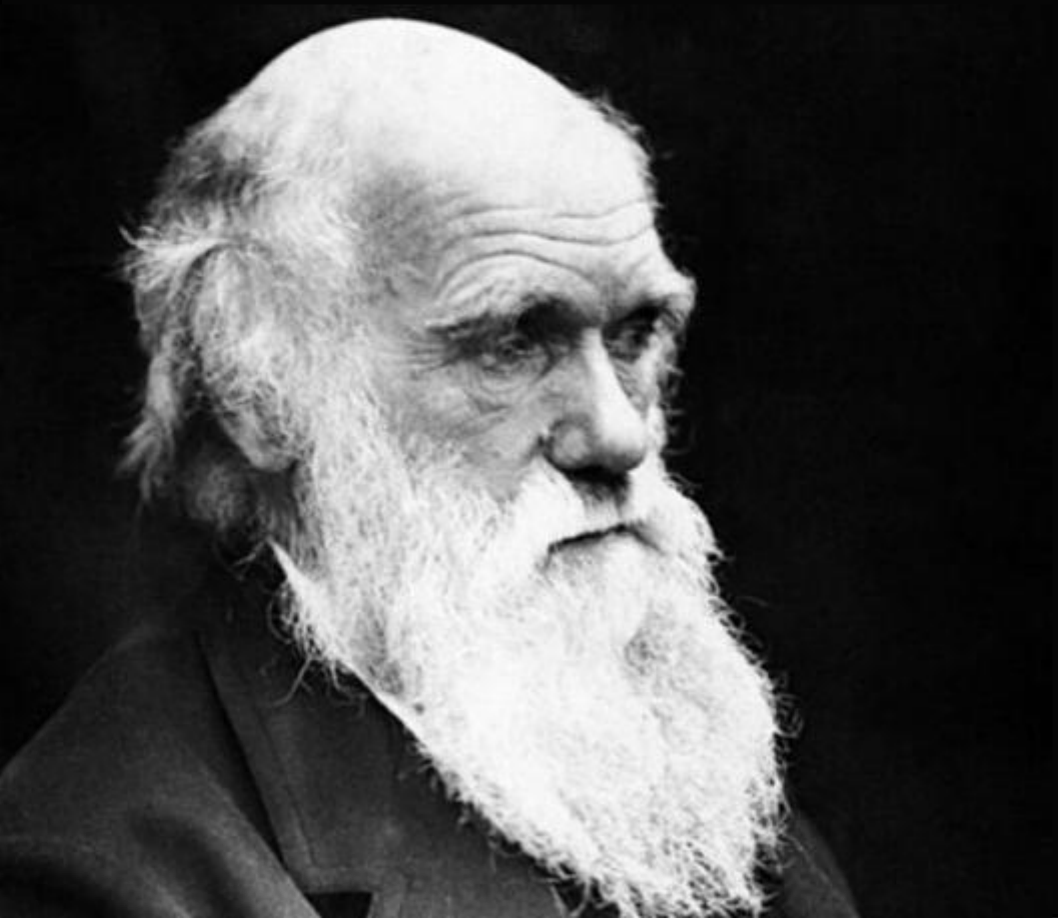
Eco-evolutionary dynamics II

Coevolution

Fabrizio Mafessoni, Biologia Evoluzionistica 2025/2026, Università di Trieste



Eco-evolutionary dynamics II

advanced

Coevolution

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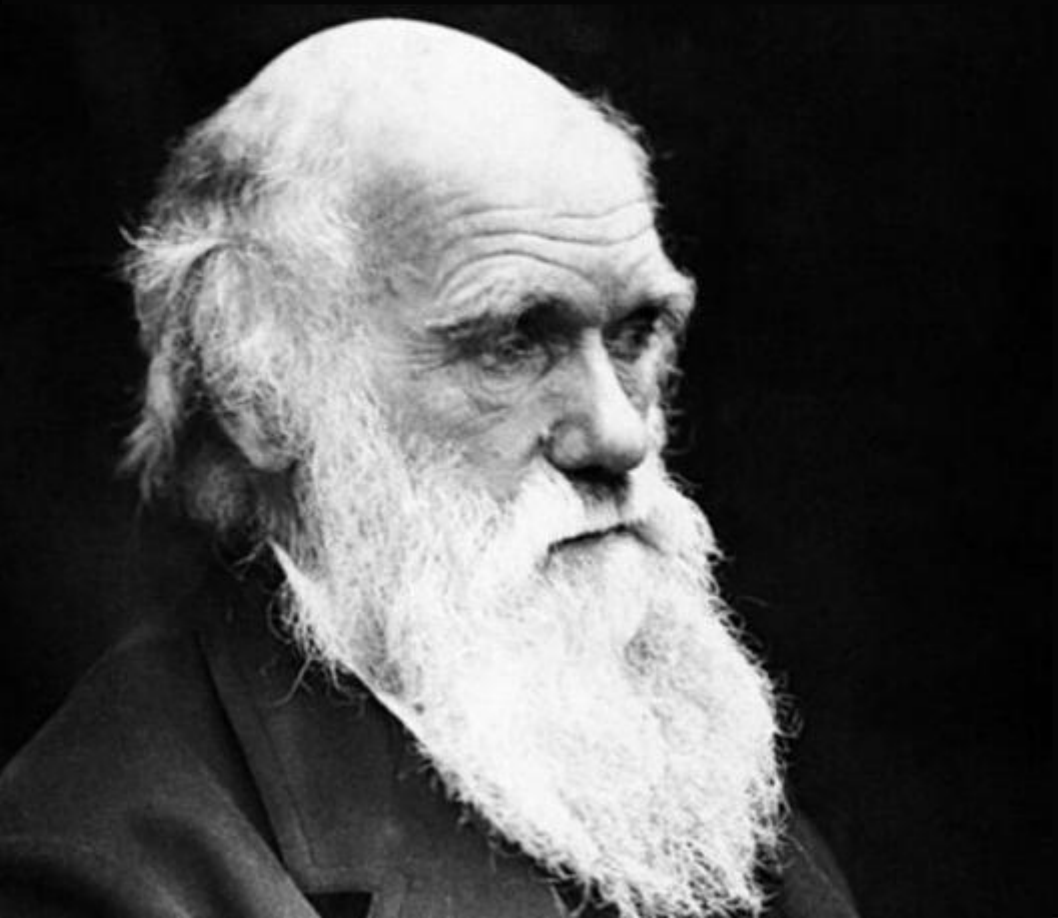


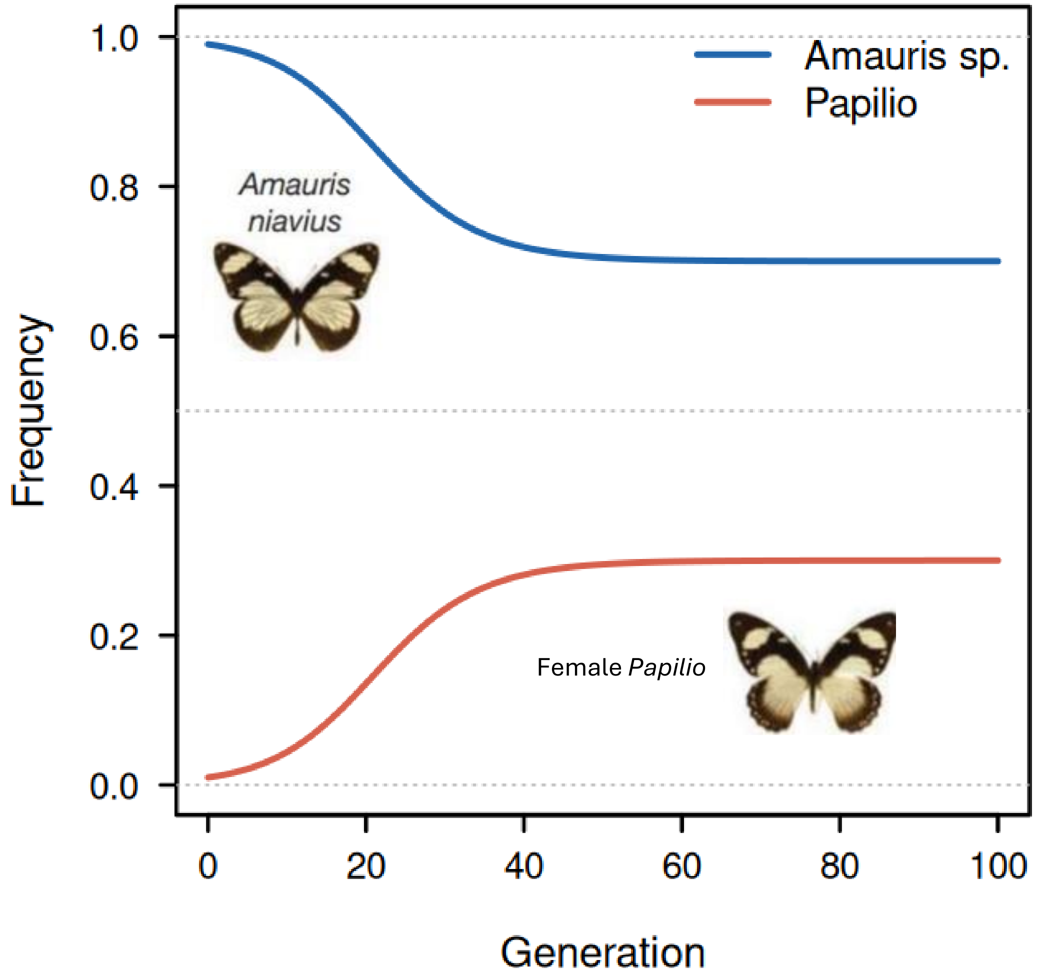
In the previous episode..

Not all ❤

Batesian mimicry: venomous vs not venomous

Evolutionary dynamics: Batesian mimicry

Mimics can invade but it cannot spread to much since birds stops avoiding eating butterflies, leading to «Hawk and Dove» dynamics



Amauris niavius

Female *Papilio*

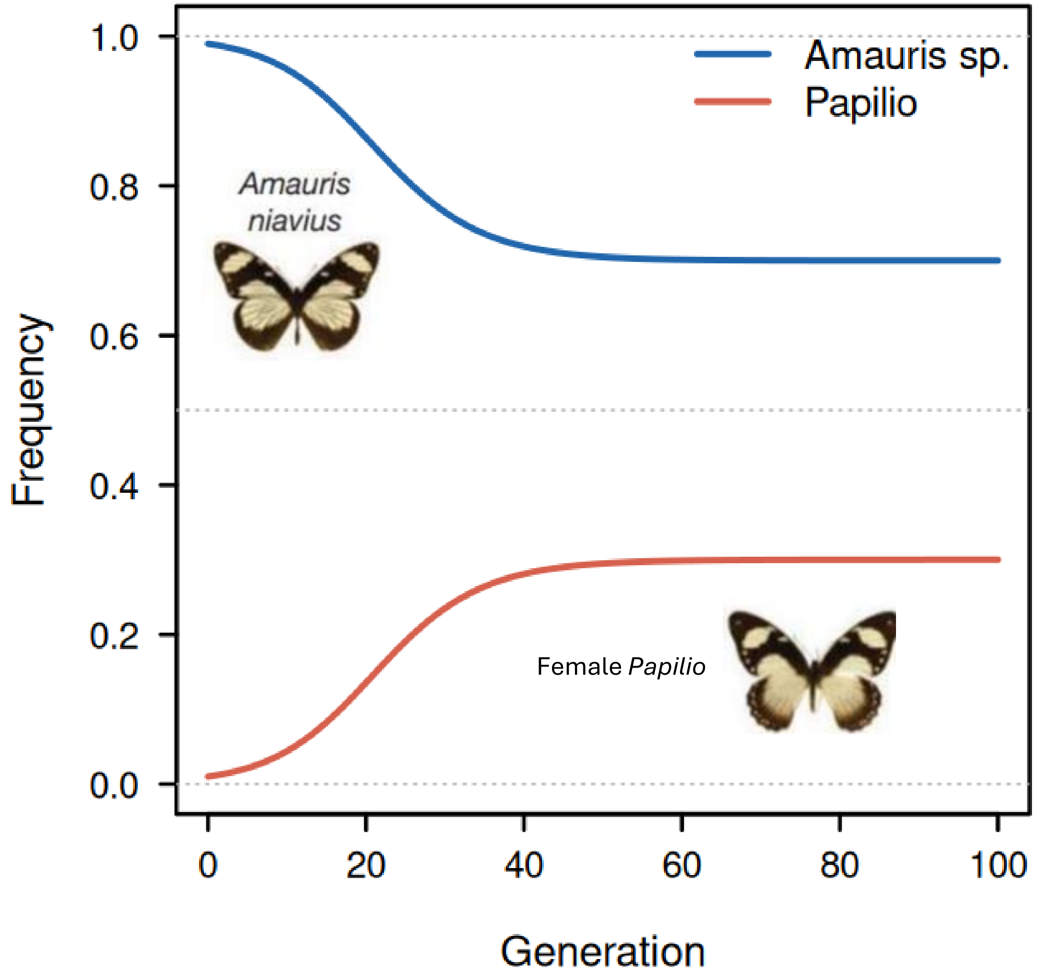
Amauris niavius

Female *Papilio*

	Model	**Mimic**
Model	0.85, 0.85	0.75, 1
Mimic	1, 0.75	0.4, 0.4

Evolutionary dynamics: Batesian mimicry

Mimics can invade but it cannot spread to much since birds stops avoiding eating butterflies, leading to «Hawk and Dove» dynamics



How was this called?

Amauris niavius

Female *Papilio*

Amauris niavius

Female *Papilio*

	Model	**Mimic**
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Evolutionary dynamics: Batesian mimicry

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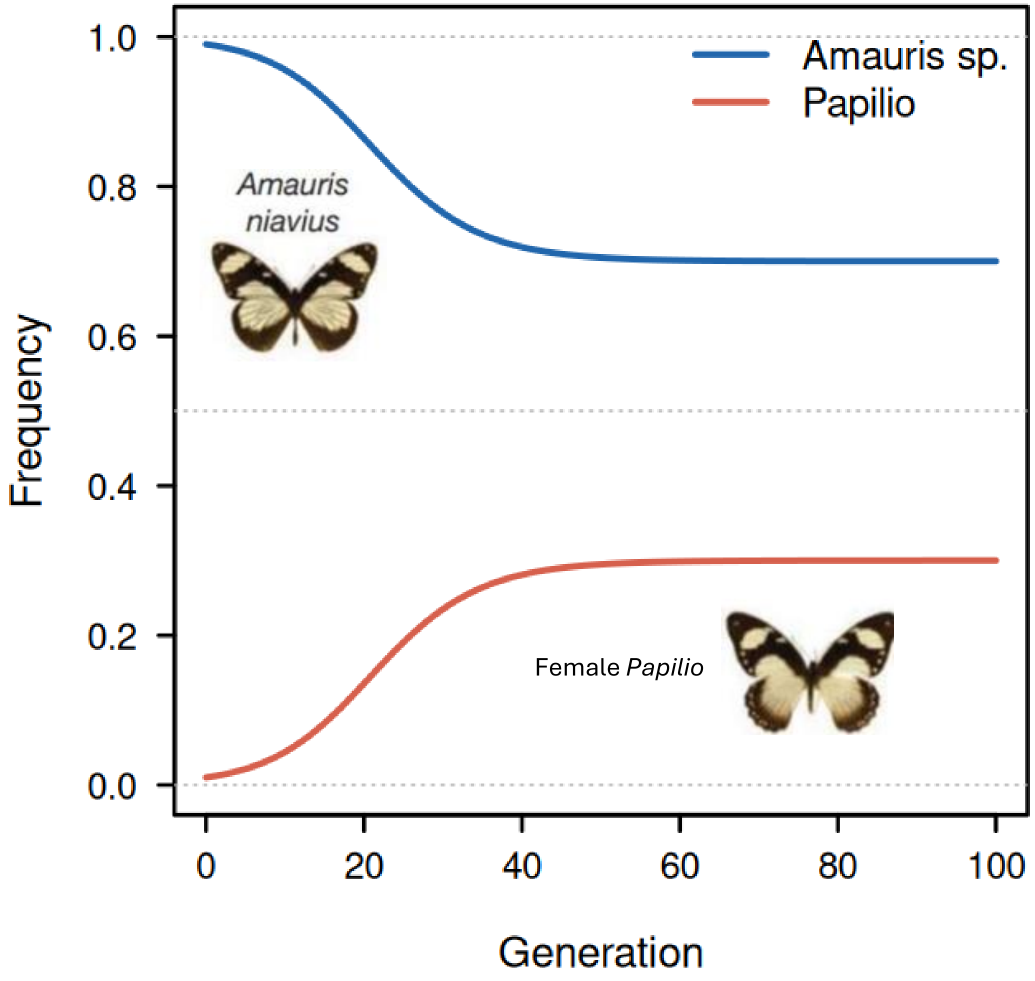


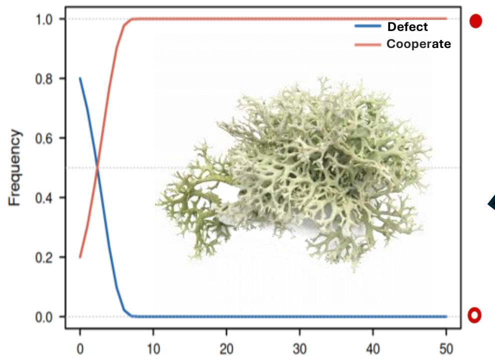
○ Unstable equilibrium 100% models

● Stable equilibrium (ESS)

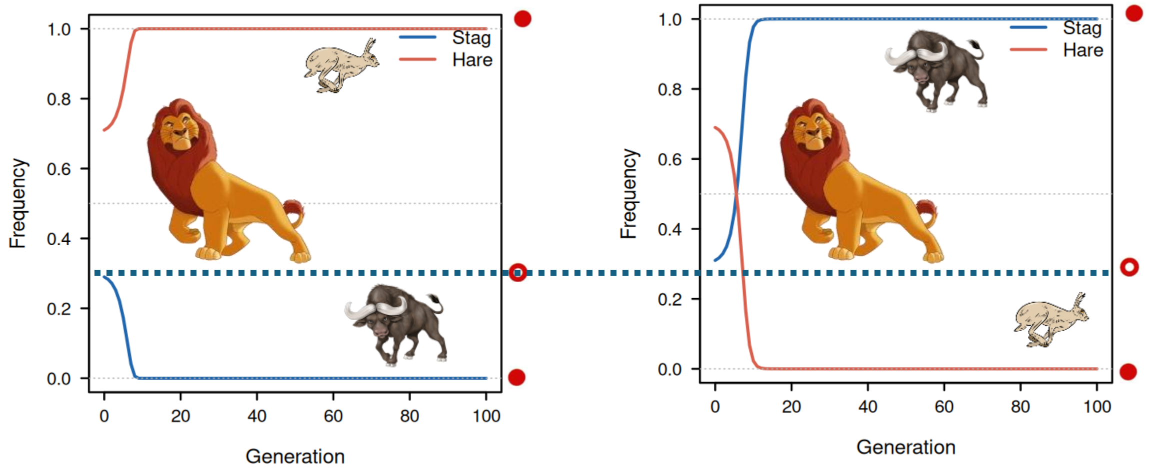
○ Unstable equilibrium 0% Mimics

	Model	**Mimic**
Model	0.85, 0.85	0.75, 1
Mimic	1, 0.75	0.4, 0.4

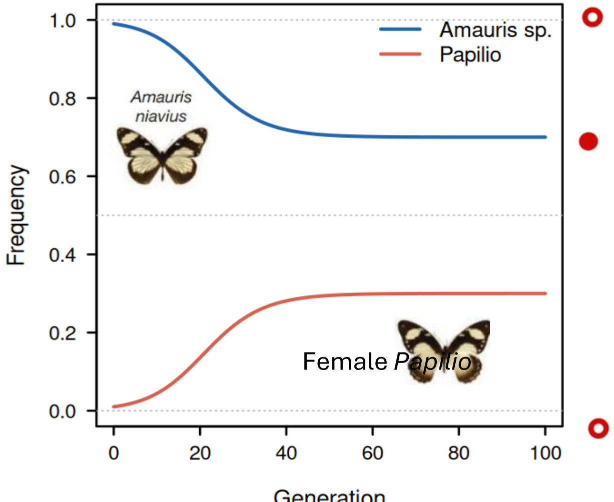
e.g. Prisoner’s dilemma,
obligated mutualism

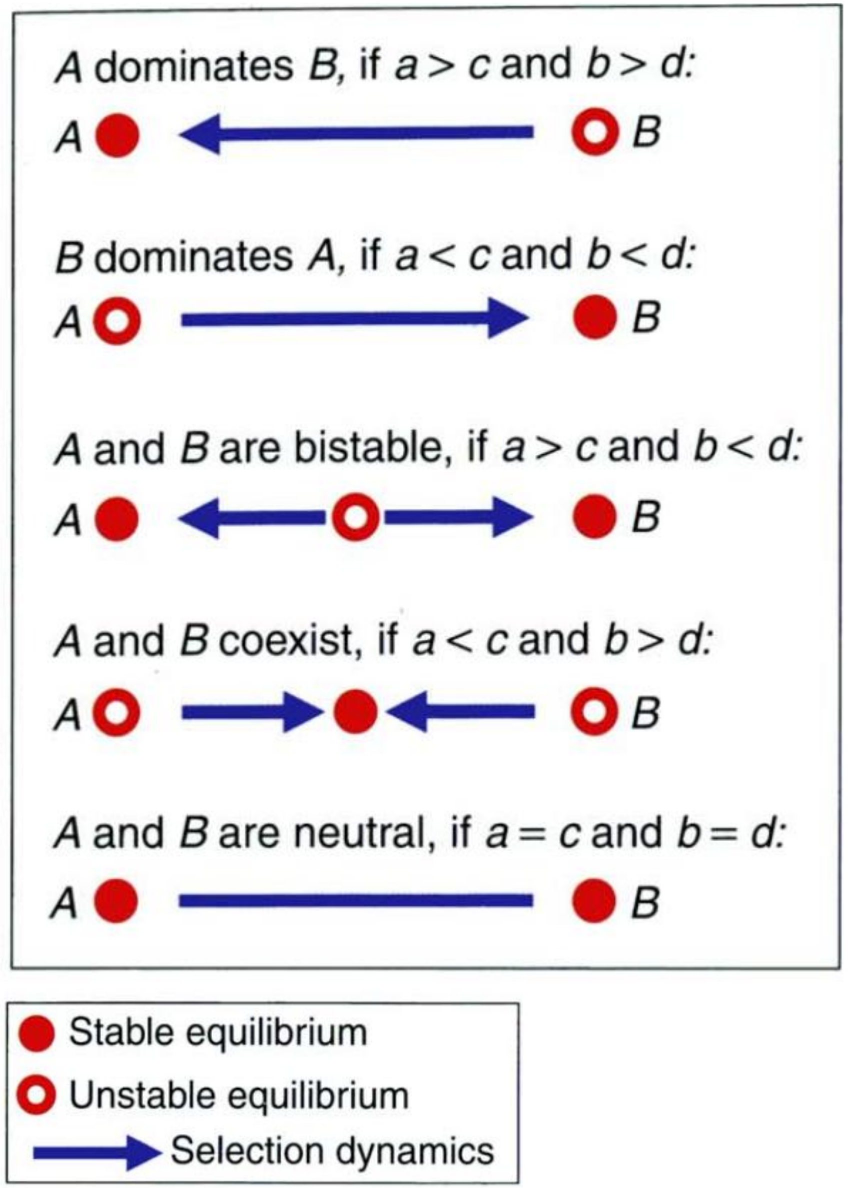


e.g. Stag-hunt,
coordination games



e.g. Hawk vs Doves,
Batesian Mimicry




A dominates B, if $a > c$ and $b > d$:

B dominates A, if $a < c$ and $b < d$:

A and B are bistable, if $a > c$ and $b < d$:

A and B coexist, if $a < c$ and $b > d$:

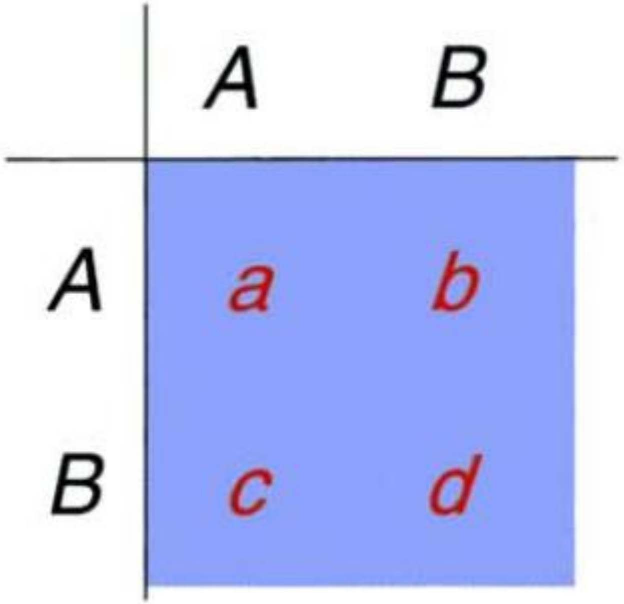
A and B are neutral, if $a = c$ and $b = d$:

● Stable equilibrium

○ Unstable equilibrium

→ Selection dynamics

The general structure of 2-players’ games



	A	B
A	a	b
B	c	d

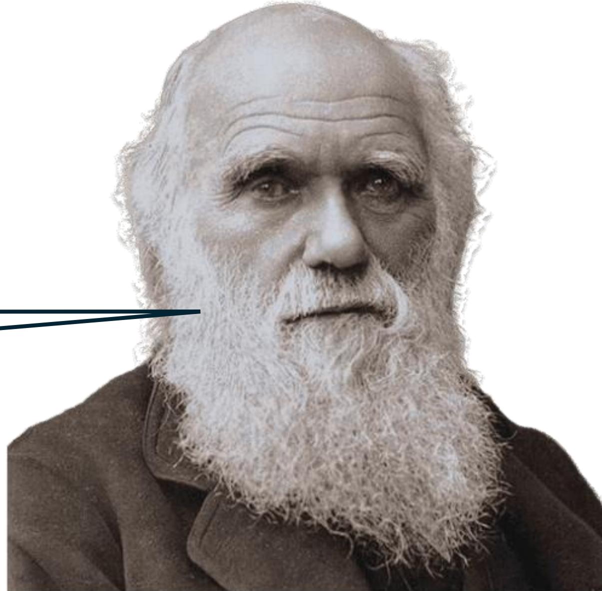
x_A = frequency of A=1- x_B

?

Fitness of strategy A

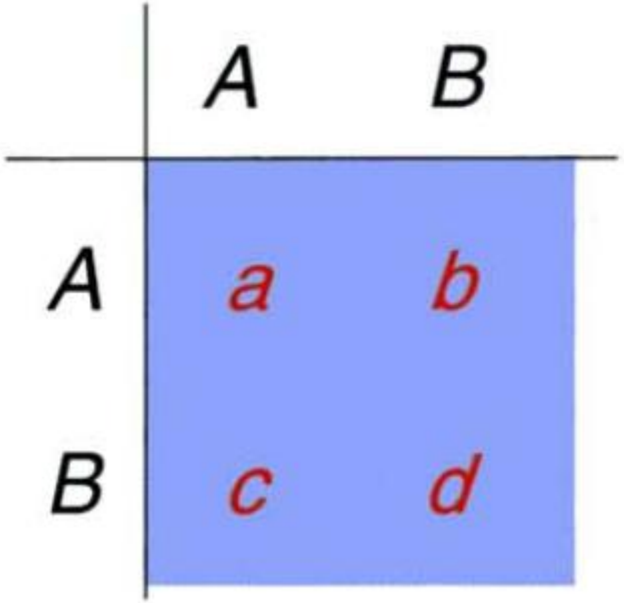
Fitness of strategy B

Average fitness



The general structure of 2-players’ games

x_A = frequency of A=1- x_B



	A	B
A	a	b
B	c	d

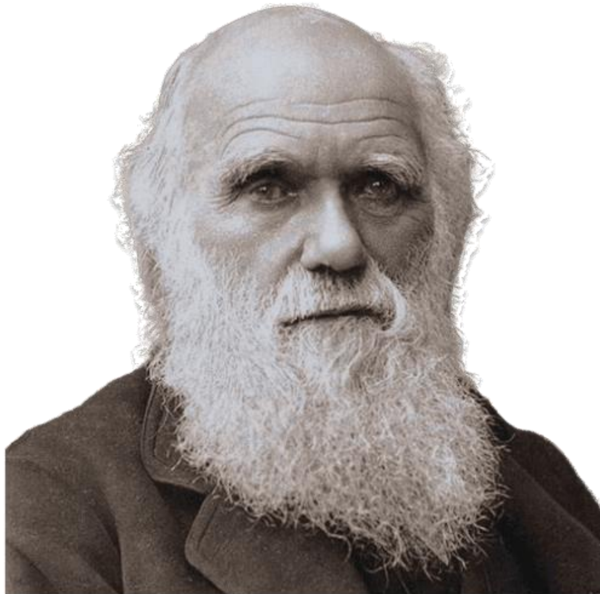
$$f_A = a x_A + b x_B$$

Fitness of strategy A

?

