Why Impossible Things Happen so Often?

The emergence of Macroscopic Complex Objects from Microscopic Noise

N.Shnerb Y.Louzoun E.Bettelheim S.Solomon

Henri Atlan (biology),

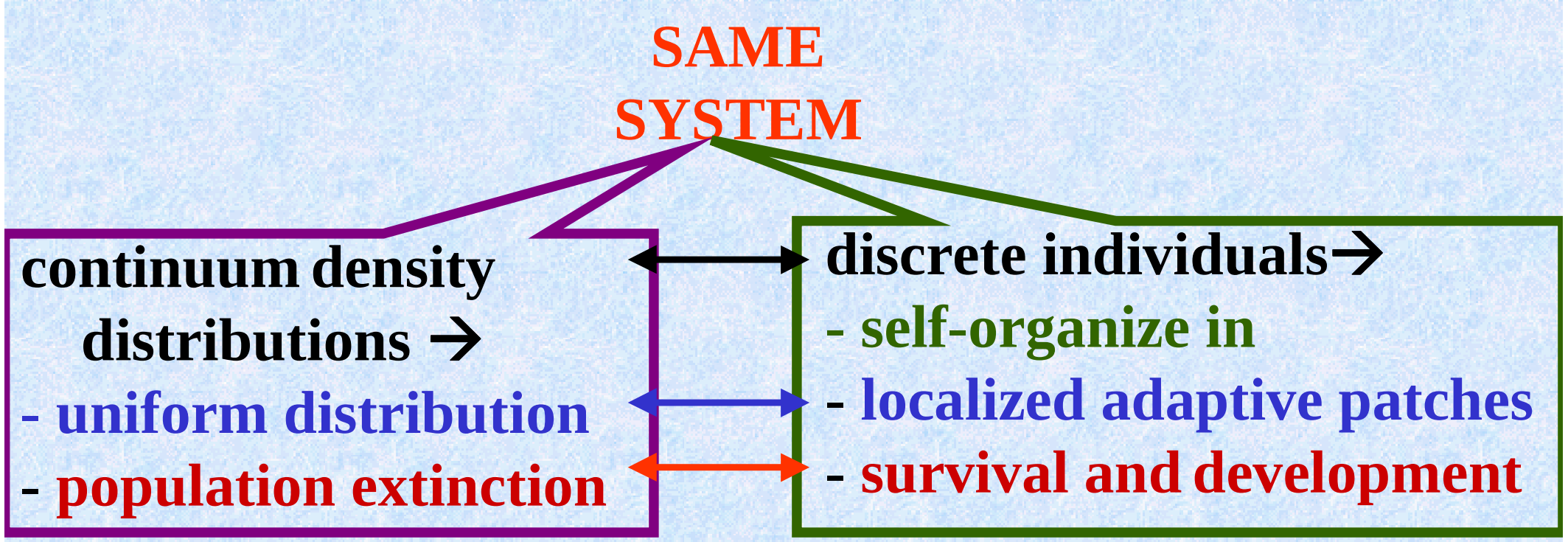
J Goldenberg (marketing),

H and M Levy (finance econo),

Irun Cohen (immunology),

Hanoch Lavee (desert/ecology)

D Mazursky (Soc. Sci., Bus. Adm.)



Explain complexity as emerging from the interactions of two very (trivially?) simple kinds of particles

A and B

"Life" (as opposed to uneventful uniform state) Emerges and thrives even **in most improbable conditions.**

Plan of the Talk

- Definition of the System
- Continuum predictions
- Actual Behavior ; Role of discreteness ; Movie

- Euristic Explanation
  emergence of adaptive collective objects
- Interpretation in various fields
  (life emergence, ecology, immunology, society, marketing, finance)
- Pedagogical Sketch of RG flow and Rigorous exact proof

