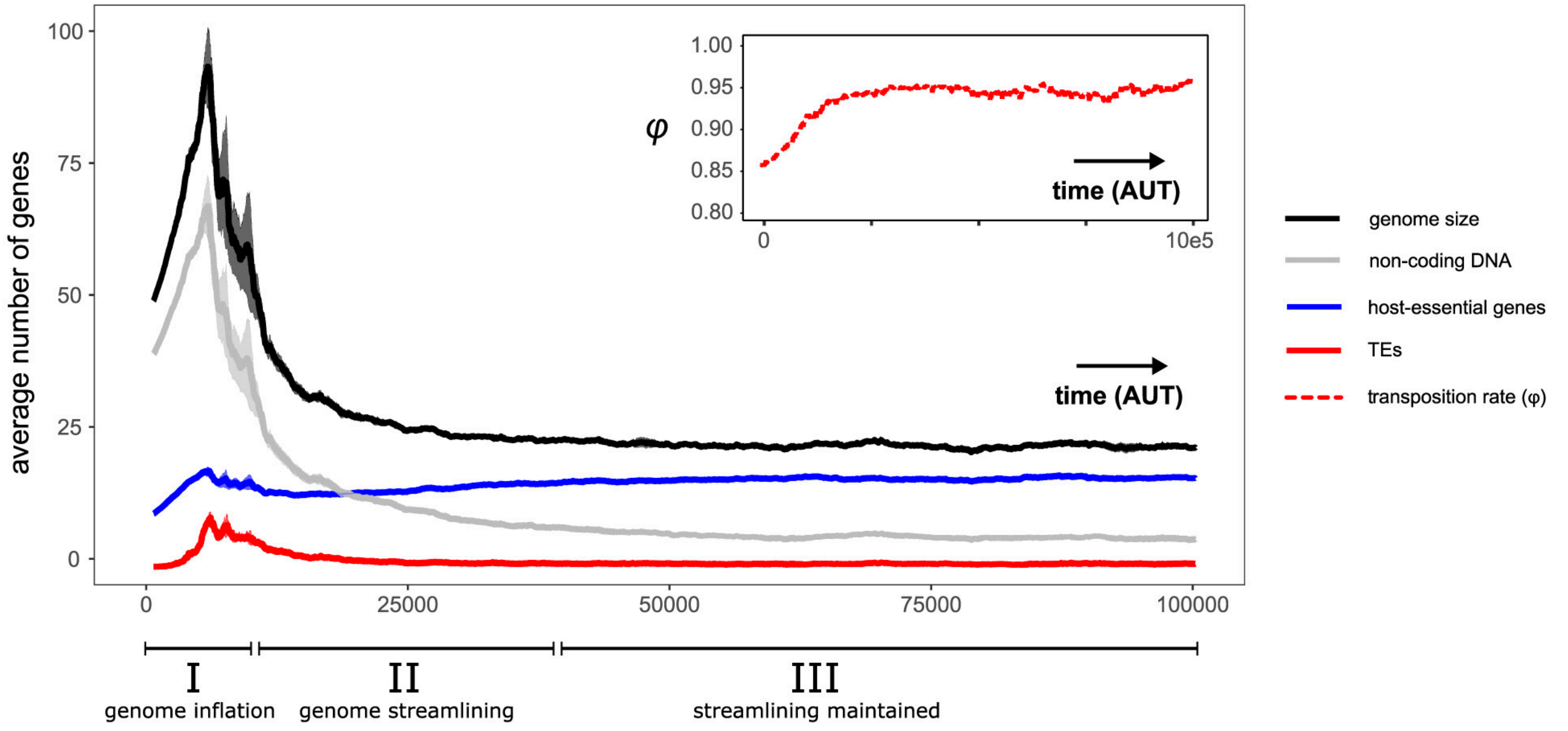
Genomes become more “streamlined” (smaller, less non-coding DNA)

This became a whole new story!

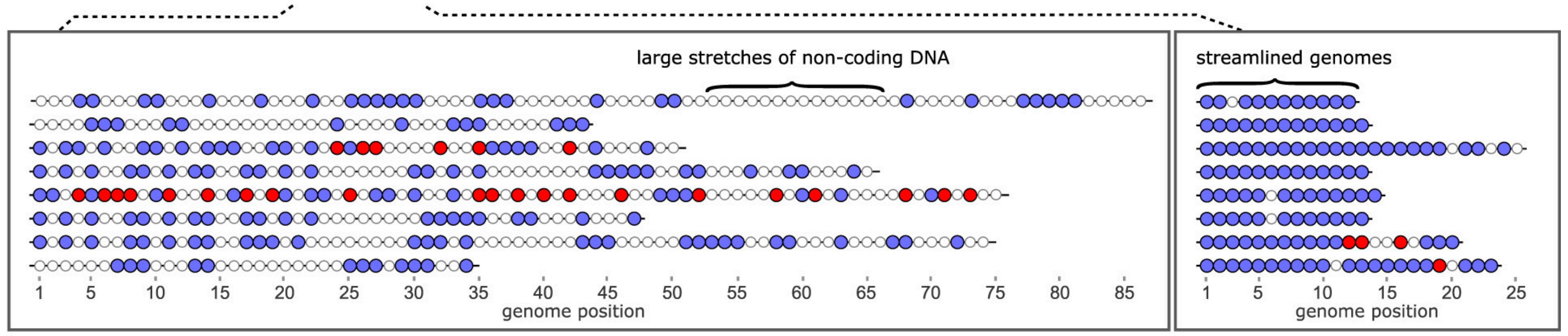
(*a*) **Individual-based model of TE/host co-evolution**



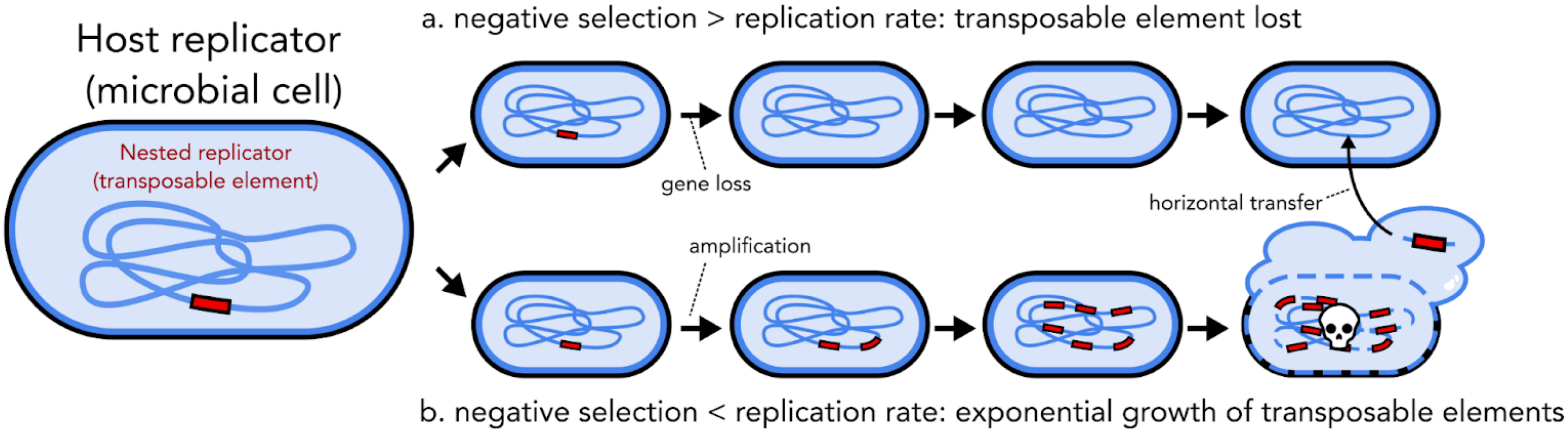
(*a*) **Genome streamlining evolves in spatially structured populations**



(*b*) **Examples of genomes before and after streamlining**



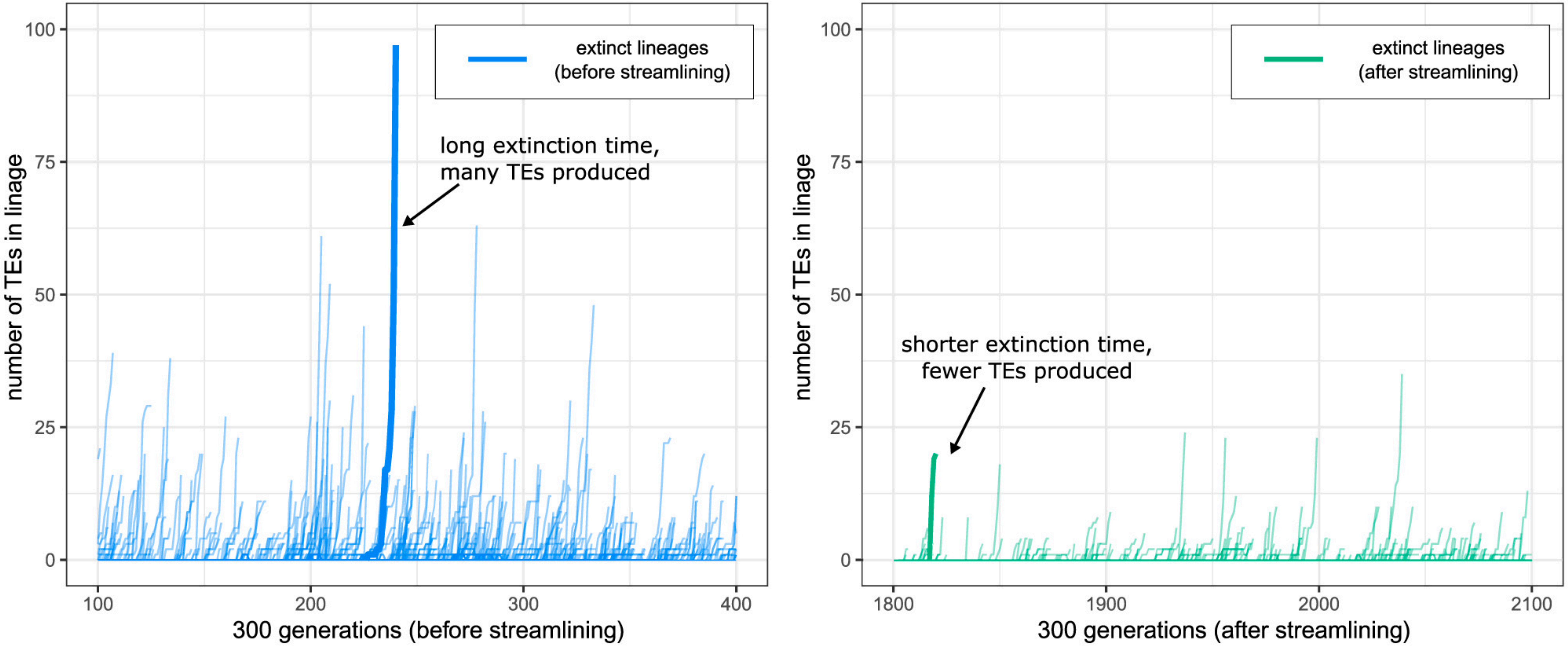
How long does it take to go extinct?



time-to-extinction

Evolving a shorter extinction time…?

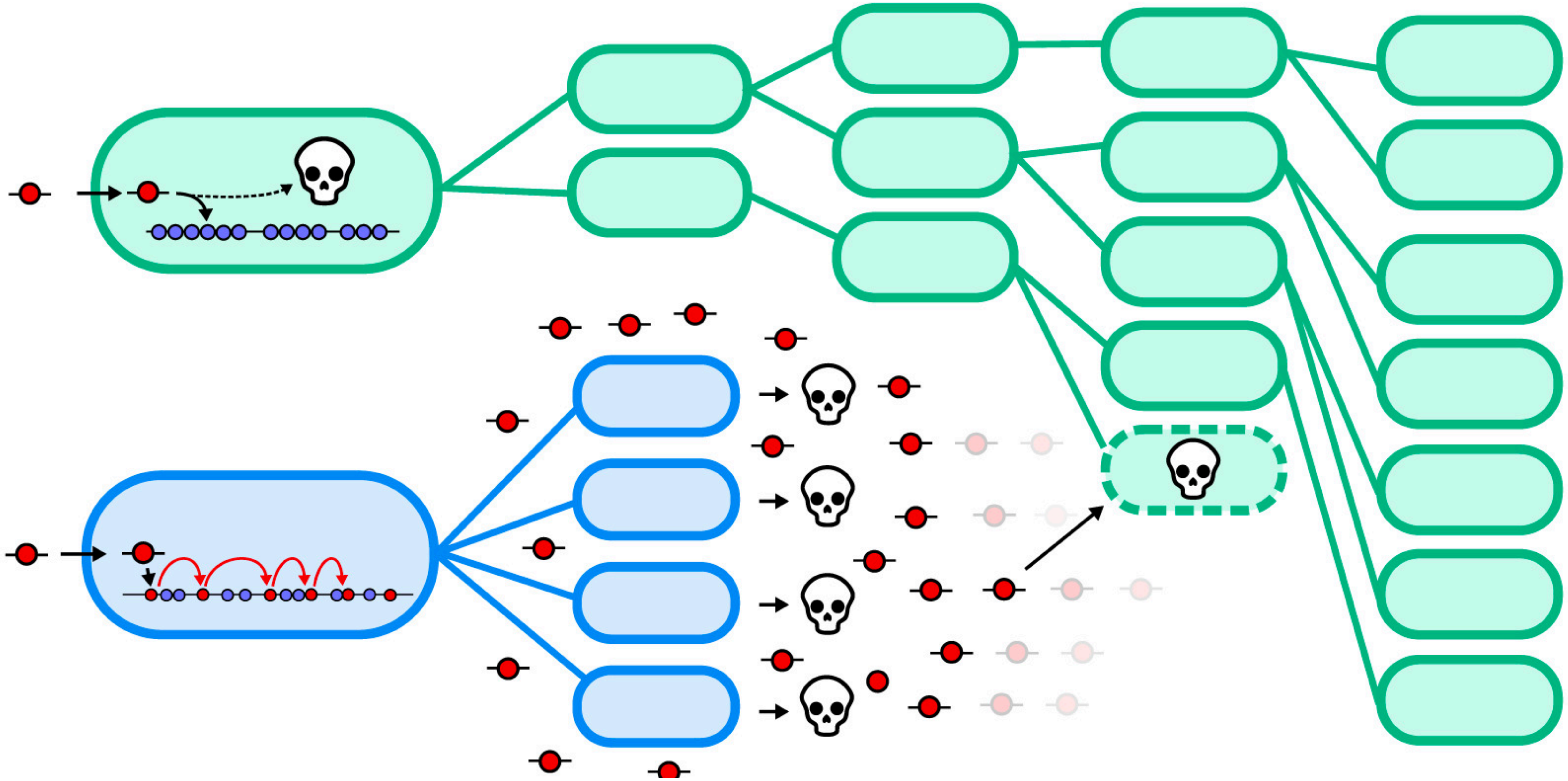
(*a*) **TE-abundance in extinct lineages before and after streamlining**



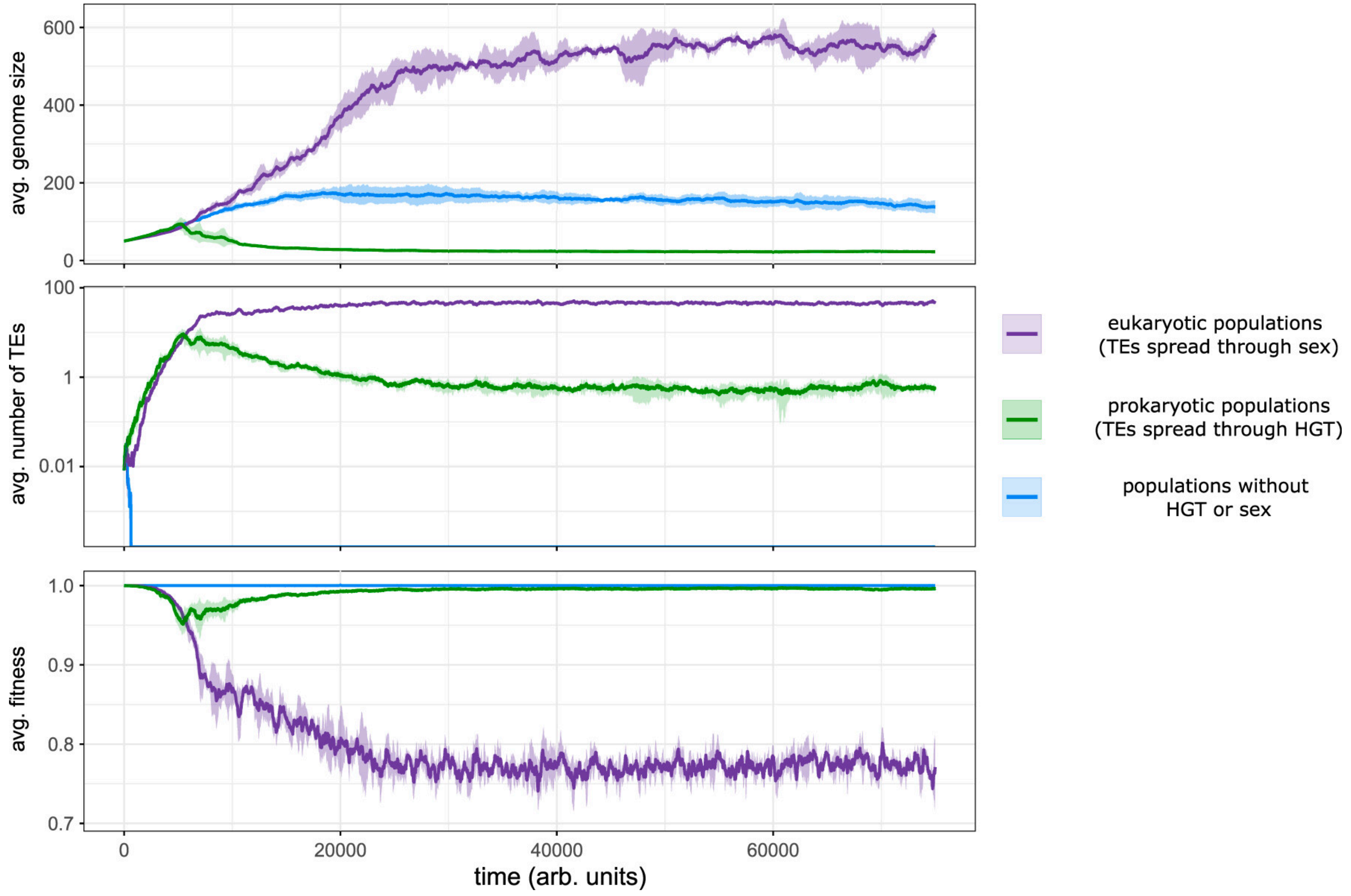
But wait, isn’t it better to take very long to go extinct?!

Immediately dying pays off: altruistic suicide!

(*c*) **Streamlined genomes outcompete non-streamlined genomes by preventing TE proliferation (cartoon)**



Not the same with sex!



- With sex, a TE doesn’t need to “jump into a gene” to transfer to another lineage!
- So streamlining doesn’t benefit the cells either!
- HGT and sex are **very** distinct processes!