Fitness of strategy B

Average fitness



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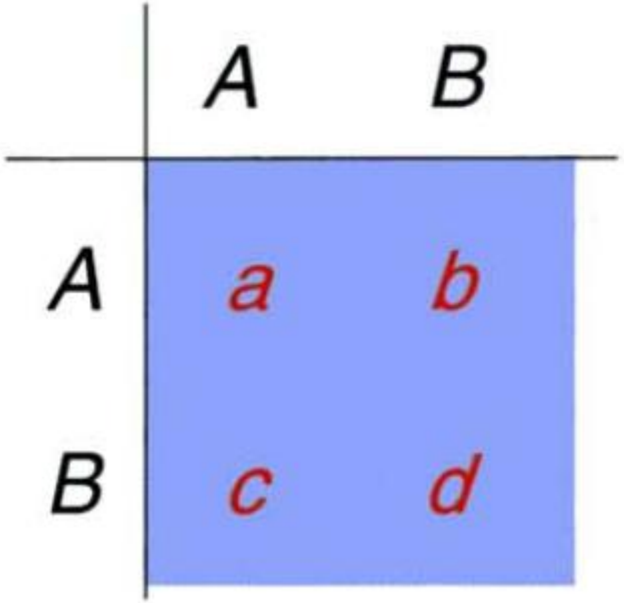


$$\begin{array}{c|cc} & A & B \\ \hline A & a & b \\ B & c & d \end{array}$$

x_A = frequency of A=1- x_B

$$f_A = a x_A + b x_B$$

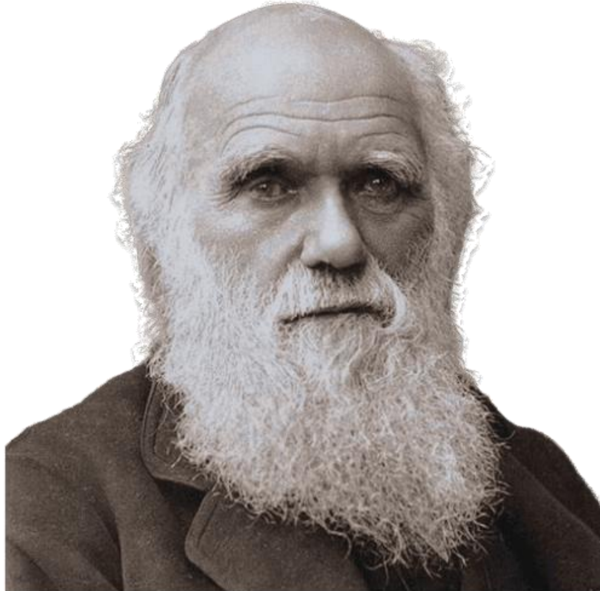
Fitness of strategy A

$$f_B = c x_A + d x_B$$

Fitness of strategy B

?

Average fitness



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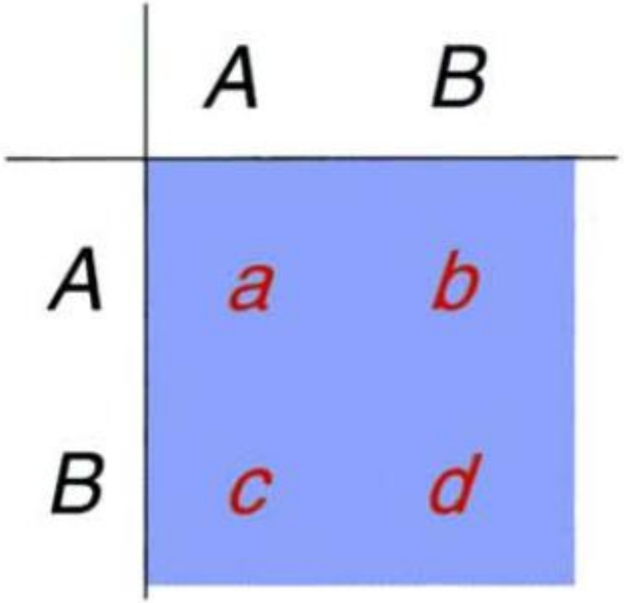


	A	B
A	a	b
B	c	d

$$f_A = ax_A + bx_B$$

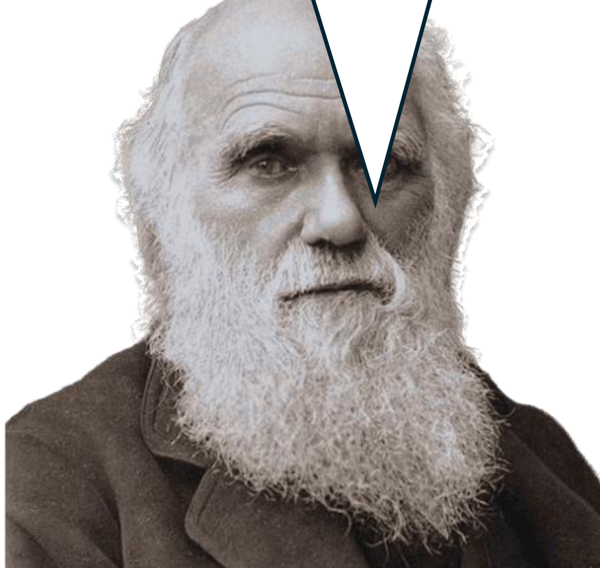
Fitness of strategy A

$$f_B = cx_A + dx_B$$

Fitness of strategy B

$$\phi = x_A f_A(\vec{x}) + x_B f_B(\vec{x})$$

Average fitness



Now let’s just say that the frequency of A (x_A) increases if its fitness is higher than the average fitness (and viceversa)

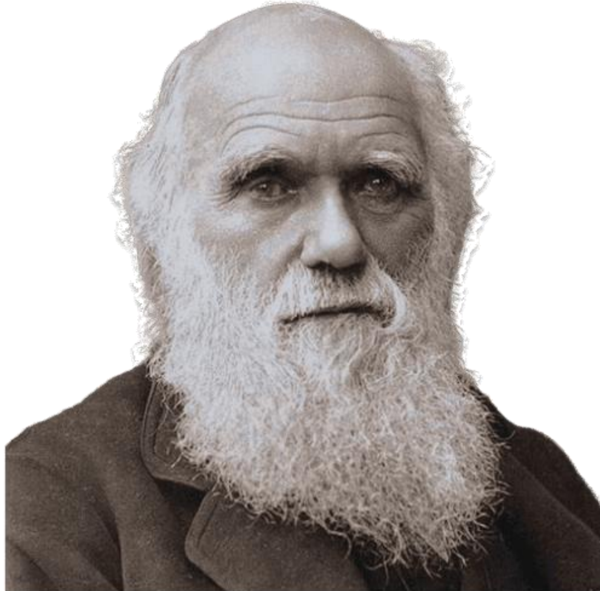
Example: let’s assume frequency of A x_A=0.5

	A	**B**
A	a=2	b=0
B	c=0	d=1

$$f_A = a x_A + b x_B$$ Fitness of strategy A

$$f_B = c x_A + d x_B$$ Fitness of strategy B

$$\phi = x_A f_A(\vec{x}) + x_B f_B(\vec{x})$$ Average fitness



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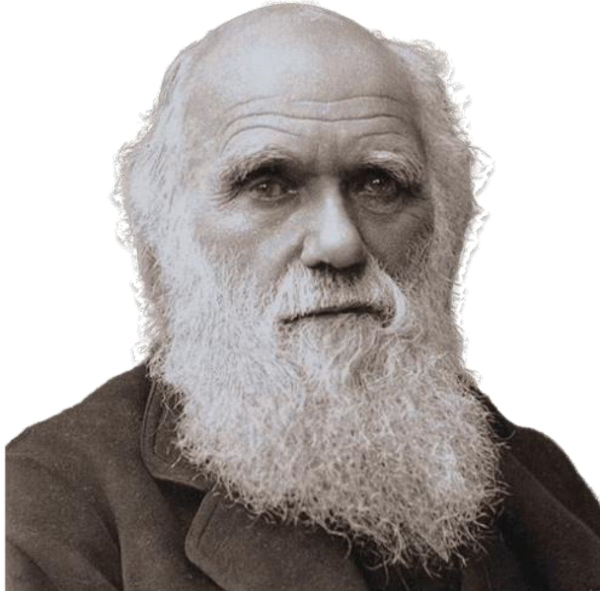
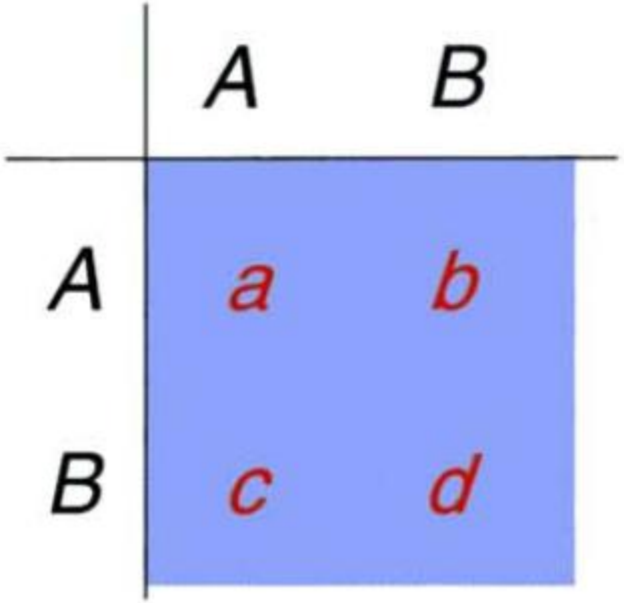


The general structure of 2-players’ games



	A	B
A	a	b
B	c	d

Change in frequency of A

frequency of A

fitness of A

Average fitness

$$\dot{x}_A = x_A[f_A(\vec{x}) - \phi]$$

$$f_A = a x_A + b x_B$$

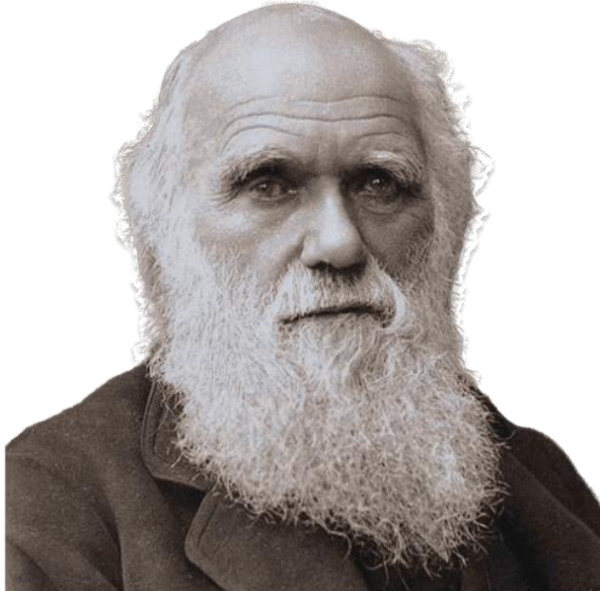
Fitness of strategy A

$$f_B = c x_A + d x_B$$

Fitness of strategy B

$$\phi = x_A f_A(\vec{x}) + x_B f_B(\vec{x})$$

Average fitness



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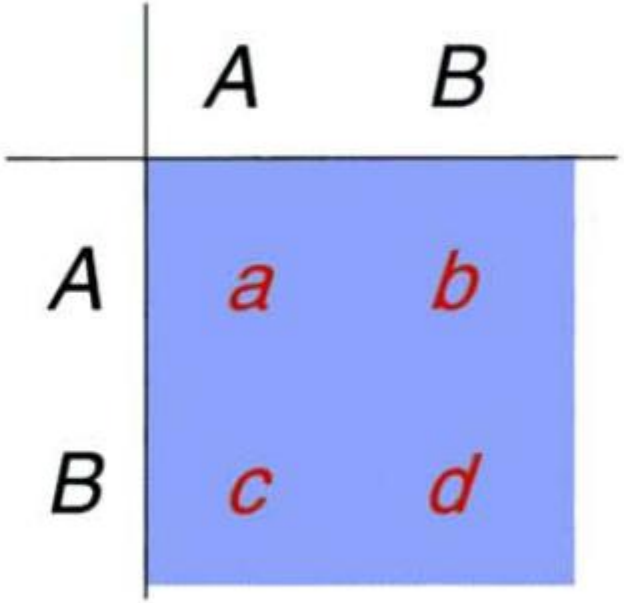

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Change in frequency of A

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Average fitness

$$\dot{x}_A = x_A[f_A(\vec{x}) - \phi]$$

$$\dot{x}_B = x_B[f_B(\vec{x}) - \phi]$$

$f_A = ax_A + bx_B$ Fitness of strategy A

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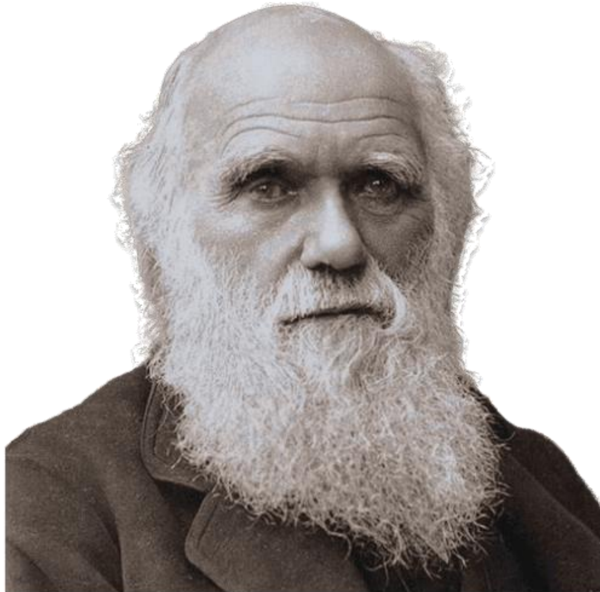
$\phi = x_A f_A(\vec{x}) + x_B f_B(\vec{x})$ Average fitness



Example: let's assume frequency of A x_A=0.5

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$$f_A = a x_A + b x_B$$

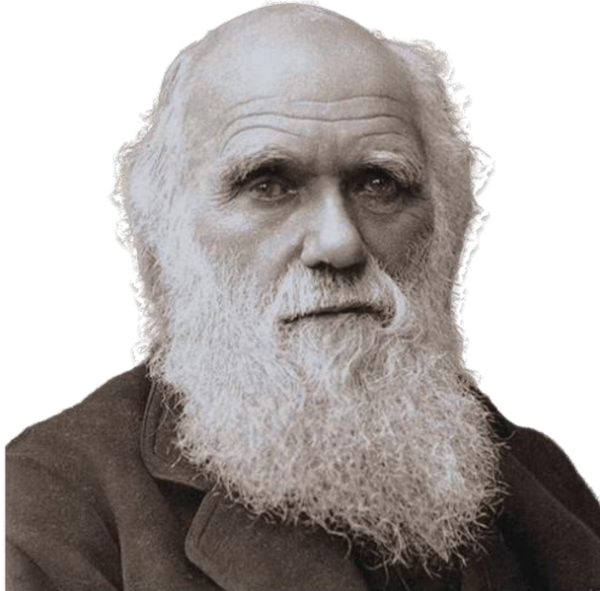
Fitness of strategy A?

$$f_B = c x_A + d x_B$$

Fitness of strategy B?

$$\phi = x_A f_A(\vec{x}) + x_B f_B(\vec{x})$$

Average fitness?



Example: let's assume frequency of A $x_A=0.5$

	A	**B**
A	a=2	b=0
B	c=0	d=1

$$f_A = a x_A + b x_B$$

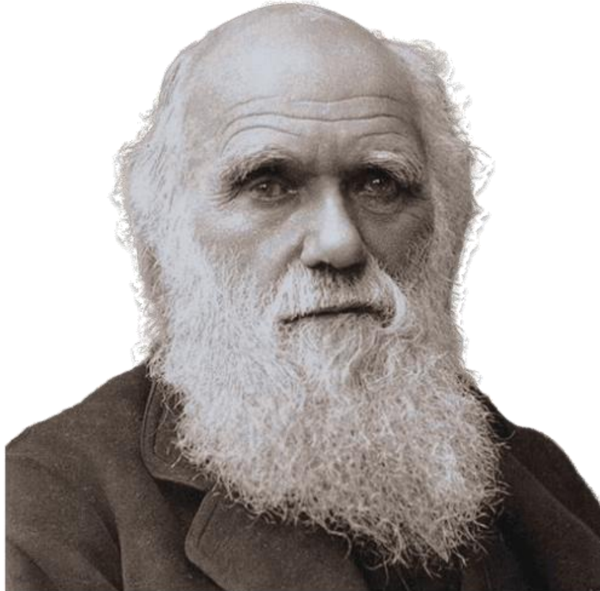
Fitness of strategy A=1+0

$$f_B = c x_A + d x_B$$

Fitness of strategy B=0+0.5

$$\phi = x_A f_A(\vec{x}) + x_B f_B(\vec{x})$$

Average fitness?



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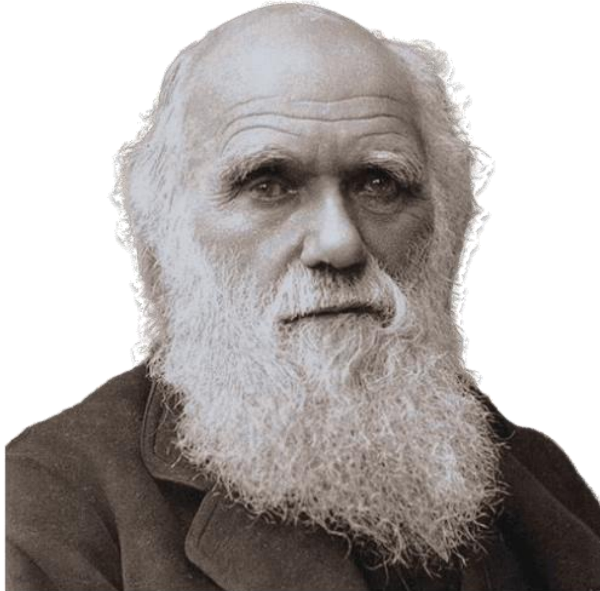
Fitness of strategy B=0+0.5

$$\phi = x_A f_A(\vec{x}) + x_B f_B(\vec{x})$$

Average fitness=0.5+0.25=0.75



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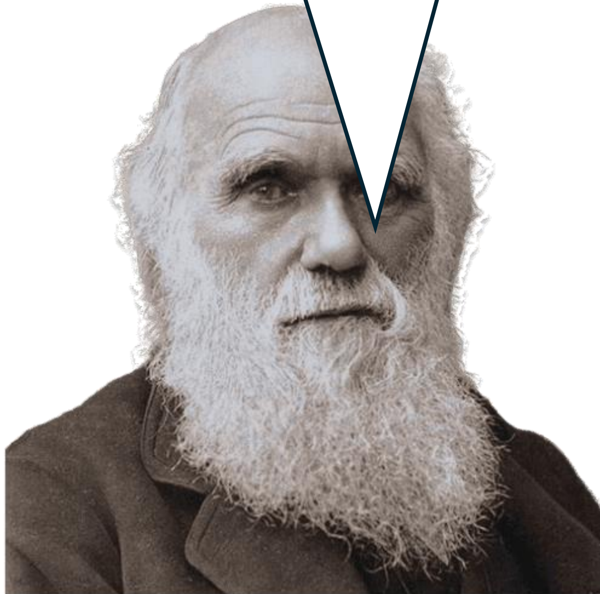


Now let’s just say that the frequency of A (x_A) increases if its fitness is higher than the average fitness (and viceversa)

Example: let's assume frequency of A x_A=0.5

	A	**B**
A	a=2	b=0
B	c=0	d=1

x_A grows if $[f_A(\vec{x}) - \phi]$ >0

$$f_A = a x_A + b x_B$$

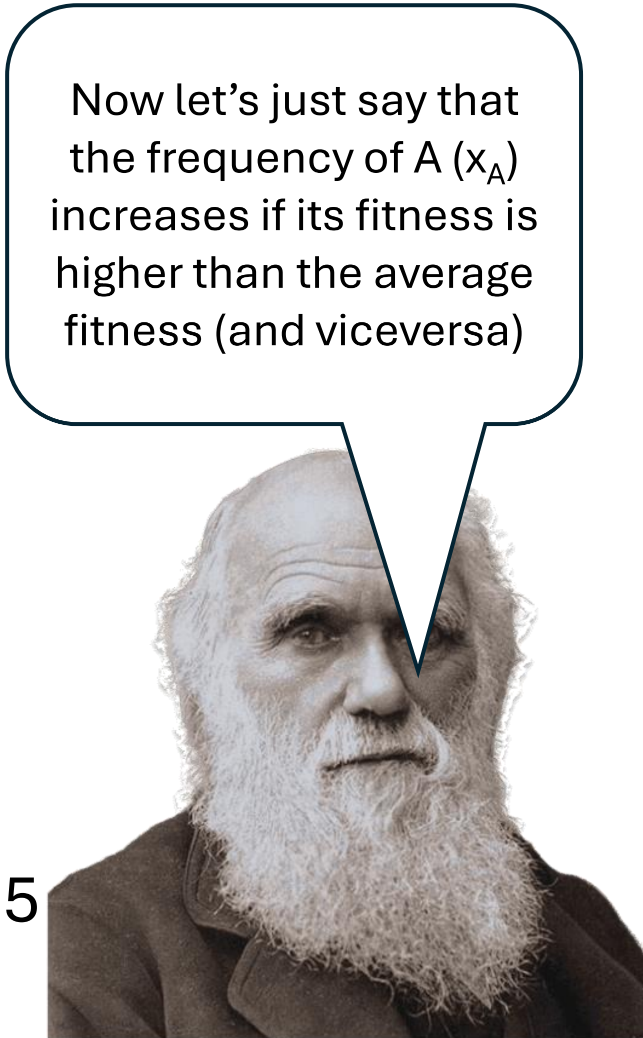
Fitness of strategy A=1+0

$$f_B = c x_A + d x_B$$

Fitness of strategy B=0+0.5

$$\phi = x_A f_A(\vec{x}) + x_B f_B(\vec{x})$$

Average fitness=0.5+0.25=0.75



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Change in frequency of A

frequency of A

fitness of A

Average fitness

$$\dot{x}_A = x_A[f_A(\vec{x}) - \phi]$$

$$f_A = ax_A + bx_B$$

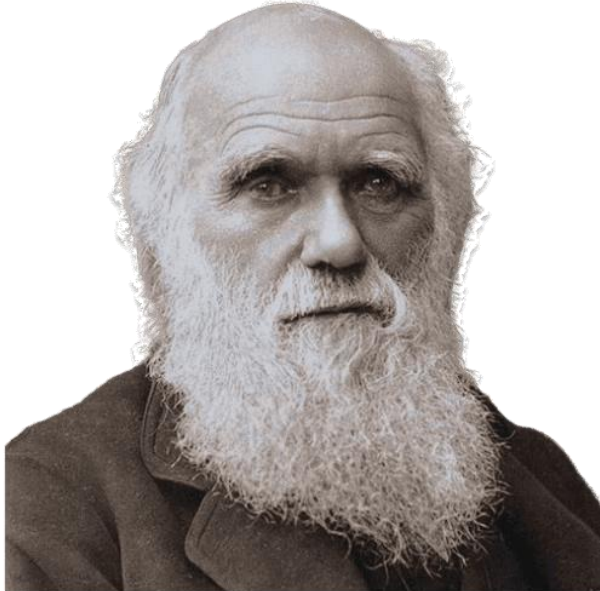
Fitness of strategy A=1+0

$$f_B = cx_A + dx_B$$

Fitness of strategy B=0+0.5

$$\phi = x_A f_A(\vec{x}) + x_B f_B(\vec{x})$$

Average fitness=0.5+0.25=0.75



Example: let’s assume frequency of A x_A=0.5

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A	a=2	b=0
B	c=0	d=1

Change in frequency of A

frequency of A

fitness of A

Average fitness

$$\dot{x}_A = x_A[f_A(\vec{x}) - \phi]$$

=0.5*[1-0.75]=0.125

$$f_A = a x_A + b x_B$$

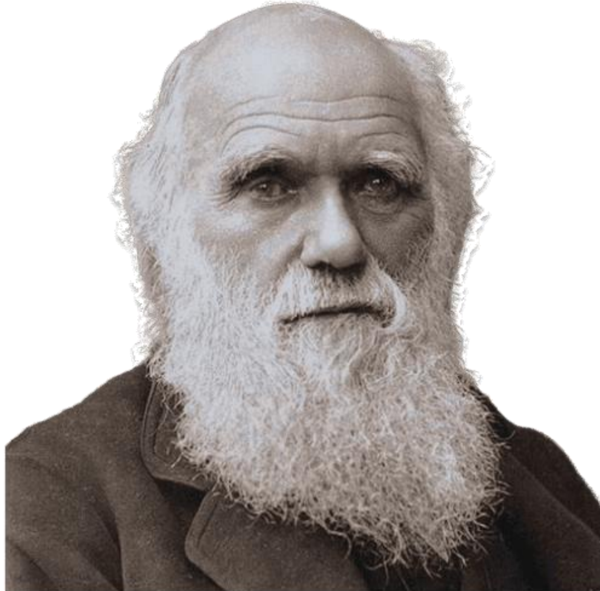
Fitness of strategy A=1+0

$$f_B = c x_A + d x_B$$

Fitness of strategy B=0+0.5

$$\phi = x_A f_A(\vec{x}) + x_B f_B(\vec{x})$$

Average fitness=0.5+0.25=0.75



Example: let's assume frequency of A x_A=0.5

	A	B
A	a=2	b=0
B	c=0	d=1

Change in frequency of A

frequency of A

fitness of A

Average fitness

$$\dot{x}_A = x_A[f_A(\vec{x}) - \phi]$$

=0.5*[1-0.75]=0.125

$$\dot{x}_B = x_B[f_B(\vec{x}) - \phi]$$

?

$$f_A = ax_A + bx_B$$

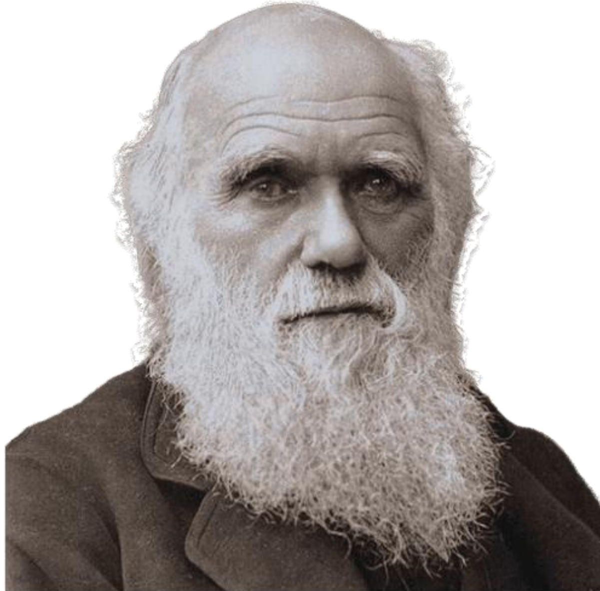
Fitness of strategy A=1+0

$$f_B = cx_A + dx_B$$

Fitness of strategy B=0+0.5

$$\phi = x_A f_A(\vec{x}) + x_B f_B(\vec{x})$$

Average fitness=0.5+0.25=0.75



Example: let’s assume frequency of A x_A=0.5

	A	**B**
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Change in frequency of A

frequency of A

fitness of A

Average fitness

$$\dot{x}_A = x_A[f_A(\vec{x}) - \phi]$$ =0.5*[1-0.75]=0.125

$$\dot{x}_B = x_B[f_B(\vec{x}) - \phi]$$ =0.5*[0.5-0.75]=-0.125

$$f_A = ax_A + bx_B$$

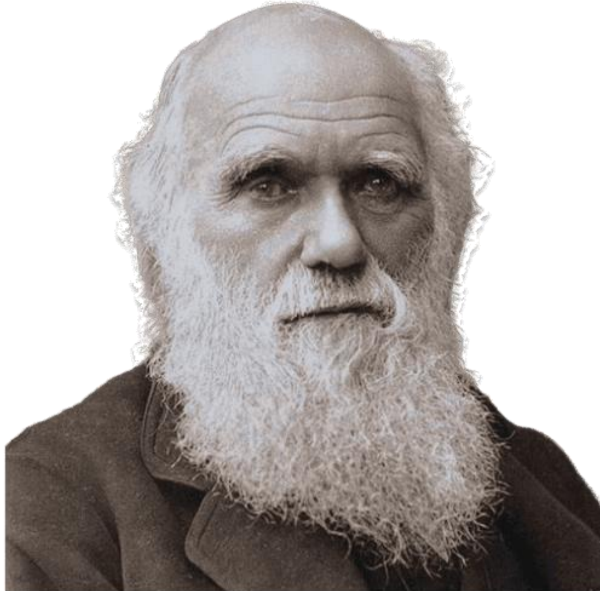
Fitness of strategy A=1+0

$$f_B = cx_A + dx_B$$

Fitness of strategy B=0+0.5

$$\phi = x_A f_A(\vec{x}) + x_B f_B(\vec{x})$$

Average fitness=0.5+0.25=0.75



The general structure of 2-players' games

	A	B
A	a	b
B	c	d

Change in frequency of A

frequency of A

fitness of A

Average fitness

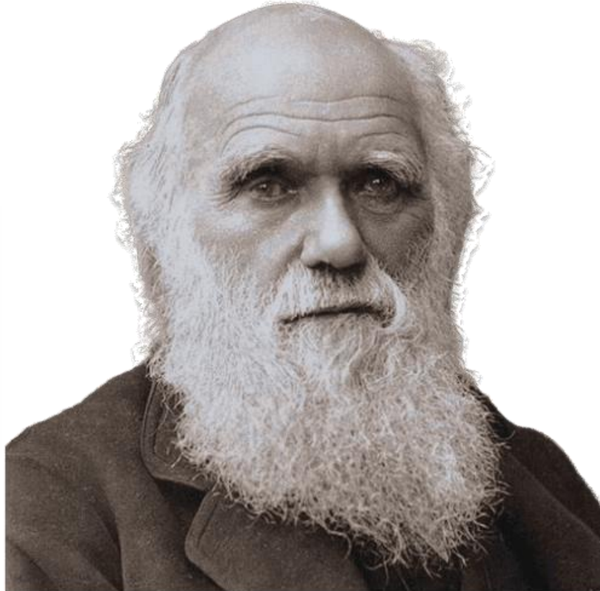
$$\dot{x}_A = x_A[f_A(\vec{x}) - \phi]$$

$$\dot{x}_B = x_B[f_B(\vec{x}) - \phi]$$

Simplifying and taking ***x=xA=1-xB***

$$\dot{x} = x(1-x)[(a-b-c+d)x+b-d].$$

Replicator's equation



e.g. Prisoner’s dilemma,
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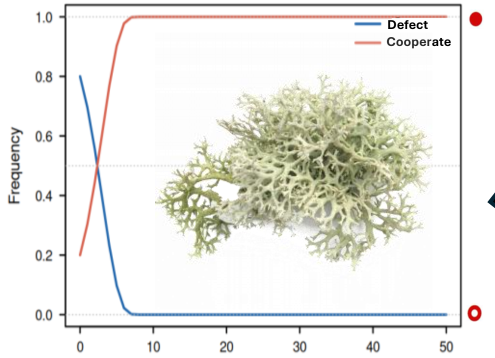