- Applications:
  - HIV
  - Globalization,
  - desert reclaim

Anticlimactic, ready to stop when time is up

Imagine an area inhabited by a population of **eternal agents *A***,

- which spread out **uniformly with average density** $a(x,t)=a_0$

and

- move around randomly, with **diffusion coefficient** D_a

Imagine now a race of **mortals *B***, hopping at a **diffusion rate** D_b

over the same area with **initial uniform density** $b(x,t=0)= b_0$

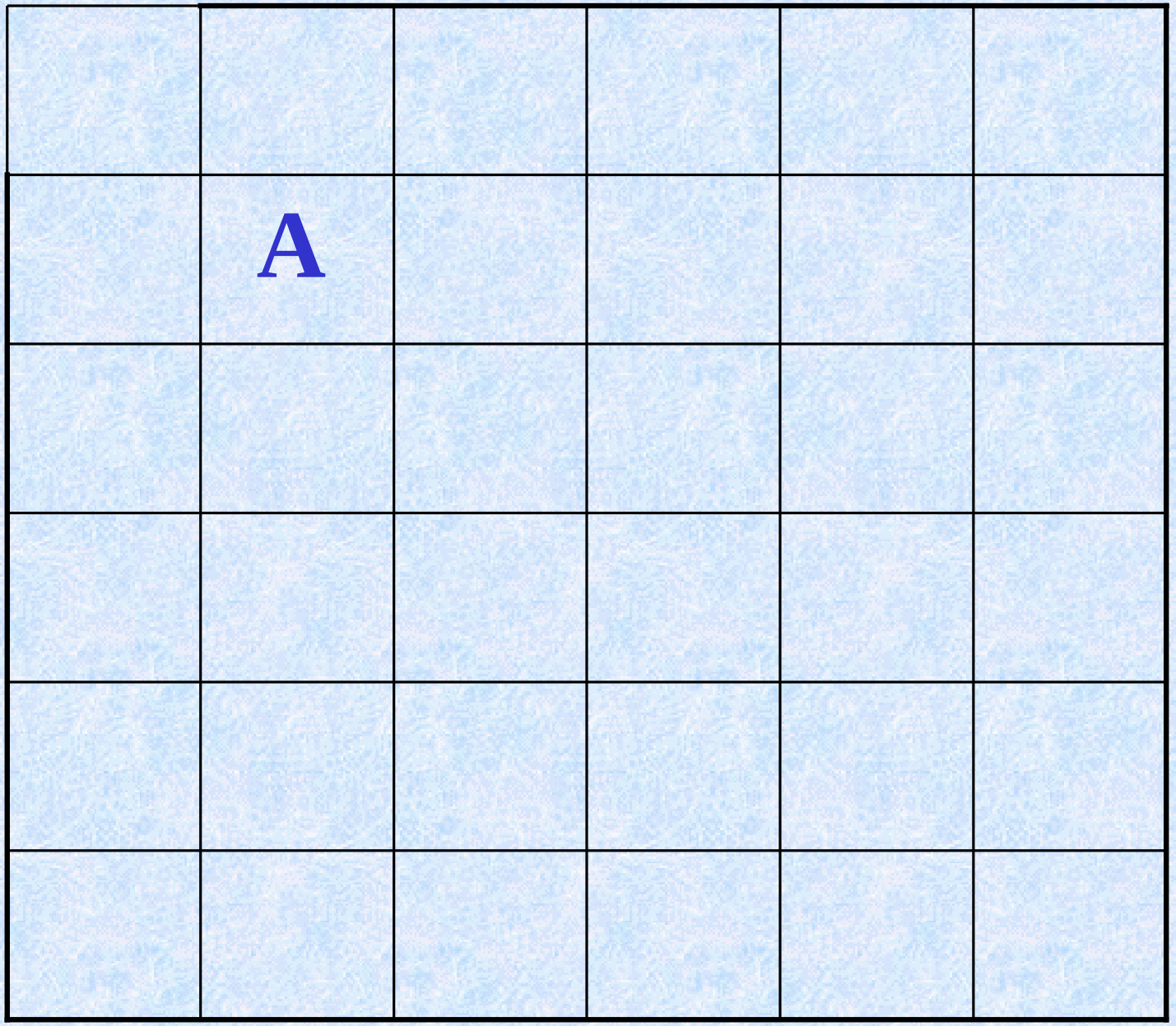
The *B*'s **die at a constant death rate** μ: $B \rightarrow \varnothing$