Utrecht University

Part 1 - Horizontal- vs vertical transmission of genes and microbiomes

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}
else{
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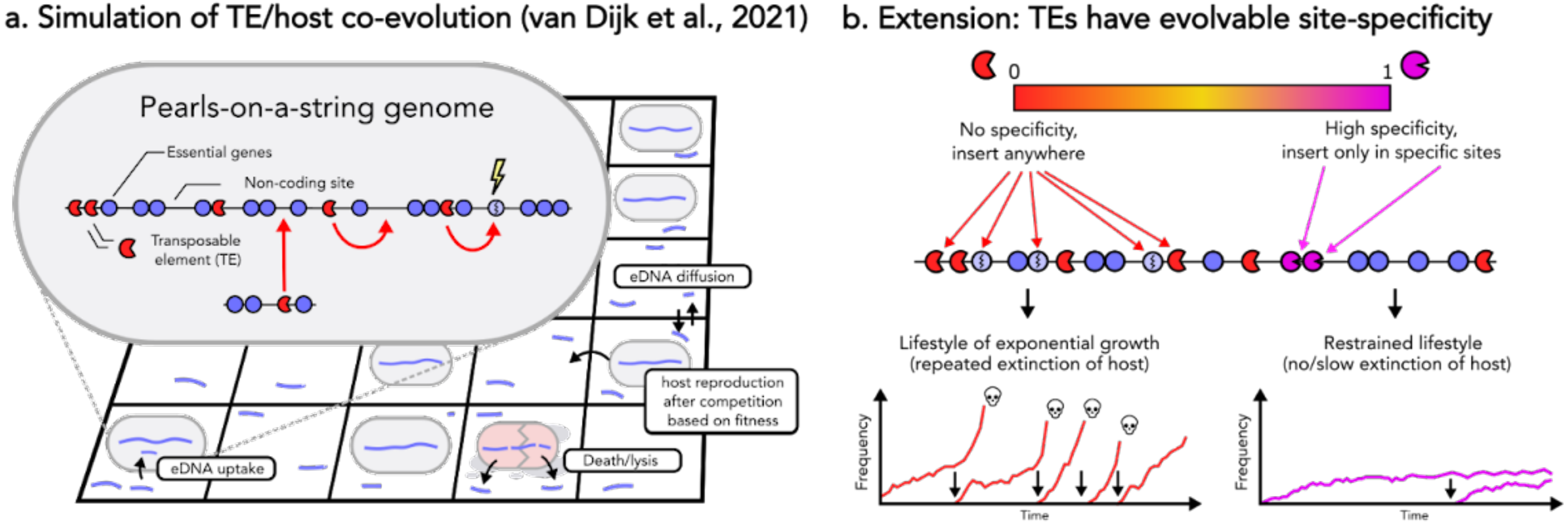
}
```

What about more “clever" TEs?

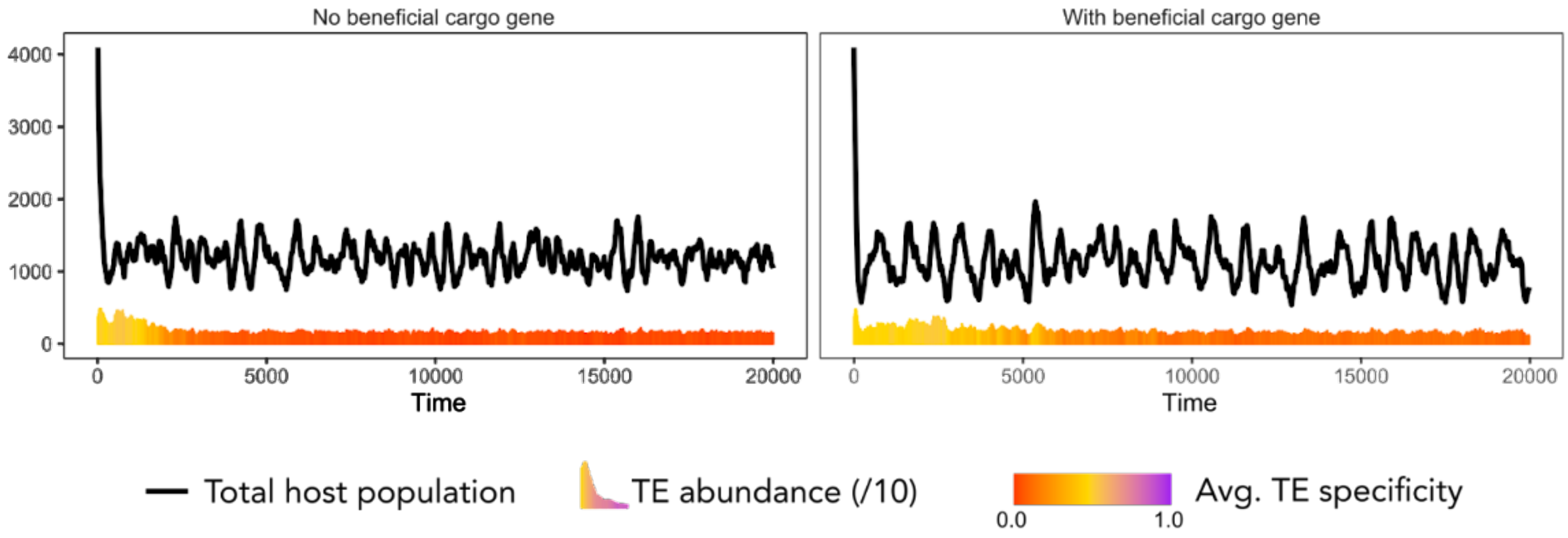

a. Simulation of TE/host co-evolution (van Dijk et al., 2021)
b. Extension: TEs have evolvable site-specificity

Every “pearl" carries a insertion-site parameter between 0 and 1
Every transposon additionally has a target-site between 0 and 1
Highly specific TEs care about this (has to match). Non-specific TEs don’t.

Transposons can evolve to be nasty (non-specific) or show some restraint (high specificity)

Dynamics with/without a beneficial gene

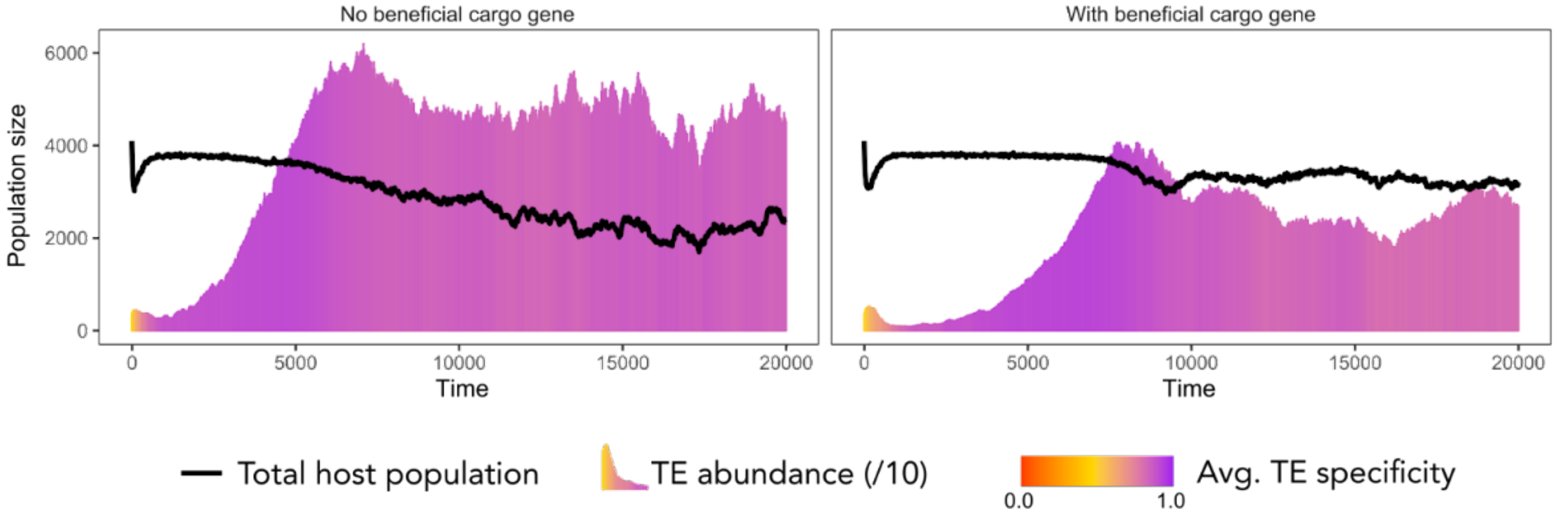
- Every TE copy reduces fitness with 0.02 (starting from 1)
- Optionally, having 1 or more copies give a 0.12 fitness benefit (cargo gene)



This is with high uptake of eDNA

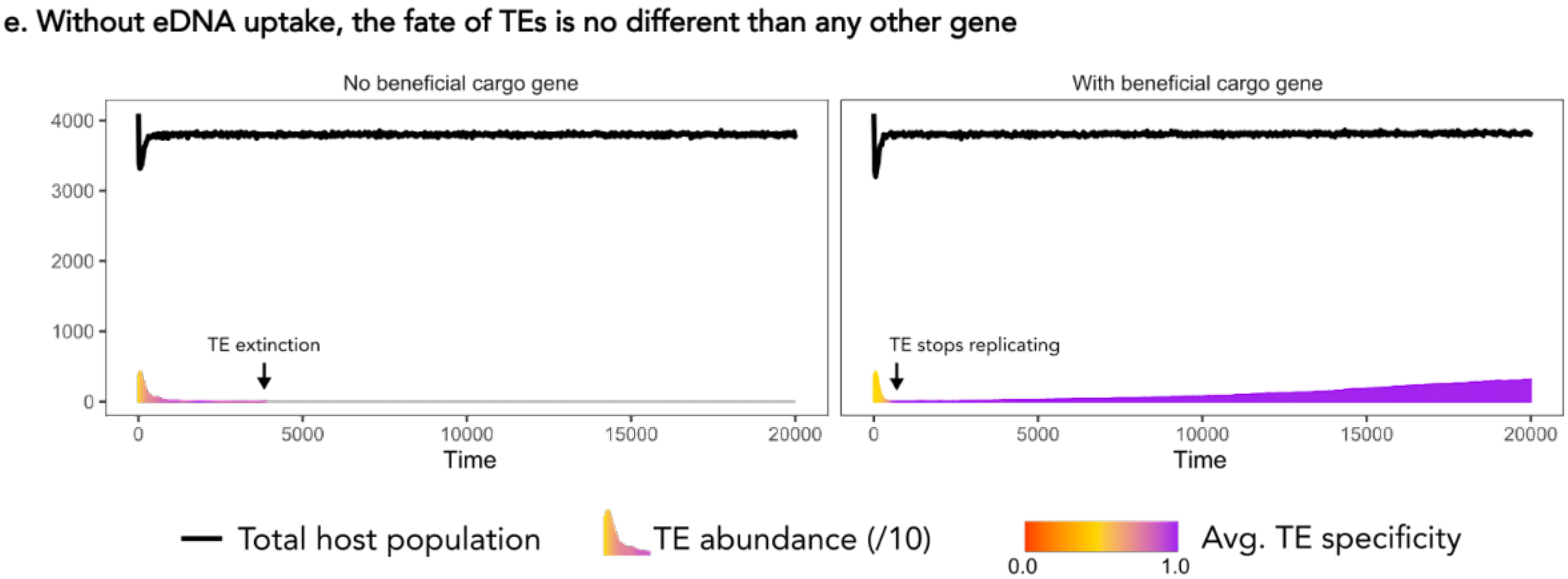
With lower HGT rates…

d. At low eDNA uptake (0.01), highly specific TEs evolve



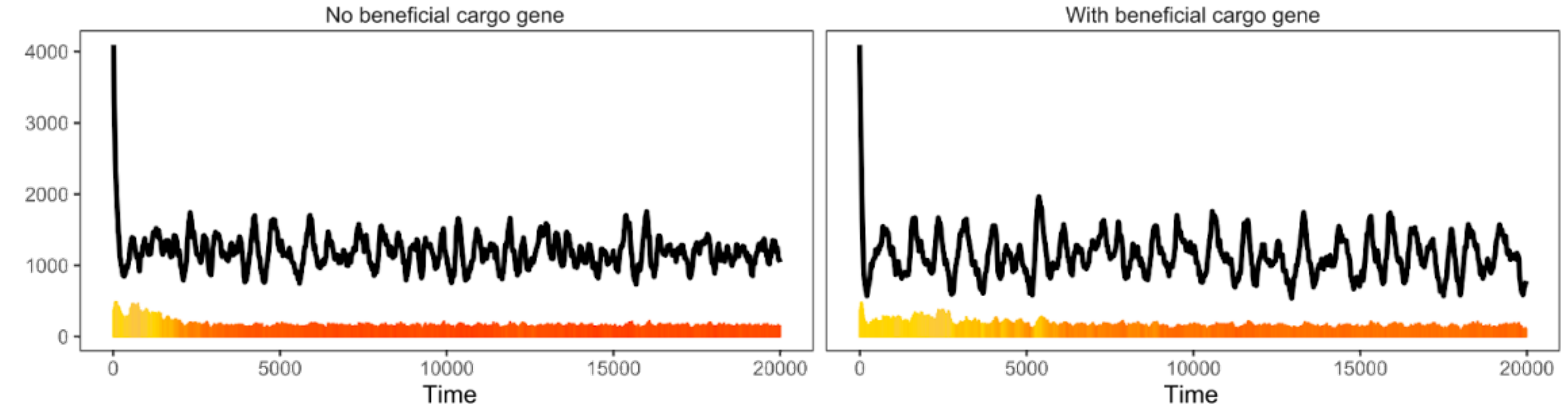
Can anyone guess why this didn’t happen at high HGT?

Without HGT altogether

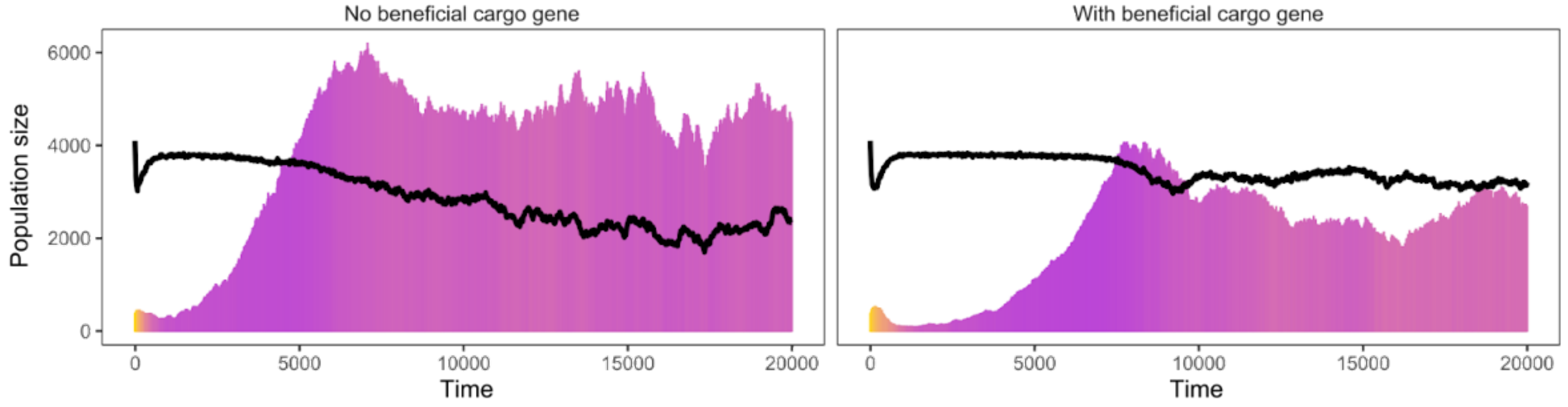


Side-by-side comparison

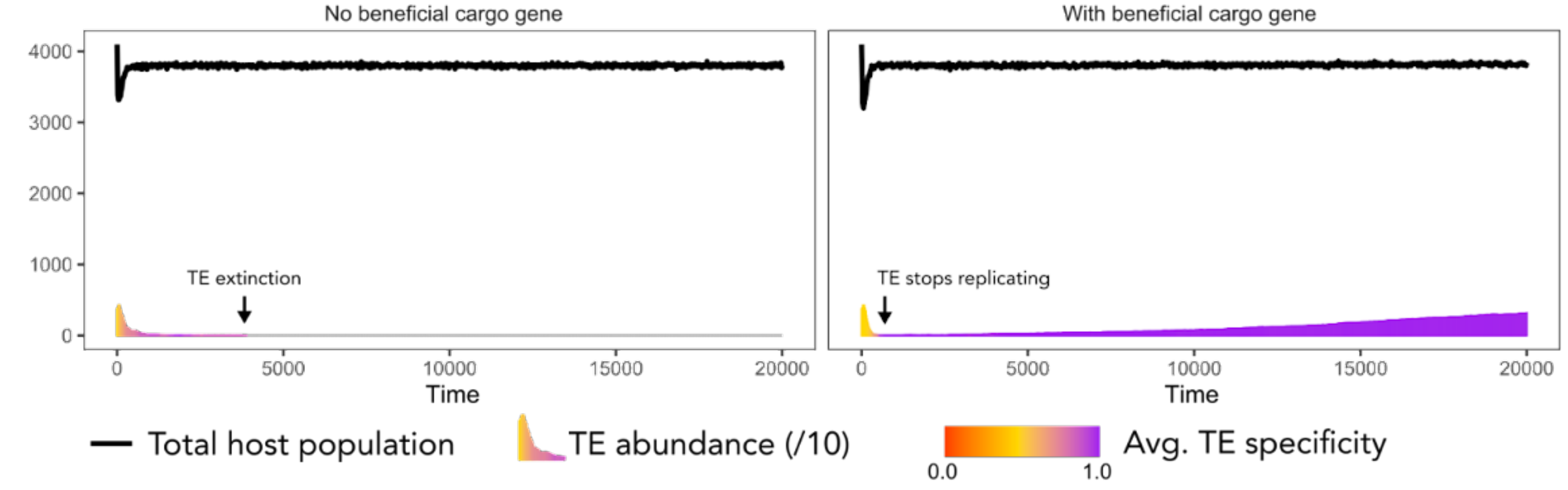
c. With high eDNA uptake (0.05), non-specific TEs evolve regardless of the beneficial cargo gene

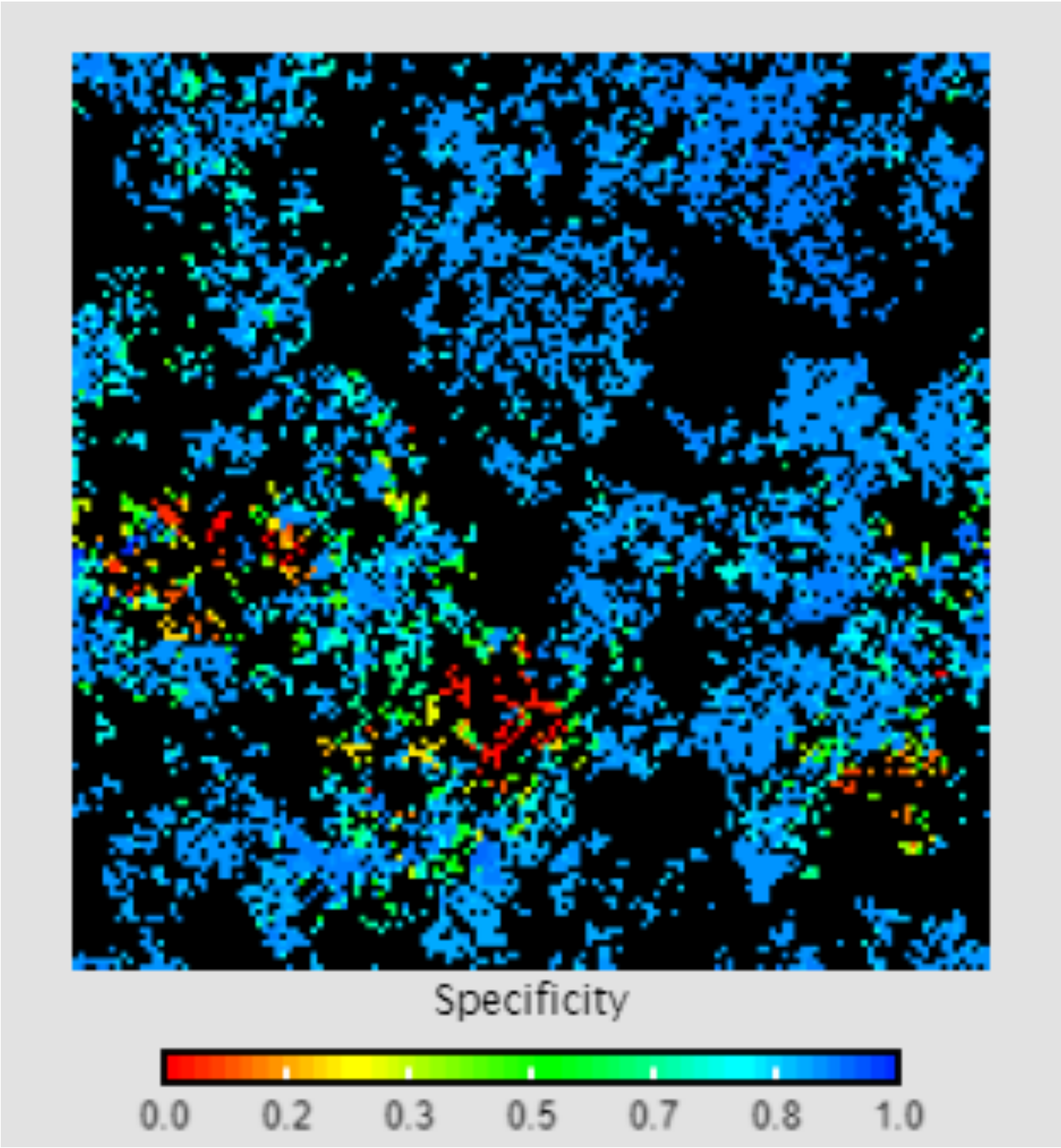


d. At low eDNA uptake (0.01), highly specific TEs evolve



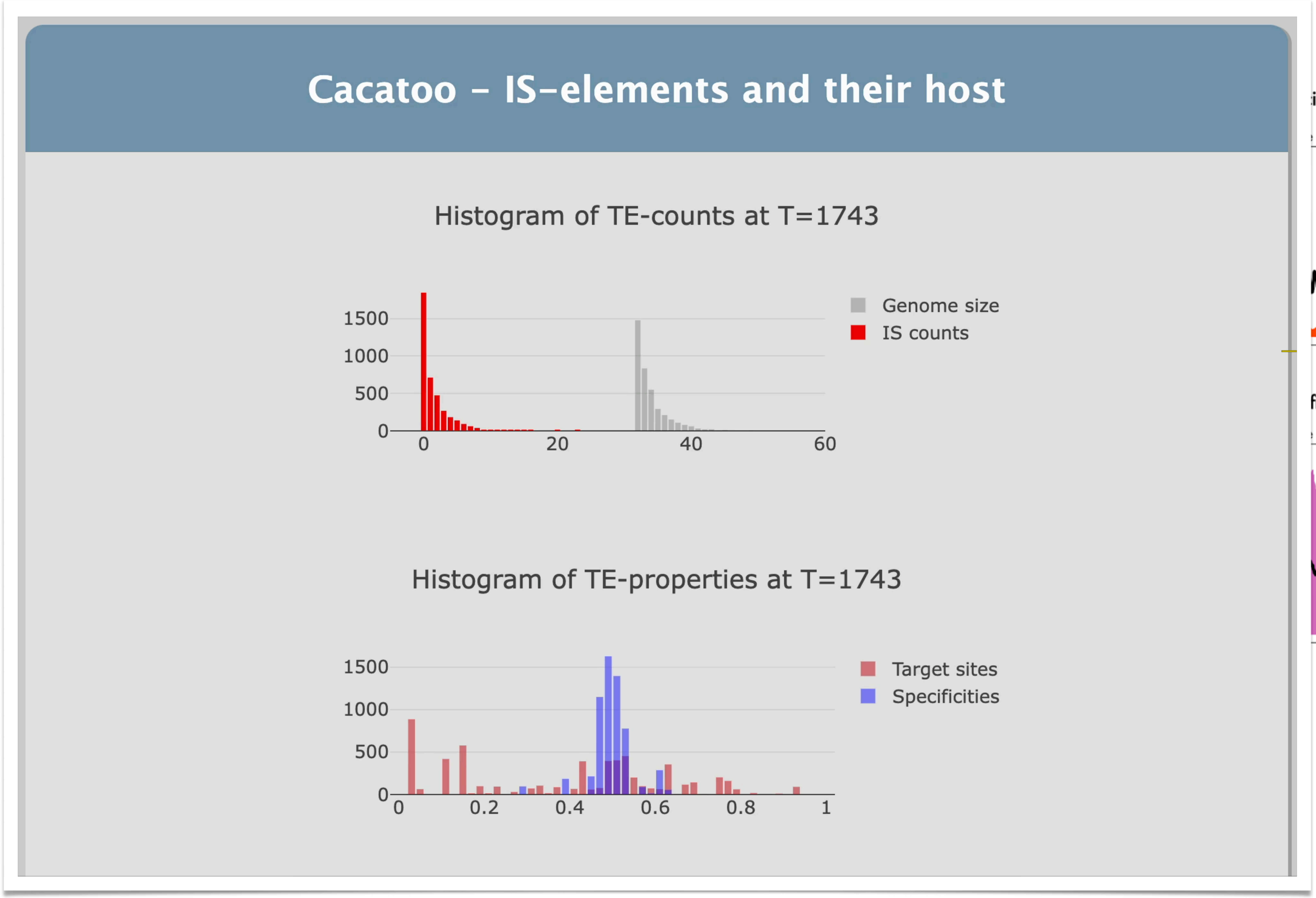
e. Without eDNA uptake, the fate of TEs is no different than any other gene





Both types coexist...? :o

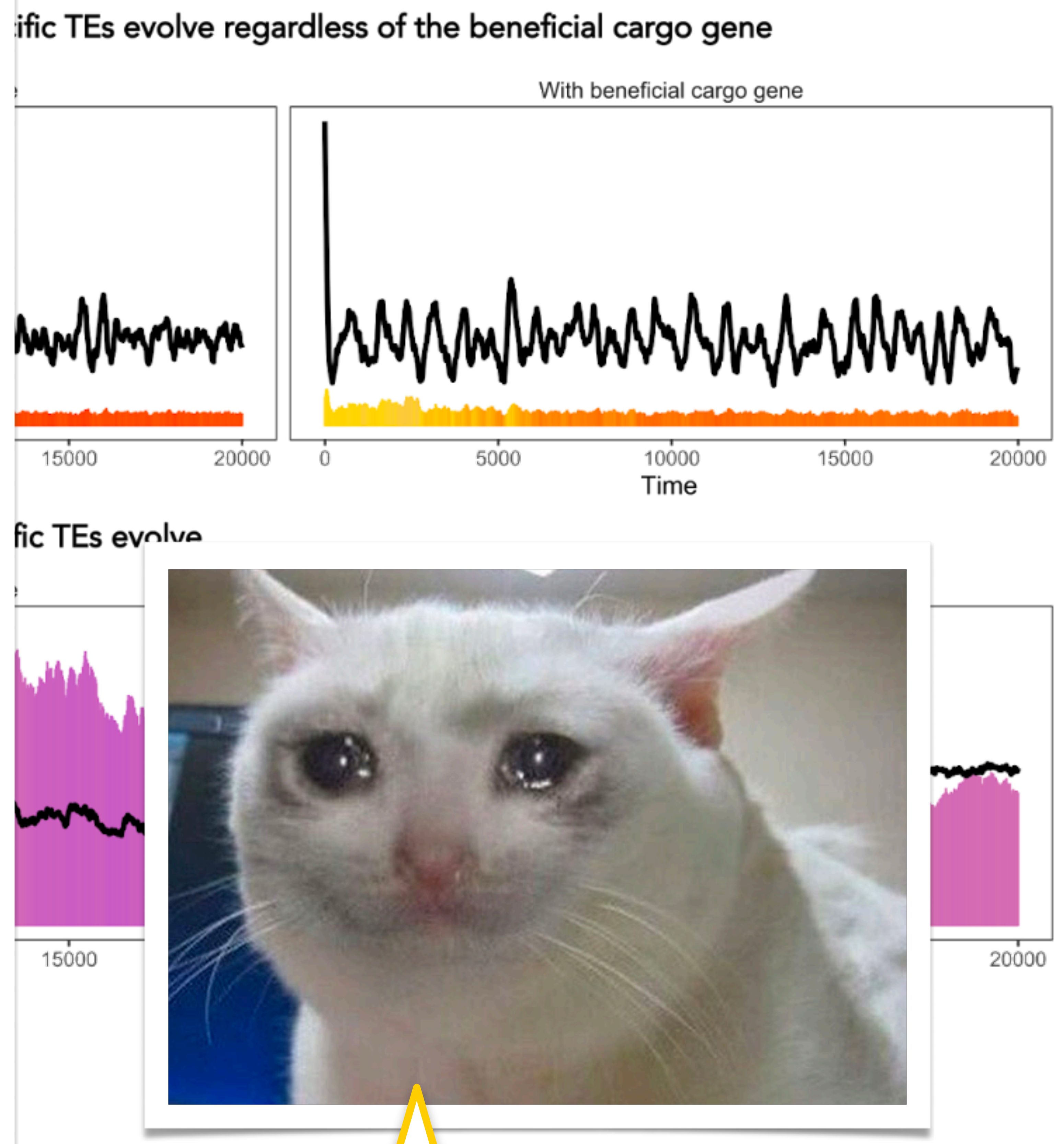
Sneak peak:



Cacatoo – IS-elements and their host

Histogram of TE-counts at T=1743

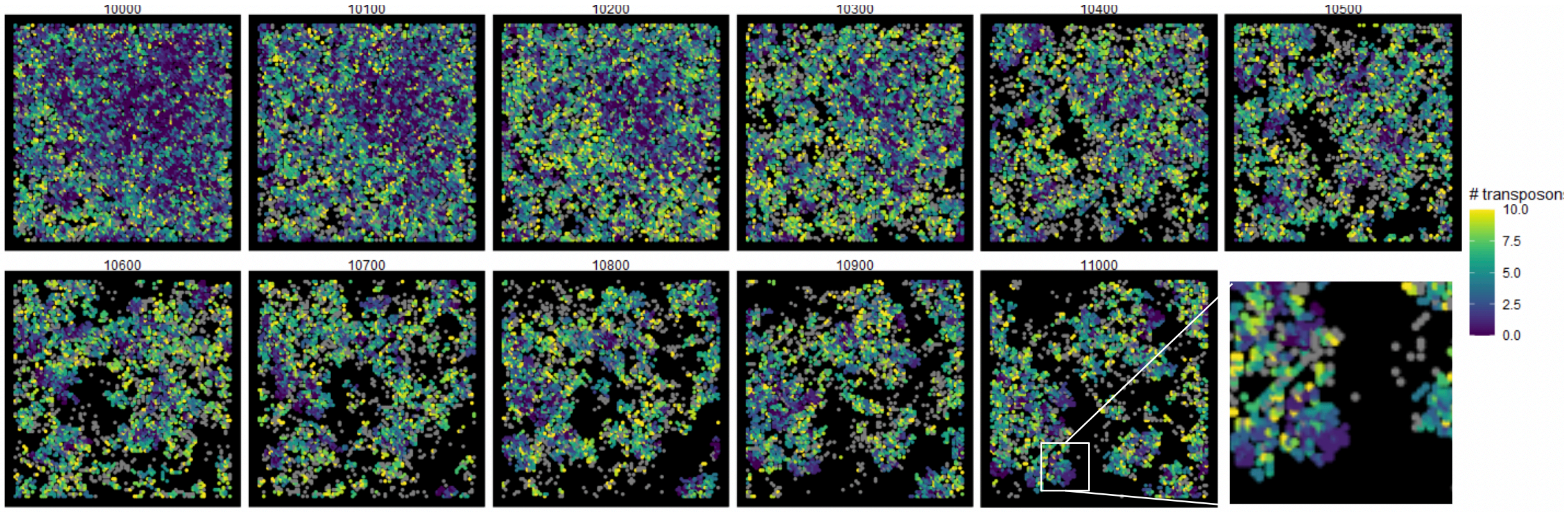
Histogram of TE-properties at T=1743



Help pls!