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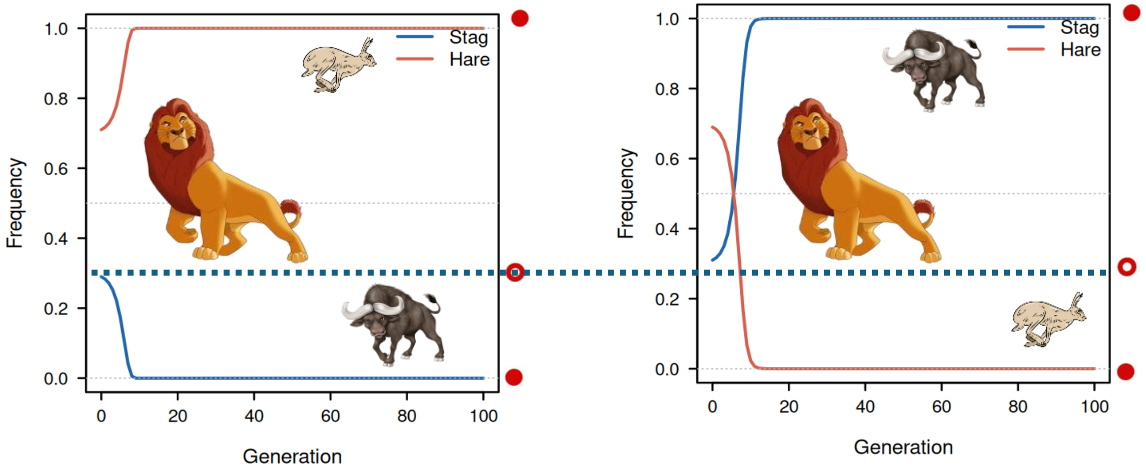

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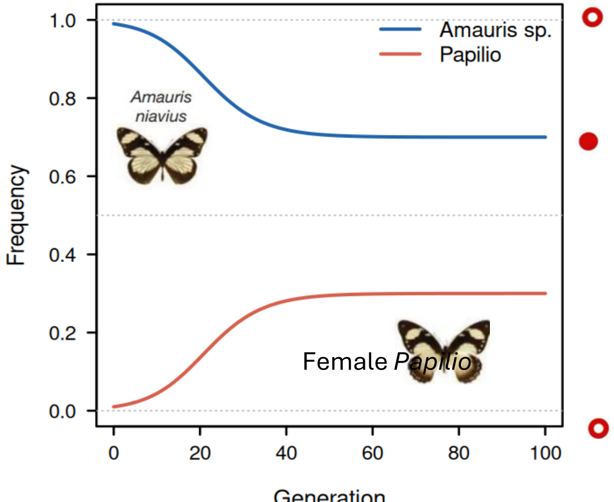
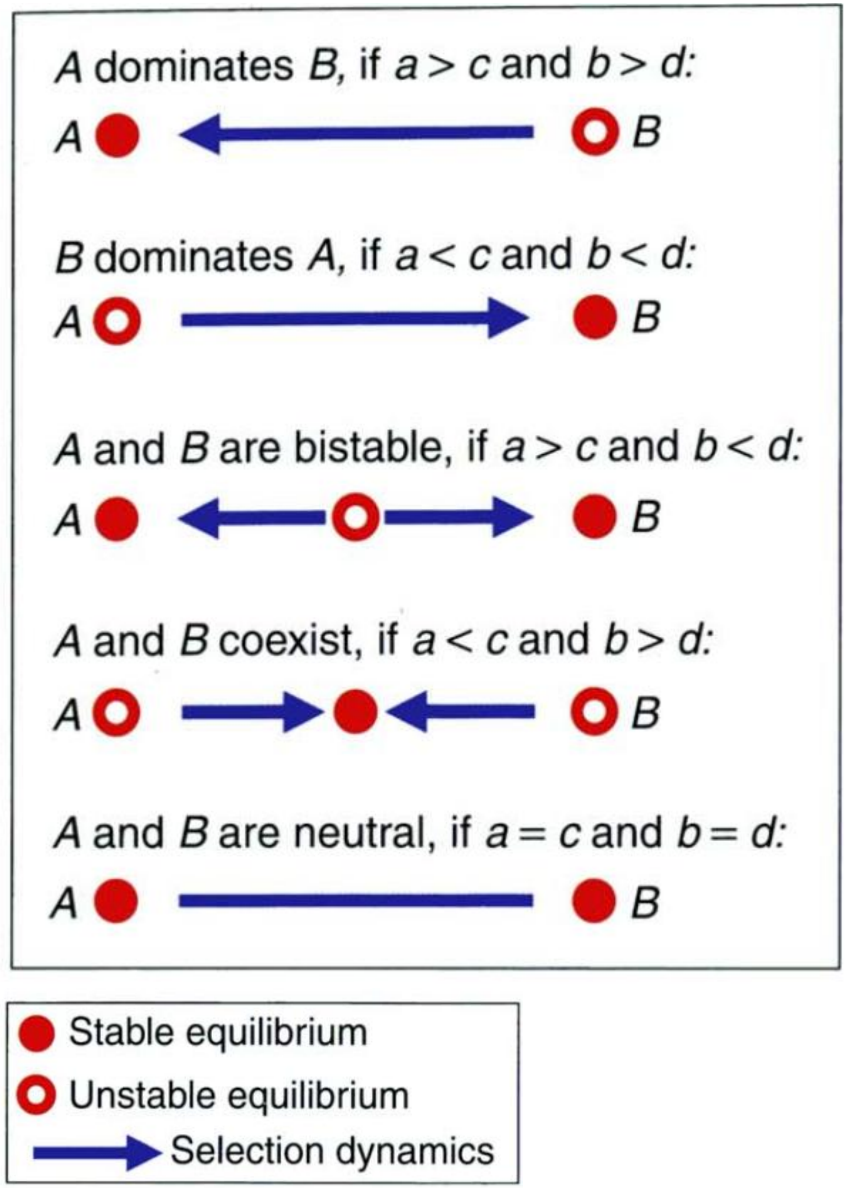




A dominates *B*, if *a* > *c* and *b* > *d*:

A ● ← ○ *B*

B dominates *A*, if *a* < *c* and *b* < *d*:

A ○ → ● *B*

A and *B* are bistable, if *a* > *c* and *b* < *d*:

A ● ← ○ → ● *B*

A and *B* coexist, if *a* < *c* and *b* > *d*:

A ○ → ● ← ○ *B*

A and *B* are neutral, if *a* = *c* and *b* = *d*:

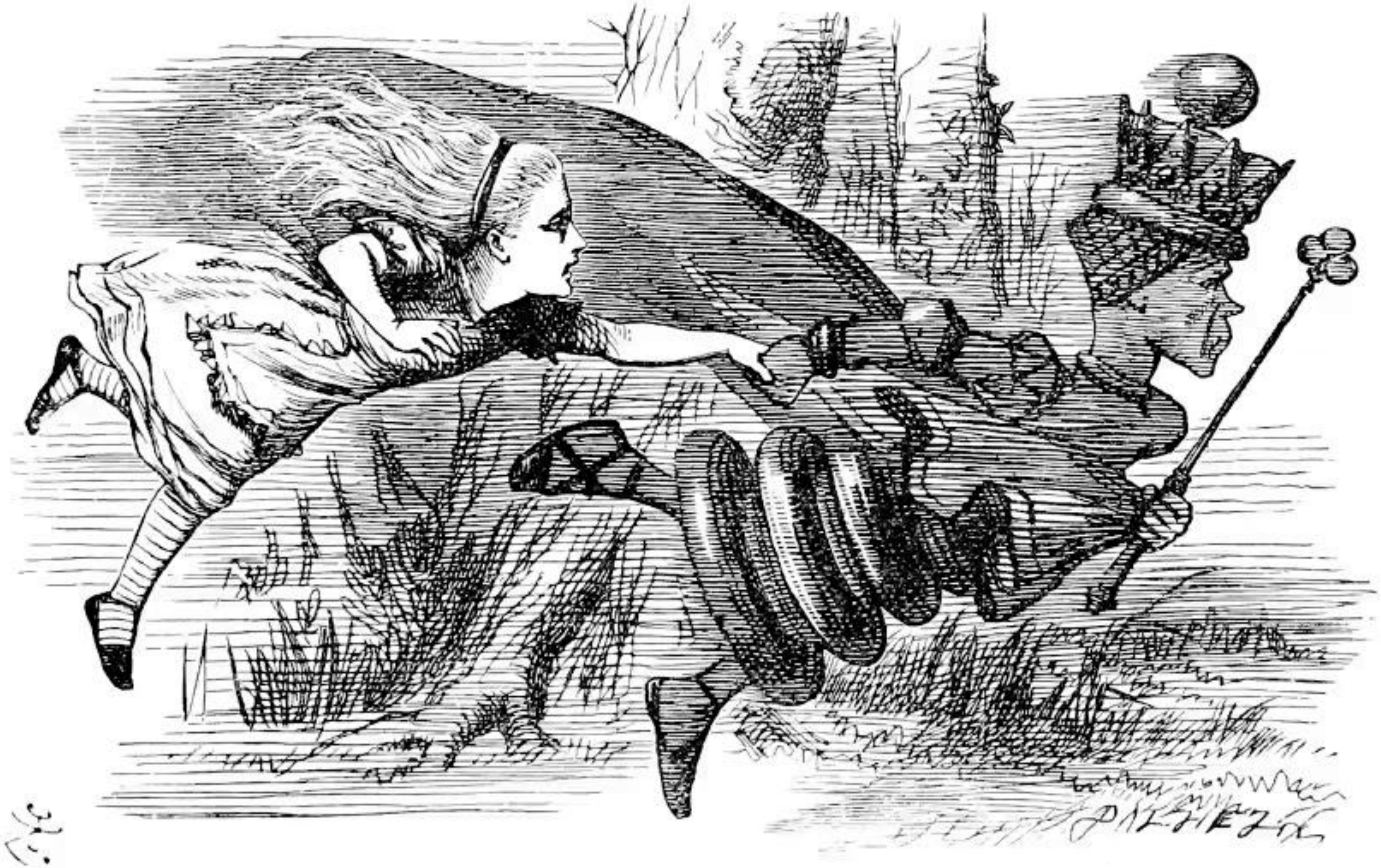
A ● — ● *B*

● Stable equilibrium
○ Unstable equilibrium
→ Selection dynamics

But what if species are more different than this?
(different payoff matrices)

"Now, here, you see, it takes all the running you can do, to keep in the same place.
If you want to get somewhere else, you must run at least twice as fast as that!"

Lewis Carroll's Red Queen in Through the Looking-Glass



Red queen dynamics
“It takes all the running you can do, to keep in the same place.”

The **Red Queen hypothesis** (coined by Leigh Van Valen) means:

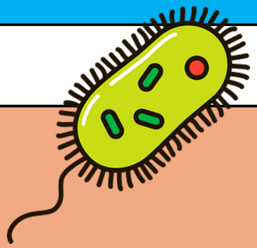
- Species must **constantly evolve** just to **maintain their relative fitness**
- Because:
  - competitors evolve
  - parasites evolve
  - environments change
- Evolution as a **continuous arms race**

Classic examples

- Host ↔ parasite coevolution
- Predator ↔ prey
- Immune system ↔ pathogens

Red queen cycles emerge when a pathogen and a host trying to escape a pathogen coevolve

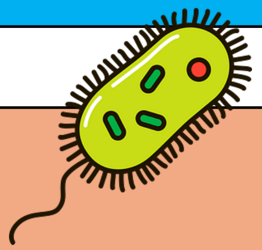
	**Parasite Strain A**	**Parasite Strain B**
Host Resistant to A	1	0
Host Resistant to B	0	1

	**Host Resistant to A**	**Host Resistant to B**
Parasite Strain A	0	1
Parasite Strain B	1	0

Red queen cycles emerge when a pathogen and a host trying to escape a pathogen coevolve

	**Parasite Strain A**	**Parasite Strain B**
Host Resistant to A	1	0
Host Resistant to B	0	1

The pathogen wins when it infects a host that is resistant to a different strain (e.g. Pathogen Strain A vs Host Resistant to B)

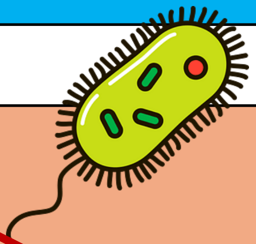
	**Host Resistant to A**	**Host Resistant to B**
Parasite Strain A	0	1
Parasite Strain B	1	0

Hosts resistant to the variant A of the pathogen wins when they are attacked by it (payoff 1) while the pathogen loses (payoff 0)

Red queen cycles emerge when a pathogen and a host trying to escape a pathogen coevolve

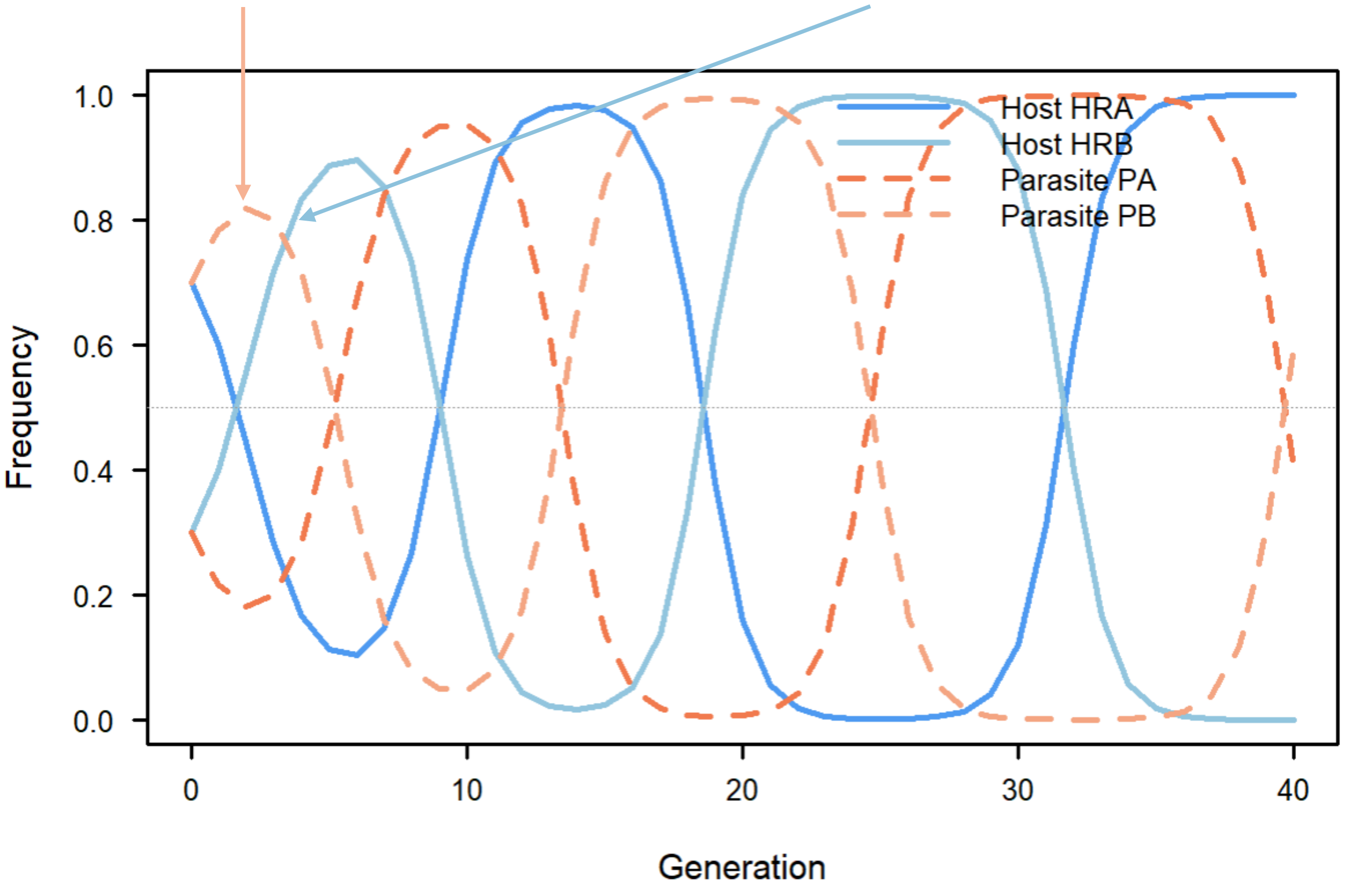
	**Parasite Strain A**	**Parasite Strain B**
Host Resistant to A	1	0
Host Resistant to B	0	1

The pathogen wins when it infects a host that is resistant to a different strain (e.g. Pathogen Strain A vs Host Resistant to B)

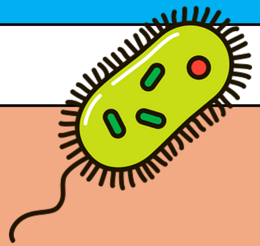
	**Host Resistant to A**	**Host Resistant to B**
Parasite Strain A	0	1
Parasite Strain B	1	0

Hosts resistant to the variant A of the pathogen wins when they are attacked by it (payoff 1) while the pathogen loses (payoff 0)

Red queen cycles emerge when a pathogen and a host trying to escape a pathogen coevolve

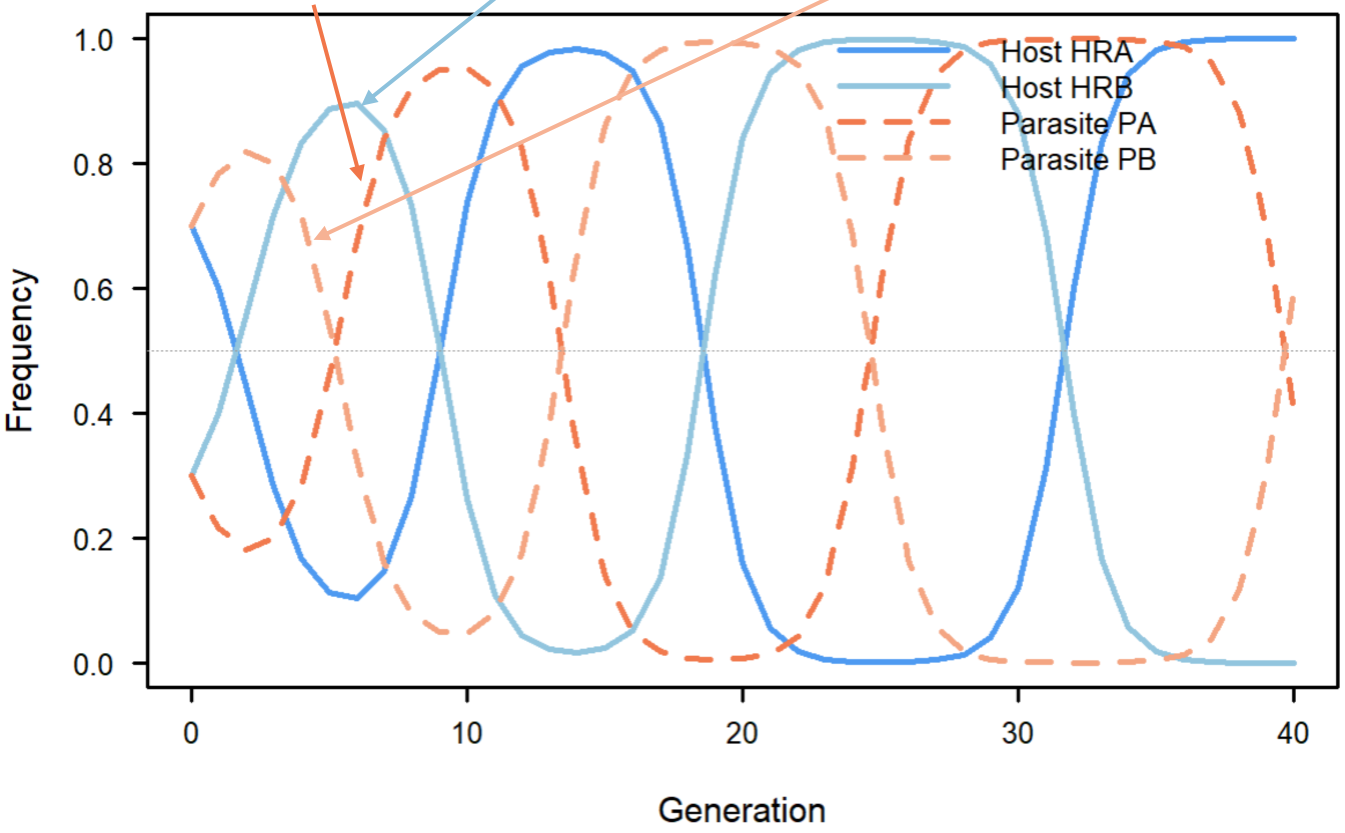

Parasite B is common, Host resistance to B increases

	**Parasite Strain A**	**Parasite Strain B**
Host Resistant to A	1	0
Host Resistant to B	0	1

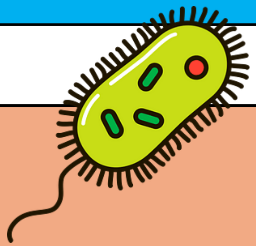
	**Host Resistant to A**	**Host Resistant to B**
Parasite Strain A	0	1
Parasite Strain B	1	0

Red queen cycles emerge when a pathogen and a host trying to escape a pathogen coevolve

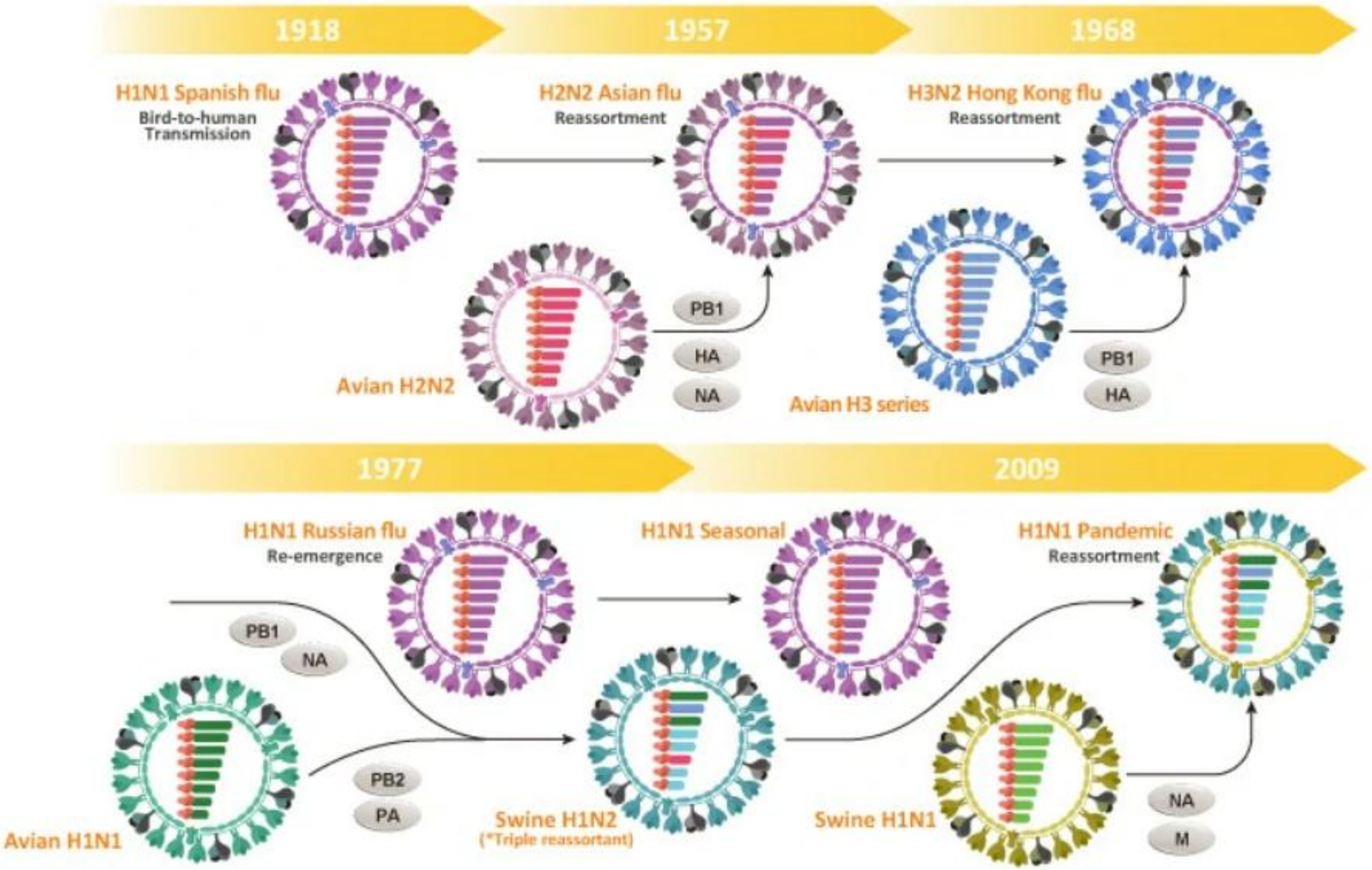
When host resistance to B is high, Pathogen B decreases and A increases



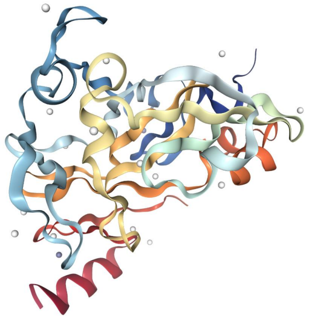
	**Parasite Strain A**	**Parasite Strain B**
Host Resistant to A	1	0
Host Resistant to B	0	1

	**Host Resistant to A**	**Host Resistant to B**
Parasite Strain A	0	1
Parasite Strain B	1	0

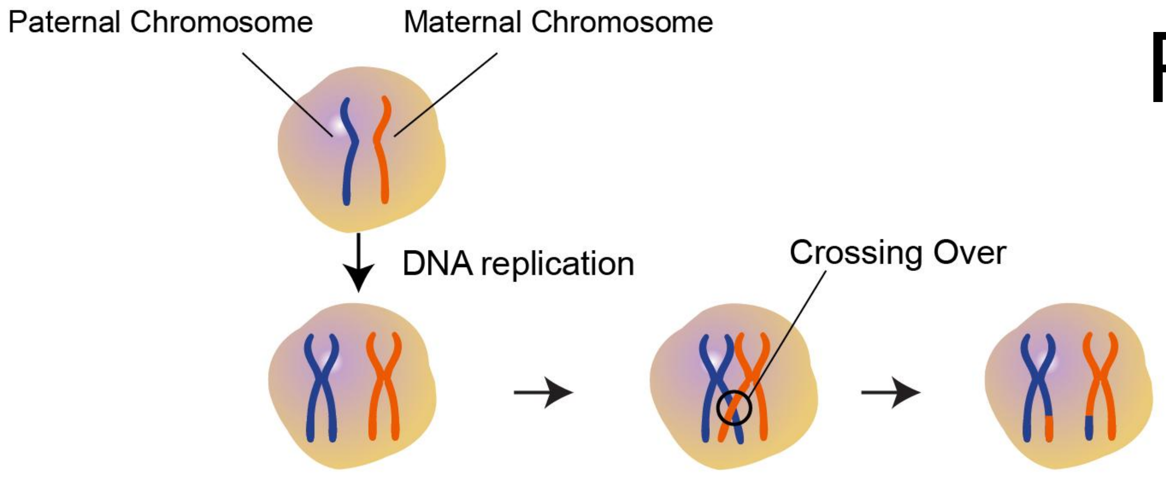
The influenza virus displays typical Red Queen dynamics