and

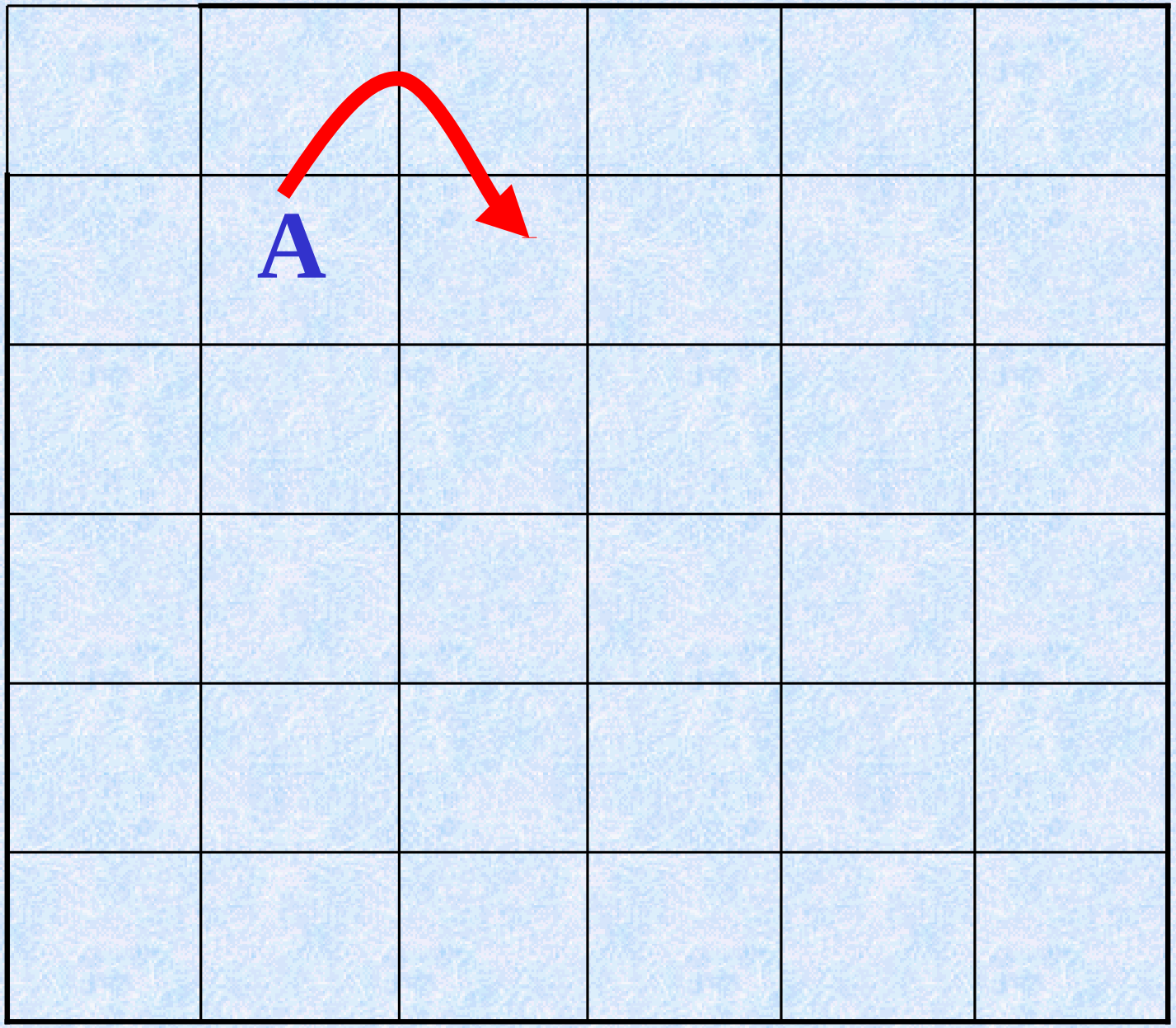
proliferate (generate a new B) [when they are at the same location with a "catalyst," *A*],