Conclusions Part Ib

- Unclear why TEs are often associated with useful (or at least ecologically relevant) genes. What are my models missing?
- The “resource” on which TEs grow is non-coding DNA
- Reducing non-coding DNA can prevent TEs from taking over
- This requires group-level effects (it only works in space)

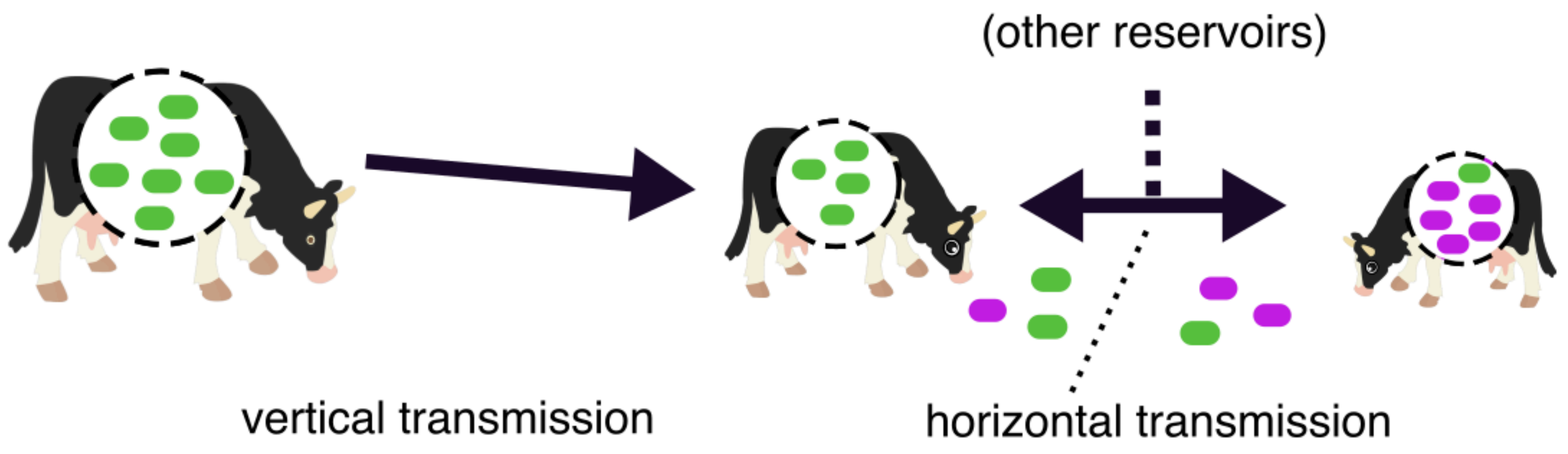


Intermezzo: setting yourself up for surprise

- As a modeller, it can be hard to decide what **NOT** to put into your model (especially if you enjoy programming, talking to you ALKMINI!!1!)
- But: by adding complexity and degrees of freedom, you allow a model to surprise you!
- This <u>can</u> reduce your bias (we can't nullify it of course, but it helps)

Horizontal vs. vertical transmission of microbiomes, similar rules?

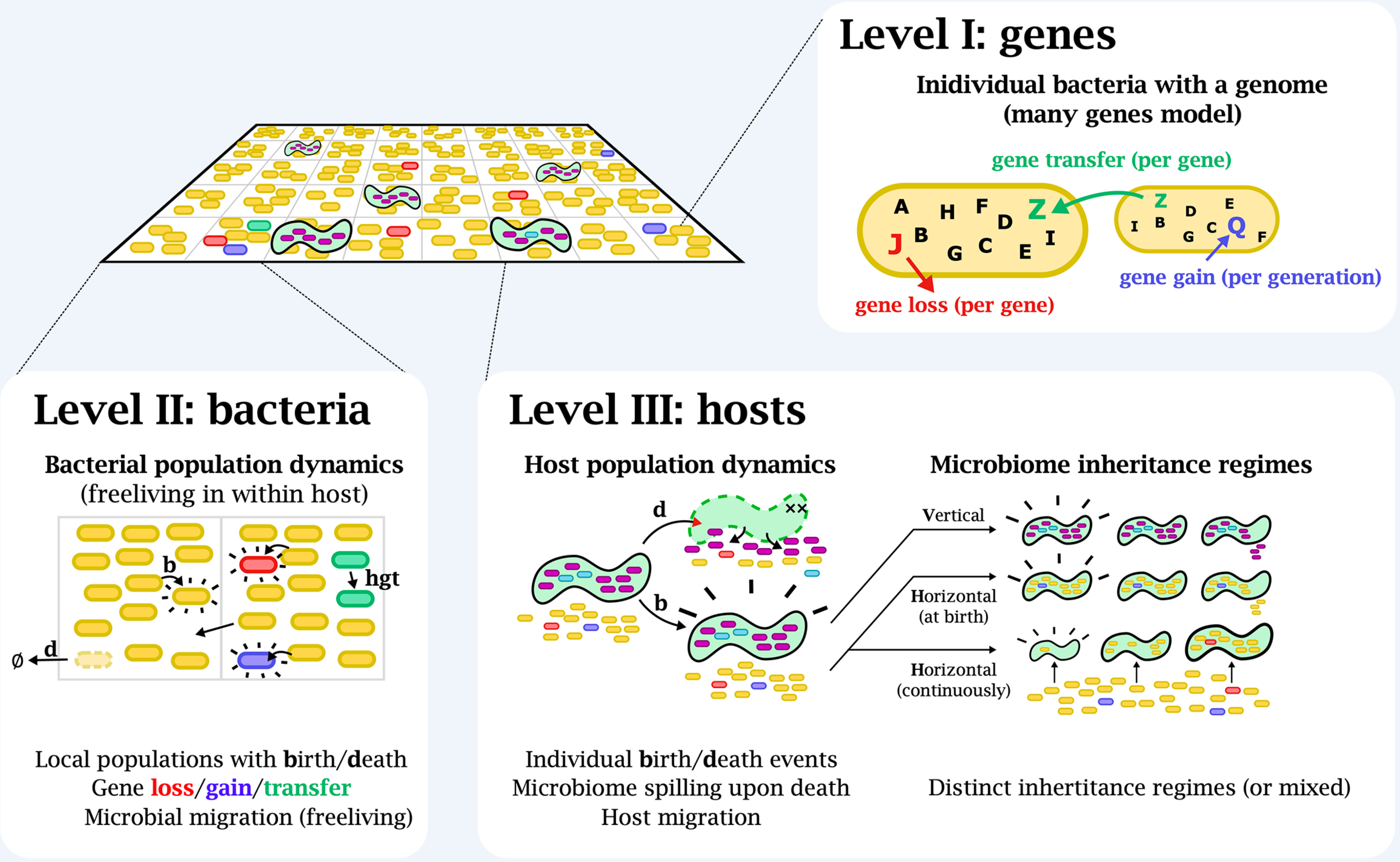
- Microbiomes can be inherited from your parents (vertical inheritance) or from the environment (horizontal inheritance)
- Do the rules we found for HGT apply here too?



Getting microbes from your mom, or from the environment

IBM of symbiont-host coevolution

(three nested biological entities: genes, bacteria, and hosts)



Level I: genes

Inidividual bacteria with a genome (many genes model)

Level II: bacteria

Bacterial population dynamics
(freeliving in within host)

Local populations with **b**irth/**d**eath
Gene loss/gain/transfer
Microbial migration (freeliving)

Level III: hosts

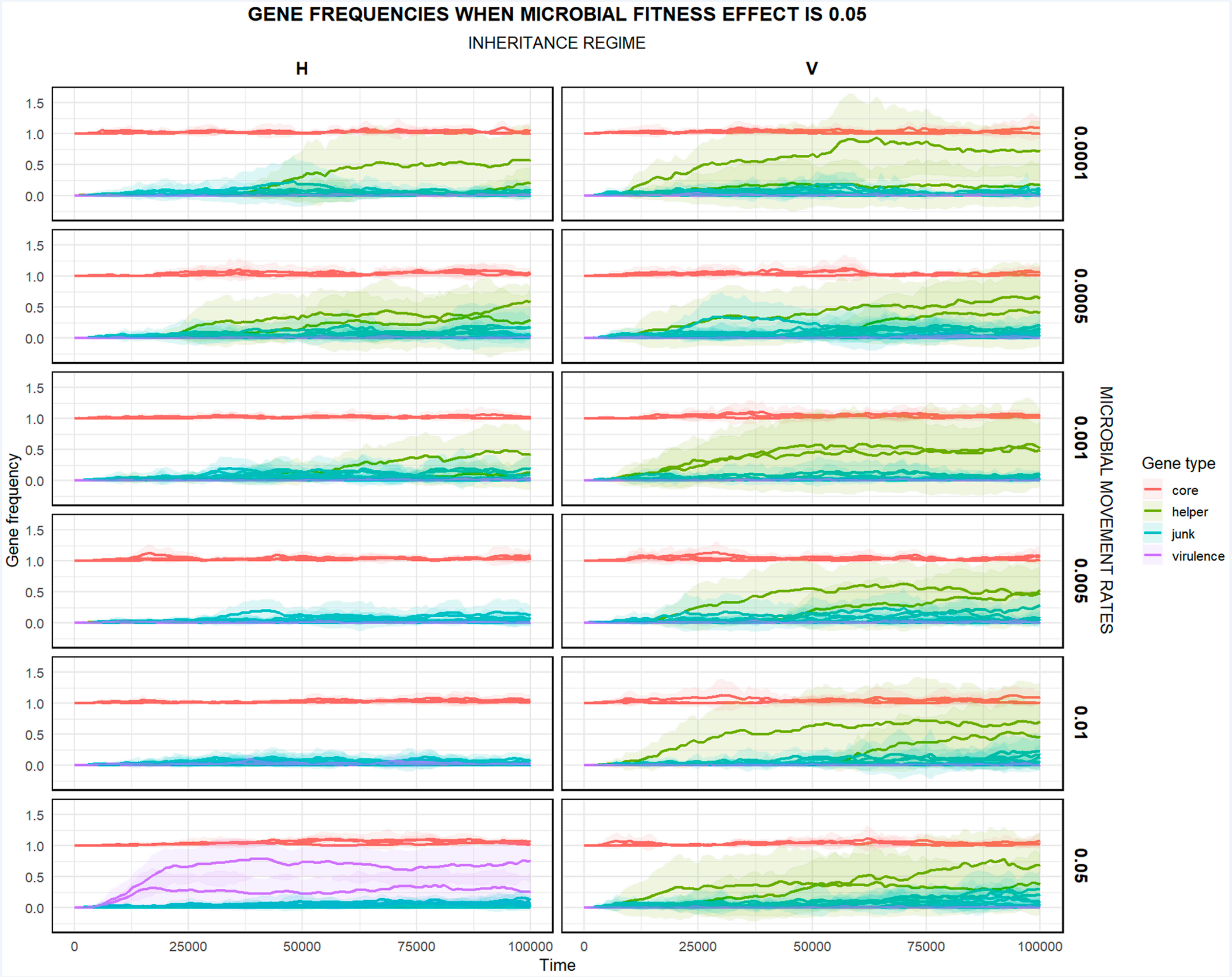
Host population dynamics

Microbiome inheritance regimes

Individual birth/death events
Microbiome spilling upon death
Host migration

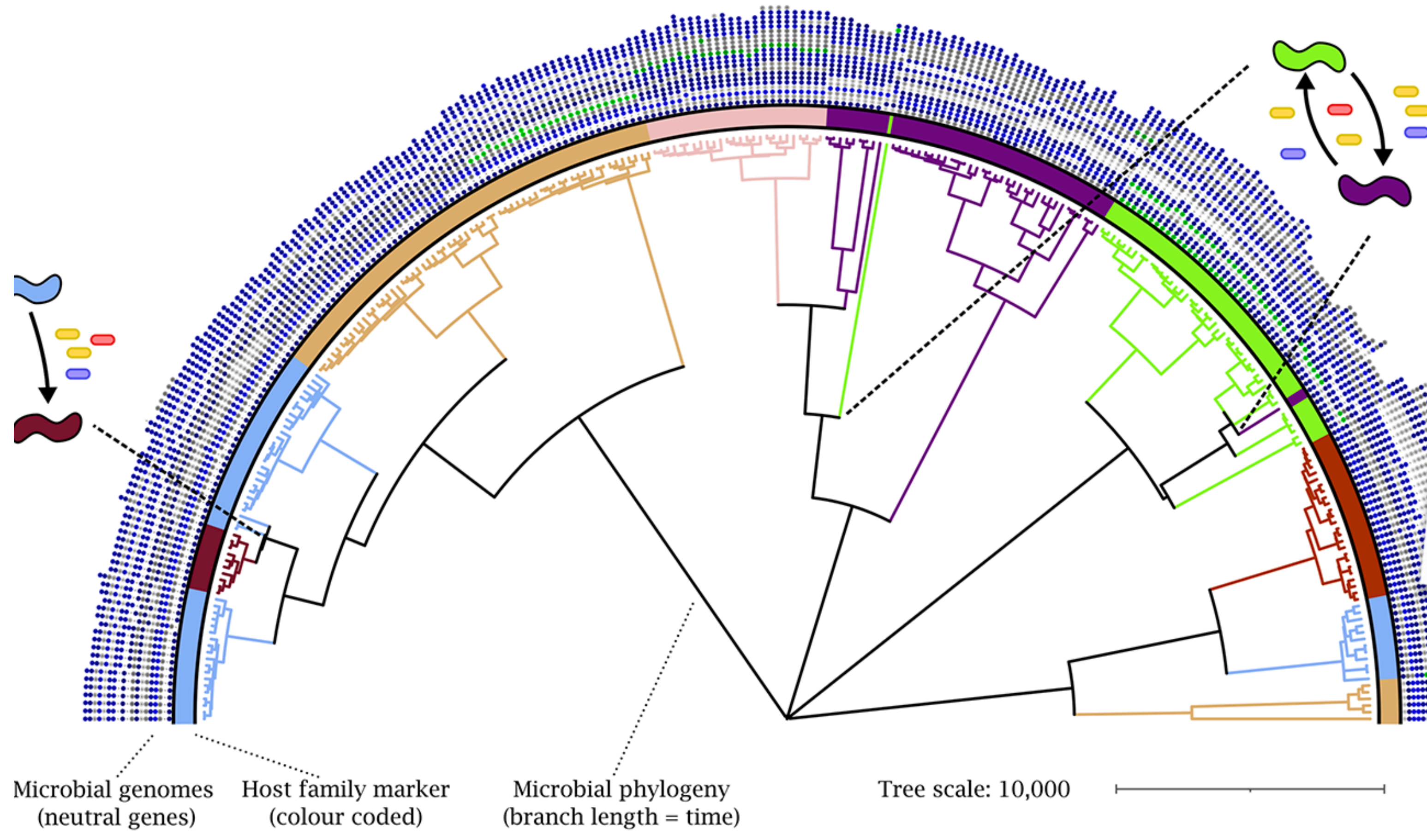
Distinct inhertittance regimes (or mixed)

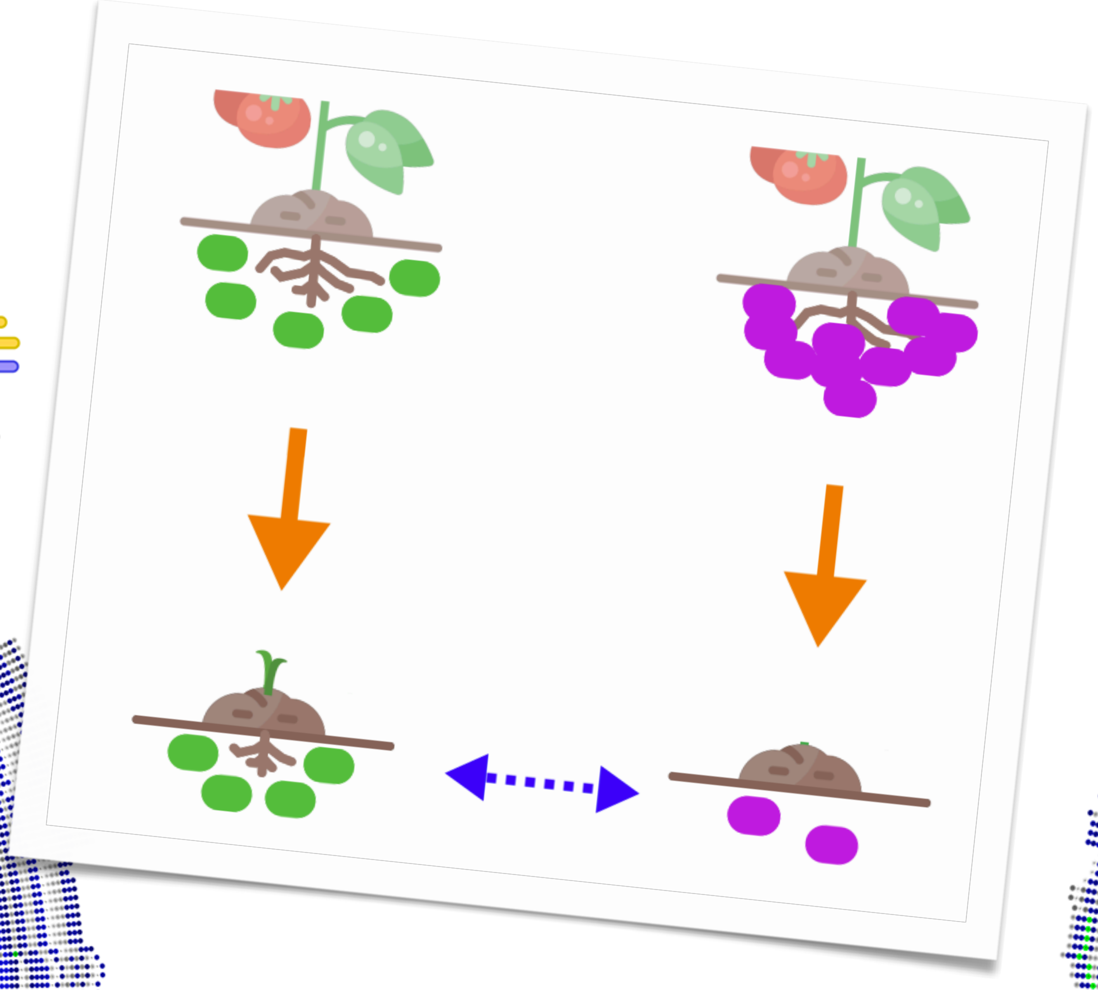
Virulence only emerges with horizontal (H) transmission, and only when microbial migration is high



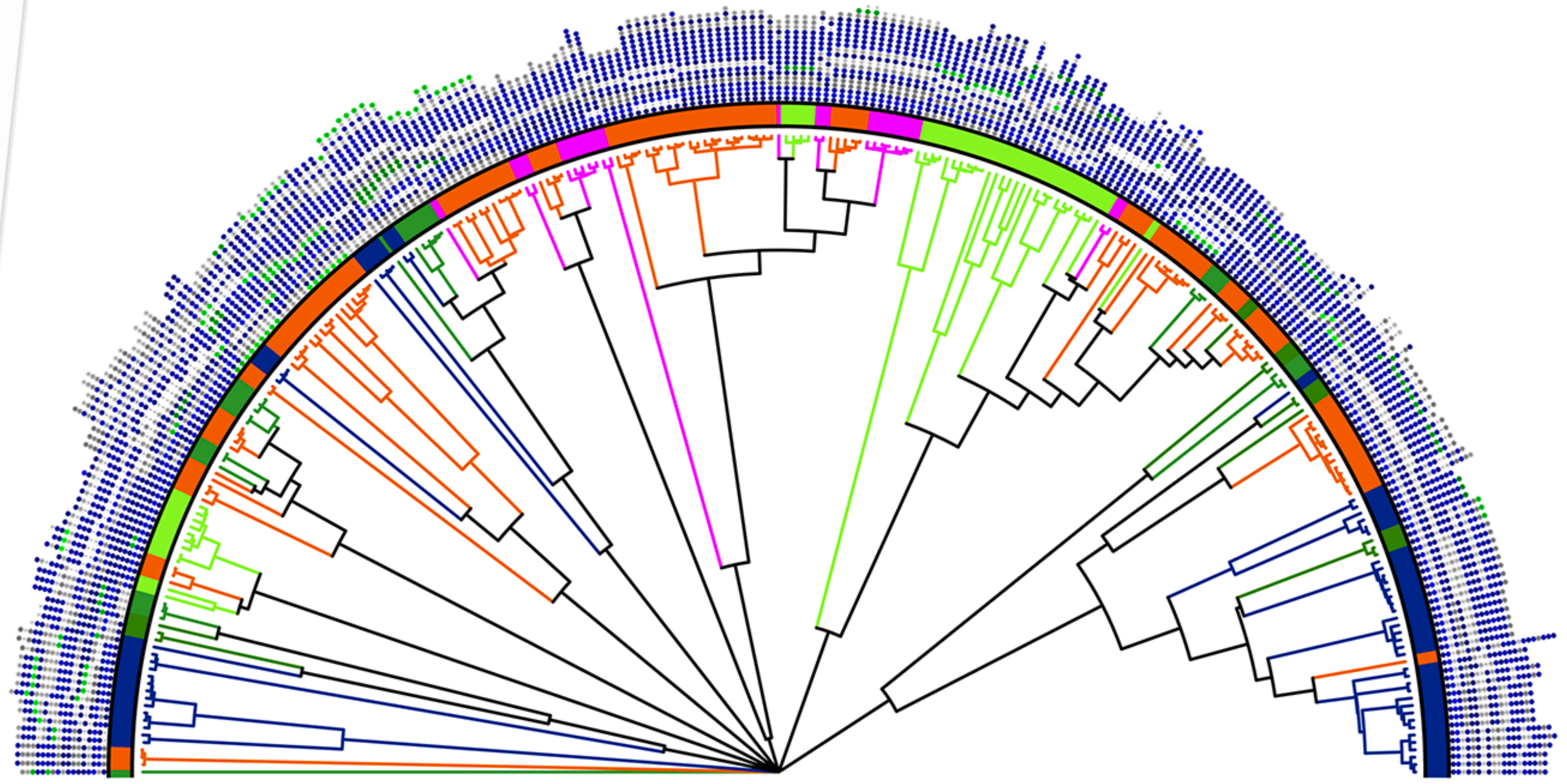
Environmental and **horizontal transmission** are different things!