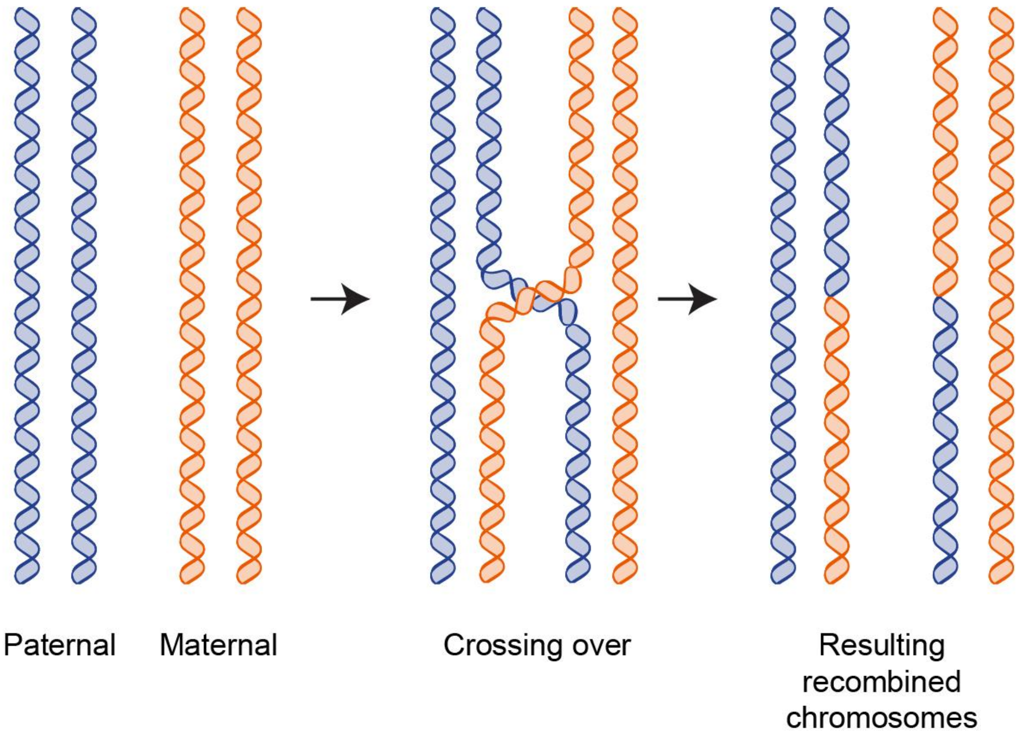
Red Queen and intra-genomic dynamics



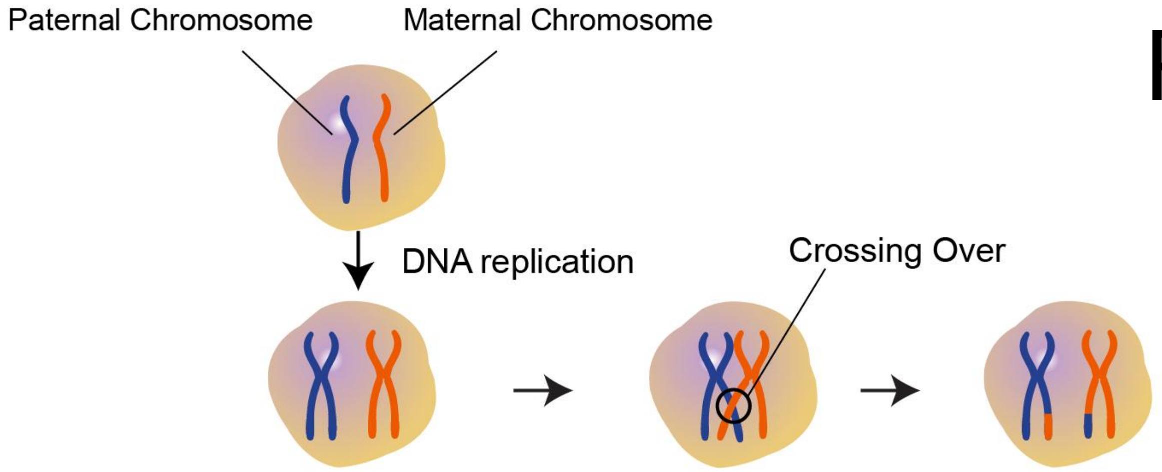
Recombination is necessary for meiosis and occurs in hotspots



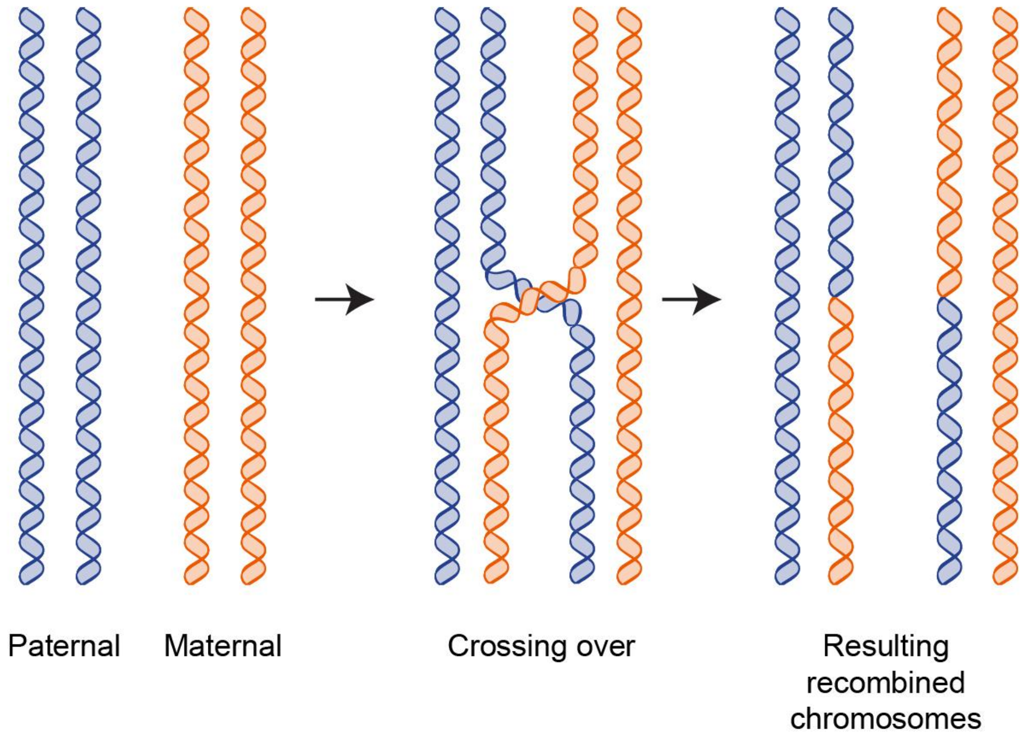
Recombination between 2 homologous chromosomes



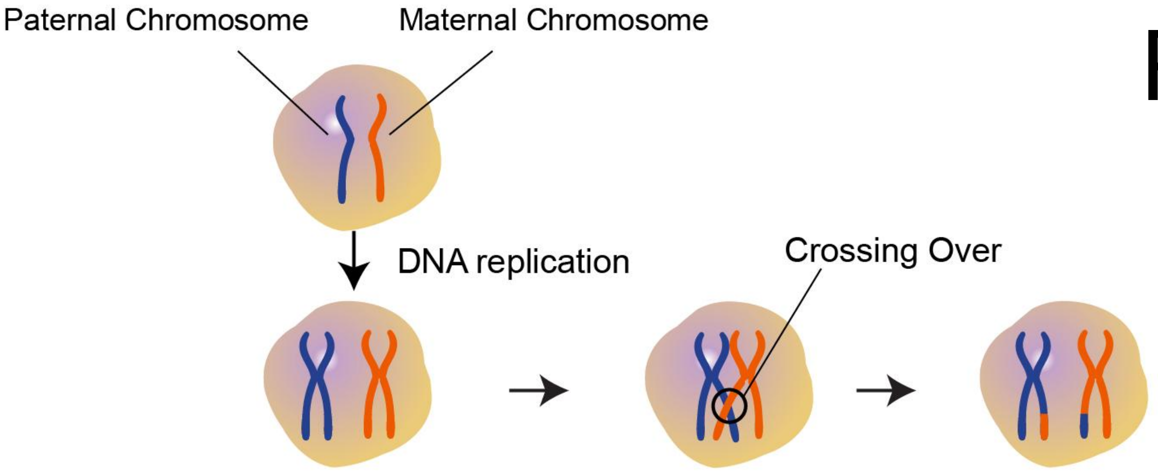
Recombination is necessary for meiosis and occurs in hotspots



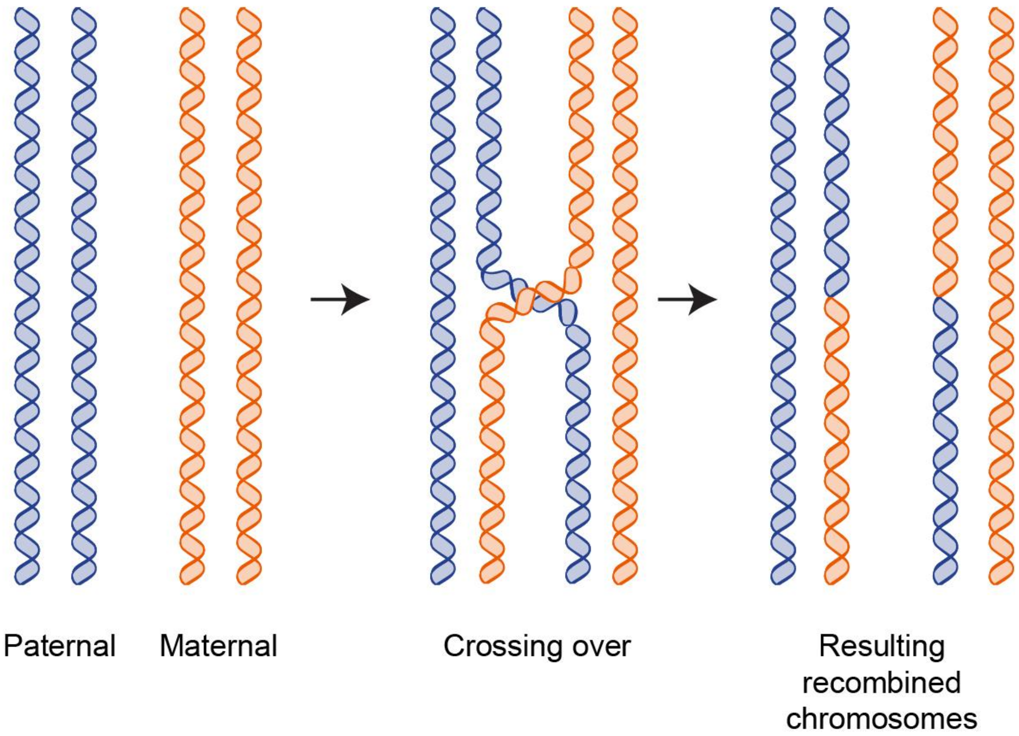
Recombination between 2 homologous chromosomes



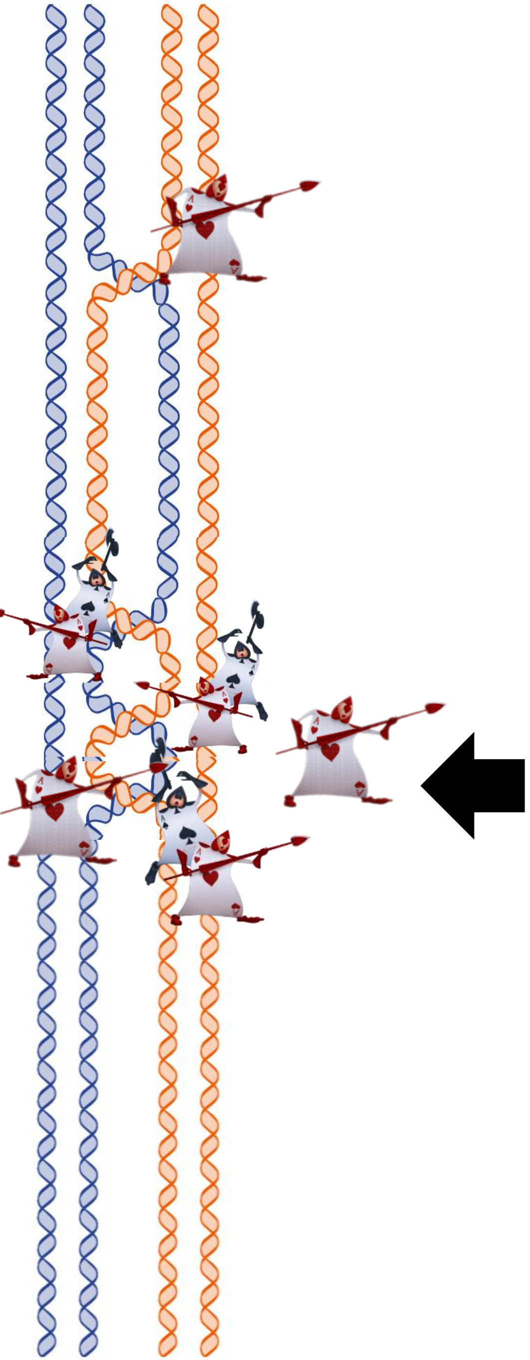
Recombination is necessary for meiosis and occurs in hotspots



Recombination between 2 homologous chromosomes

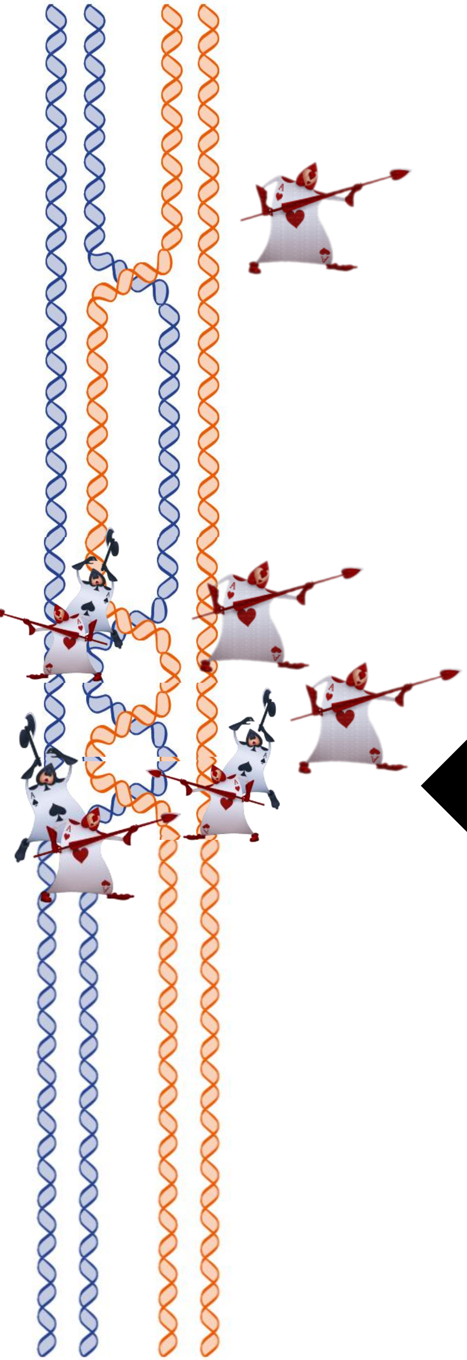


Recombination is necessary for meiosis and occurs in hotspots



Much more frequent
recombination events
in certain regions
(and heads chopped off)

Recombination is necessary for meiosis and occurs in hotspots

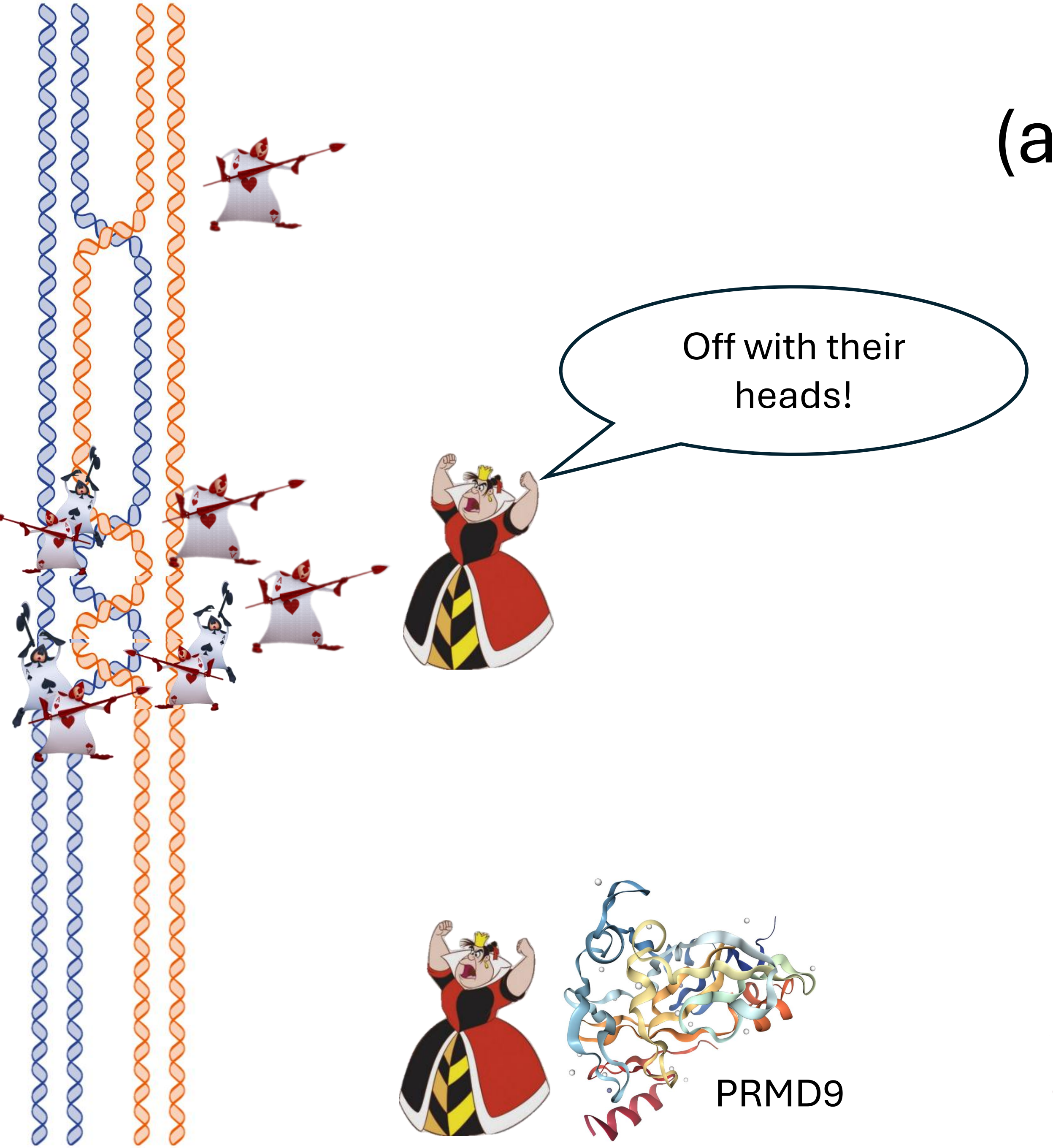


Much more frequent recombination events (and heads chopped off) in certain regions (*recombination hotspots*)

PRMD9

Motif bound by PRMD9

PRMD9 recognizes certain motifs guiding recombination (and those motifs get chopped off!)



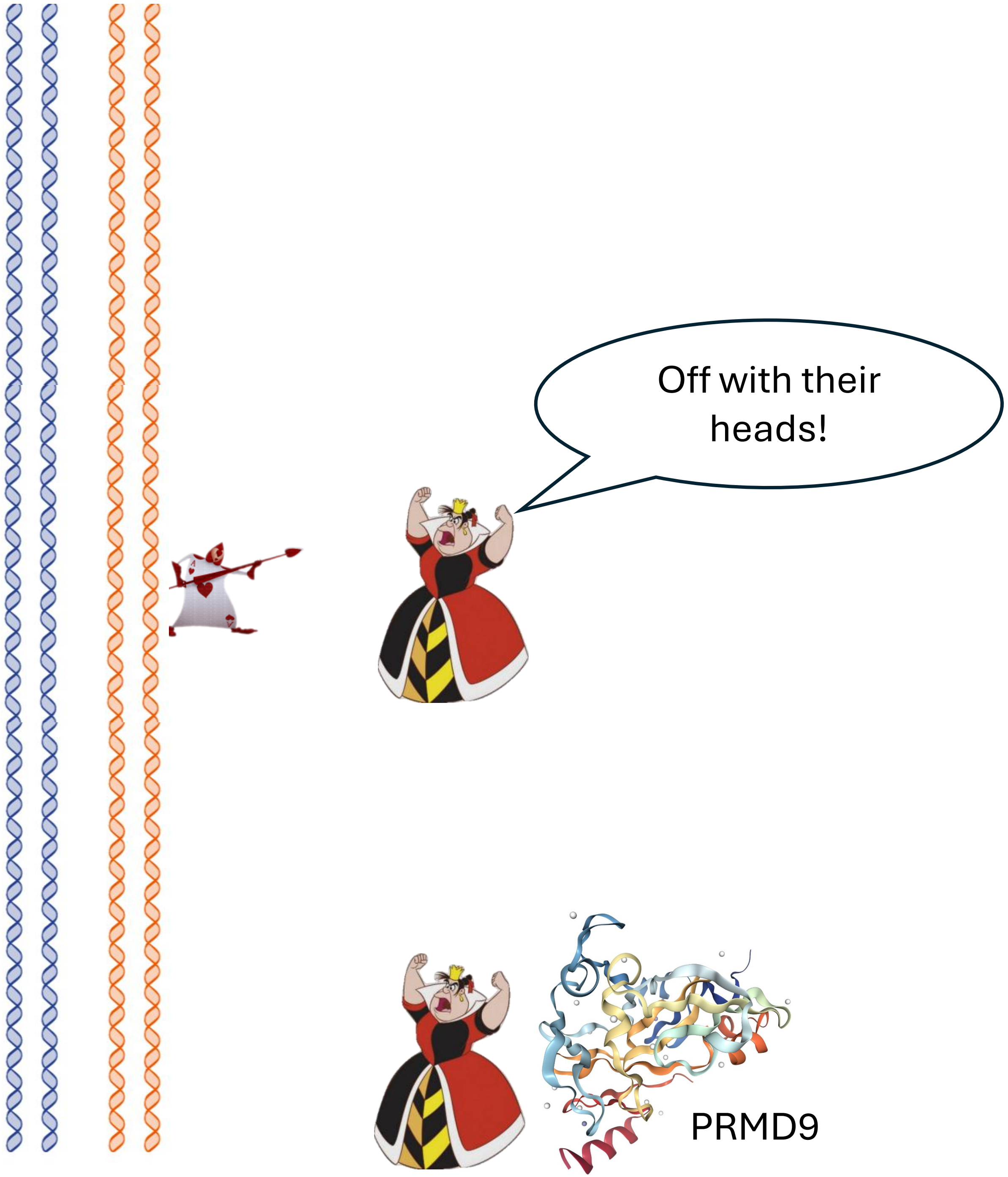

Motif bound by PRMD9

What happens then?


PRMD9


Motif bound by PRMD9

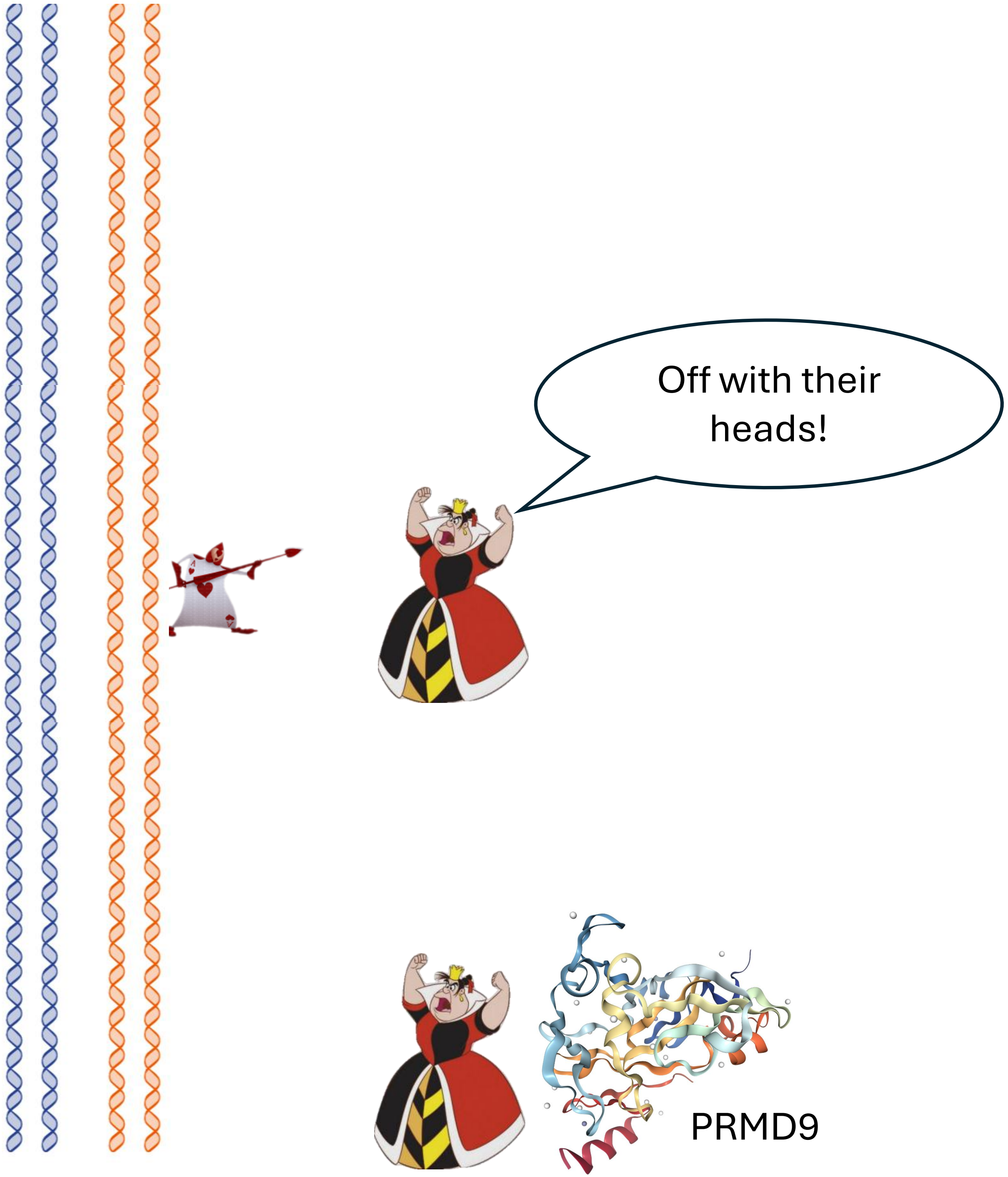
Loss of motif throughout the genome



Motif bound by PRMD9

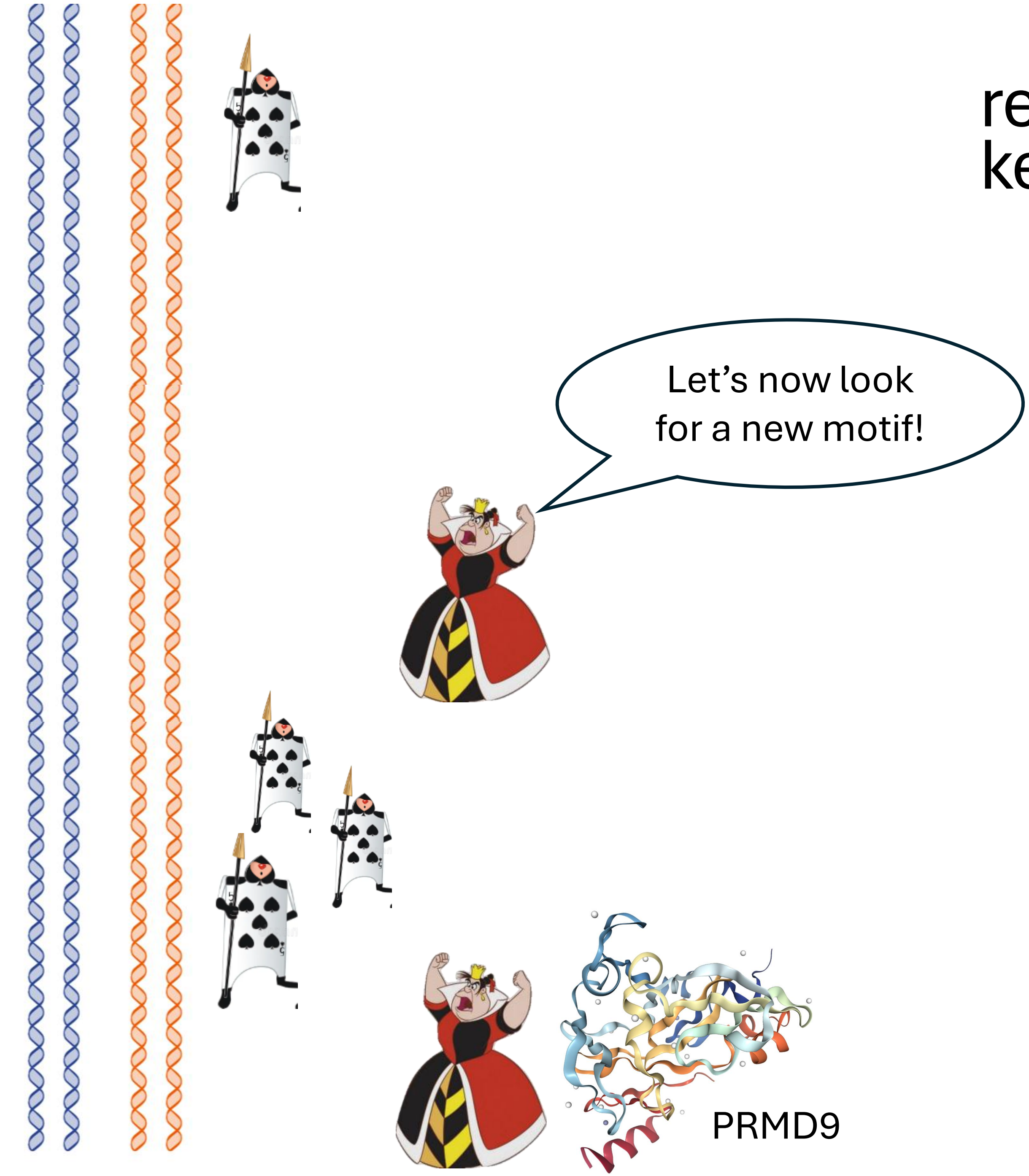
Loss of motif throughout the genome

Less cross-overs → impaired meiosis
Hill-Robertson effect (less effective natural selection due to excessive linkage)



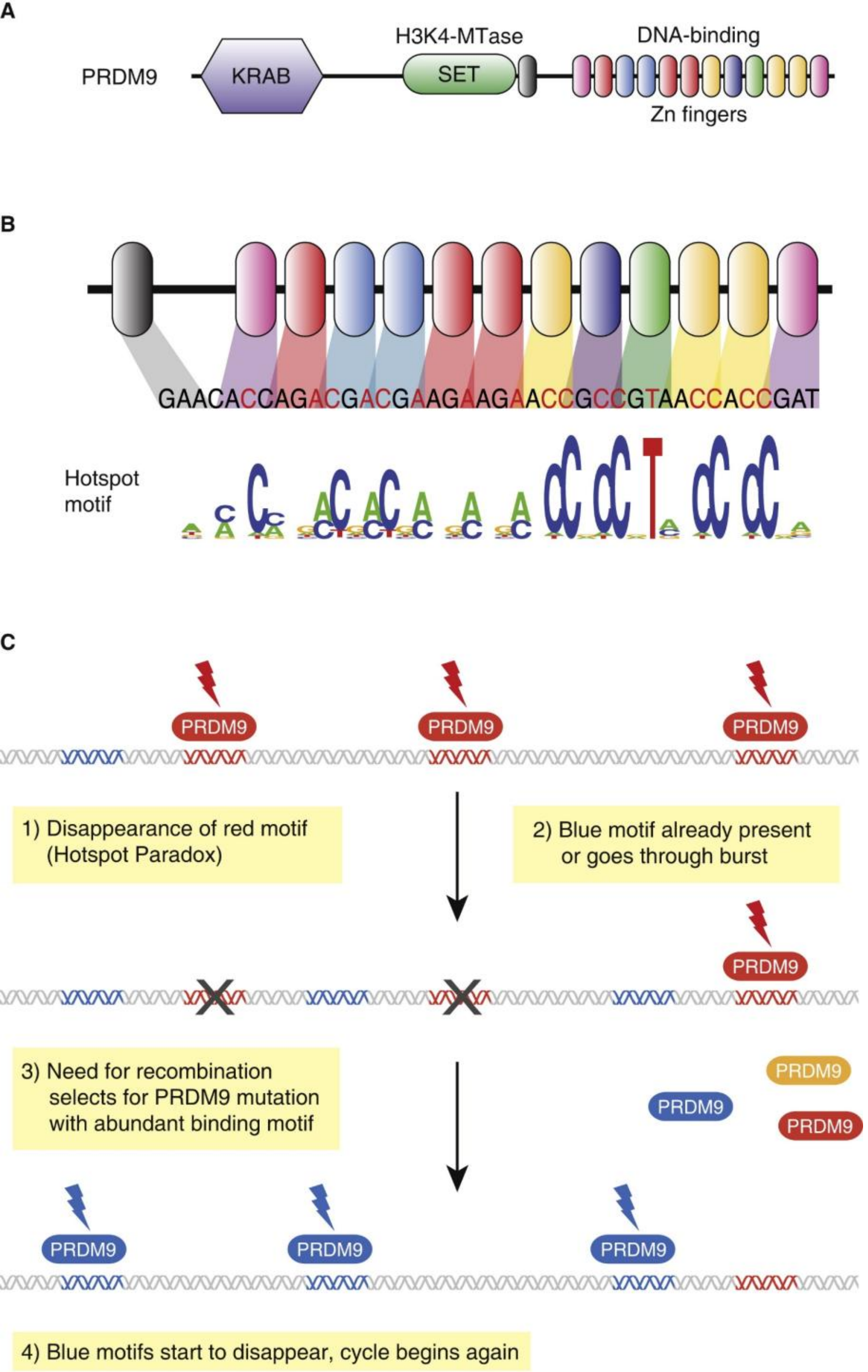

Motif bound by PRMD9

A mutation in PRMD9 will guide recombination to new motifs/hotspots keeping «sexual reproduction feasible»




Motif bound by PRMD9

PRDM9: Red Queen at the Molecular Level



1) Disappearance of red motif (Hotspot Paradox)

2) Blue motif already present or goes through burst

3) Need for recombination selects for PRDM9 mutation with abundant binding motif

4) Blue motifs start to disappear, cycle begins again

Current Biology

- **Function**: PRDM9 determines where meiotic crossovers occur by binding specific DNA sequence motifs
- **The paradox**: Crossover activity at motifs → erosion of those same motifs through gene conversion → loss of PRDM9 binding sites
- **Frequency-dependent selection**:
  - Common PRDM9 alleles deplete their target motifs → meiotic failure → sterility
  - Rare PRDM9 alleles find abundant target motifs → normal meiosis → reproductive success
- **Self-defeating success**: Each PRDM9 variant literally destroys the genomic substrate it depends on
- **Red Queen cycling**: Selection continuously favors whichever PRDM9 variant can bind to currently abundant (un-eroded) motifs
- **Empirical signature**: Extreme allelic diversity in natural populations, rapid molecular evolution, population-specific recombination hotspots
- **Why it matters**: Shows Red Queen dynamics operating at the level of individual genes, not just species interactions

PRDM9: Molecular Mechanism & Cool Facts

- **Protein structure**: Zinc-finger array determines DNA binding specificity — small changes in finger sequence = completely new binding preferences
- **Biochemical cascade**: PRDM9 binding → H3K4me3 histone marks → chromatin opening → SPO11 recruitment → DNA double-strand breaks → crossover initiation
- **Evolutionary speed**: PRDM9 zinc-finger domains evolve 5-10x faster than typical protein-coding genes — among the fastest evolving sequences in mammalian genomes
- **Species differences**:
    - Humans: ~25,000-50,000 hotspots, highly variable between individuals
    - Mice: PRDM9 knockout = sterile males, but females can compensate
    - Birds/plants: Lost PRDM9 entirely — use different hotspot mechanisms
- **Population genetics**: >200 human PRDM9 alleles identified worldwide, most populations have 10-20 common variants in circulation simultaneously
- **Hotspot lifespan**: Individual recombination hotspots estimated to persist only ~100,000-200,000 years before being eroded away
- **Clinical relevance**: PRDM9 variants linked to recombination rate variation, male infertility, and potentially autism spectrum disorders
- **Evolutionary mystery**: Why maintain this self-destructive system? Why not evolving PRDM9-independent mechanisms like most other species?



How does it work in other species?

Birds & Most Vertebrates:

- Hotspots occur at **promoters** and **CpG islands** — transcription start sites, where open chromatin at active regulatory elements provides access for recombination machinery
- **Evolutionarily stable**: Hotspots conserved for tens of millions of years because promoter sequences can't be eroded without destroying gene function
- Trade-off: Recombination occurs **near genes** rather than in neutral regions

Plants (Arabidopsis, crops):

- Similar to birds — hotspots at **transcriptional start sites** and regulatory elements, thus no PRDM9 dependency, stable hotspot locations over evolutionary time
- Some species also show hotspots in **intergenic regions** with specific chromatin marks

Fungi (yeast, Neurospora):

- Multiple mechanisms: some use **transcription-coupled** recombination, with open chromatin structure driving hotspot location. However more variable — some species have evolved **anti-recombination** systems in certain genomic regions

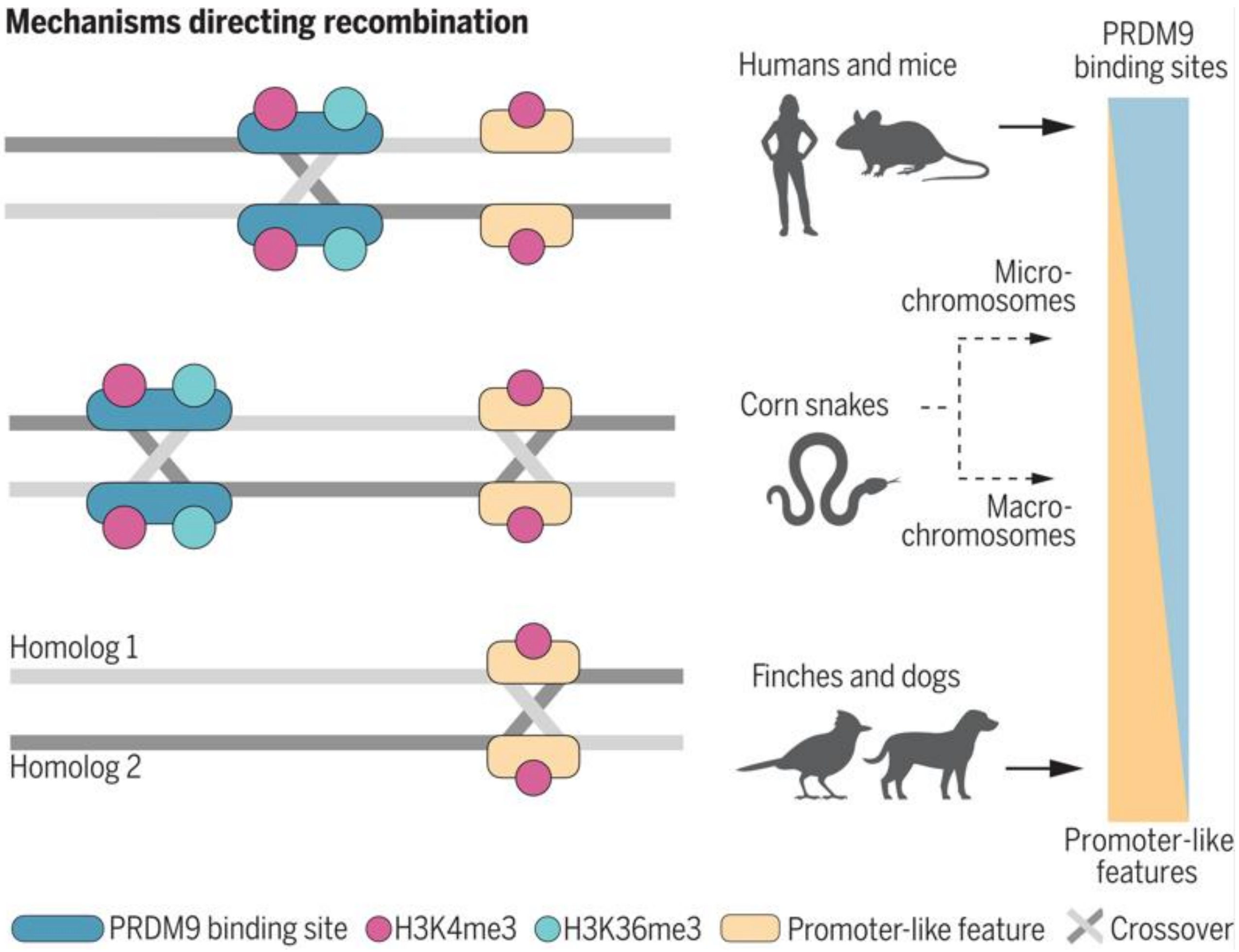
C. elegans:

- **Chromosome arms only** — recombination prohibited in centers to protect essential genes

Drosophila:

- **Males have zero recombination** — complete absence in one sex, females show regional preferences but not discrete hotspots like mammals
- **General pattern:** Most species evolved **stable, function-coupled** recombination that doesn't self-destruct — mammals with PRDM9 are the evolutionary outliers trapped in Red Queen dynamics

The evolutionary trade-off

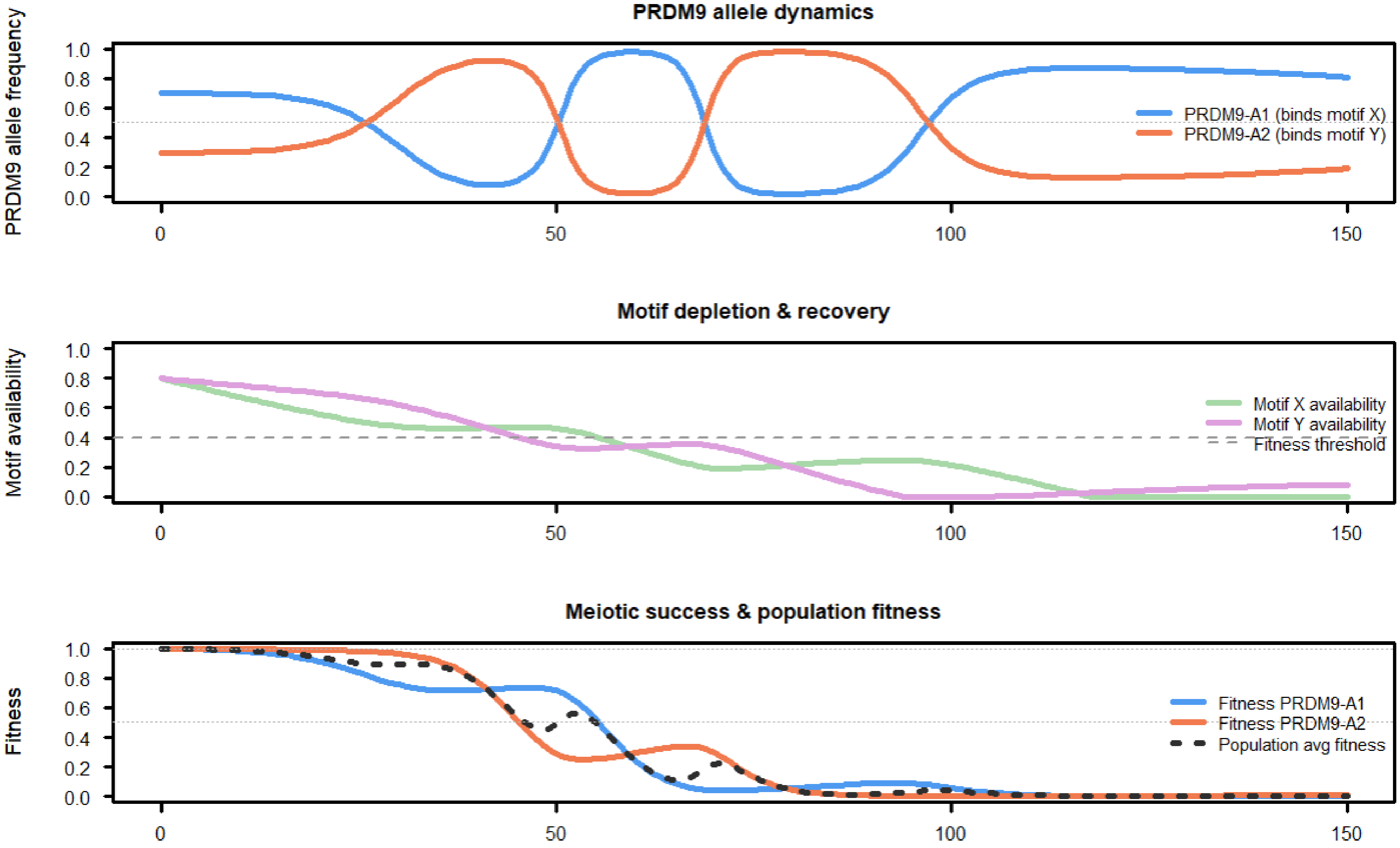


- **PRDM9 system (mammals)**:
- ✓ Directs recombination **away from genes** (safer)
- ✗ Self-destructive, requires constant Red Queen cycling
- **Promoter-based system (birds/plants)**:
- ✓ Stable over evolutionary time, no Red Queen needed
- ✗ Recombination **near genes** (potentially more disruptive)
- **Why the difference?**
- Most vertebrates **lost PRDM9** and evolved stable promoter-based systems. PRDM9 is widely conserved across vertebrates yet has been lost numerous times Yale — suggesting the Red Queen system, while functional, is **evolutionarily costly** and most lineages eventually escape it.
- Mammals seem to be "stuck" in the Red Queen trap, possibly because once you depend on PRDM9, losing it causes **meiotic failure** until alternative mechanisms evolve.

Hoge et al.,2024, *Science*

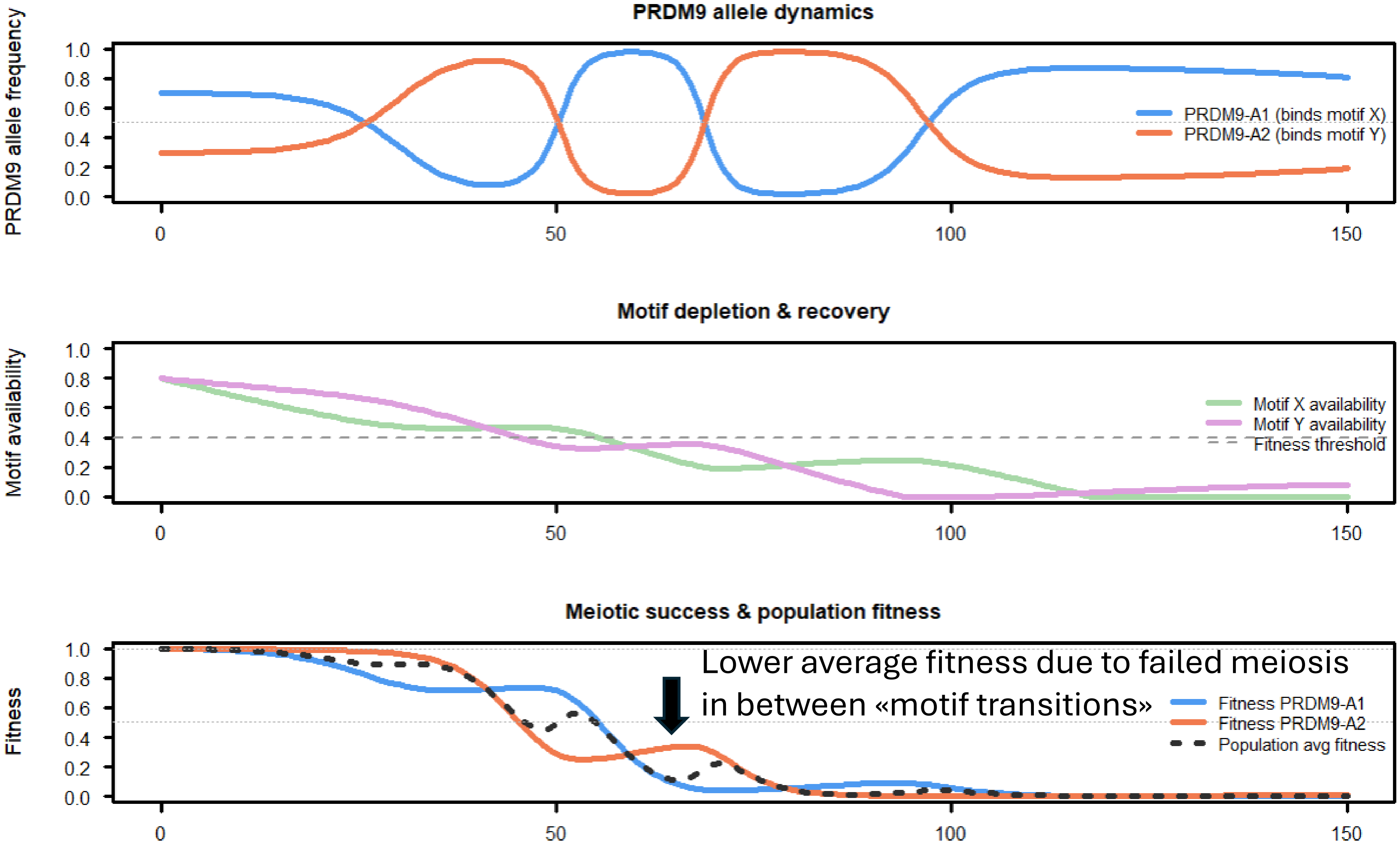
- **Canids are the remarkable exception among mammals** — they lost PRDM9 and evolved the stable promoter-based system like birds and plants.
- **Convergent evolution**: They independently evolved the **same stable system** as birds — hotspots at promoters and CpG islands rather than PRDM9-directed hotspots
- **Functional success**: This proves the stable system **works fine in mammals** — there's no fundamental reason mammals need to be trapped in Red Queen cycling

PRDM9 Red Queen: Hotspot Self-Destruction Model

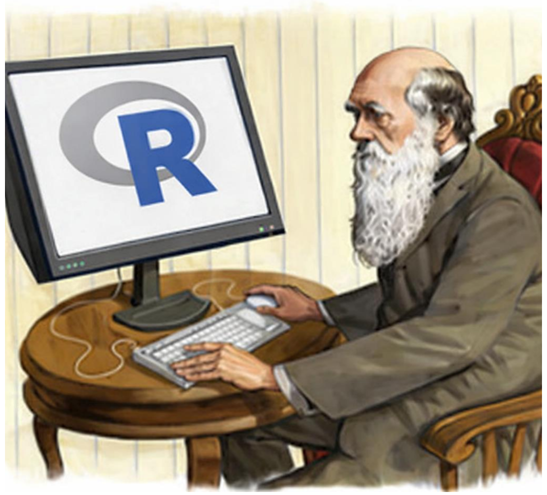


Check it out on Moodle. Motifs recover through new mutations but slowly. The model is simplistic as there are only two possible motifs.

PRDM9 Red Queen: Hotspot Self-Destruction Model



Lower average fitness due to failed meiosis in between «motif transitions»



What experiments or empirical evidence would you look for to show «Red Queen dynamics»?

If host and pathogen chase each other:

- The host likely evolved resistance to past variants of the pathogen (so past pathogens should be less infective)..

- ..but also future pathogens will probably be evolving to future versions of the host, so maybe they are less infective too

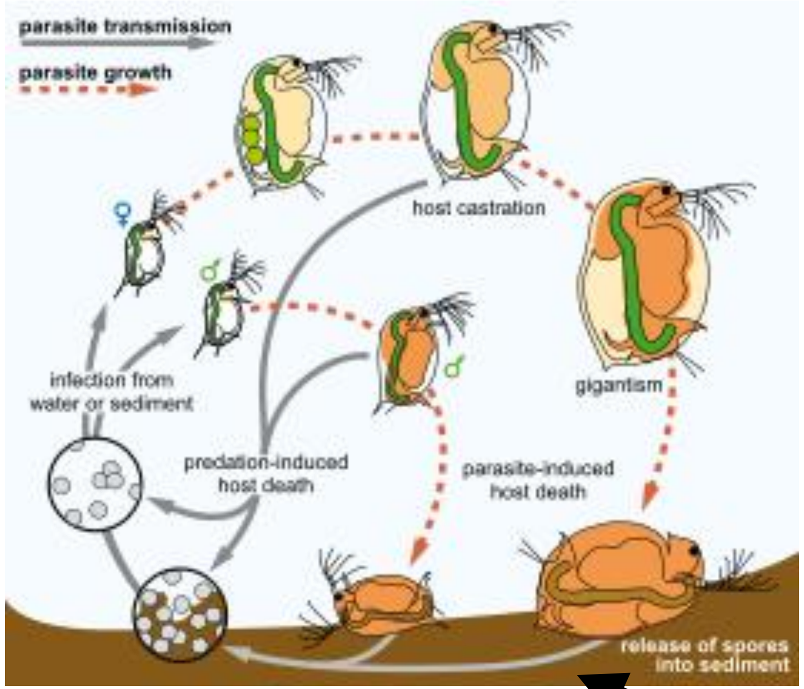
But how do I test if a host is more susceptible to contemporary pathogens than to past and future ones?

Heverlee pond, Belgium

Host–parasite 'Red Queen' dynamics archived in pond sediment

Ellen Decaestecker[1,3], Sabrina Gaba[4,5], Joost A. M. Raeymaekers[1,2], Robby Stoks[1], Liesbeth Van Kerckhoven[1], Dieter Ebert[4]* & Luc De Meester[1]*

Bacterial endoparasite
Pasteuria ramosa



Bacteria and dormant Daphnias from sediments up to 39 year old

Daphnia magna

Decaestecker et al.,2007, *Nature*

Host–parasite ‘Red Queen’ dynamics archived in pond sediment

Ellen Decaestecker[1,3], Sabrina Gaba[4,5], Joost A. M. Raeymaekers[1,2], Robby Stoks[1], Liesbeth Van Kerckhoven[1], Dieter Ebert[4]* & Luc De Meester[1]*

Bacterial endoparasite
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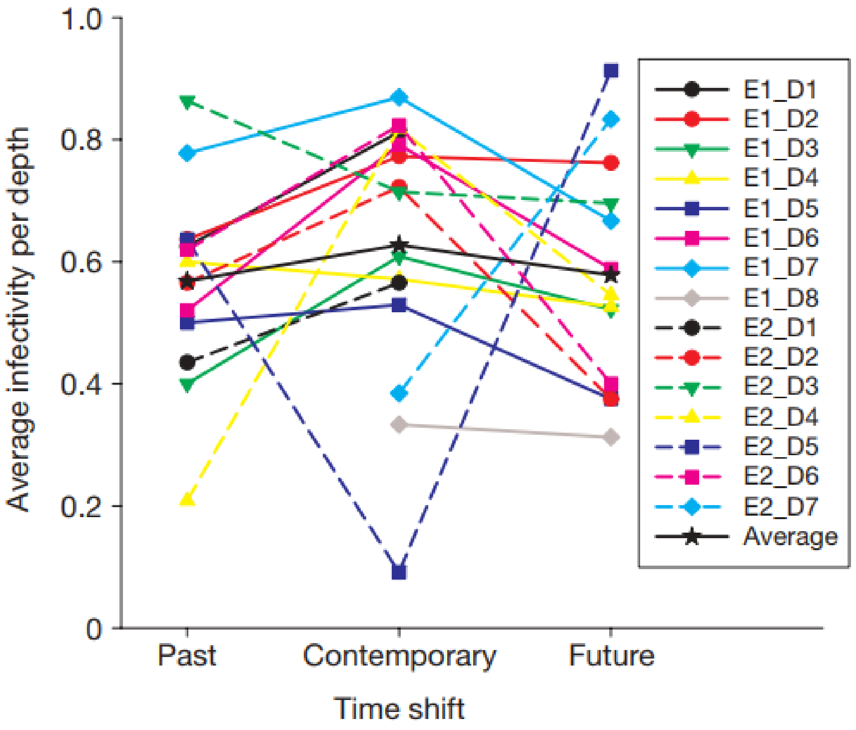

Figure 1 | Experimental results on temporal parasite adaptation. Average proportion of infected hosts when confronted with ‘past’, ‘contemporary’ and ‘future’ parasite isolates. Black stars, mean infectivity.

Daphnia magna

Decaestecker et al.,2007, *Nature*

The genomic basis of Red Queen dynamics during rapid reciprocal host–pathogen coevolution

Andrei Papkou[a,b], Thiago Guzella[c], Wentao Yang[a], Svenja Koepper[a], Barbara Pees[a], Rebecca Schalkowski[a], Mike-Christoph Barg[a], Philip C. Rosenstiel[d], Henrique Teotónio[c], and Hinrich Schulenburg[a,e,1]

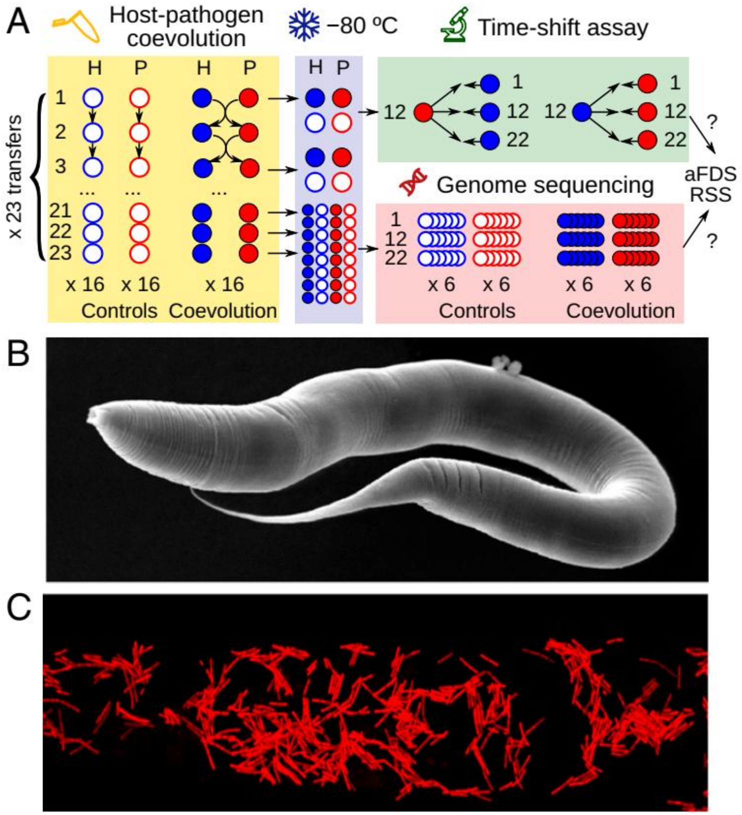

Fig. 1. A *Caenorhabditis elegans–Bacillus thuringiensis* model for experimental host–pathogen coevolution. (*A*) Overview of the general design of the evolution experiment, the subsequent phenotyping and time-shift experiment, and focus of the genomic analysis. H, host; P, pathogen. (*B*) Scanning electron micrograph of a *C. elegans* hermaphrodite. (*C*) Illustration of *B. thuringiensis*, labeled by red fluorescence, inside an infected and killed *C. elegans*.