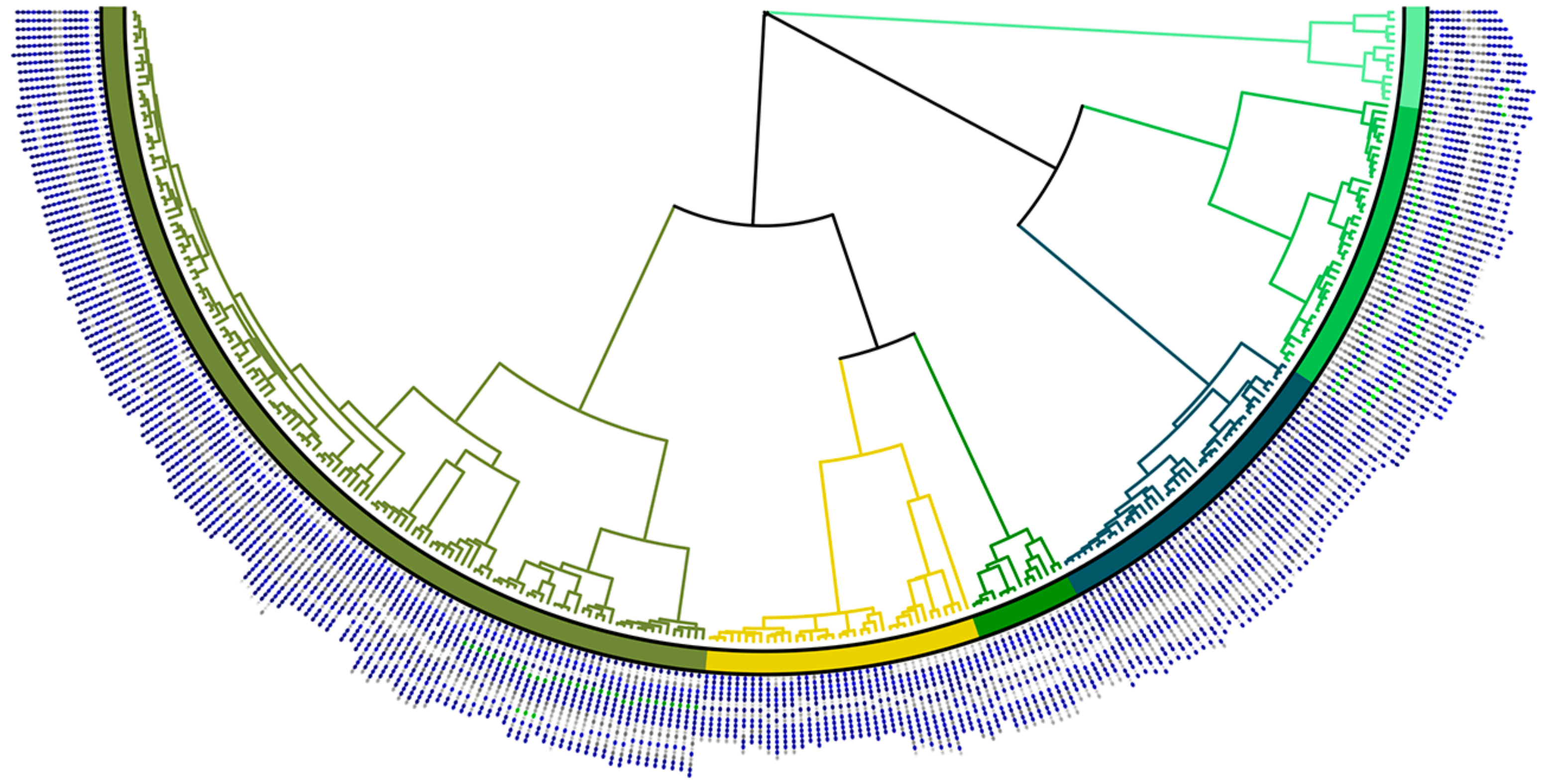
~~Horizontal~~ (low migration)



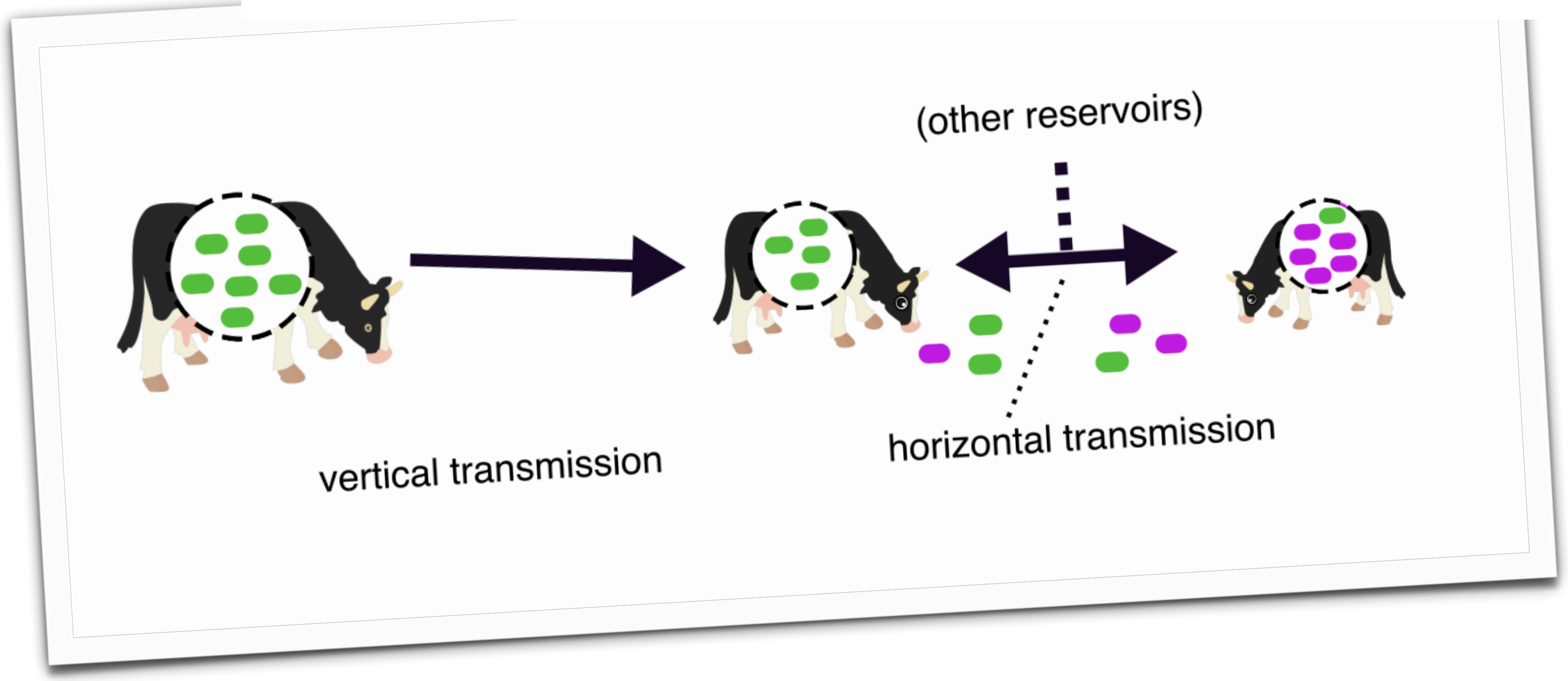


Horizontal (high migration)



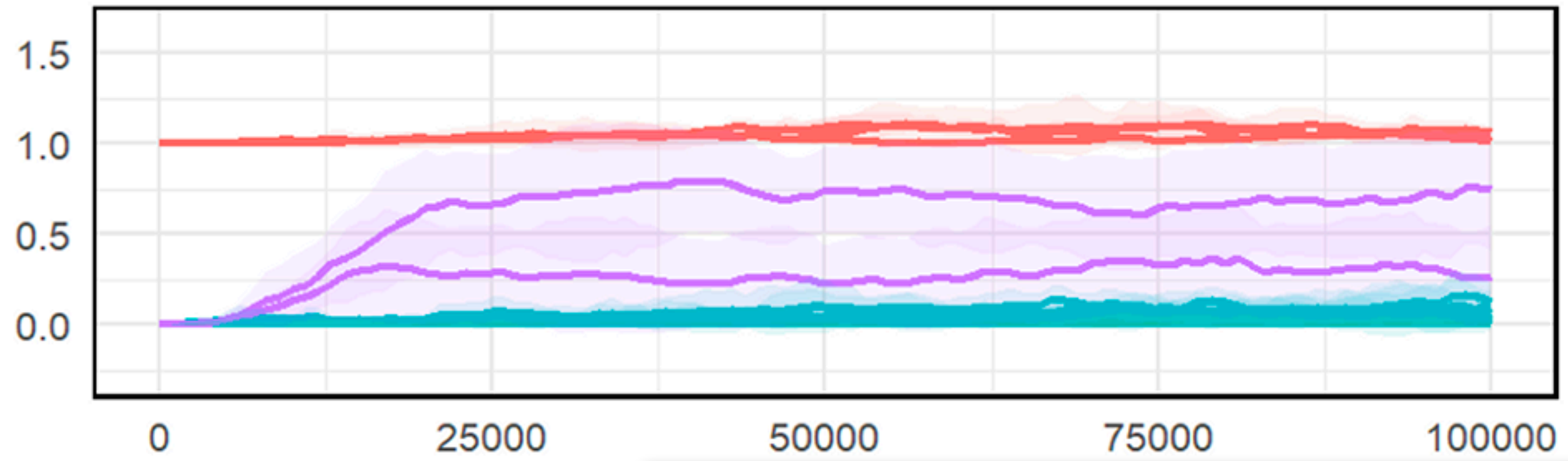


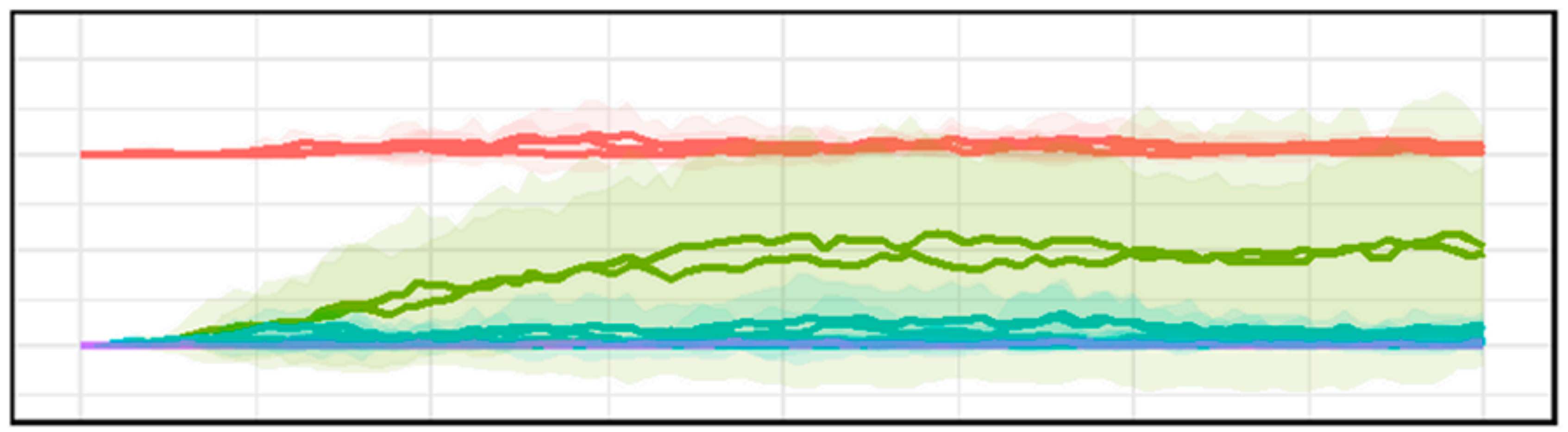
Vertical



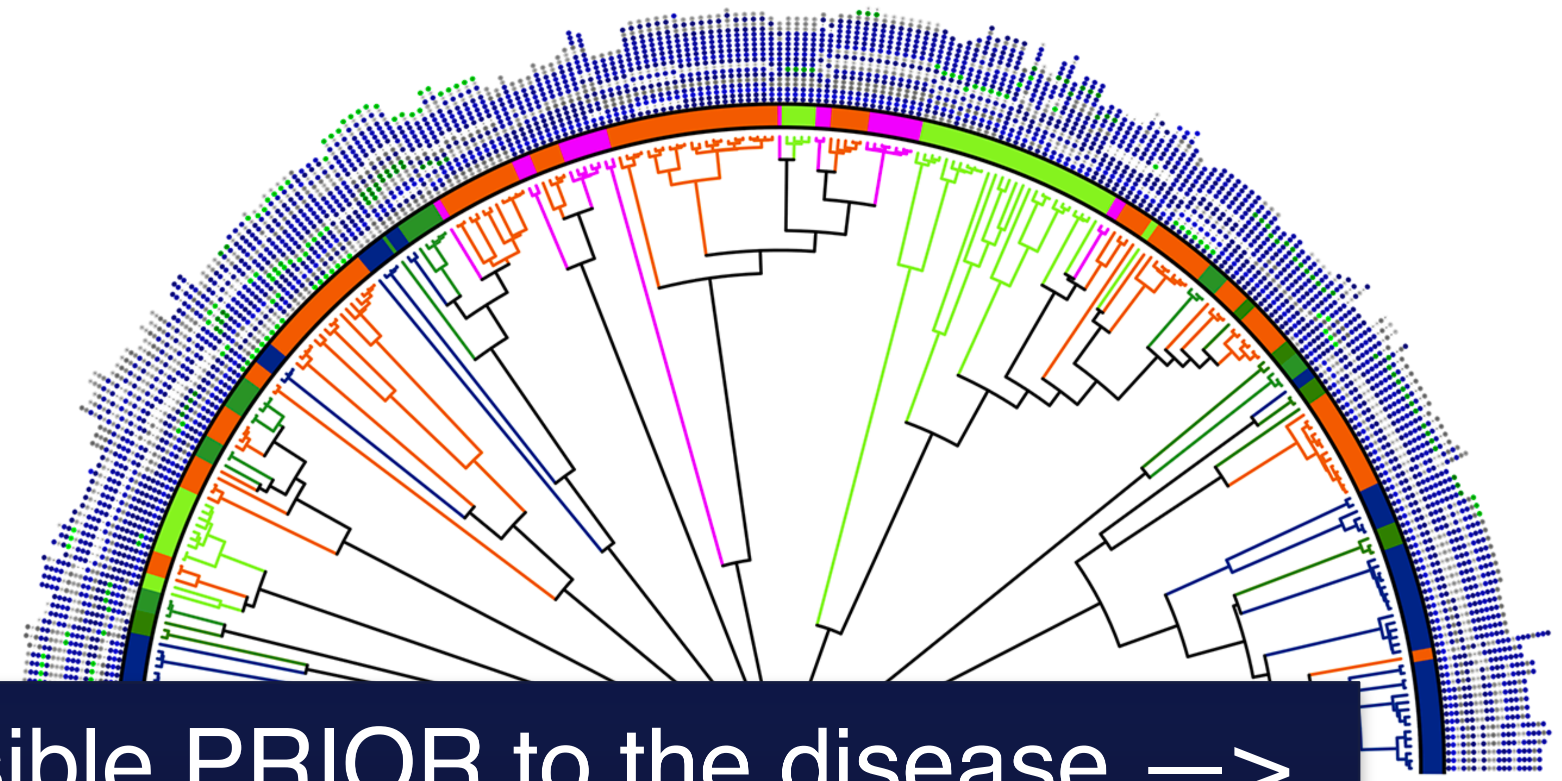
Animals need an adaptive immune system?

Once again: **horizontal** vs. **vertical** transmission modes have a massive impact on the **phylogeny**

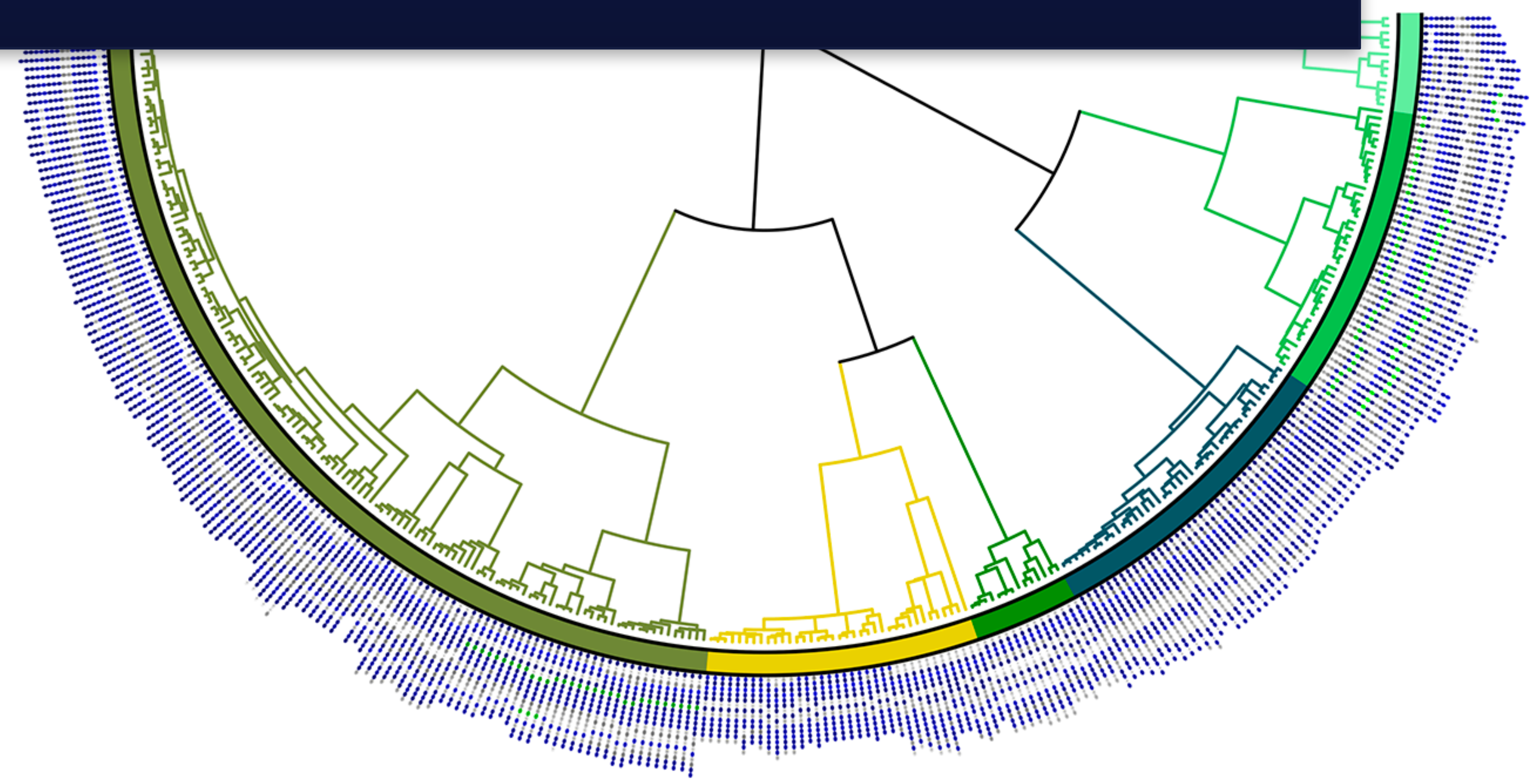




Horizontal (high migration)

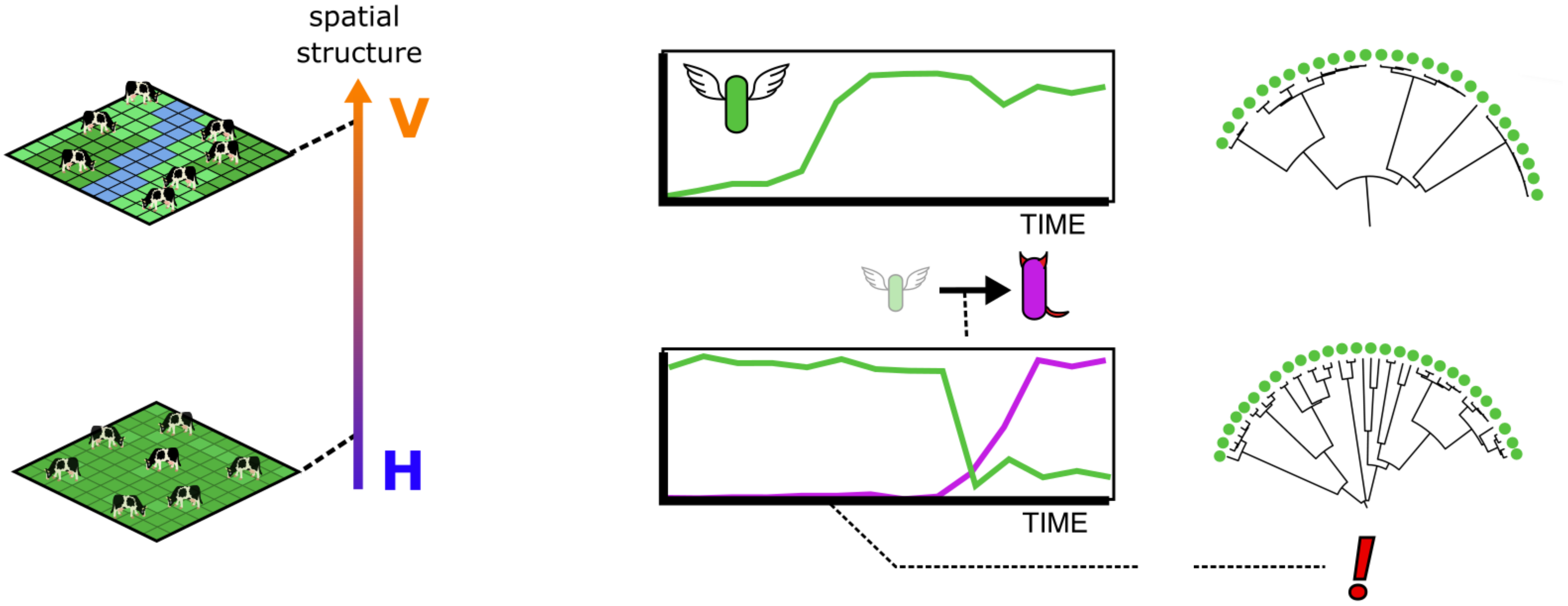


This impact on phylogeny is visible PRIOR to the disease —> it is an effect of horizontal transfer, not of the disease!

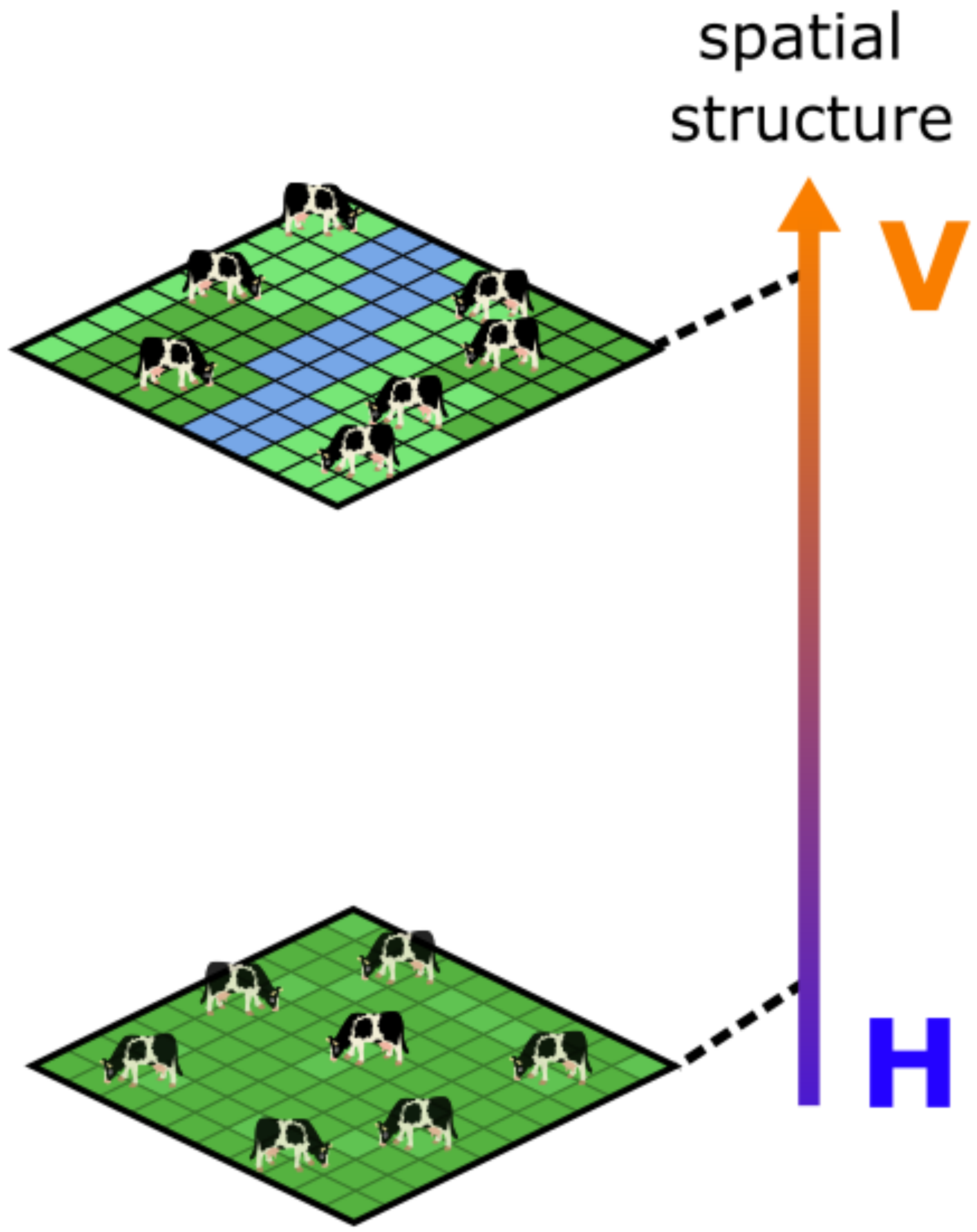


Vertical

Signatures of horizontal vs. vertical transmission, early indicators of disease?



Conclusions Part Ic



- While “horizontal transmission” and “via the environment” are often used interchangeably in the literature, **environmental transmission can be vertical in space**!
- Horizontal transmission gives lower-level entities (genes in microbes, or microbes in hosts) **an opportunity to be nasty**
- This interplay gets even more interesting when considering **more than just 2 levels** (*e.g.* genes, microbes, groups of microbes)
- Phylogenies could be **predictors of conditions that promote nastiness** (not the nastiness itself!!)
- (<u>could be</u>: this is work in process!)

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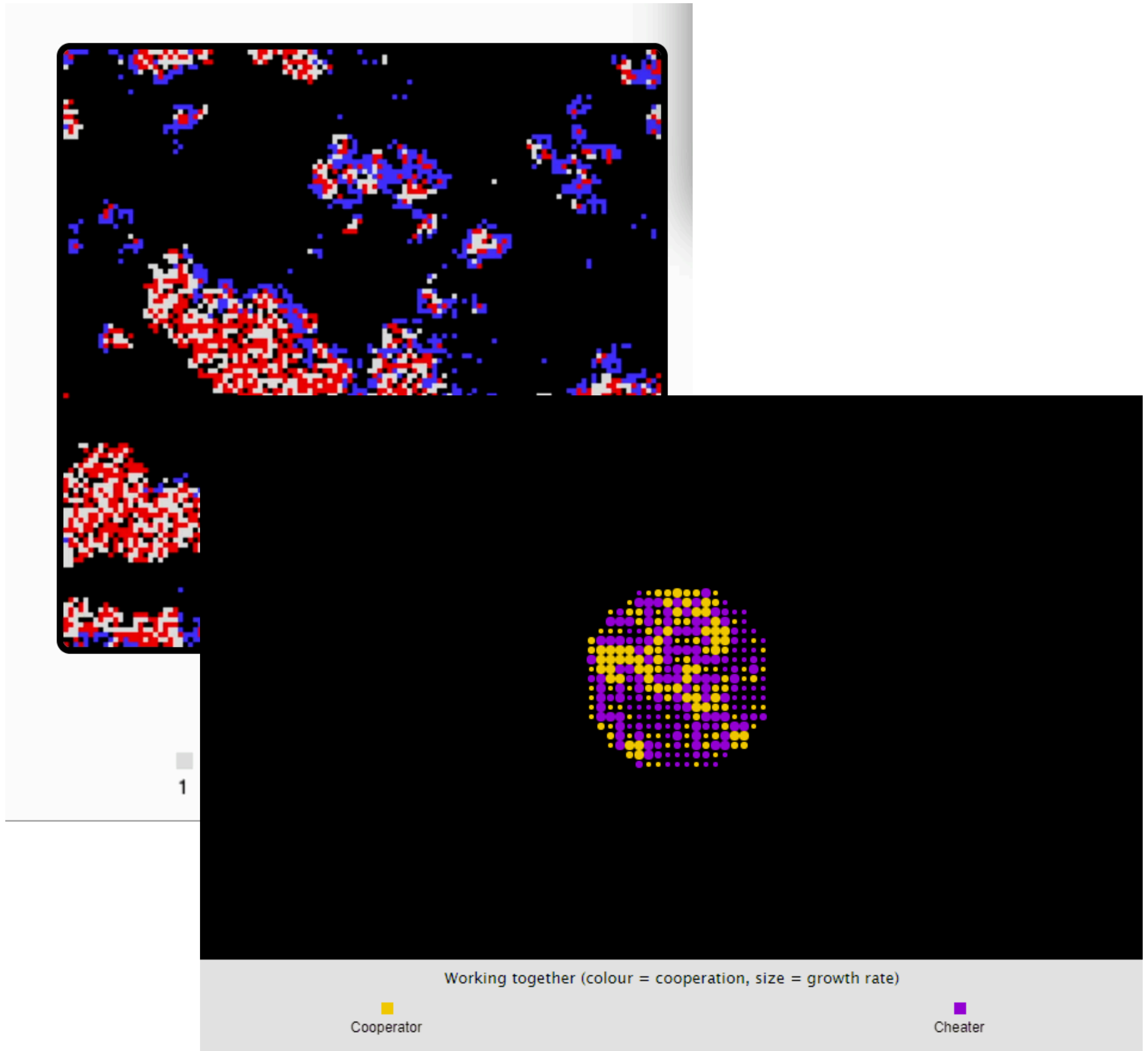
“Cheaters and cooperators”

- Altruism is common in nature
- Many papers on this, but most focus on a “cheater-cooperator” framework…
- Cooperators provide a public good. Cheaters don’t.
- Often **unstable** in well-mixed environments, but the system survives in space

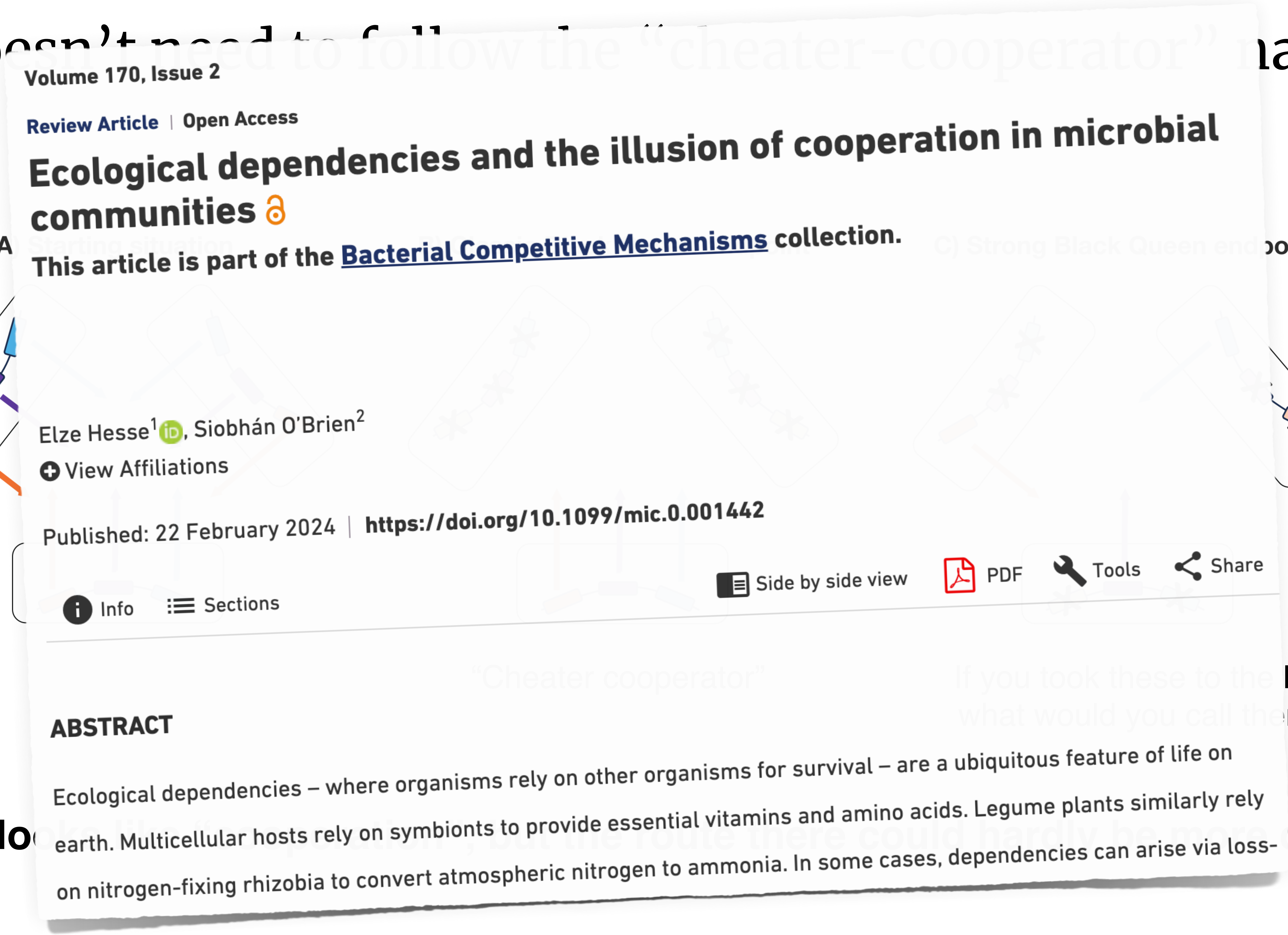


Local extinction but global persistence

Black Queen dynamics

- Benefits are not always exclusive