Papkou et al.,2019, *PNAS*

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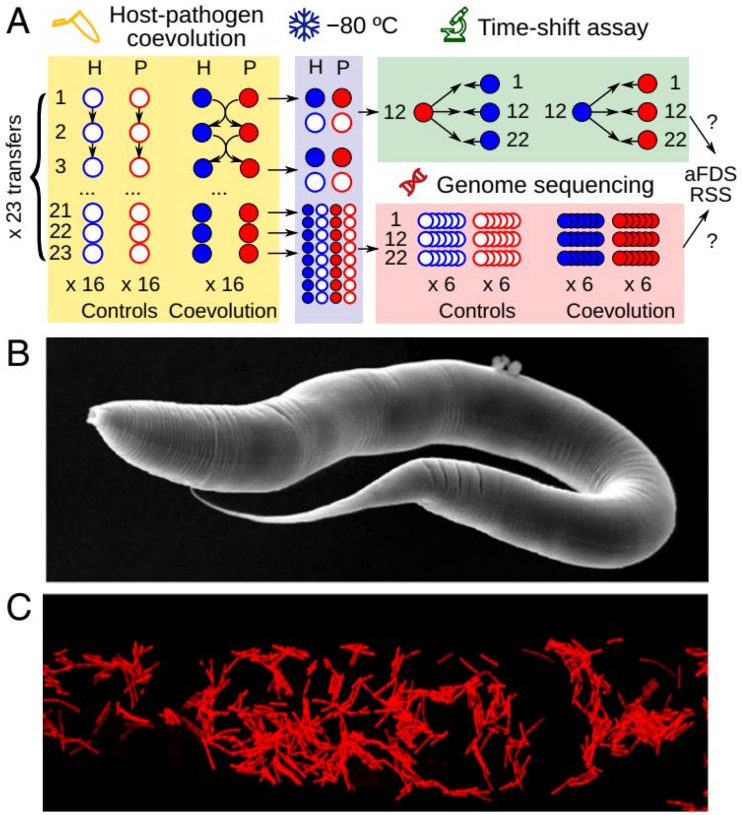

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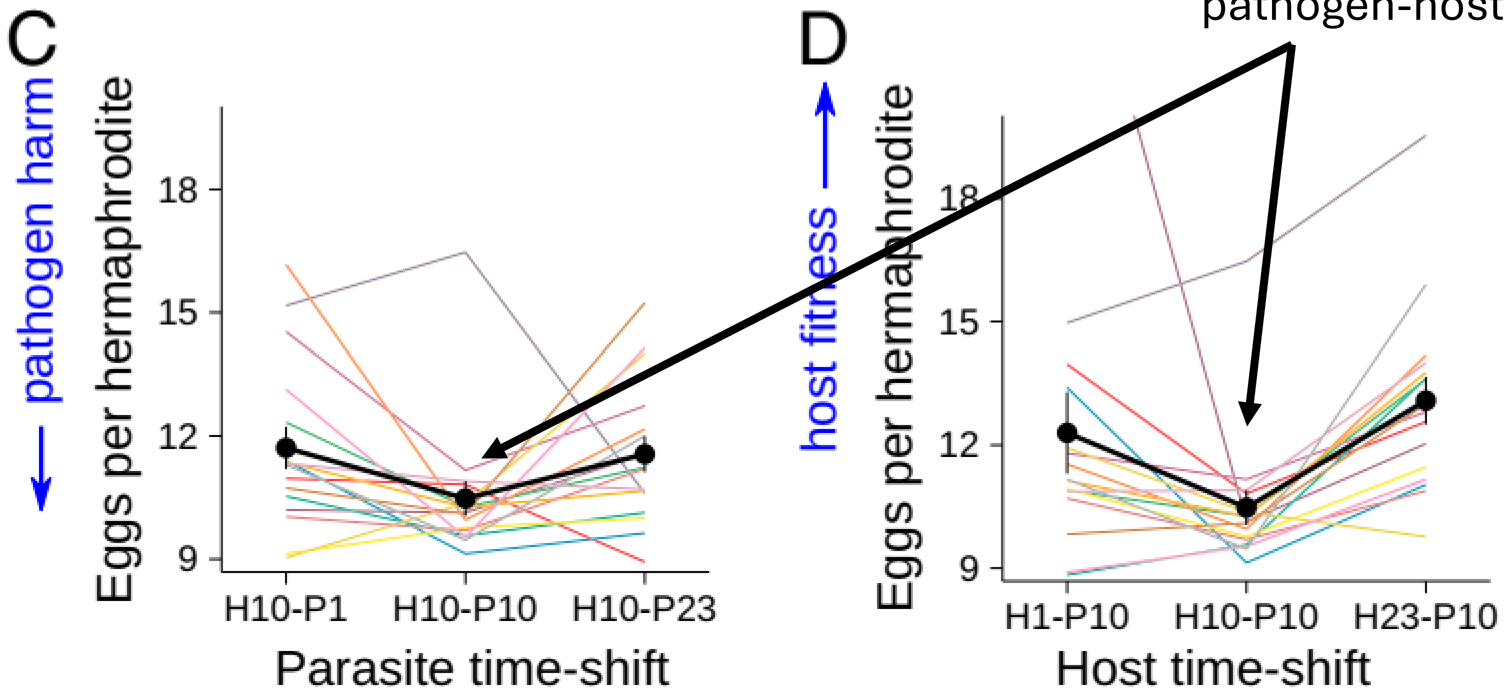
Highest infectivity for
«contemporary
pathogen-host» pairings

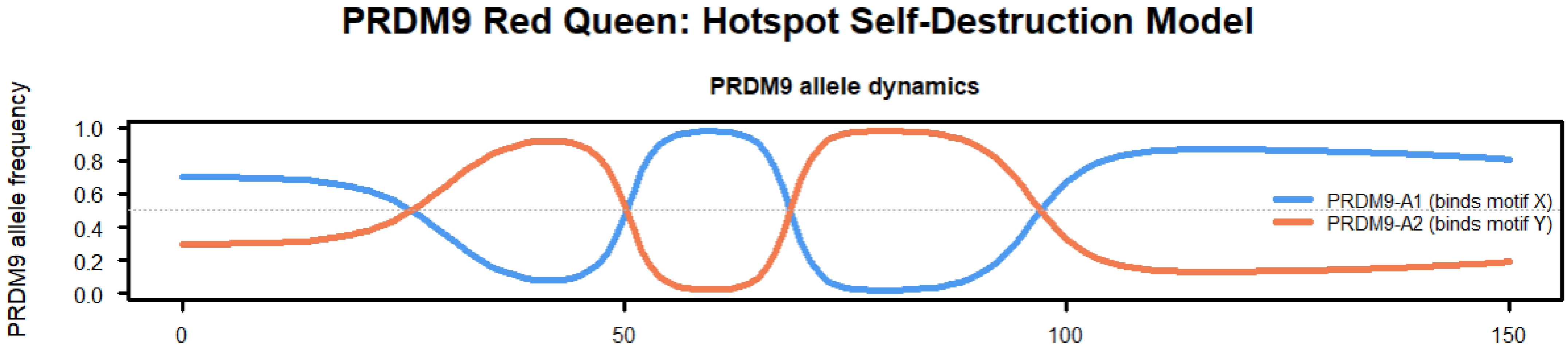


Papkou et al.,2019, *PNAS*

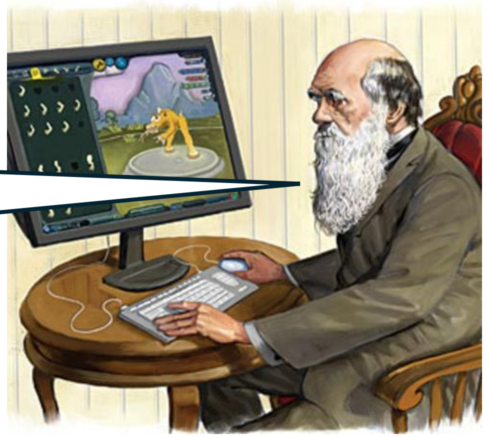
Other ideas? How could you show «Red Queen dynamics»?

And more generally, how could we use what we learned to test an evolutionary hypothesis?

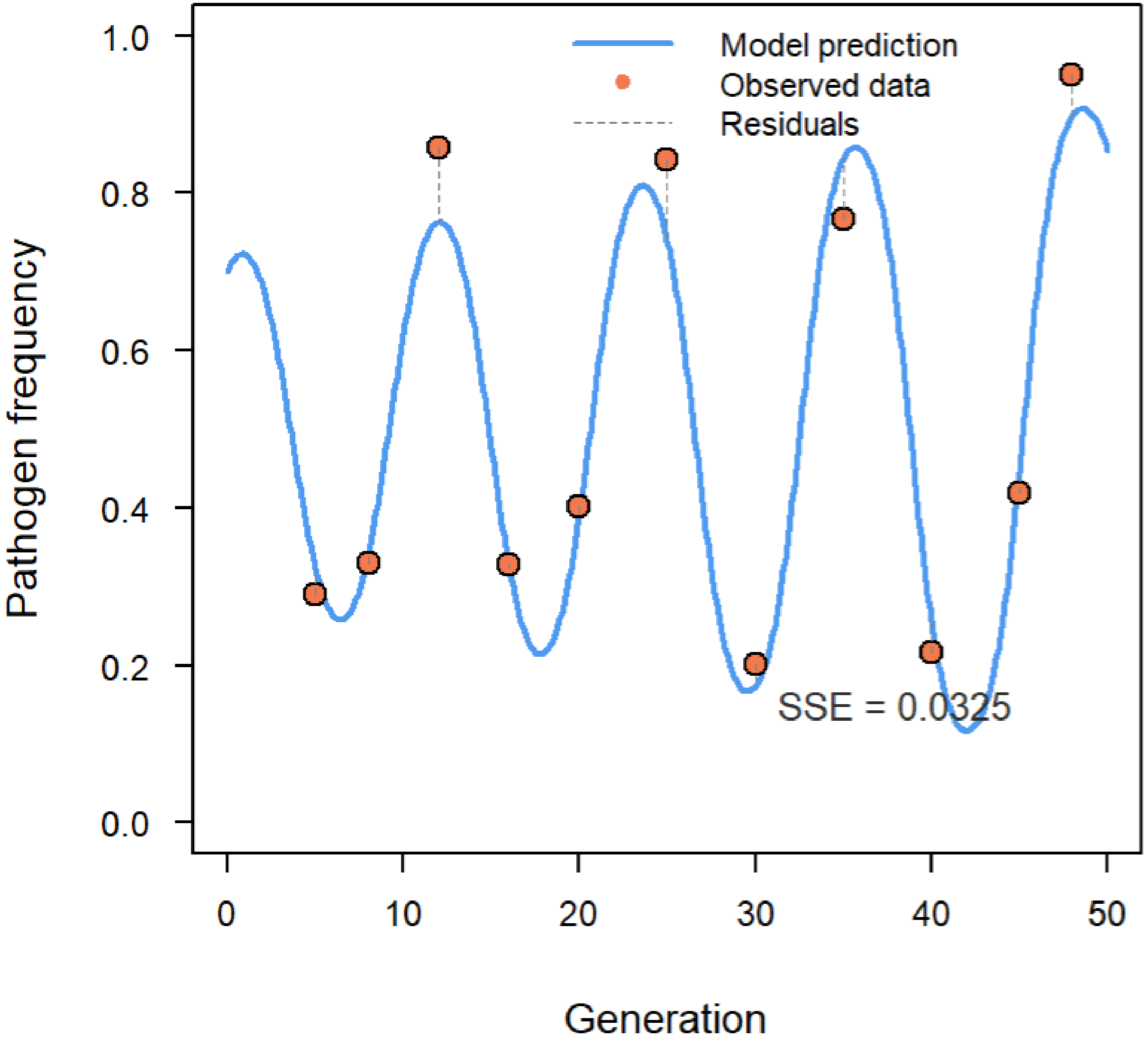
We can predict dynamics, let’s use them!



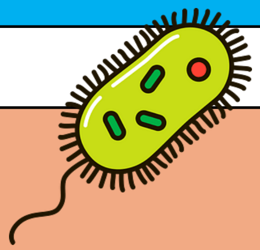
Let’s draw some trajectories and see if they «fit» some observed data over time. How would you do it?



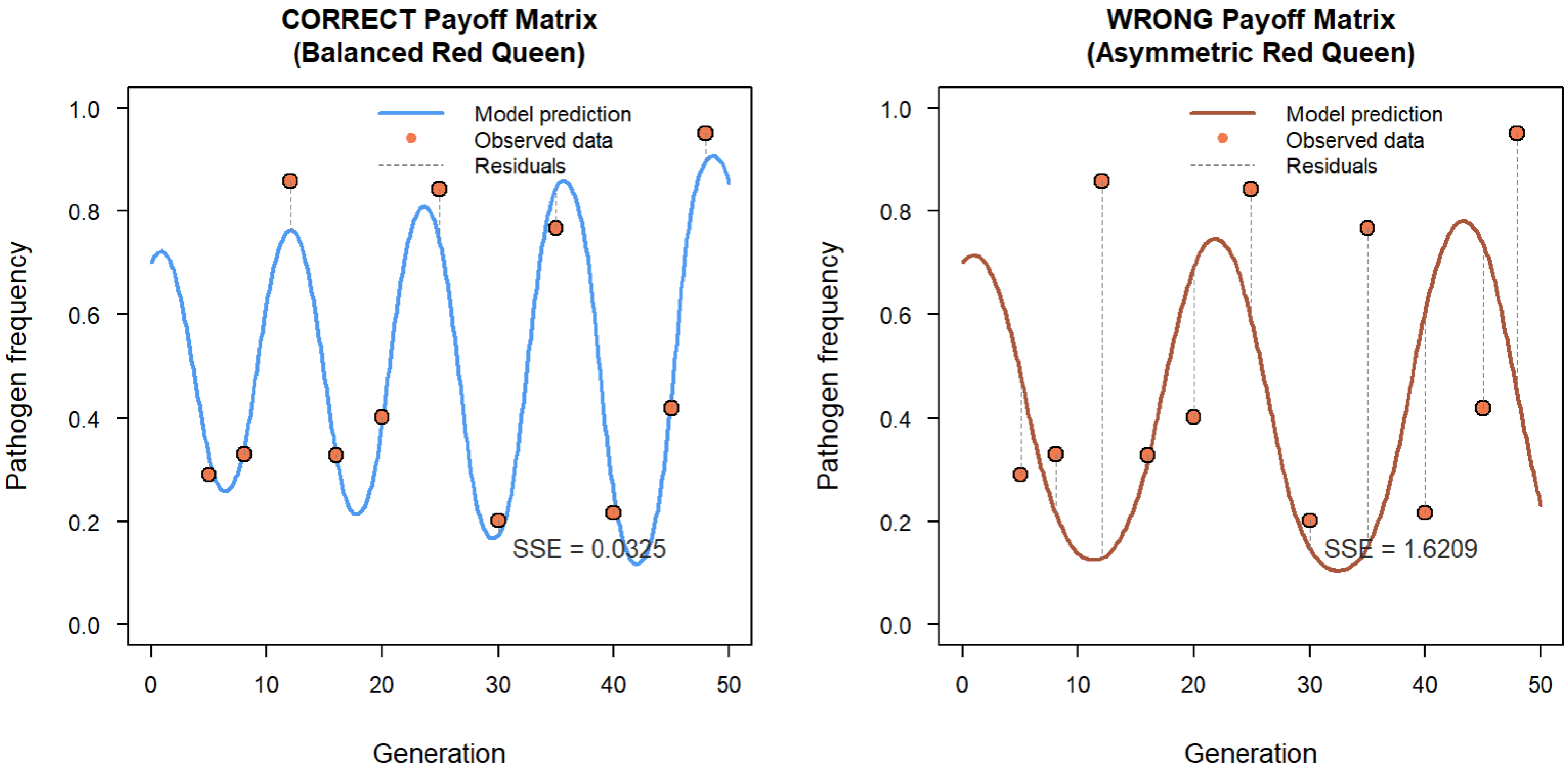
CORRECT Payoff Matrix
(Balanced Red Queen)



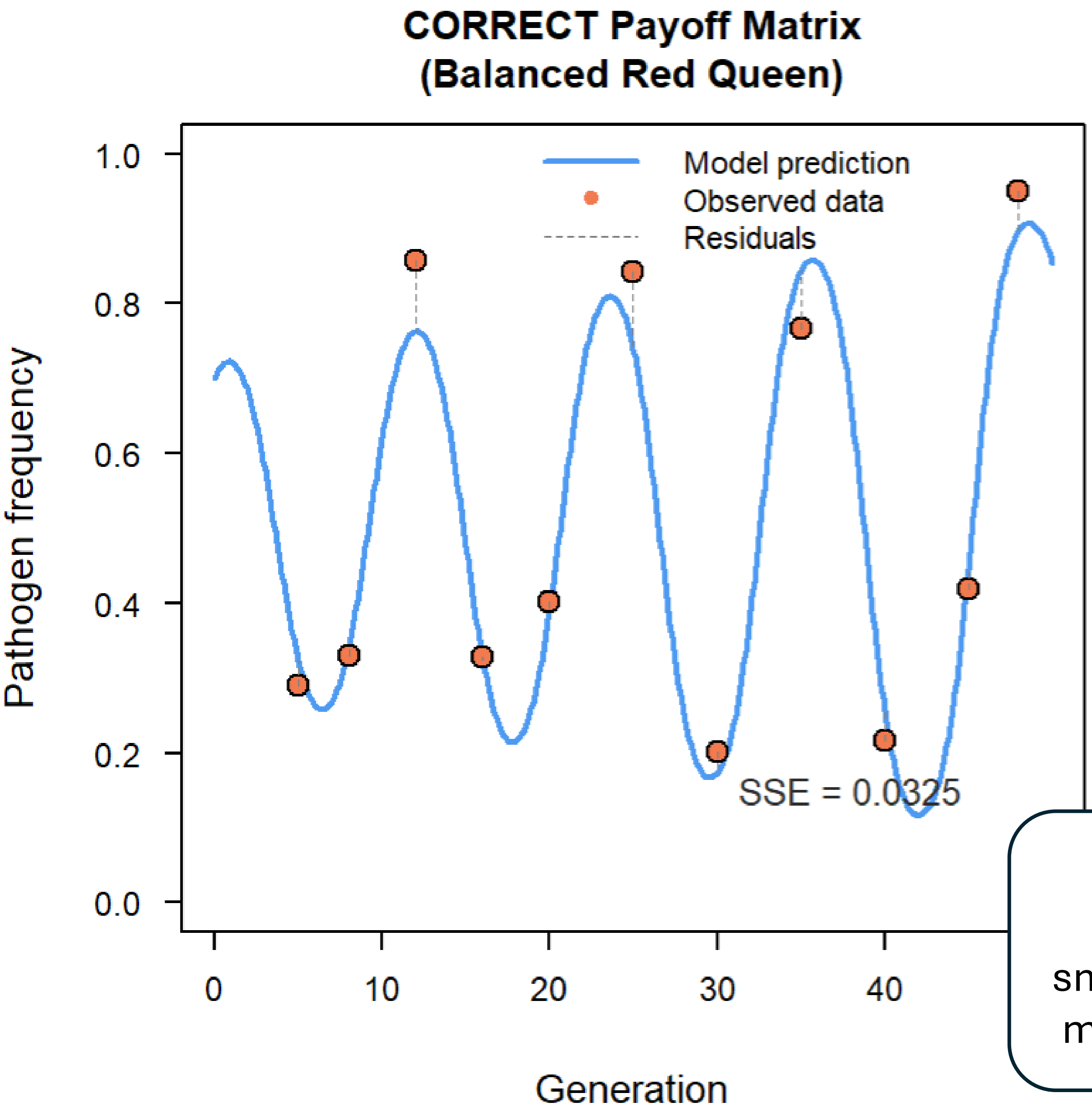
	**Parasite Strain A**	**Parasite Strain B**
Host Resistant to A	1	0
Host Resistant to B	0	1

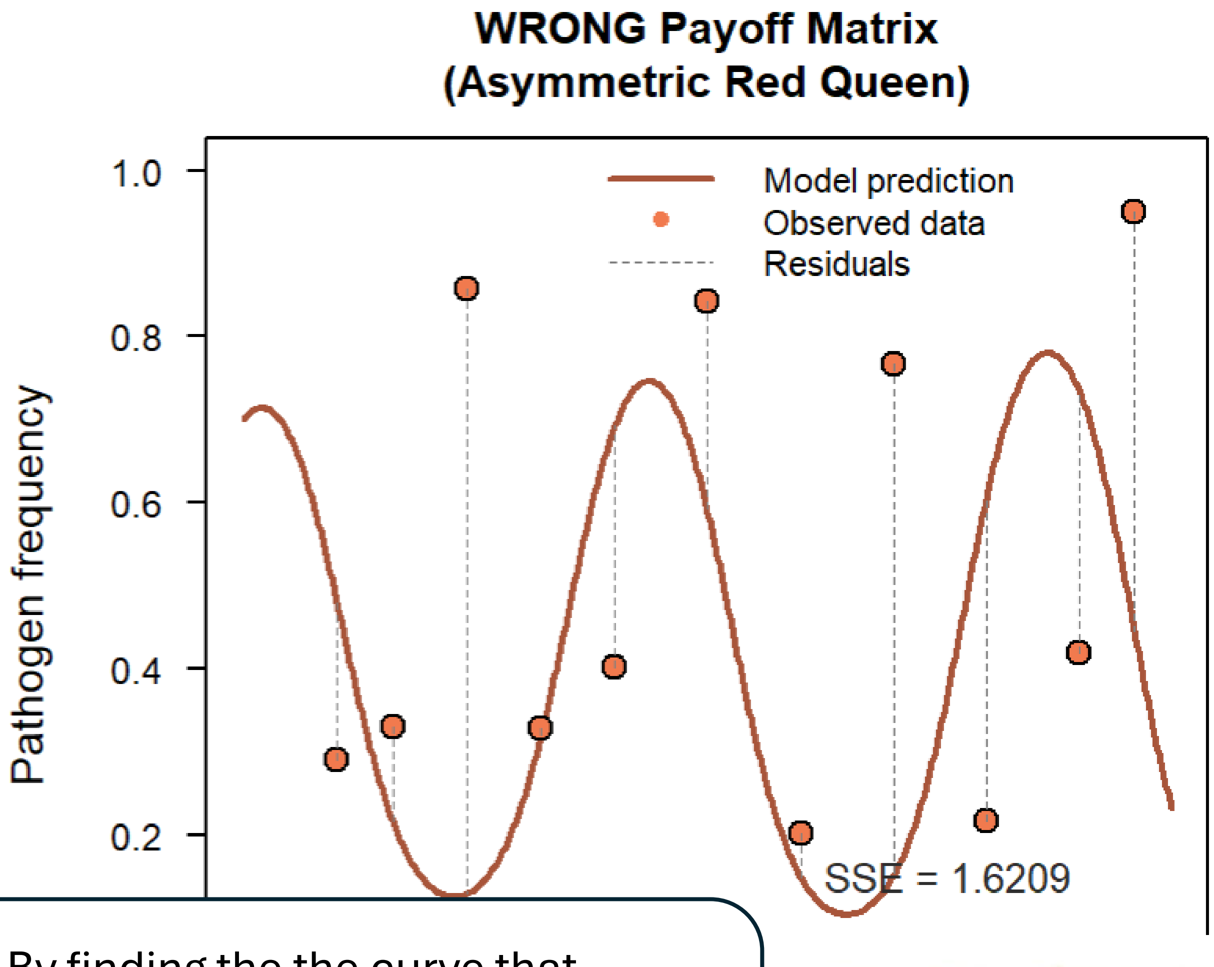
	**Host Resistant to A**	**Host Resistant to B**
Parasite Strain A	0	1
Parasite Strain B	1	0

SSE: Sum of Squared Errors: Sum[(y_Observed-y_Predicted)^2]

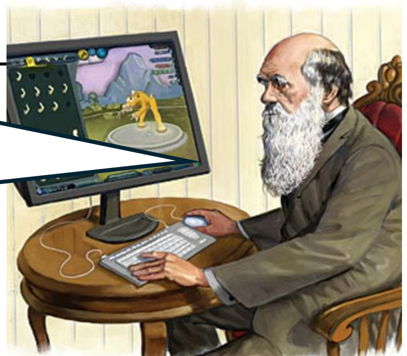


SSE: Sum of Squared Errors: $\text{Sum}[(\text{y\_Observed}-\text{y\_Predicted})^2]$





By finding the the curve that fits/represents better our data (e.g. smallest SSE) we can estimate the payoff matrix and test evolutionary hypothesis!