- Microbial communities contain many “public goods” (cellulase, peroxidase, elastase, amylase, beta-lactamase, heavy metal detoxification, wss operon (*cellulose* *__production__!*)
- Why provide this costly public service, if someone else can do it for you?
- Refers to “Game of Hearts”, where players do not want to hold hearts (-1), and especially not the queen of spades! (-13)

This doesn’t need to follow the “cheater-cooperator” narrative



Volume 170, Issue 2

Review Article | **Open Access**

Ecological dependencies and the illusion of cooperation in microbial communities

This article is part of the **Bacterial Competitive Mechanisms** collection.

Elze Hesse[1], Siobhán O’Brien[2]
View Affiliations

Published: 22 February 2024 | https://doi.org/10.1099/mic.0.001442

ABSTRACT

Ecological dependencies – where organisms rely on other organisms for survival – are a ubiquitous feature of life on earth. Multicellular hosts rely on symbionts to provide essential vitamins and amino acids. Legume plants similarly rely on nitrogen-fixing rhizobia to convert atmospheric nitrogen to ammonia. In some cases, dependencies can arise via loss-

A) Starting situation

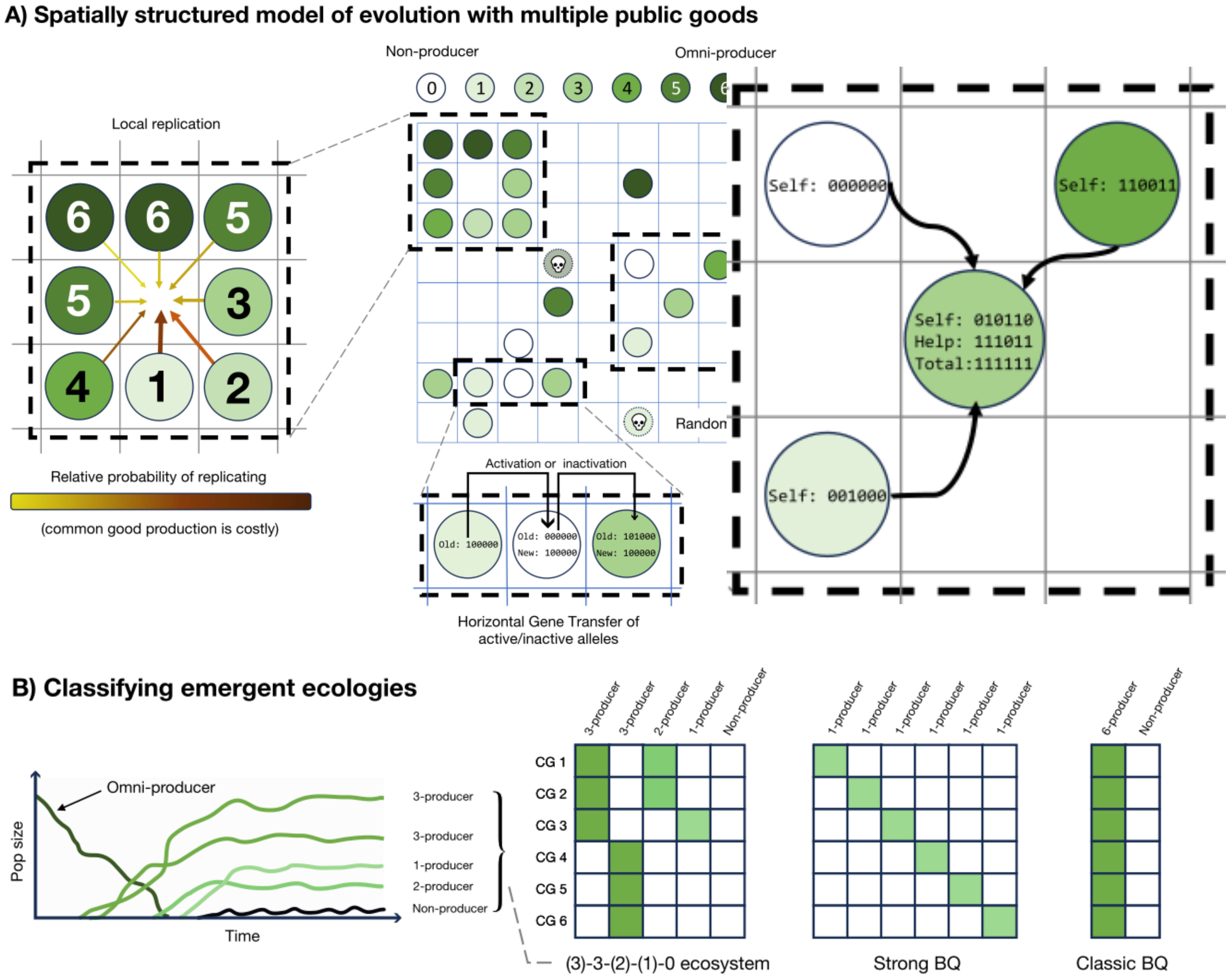
C) Strong Black Queen endpoint

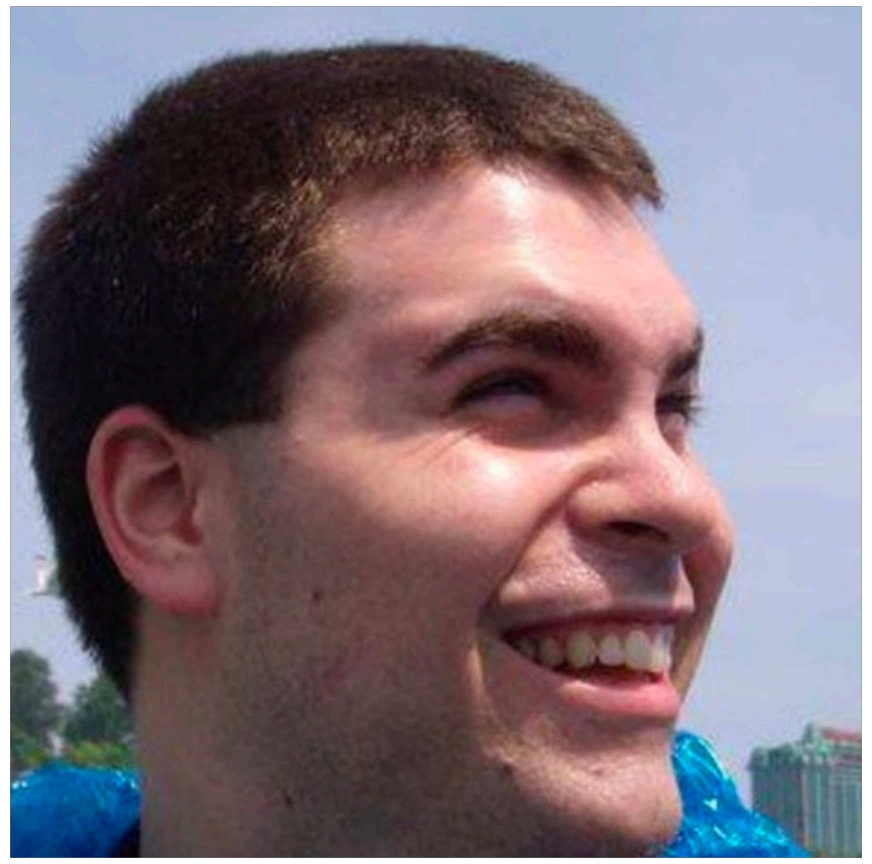
“Cheater cooperator”

If you took these to the lab, what would you call them?

Strong BQ looks like “cooperation”, but the route there could hardly be more different!

Matt Fullmer’s model of BQ dynamics





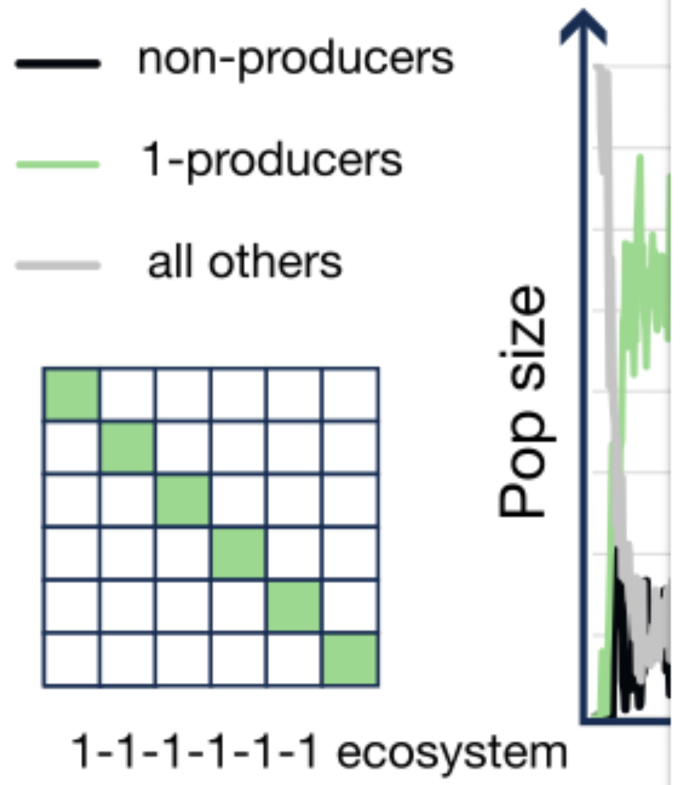
Matt Fullmer

Nobuto Takeuchi

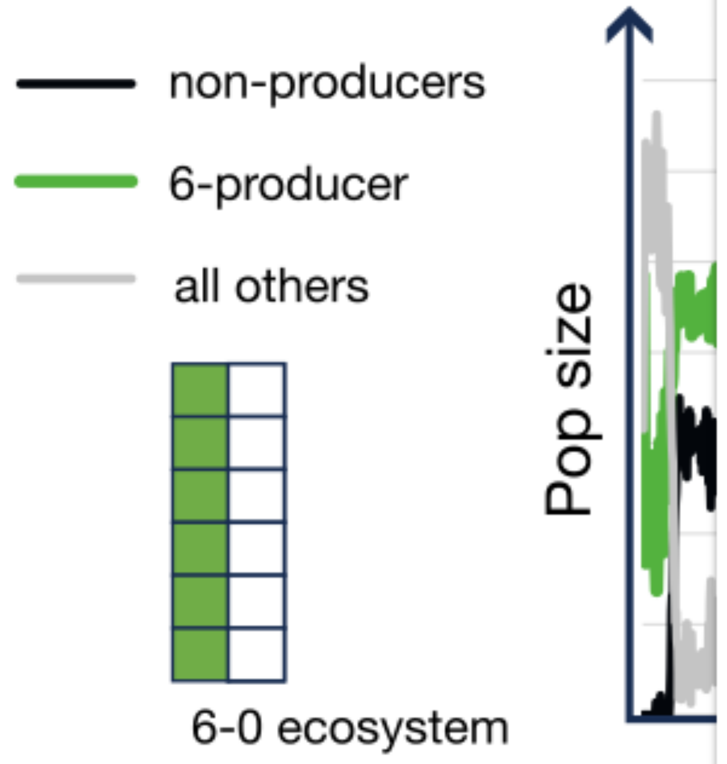
Part II: Black Queen ecosystems and a race to the bottom

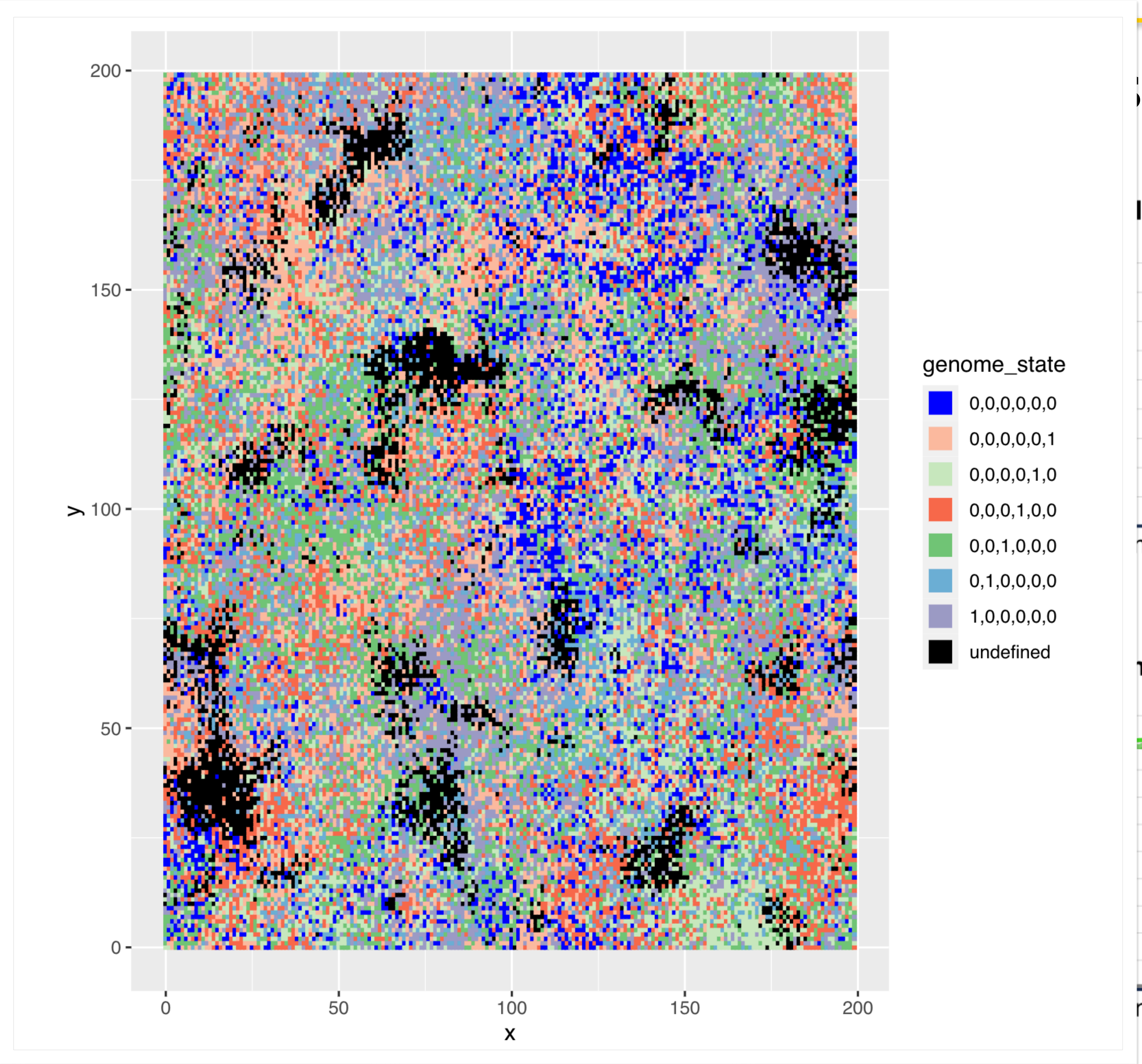
Both ' served

A) Strong Black Queen



B) Classic Black Queen





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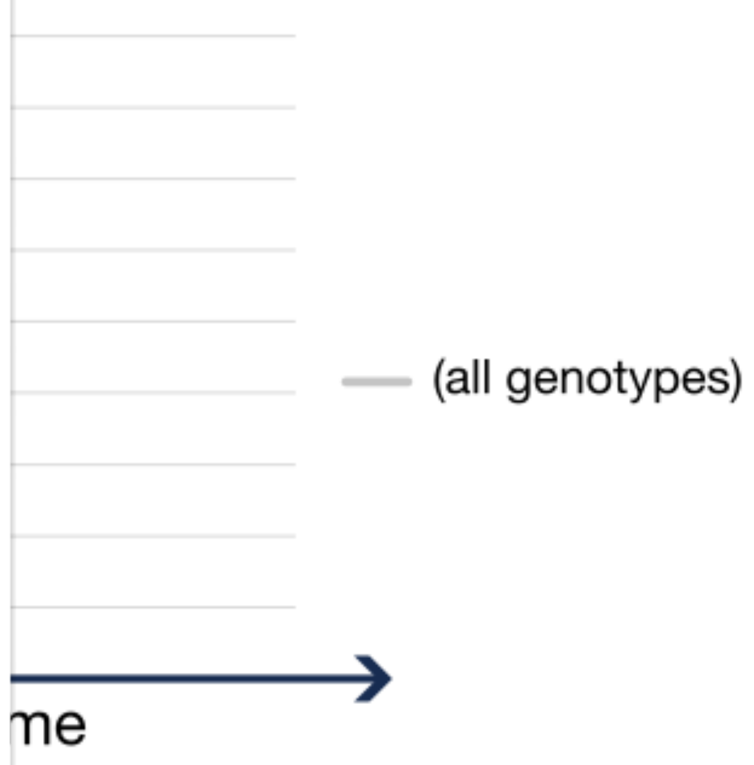
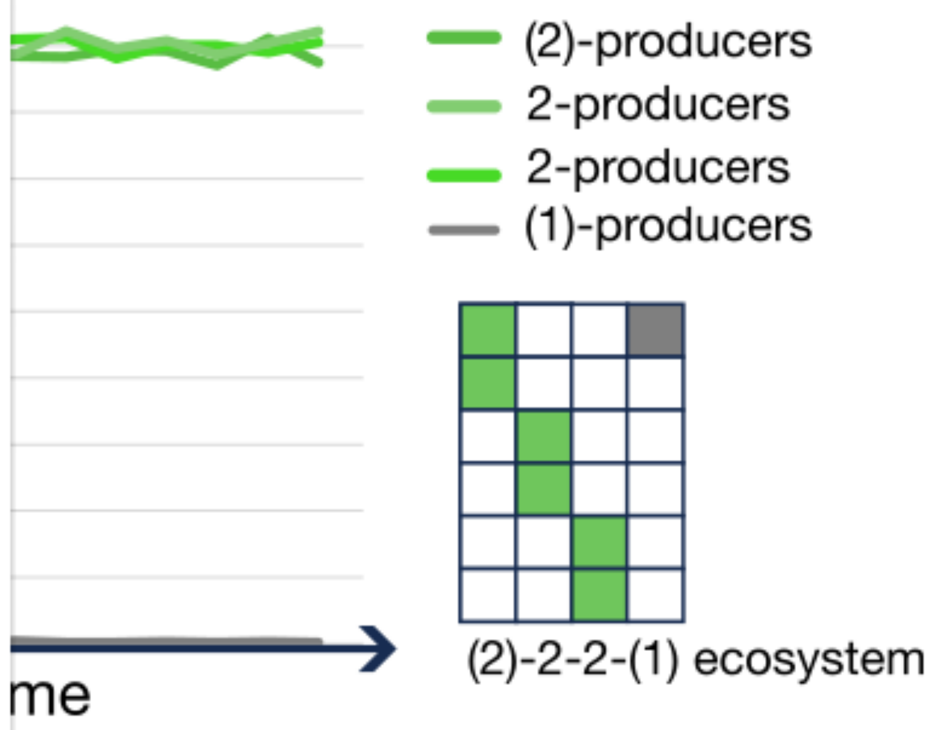
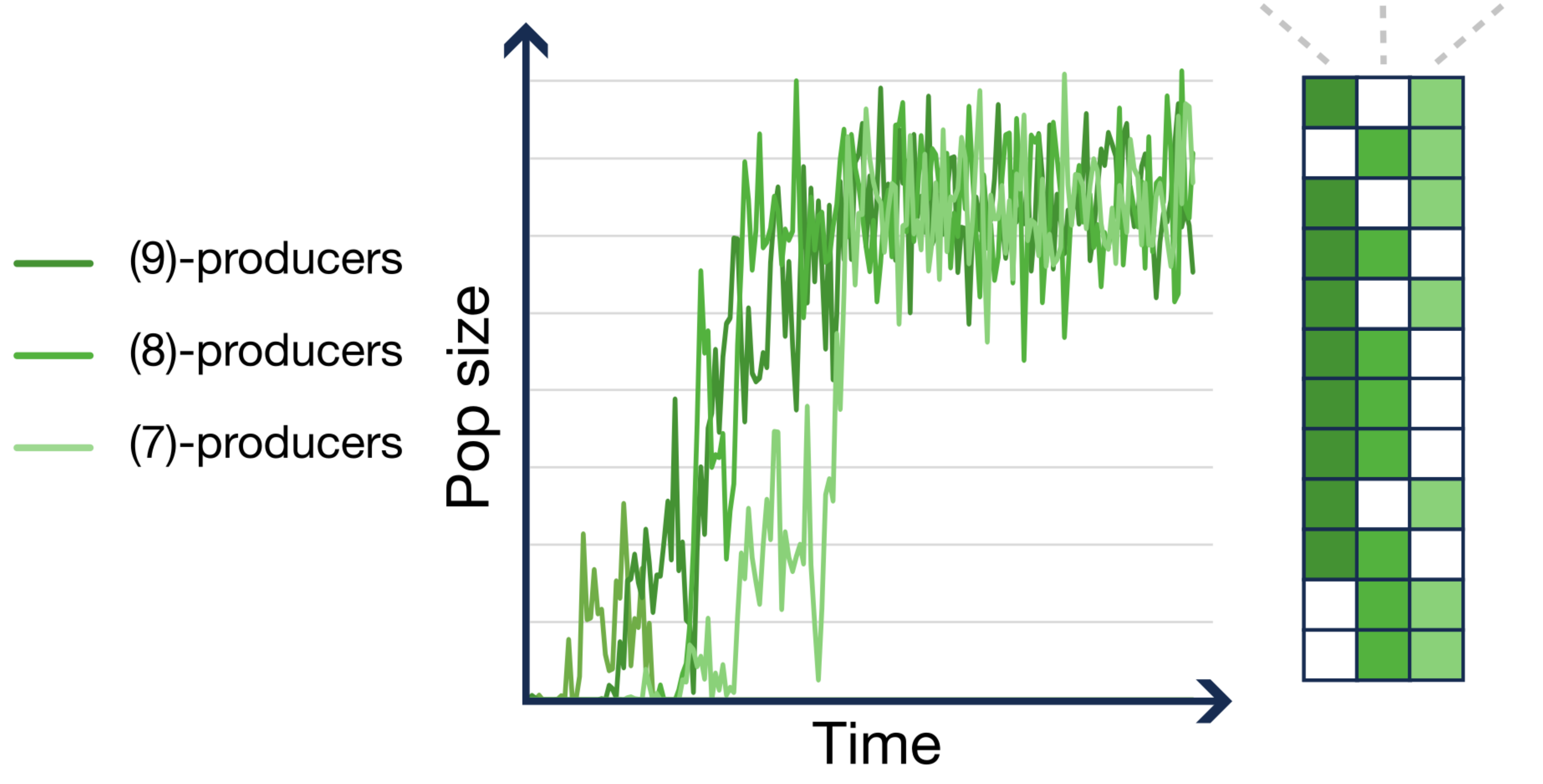


nediate ecosystems

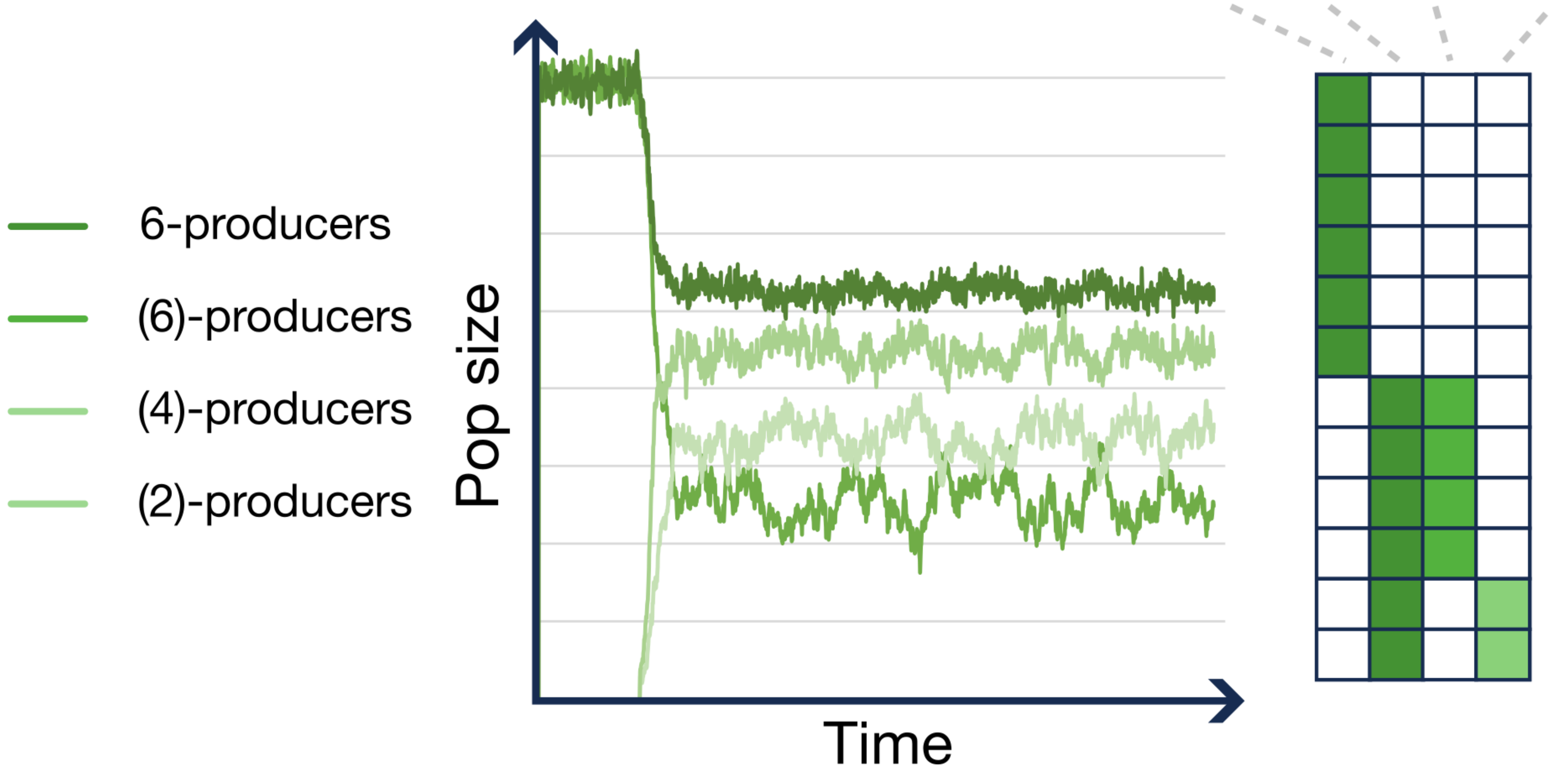


Stable yet redundant ecosystems…?

A) Ecosystem of redundant types (9)-(8)-(7)



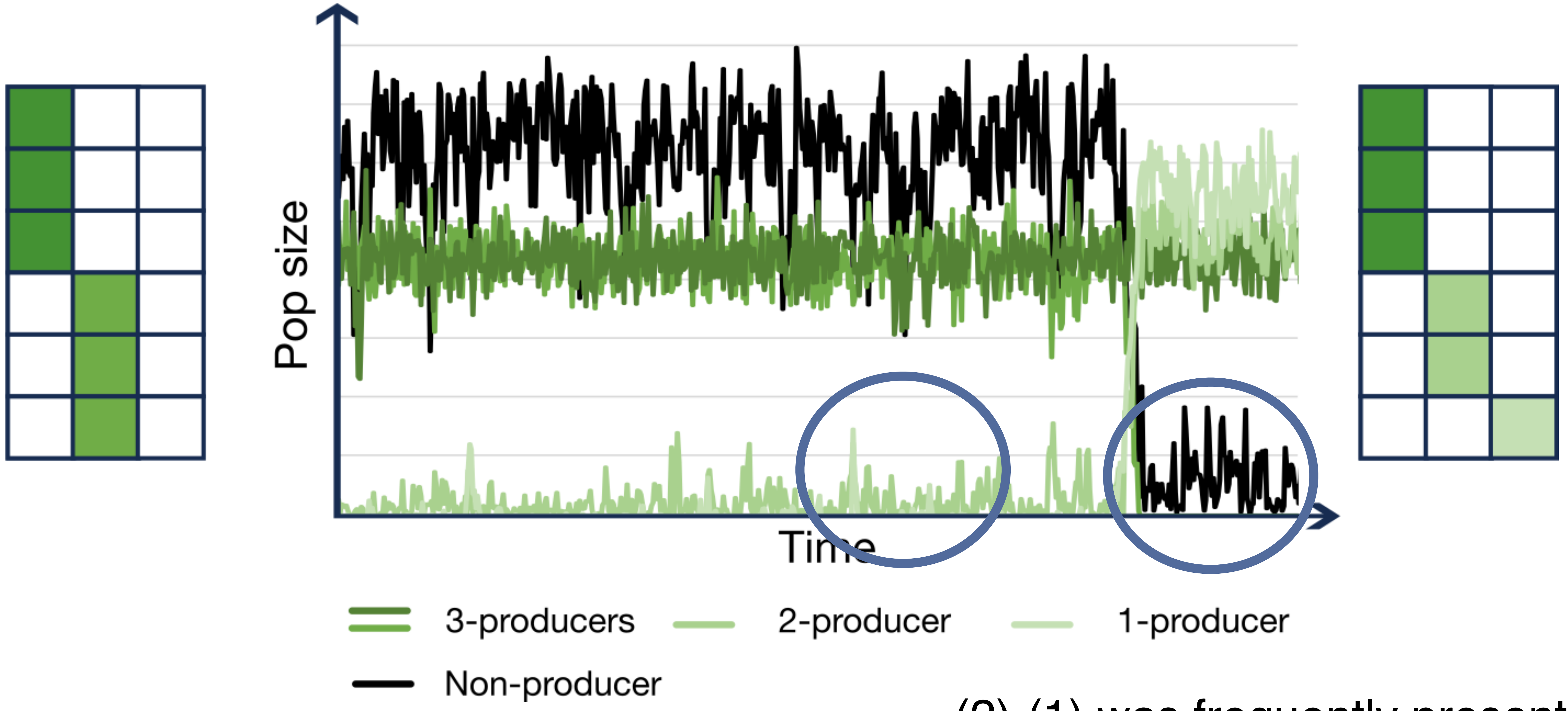
B) Ecosystem of redundant types 6-(6)-(4)-(2)



What the heck?!

Late shifts in ecosystem structure

A 3-3-0 ecosystem transitions to a 3-(2)-(1) after millions of time steps



(2)-(1) was frequently present, but failed to invade

After they DID invade, the non-producers are nearly pushed out

Why can ecosystems “be stuck”, not dividing labour, for so long?

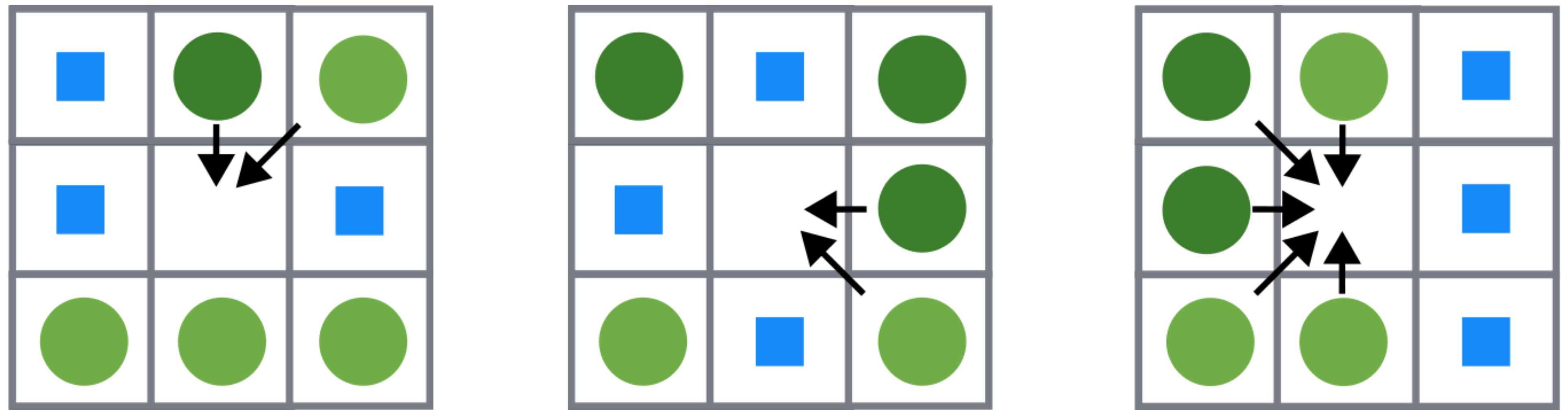
Two hypotheses:

i) Non-producers (or lesser producers) keep preventing it because of their fitness benefits

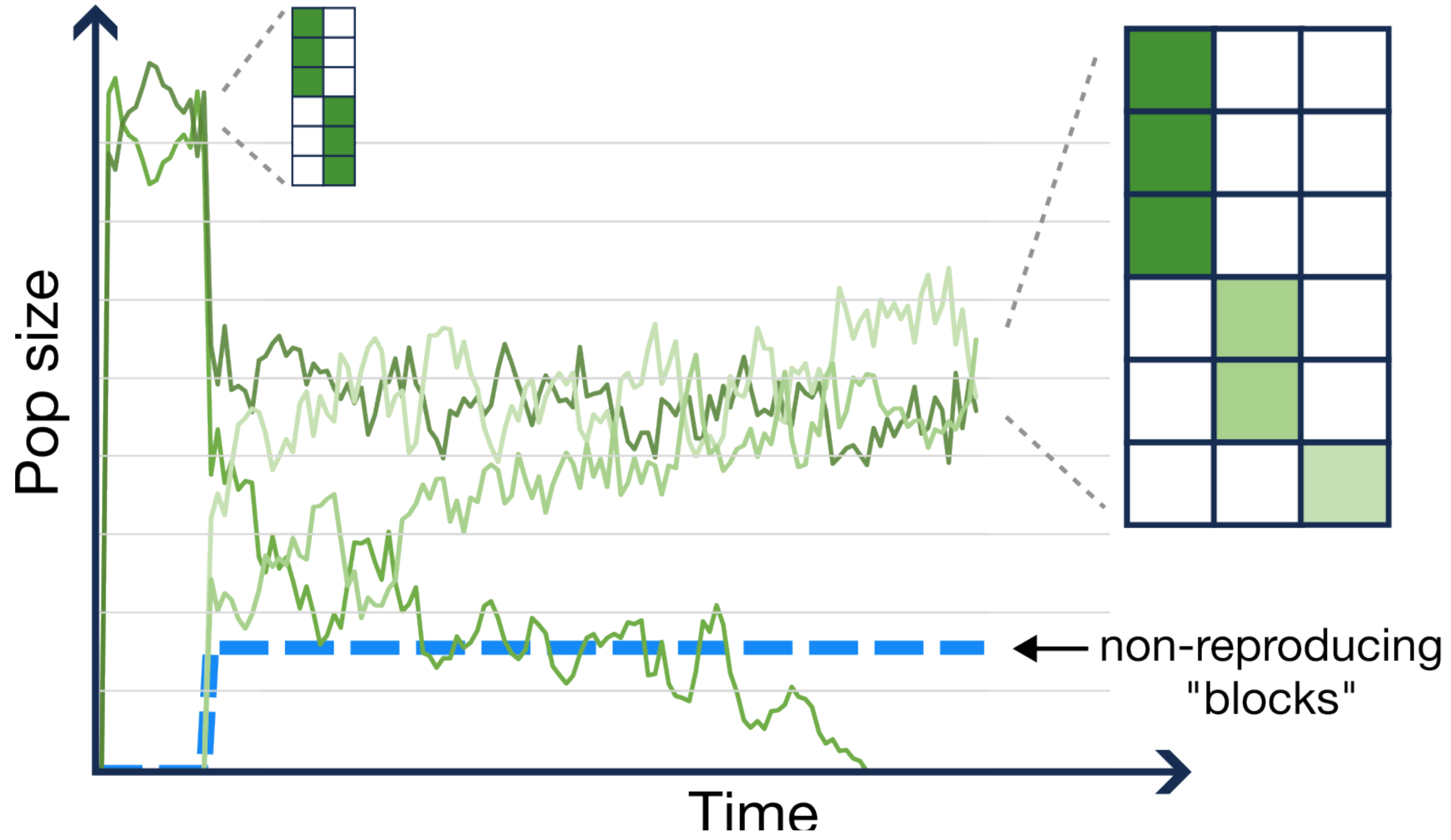
ii) Non-producers (or lesser producers) keep preventing it because **they simply take up space!**

Introducing "infertile blocks"

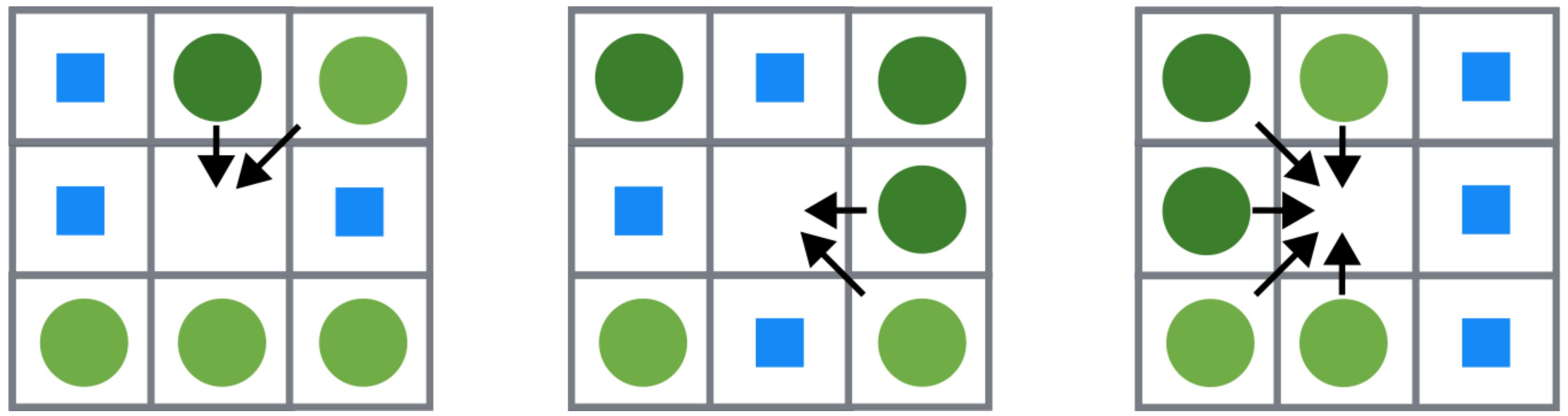
With 10% blocks, 3-3 successfully transforms into 3-2-1



Exactly the type of "experiment" that would be impossible in the lab

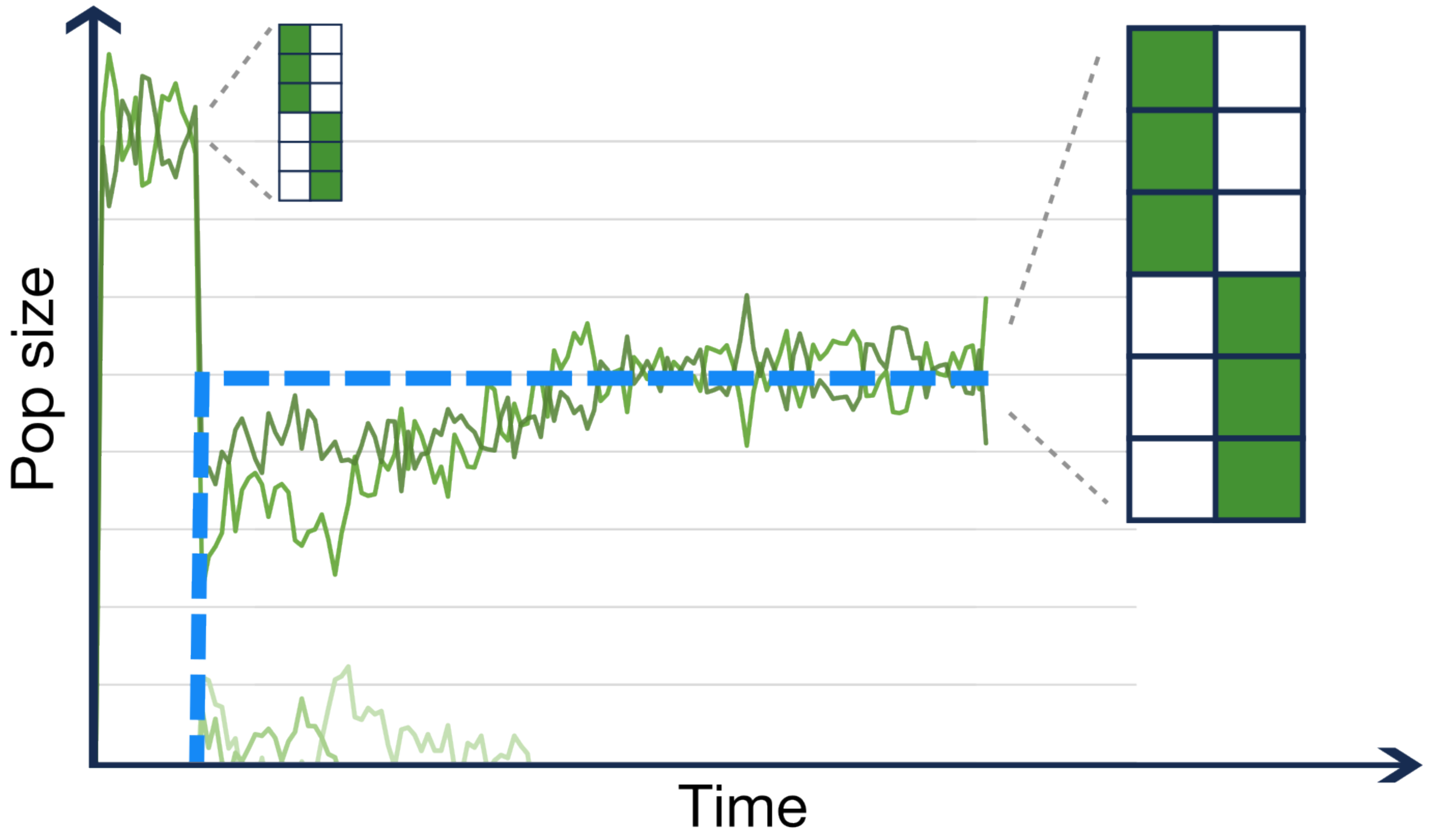


Introducing “infertile blocks”



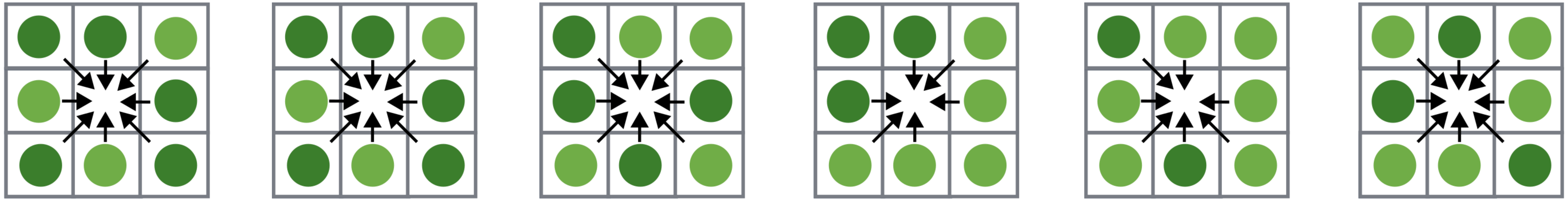
Exactly the type of “experiment” that
would be impossible in the lab

With 33% blocks, 3-3 persisted !

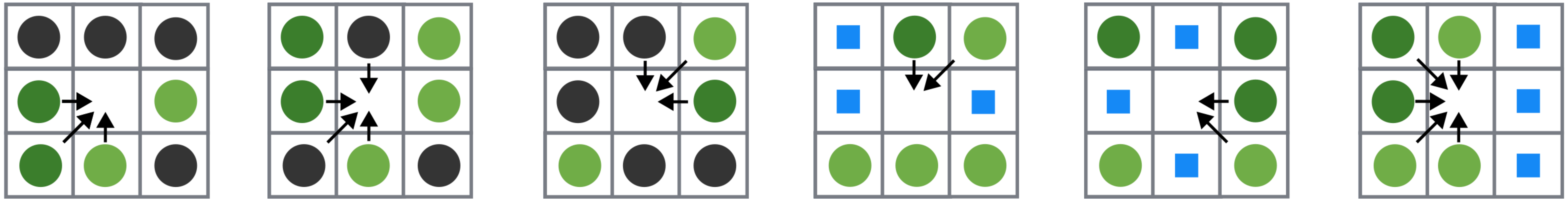


Neighbourhood certainty shapes BQ dynamics!

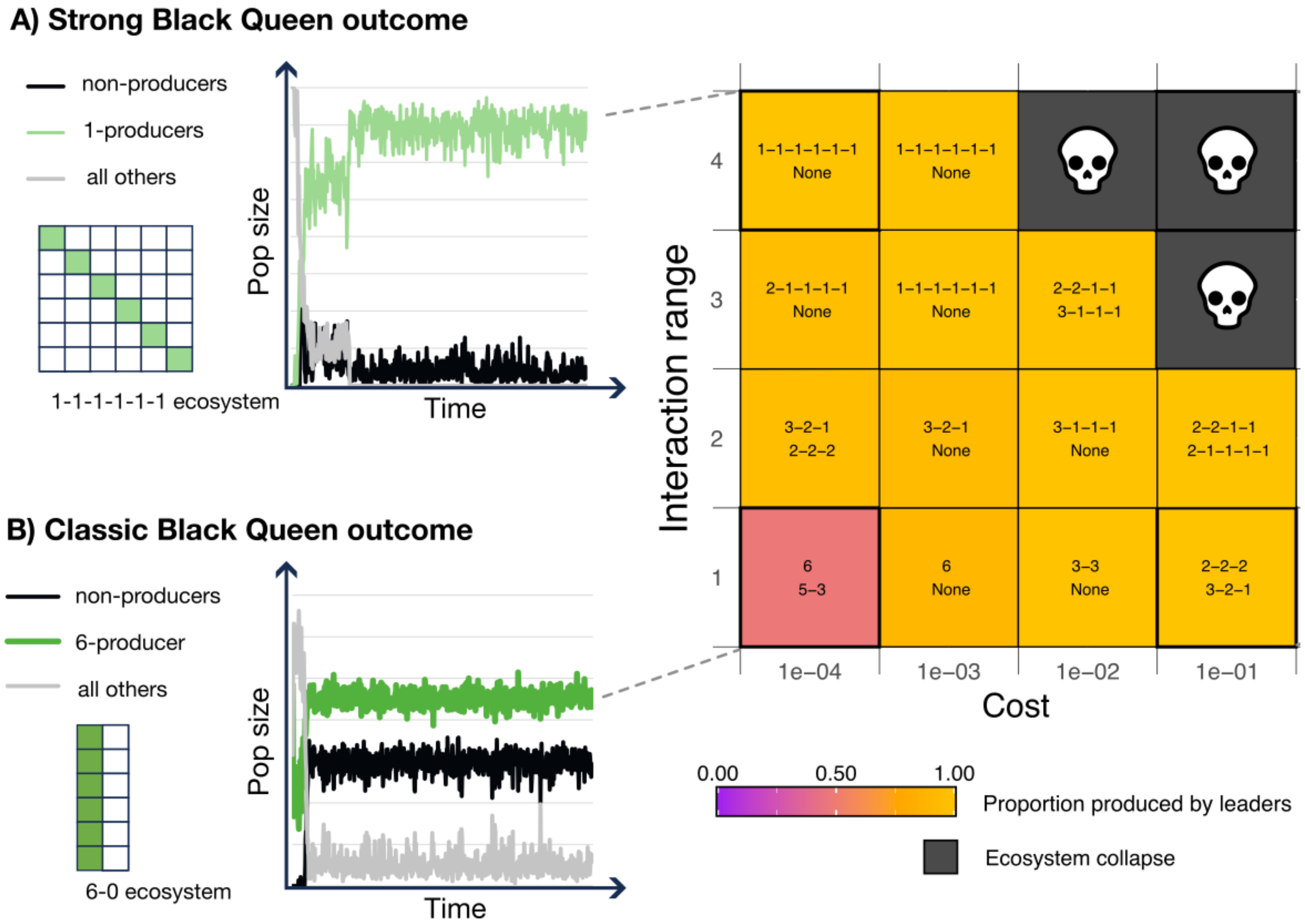
B) Without non-producers, producers can nearly always reproduce, promoting Black Queen dynamics



C) With non-producers or "blocks", producers are often reproductively isolated, stalling Black Queen dynamics



Are costs even all that relevant?



A) Strong Black Queen outcome

B) Classic Black Queen outcome

Interaction range	1e−04	1e−03	1e−02	1e−01
4	1−1−1−1−1−1 None	1−1−1−1−1−1 None	💀	💀
3	2−1−1−1−1 None	1−1−1−1−1−1 None	2−2−1−1 3−1−1−1	💀
2	3−2−1 2−2−2	3−2−1 None	3−1−1−1 None	2−2−1−1 2−1−1−1−1