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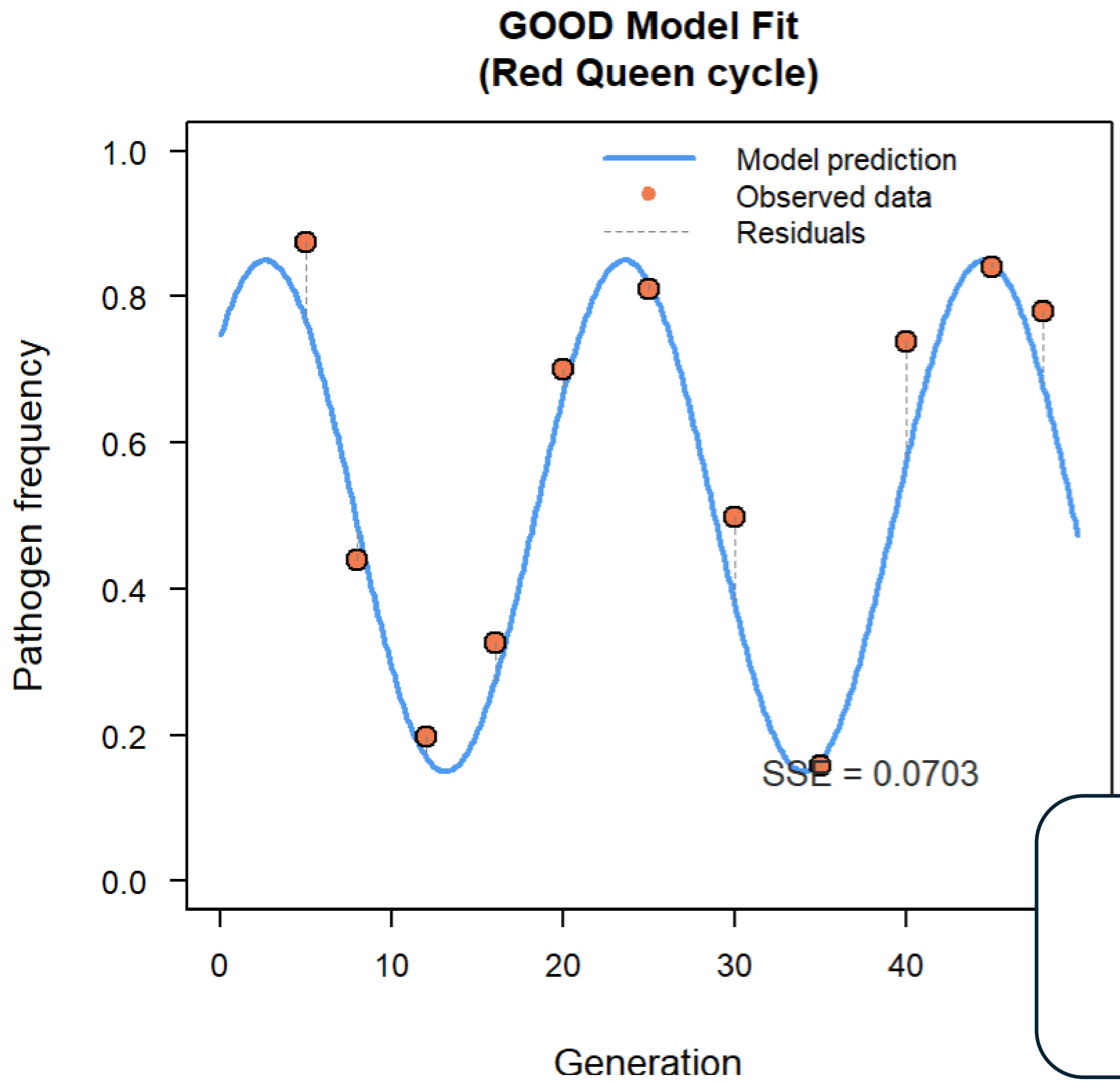
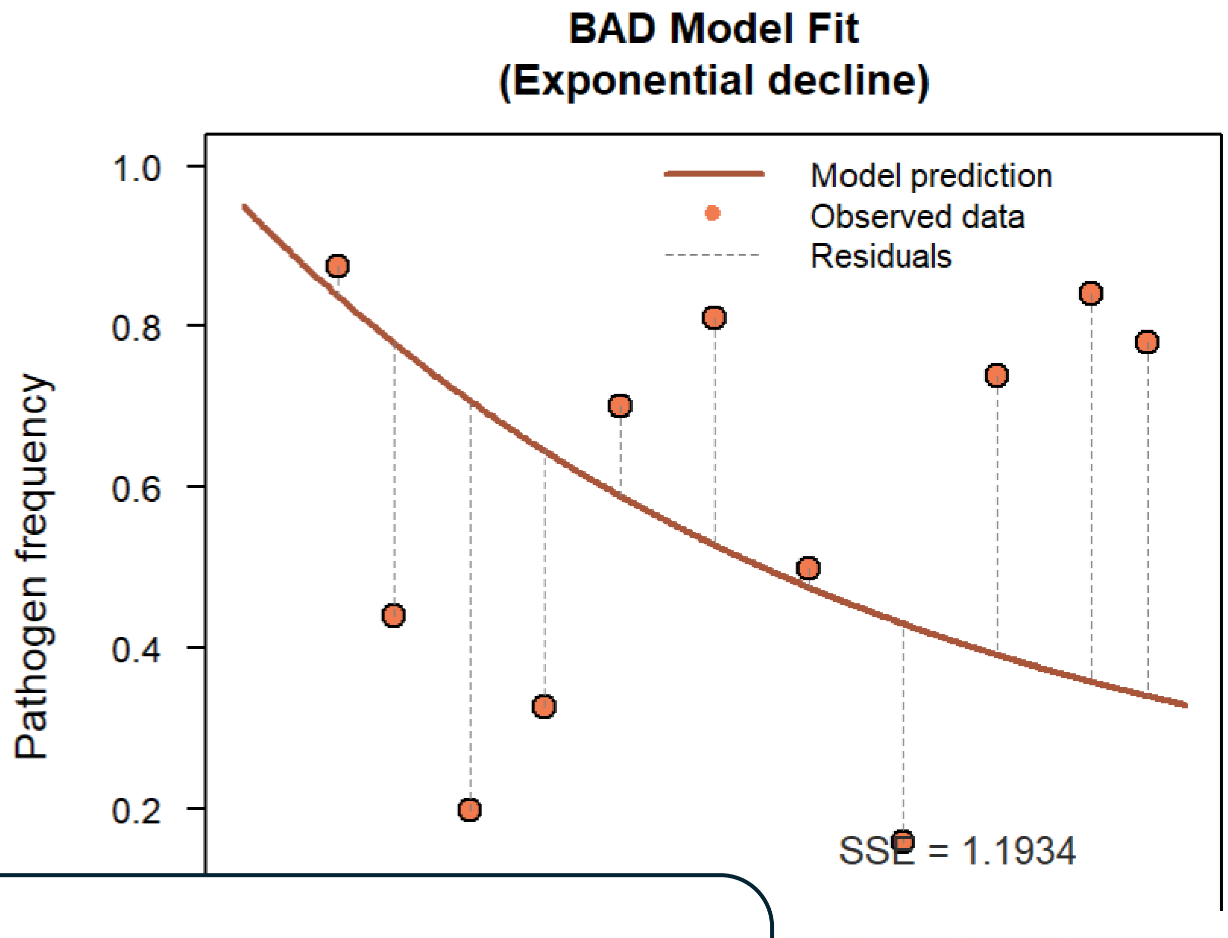
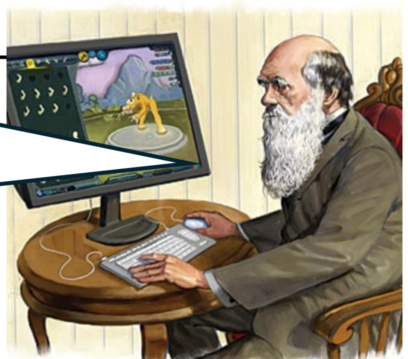




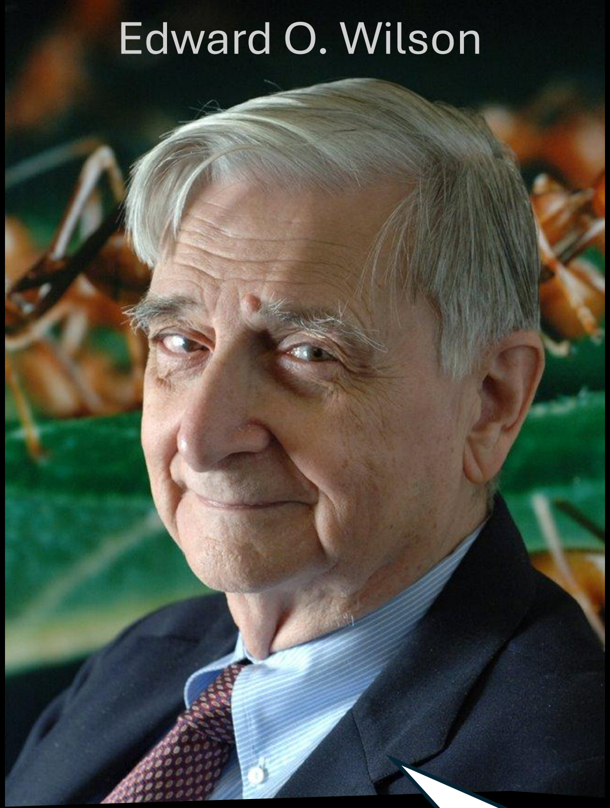
We can even compare different evolutionary models.

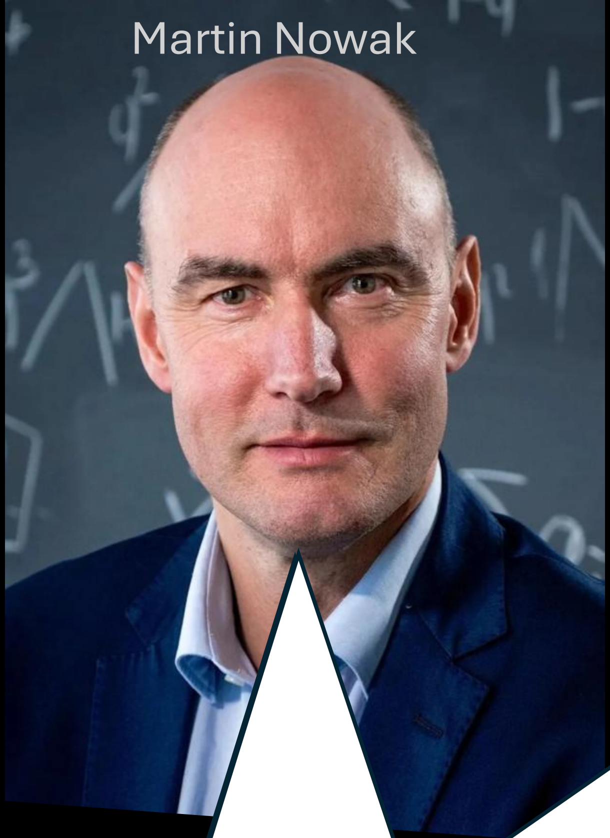


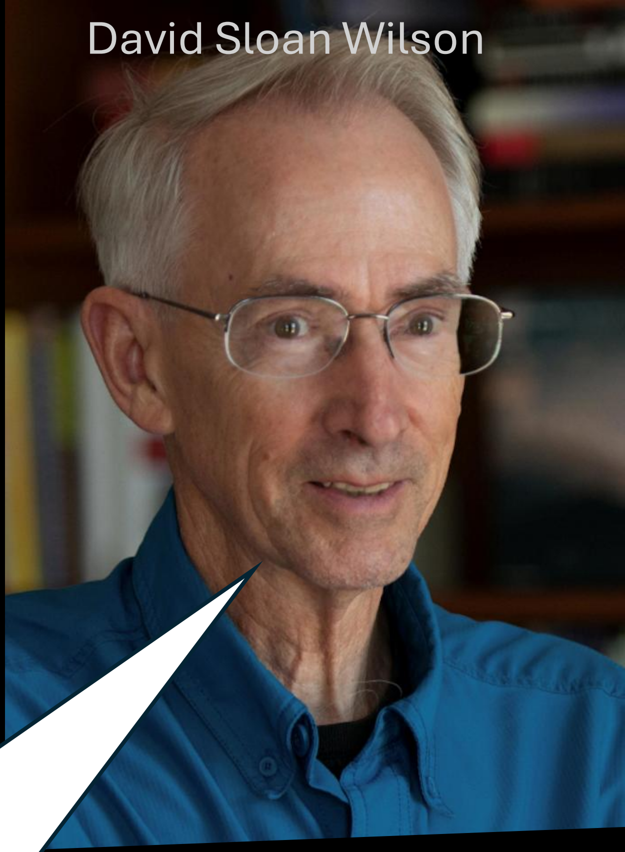
Evolutionary Game Theoreticians complaining about kin selection and inclusive fitness

*And if you care, inclusive fitness is also «dynamically insufficient». We’ll see a little bit what it means next time (which is today!)

Evolutionary Game Theoreticians complaining about kin selection and inclusive fitness


Edward O. Wilson


Martin Nowak


David Sloan Wilson

With «evolutionary game theory/evolutionary dynamics» we can instead predicts how populations change over time!

Actually I care more that it is not mathematically rigorous and it is «dynamically insufficient»*

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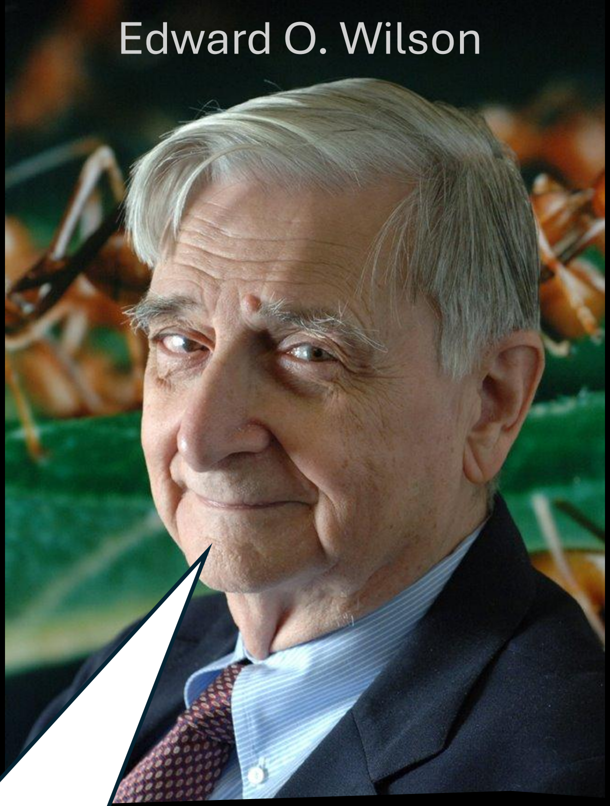
*And if you care, inclusive fitness is also «dynamically insufficient». We'll see a little bit what it means next time (which is today!)

Evolutionary Game Theoreticians complaining about kin selection and inclusive fitness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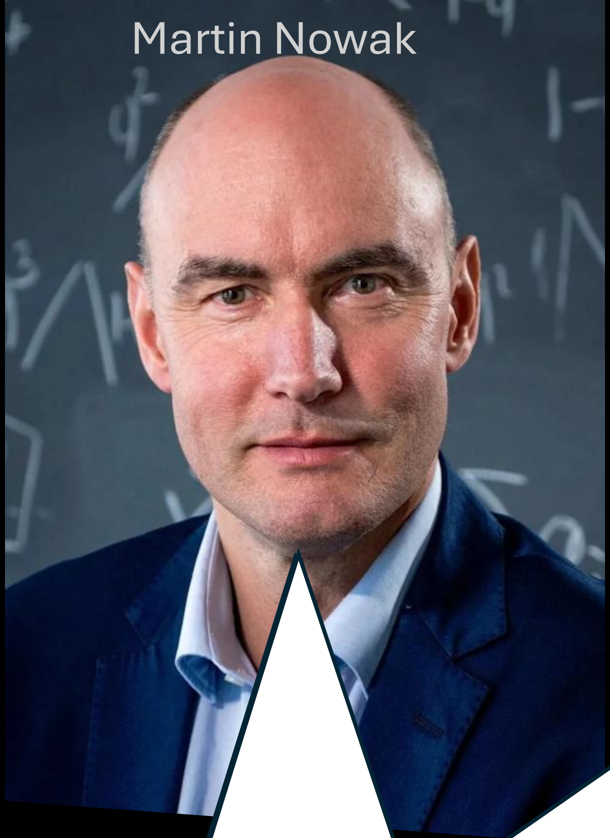
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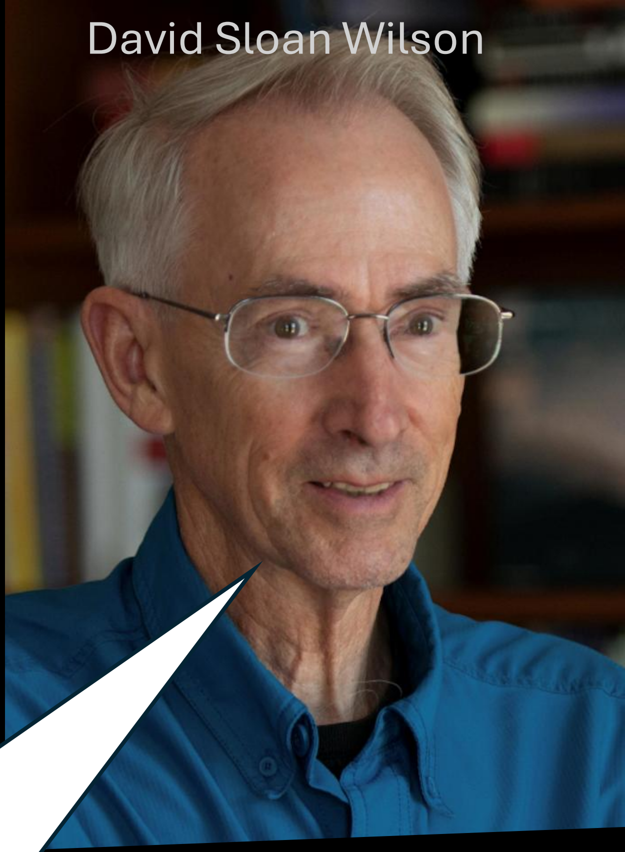

David Sloan Wilson

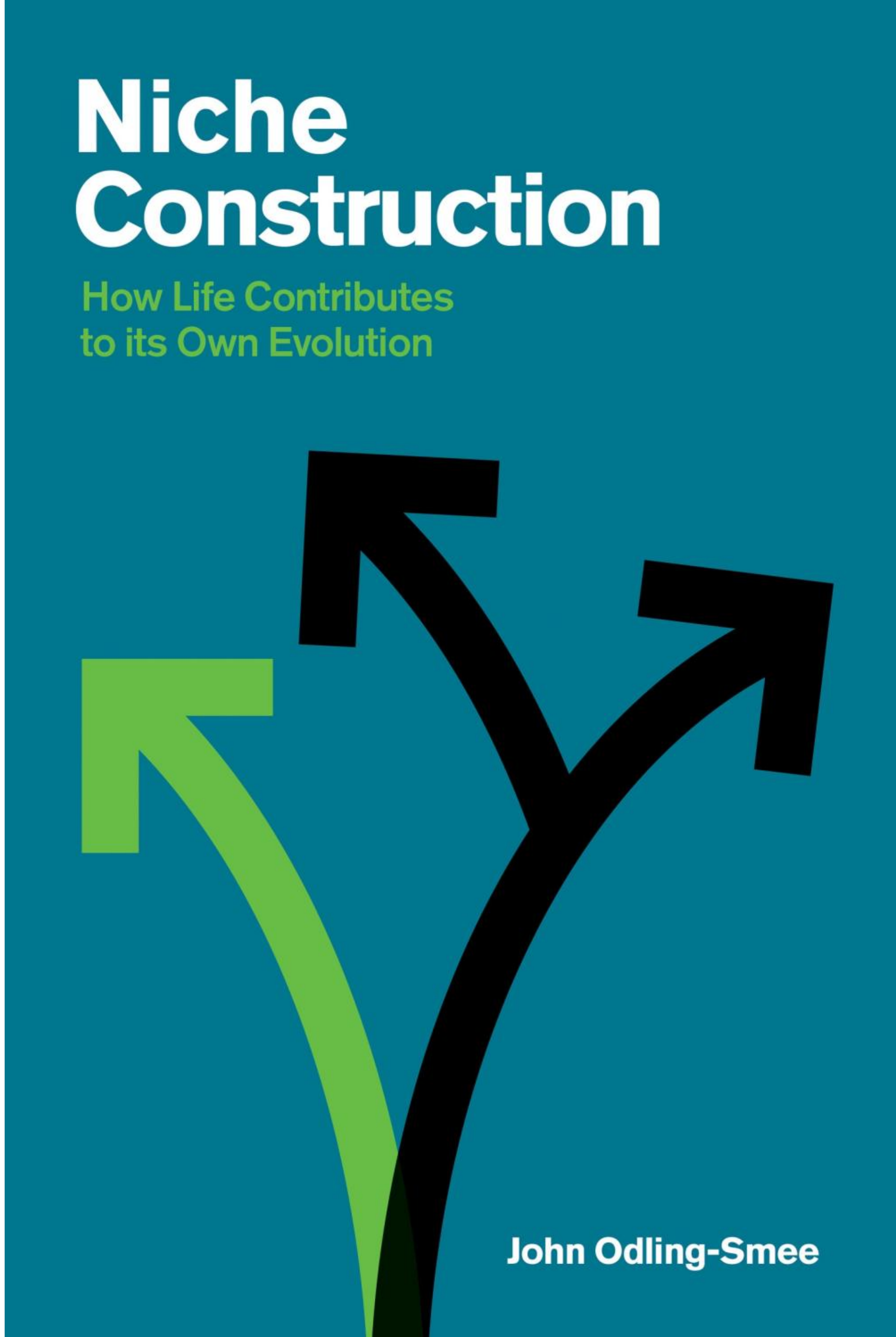
Not just a mathematical curiosity. We can use this to look at data, infer the past and make predictions (e.g. adaptation to climate change, pandemics, etc.)

Actually I care more that it is not mathematically rigorous and it is «dynamically insufficient»*

*And if you care, inclusive fitness is also «dynamically insufficient». We'll see a little bit what it means next time (which is today!)

Niche construction

- Organisms **actively modify their environment**
- These modifications **change selection pressures**
- Evolution is not just:
  - environment → organism
    but also:
  - **organism → environment → organism**
- Feedback loop between ecology and evolution
- **Standard vs Niche Construction View**
- **Standard view**
  - Environment is **external and fixed**
  - Selection acts on organisms
- **Niche construction**
  - Environment is **partly constructed by organisms**
  - Organisms **co-direct their own evolution**



Niche
Construction

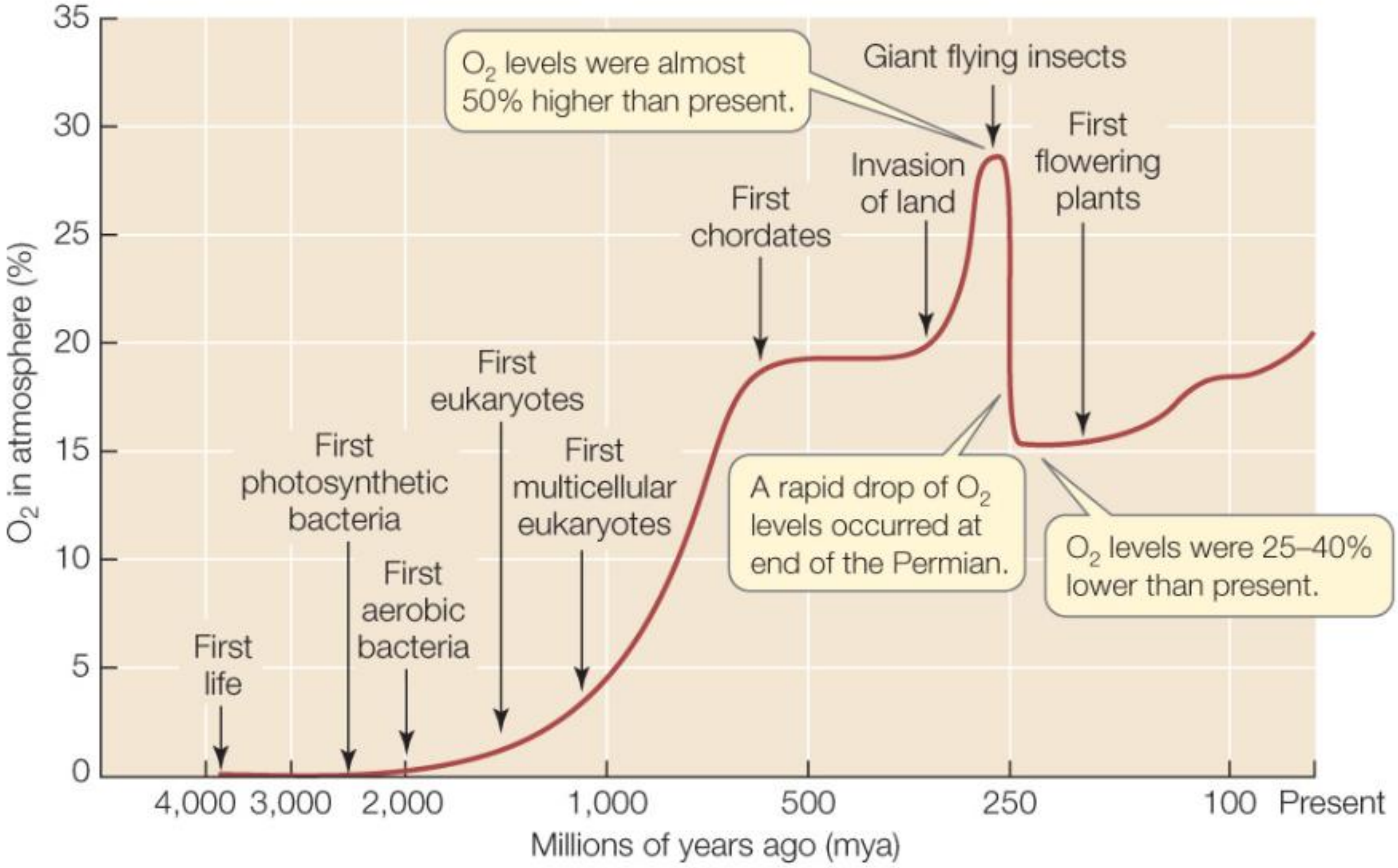
How Life Contributes
to its Own Evolution

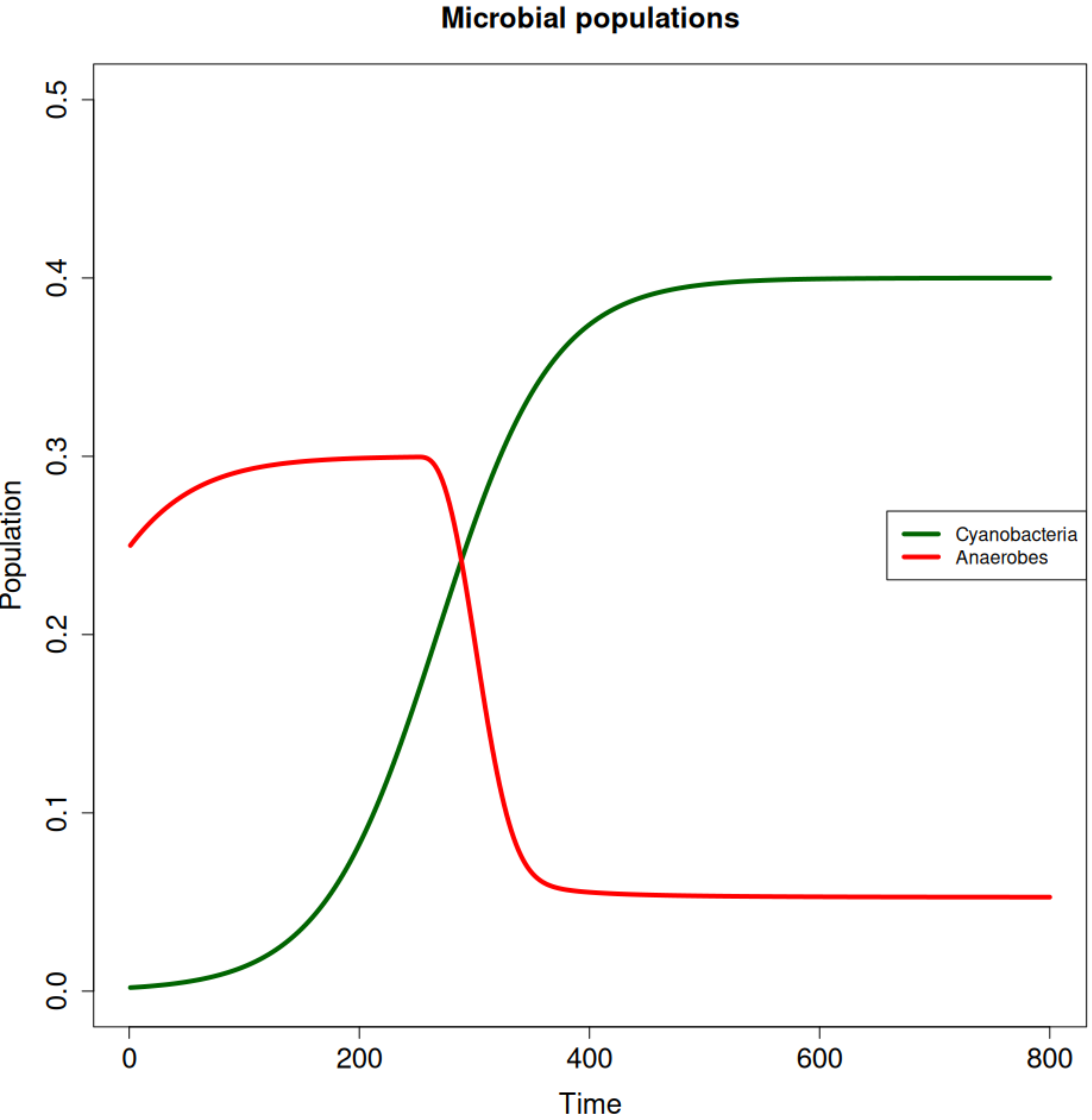
John Odling-Smee

THE GREAT OXYGENATION EVENT

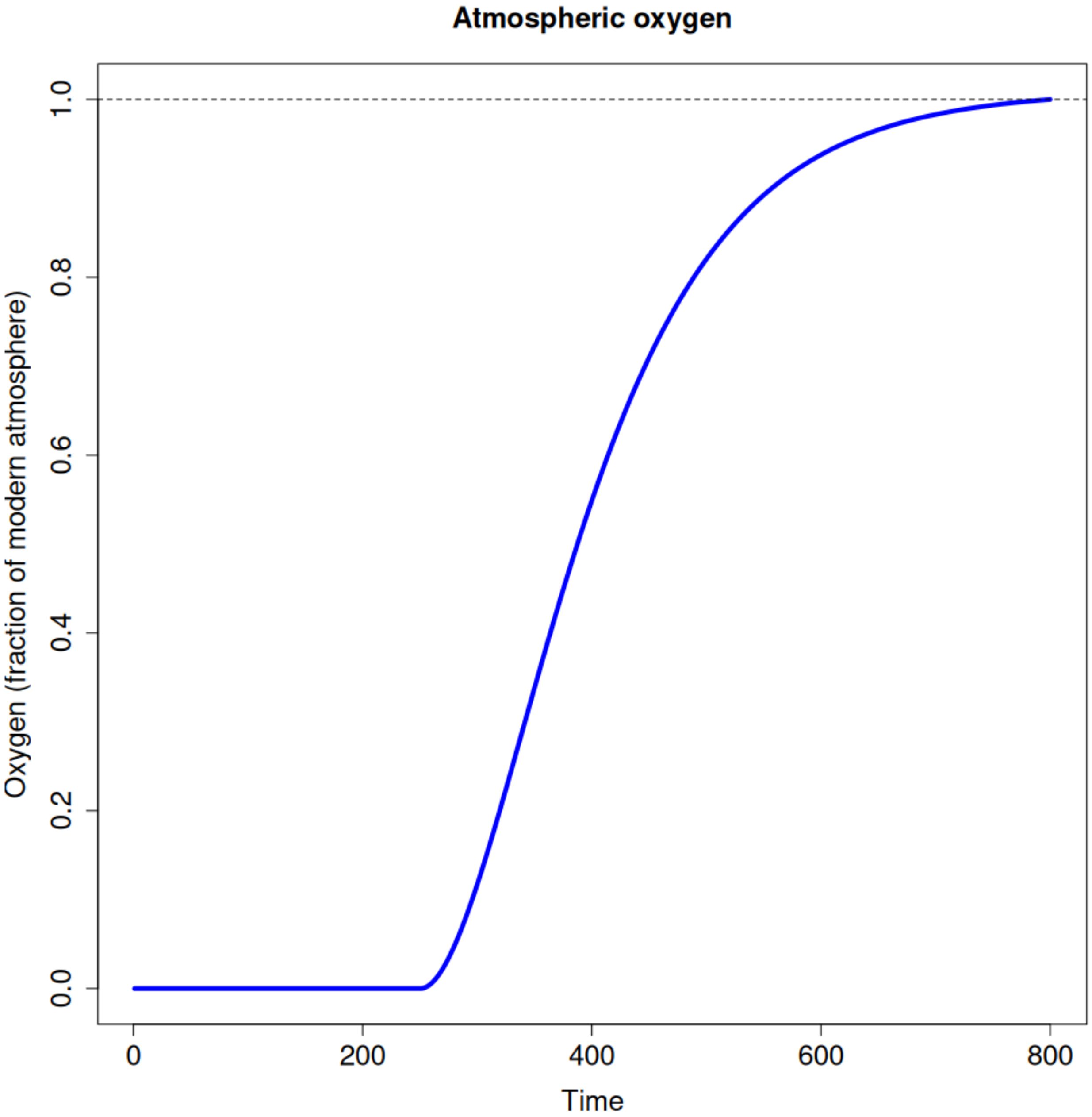
THE AIR WE BREATHE

The great oxygenation event

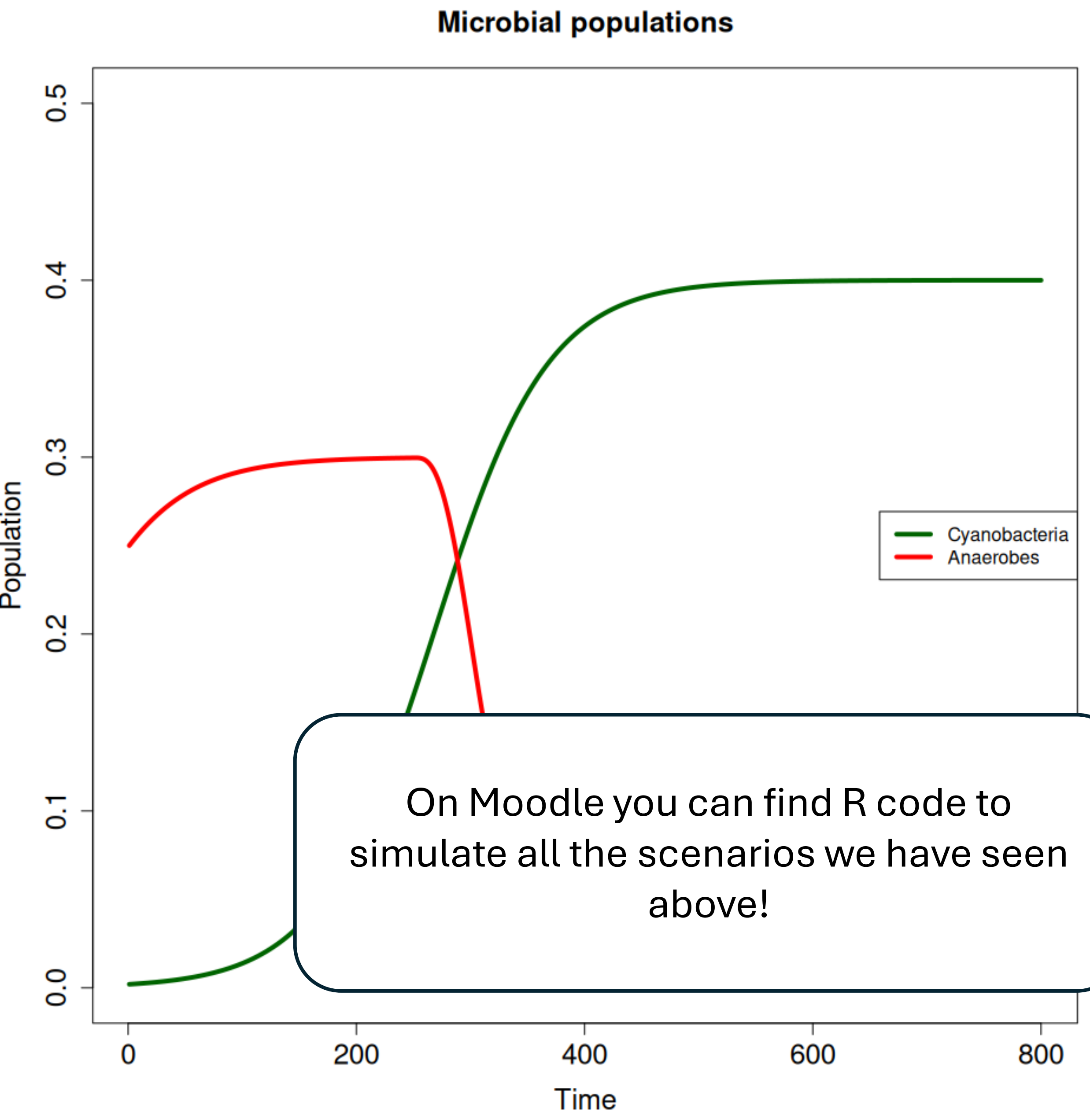


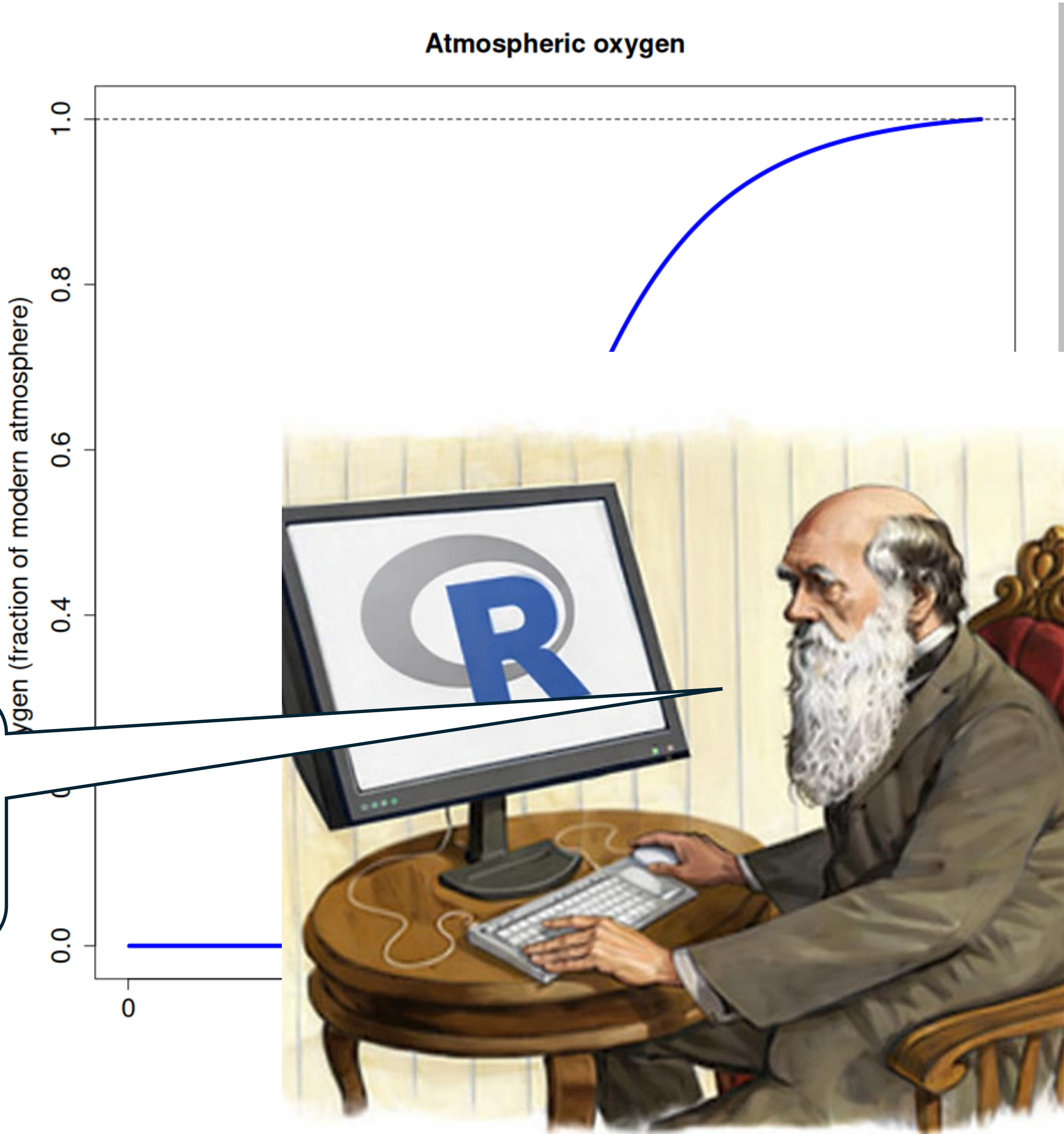


Microbial populations

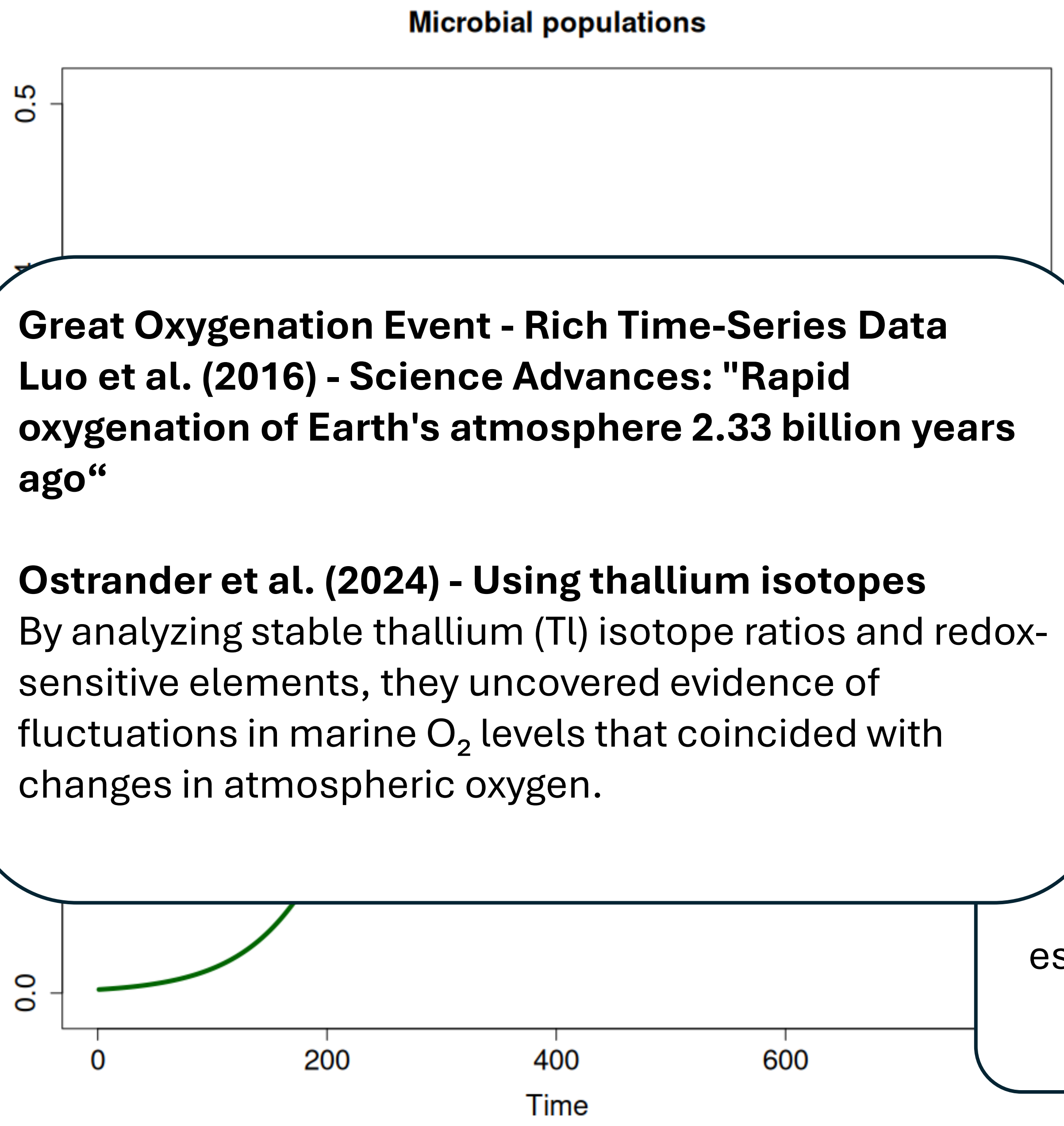


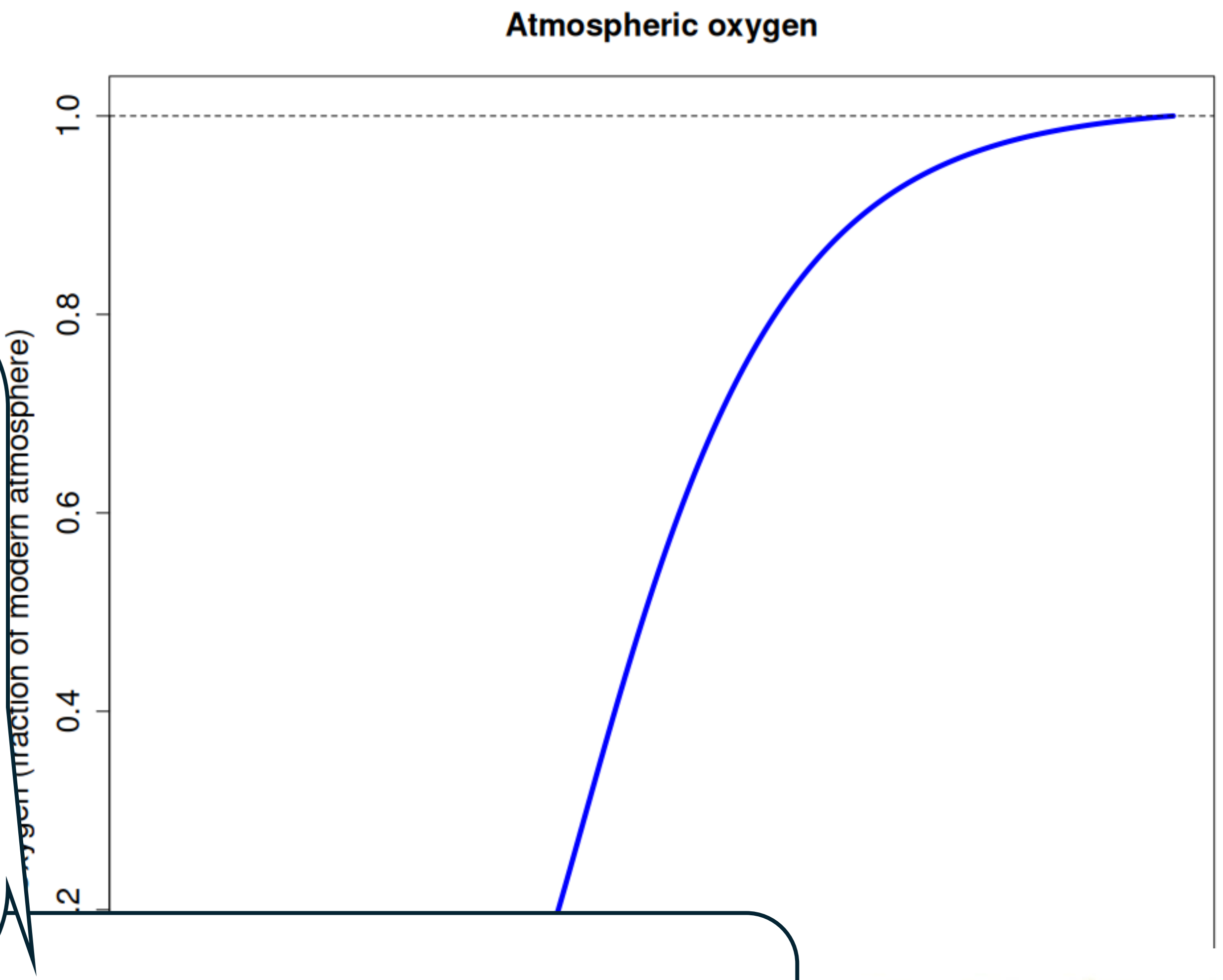
Atmospheric oxygen





On Moodle you can find R code to simulate all the scenarios we have seen above!

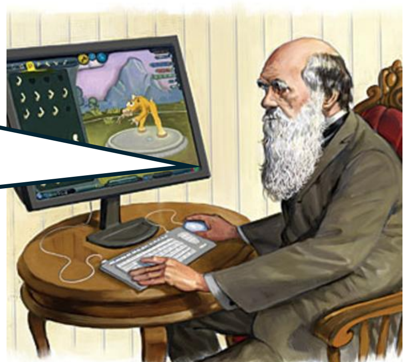




Great Oxygenation Event - Rich Time-Series Data
Luo et al. (2016) - Science Advances: "Rapid oxygenation of Earth's atmosphere 2.33 billion years ago“

Ostrander et al. (2024) - Using thallium isotopes
By analyzing stable thallium (Tl) isotope ratios and redox-sensitive elements, they uncovered evidence of fluctuations in marine O_2 levels that coincided with changes in atmospheric oxygen.

In principle, we could even think of estimating the evolutionary dynamics of these events!

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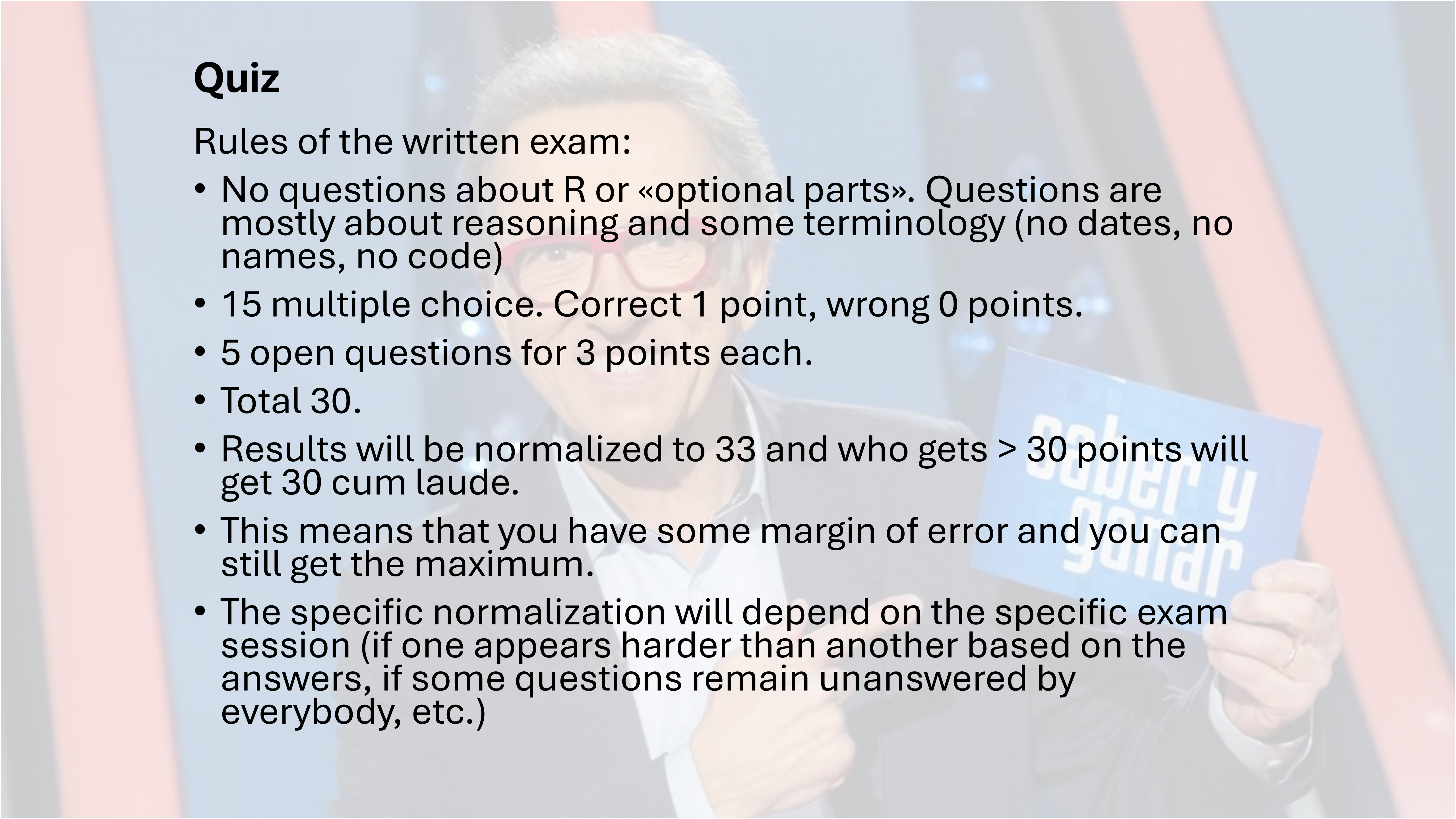
Quiz



Rules of the written exam:

- No questions about R or «optional parts». Questions are mostly about reasoning and some terminology (no dates, no names, no code)
- 15 multiple choice. Correct 1 point, wrong 0 points.
- 5 open questions for 3 points each.
- Total 30.
- Results will be normalized to 33 and who gets > 30 points will get 30 cum laude.
- This means that you have some margin of error and you can still get the maximum.
- The specific normalization will depend on the specific exam session (if one appears harder than another based on the answers, if some questions remain unanswered by everybody, etc.)

Which of these characteristics are fundamental for evolution through natural selection:

a) mutation, drift, selection
b) variability, selection, inheritance
c) selection and inheritance
d) mutation, variability and selection
e) drift, reproduction and mutation

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Darwin’s model of inheritance was:

a) Visionary as it broke up with the acquired characters view of Lamarck and was more similar to Mendelian Inheritance

b) Based on acquired characters, similarly to Lamarck

c) Based on acquired characters, but devoid of the “blending inheritance problem” that affected Lamarck’s view

d) Similar to Mendelian Inheritance, refusing acquired characters, with the exception of the “blending inheritance problem”

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Cooperation between related individuals:

- a) is evolutionarily favoured by kin selection
- b) is evolutionarily disfavoured by kin selection
- c) is evolutionarily favoured only when relatedness is higher than 0.5, also called the Hamilton's threshold
- d) evolves only in contexts different than that of Prisoner's dilemma, where defection always dominates

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In mutualistic interactions, the genomes of the species involved often:

a) gain new genes through lateral gene transfer to accomodate the symbiotic process

b) lose genes, since not necessary anymore

c) evolve their genomes to resemble that of their host to evade immune defenses

d) lose mostly non-coding regions to replicate more rapidly, while keeping all genes intact

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In the replicator's equation $dx/dt=x(1-x)[(a-b-c+d)x+b-d]$, in the context of a 2-player game with payoff matrix with entries a,b,c and d, what does the term $x(1-x)$ represents:

a) the change in strategy/allele frequencies over time in the population

b) the average fitness of the population

c) the variance in allele/strategy frequencies in the population

d) a normalization term that force frequencies to be between 0 and 1

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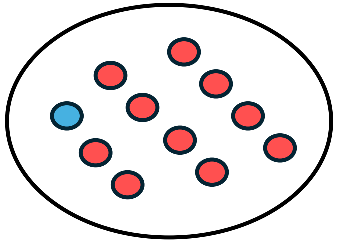
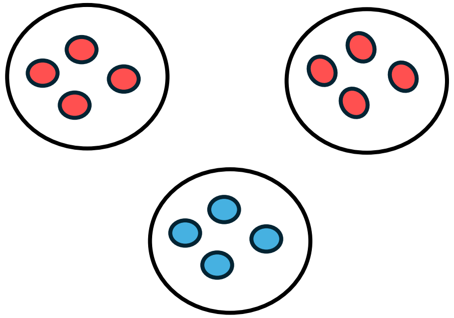
Examples of open questions:

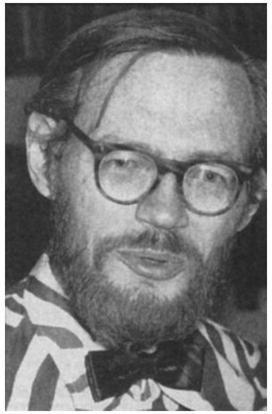
- Reciprocity, indirect reciprocity, kin selection, group selection and network selection favour the evolution of cooperation. How do they do it? Is there a common motif?

More like “one rule for cooperation”: positive assortment

evolutionary change in altruism = effect of selection within groups + effect of selection between groups







Whenever cooperators interact more often with other cooperators

Positive assortment ⬆ ⬇ ⬆

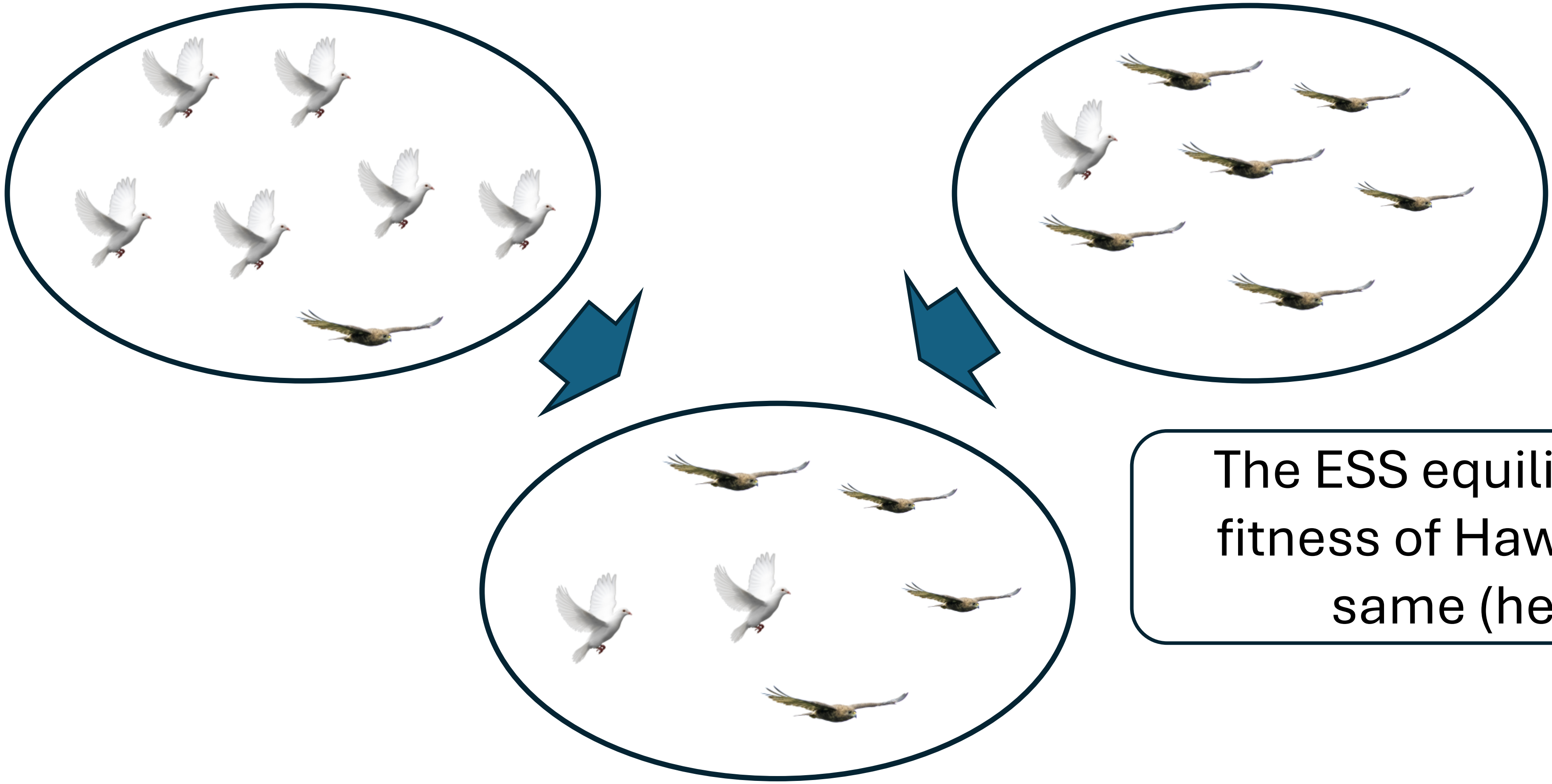
Cooperators meet less often defectors

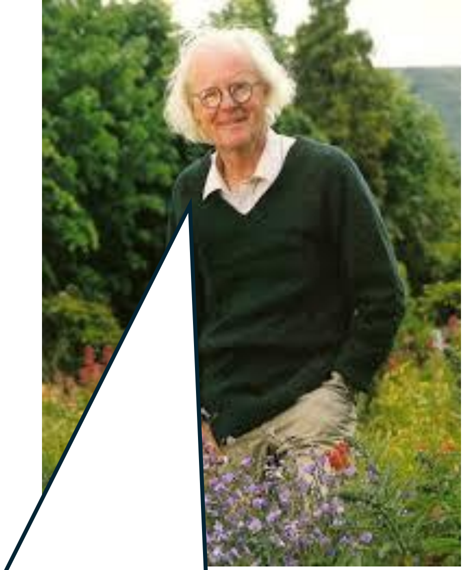
“groups” of cooperators are favoured

Examples of open questions:

- Reciprocity, indirect reciprocity, kin selection, group selection and network selection favour the evolution of cooperation. How do they do it? Is there a common motif?
- What is an Evolutionarily Stable Strategy (ESS)?
- In the Hawk vs Doves games, is there an Evolutionarily Stable Strategy? If yes, which? If no, why not?

An **Evolutionarily Stable Strategy (ESS)** is a strategy that, if adopted by almost all individuals, cannot be invaded by a rare alternative strategy.





ynard-Smith
0-2004)

The ESS equilibrium is where the fitness of Hawks and Dove is the same (here 2/3 Hawks)

	Hawk	Dove
Hawk	-1,-1	4,0
Dove	0,4	2,2