1	6 5−3	6 None	3−3 None	2−2−2 3−2−1

No! BQ can also work without any costs!

- “Selection is blind” for as long as someone provides the CG
- So without a cost, **drift** can promote dependencies!
- So once again: **fitness effect** for public goods are not the only important thing!
- The **Black** Queen hypothesis was defined as this social dilemma.
- The neutral variant has been phrased as “Grey Queen” by Ford Doolittle, but I like purple more.
- Let’s explore this a little more (WIP)

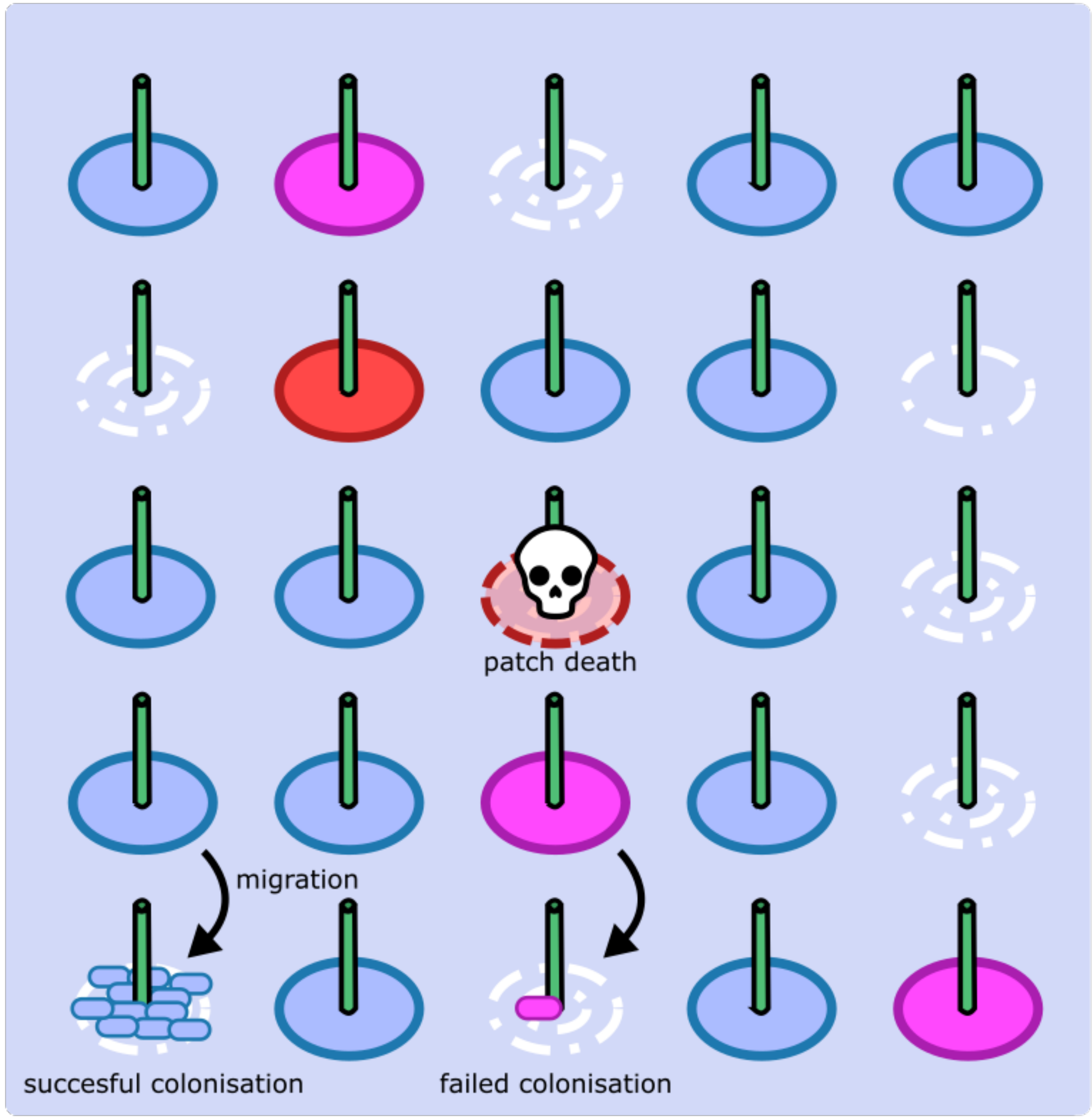
Grey Queen dynamics in patches

Within populations (patches of 200 cells)

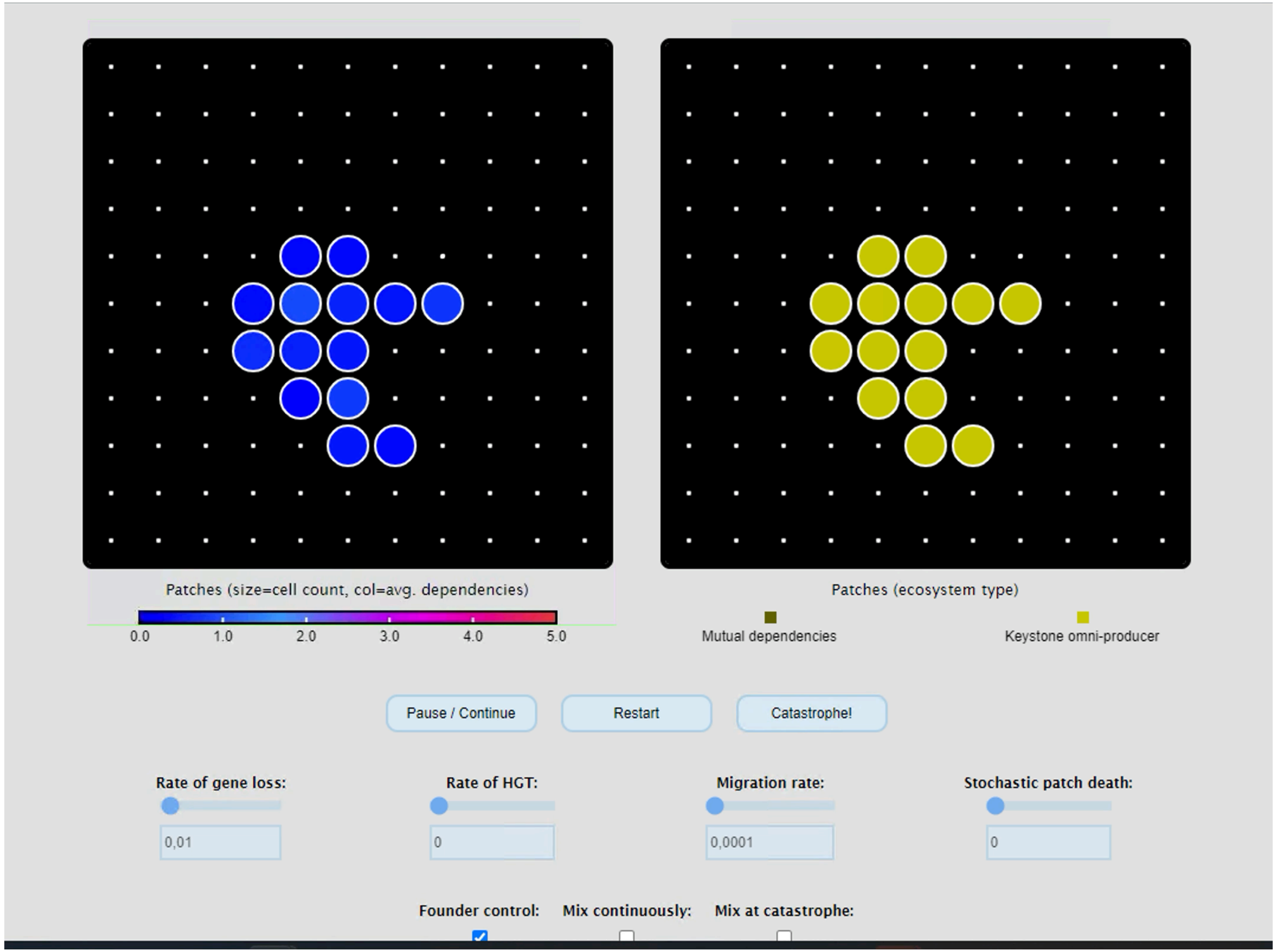
- **Strong** black queen dynamics ("evolutionary race to the bottom")
- Five **essential** public goods (A,B,C,D,E)
- Death of **random** cells
- Birth of (viable) cells
- **Gene loss** upon reproduction
- **No costs** for public good production ("Grey Queen")

Between populations (patches)

- Differential **persistence** (resilience to noise)
- Differential **spread** (colonisation capacity)
- "Survival of the systems"?



Grey Queen dynamics in patches



Rate of gene loss: 0,01

Rate of HGT: 0

Migration rate: 0,0001

Stochastic patch death: 0

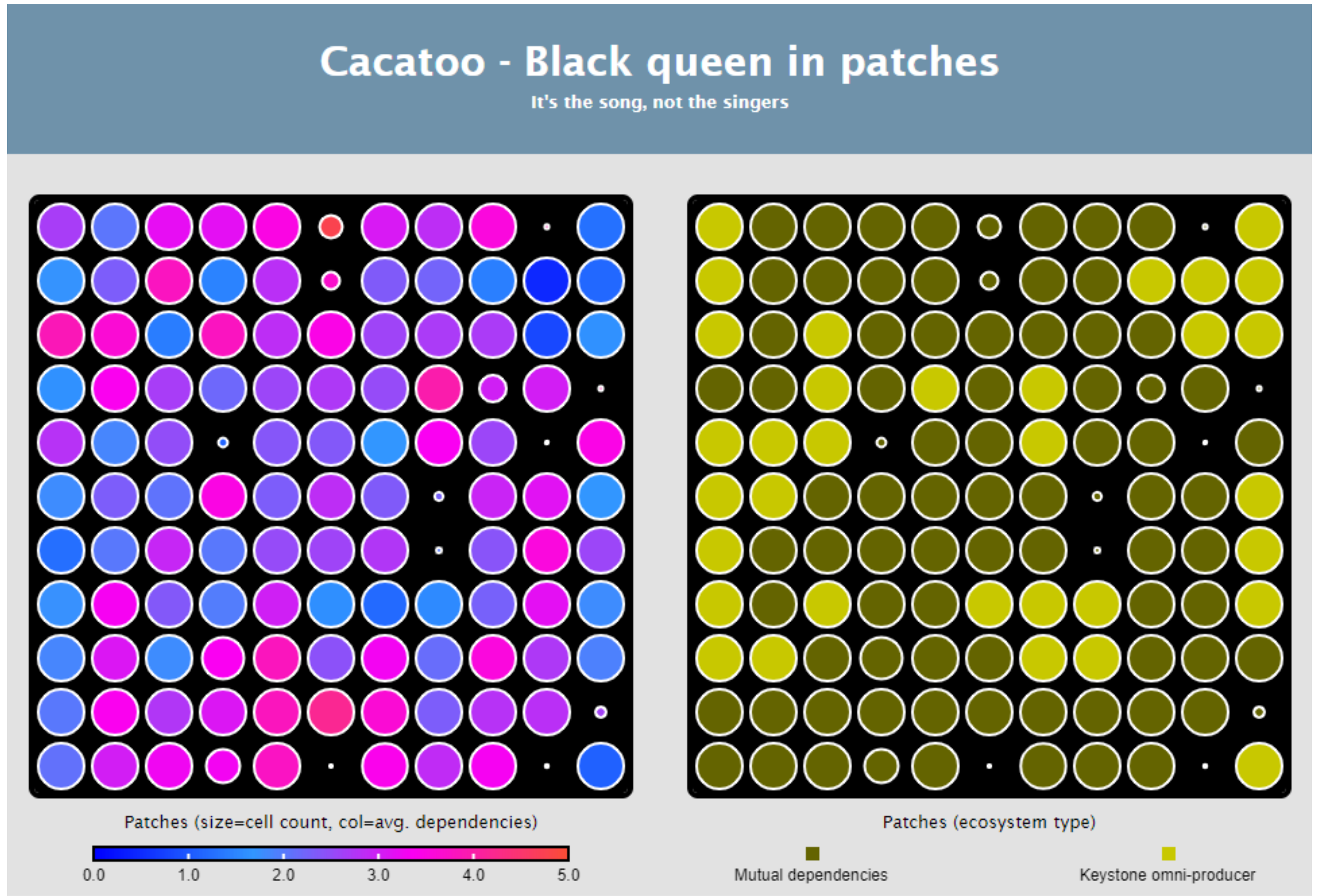
Founder control: ☑
Mix continuously: ☐
Mix at catastrophe: ☐

Community-level properties

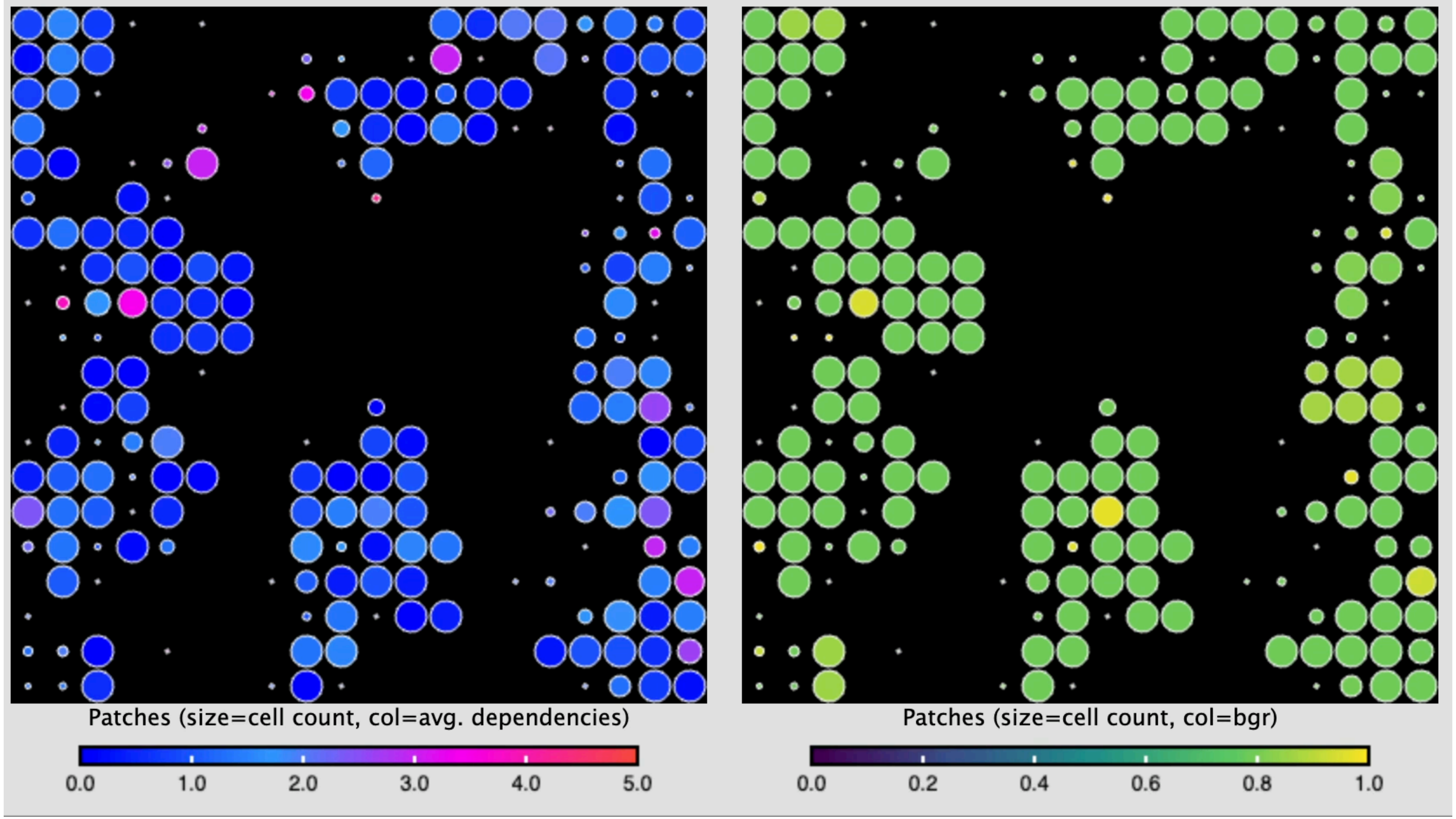
- Patches (microbial communities!) have the following properties:
  “*Aging*” → Within-population conflict ages patches
  “*Death*” → “Aged” communities die
  “*Birth*” → Founding a new colony,
  “*Rejuvenation*” → Decrease in mutual dependencies upon community-reproduction

  I.e. patch-level properties, but without “patch-level” parameters!

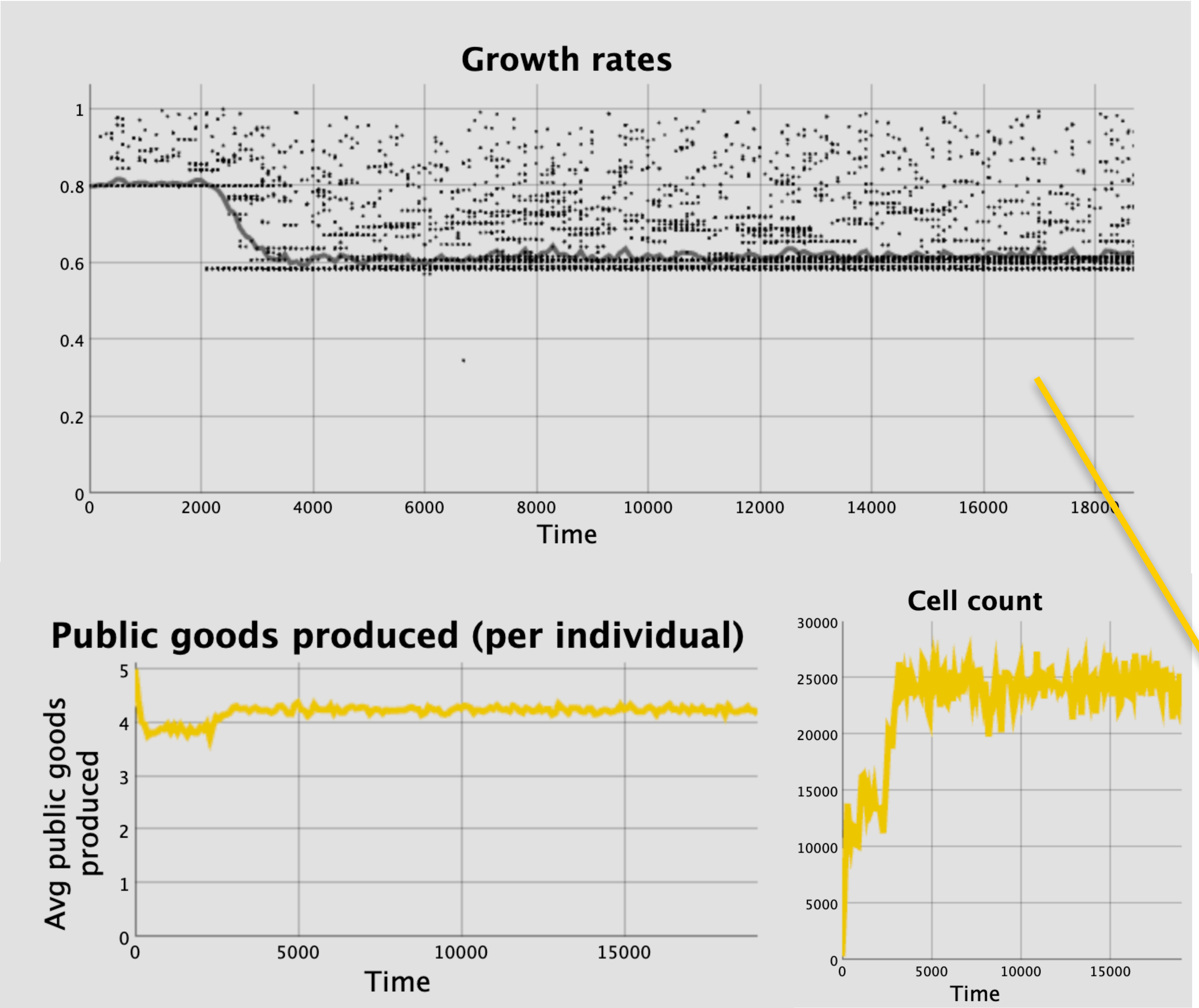
- These dynamics can avoid the evolutionary race to the bottom, sustaining public good production



Community-level selection?



Strong community-level selection!



> "Recent theoretical progress highlights that natural selection can occur based solely on differential persistence of biological entities, without the need for conventional replication. This calls for a reconsideration of how ecosystems and social systems can evolve, based on identifying system-level properties that affect their persistence."
> - Tim Lenton on "Survival of the Systems"

This still requires some unpacking.

Earlier models have only shown selection for lower growth rate when considering **limited resources**! (If we're going to use words like "cheaters", **THIS** is cheating! :P)

Conclusions Part II

- **Black Queen dynamics** can promote an evolutionary race to the bottom
- With limited interaction range, strange (“redundant”) ecosystems can form and be maintained for long evolutionary timescales
- Non-producers can “stall” the race to the bottom by taking up space -> neighbourhood uncertainty!

- BQ still happens **even when there are no costs** to producing the common goods (Grey Queen)
- If the race to the bottom ends in catastrophe, we can observe **selection for lower growth rates**, without any limiting resource!

General conclusions

- Evolutionary dynamics with horizontal transmission create **conflicts between lower- and higher-level entities**
- The conflict between levels of selection (gene and microbe, microbe and host) becomes extra interesting when including **more than two levels**
- Sometimes it can help to **not** start with the simplest model (this is how we accidentally discovered the streamlining in response to transposons!)
- While many models do not include the fact that organisms “take up space”, this turns out to be very important for ecosystem function (BQ dynamics)
- Besides spatial structure there is also **genome structure** to consider when thinking about evolution. Paulien will discuss more on this later in the course.