with a **birth rate** λ: $B + A \rightarrow B + B + A$

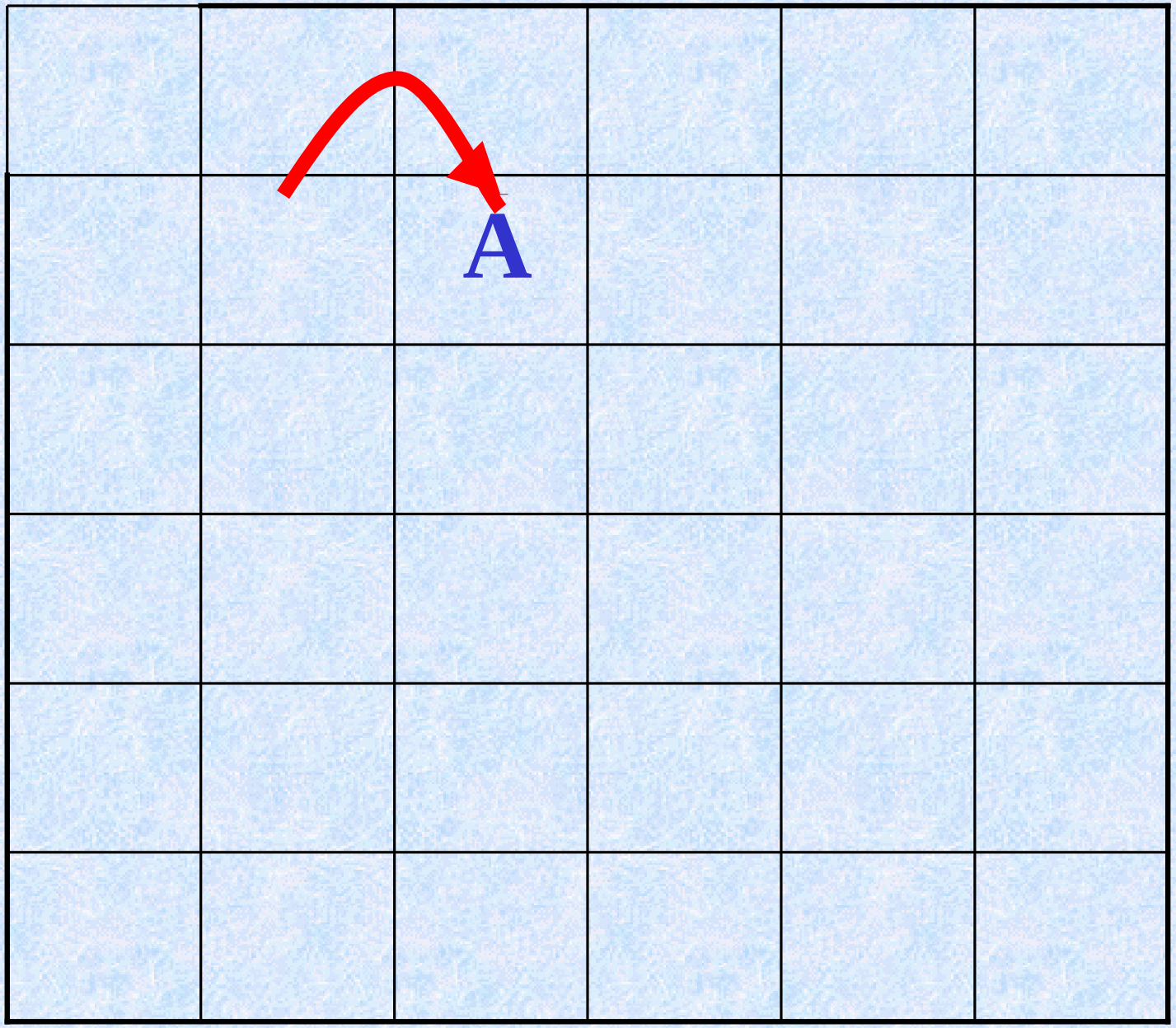
Diffusion of **A** at rate D_a



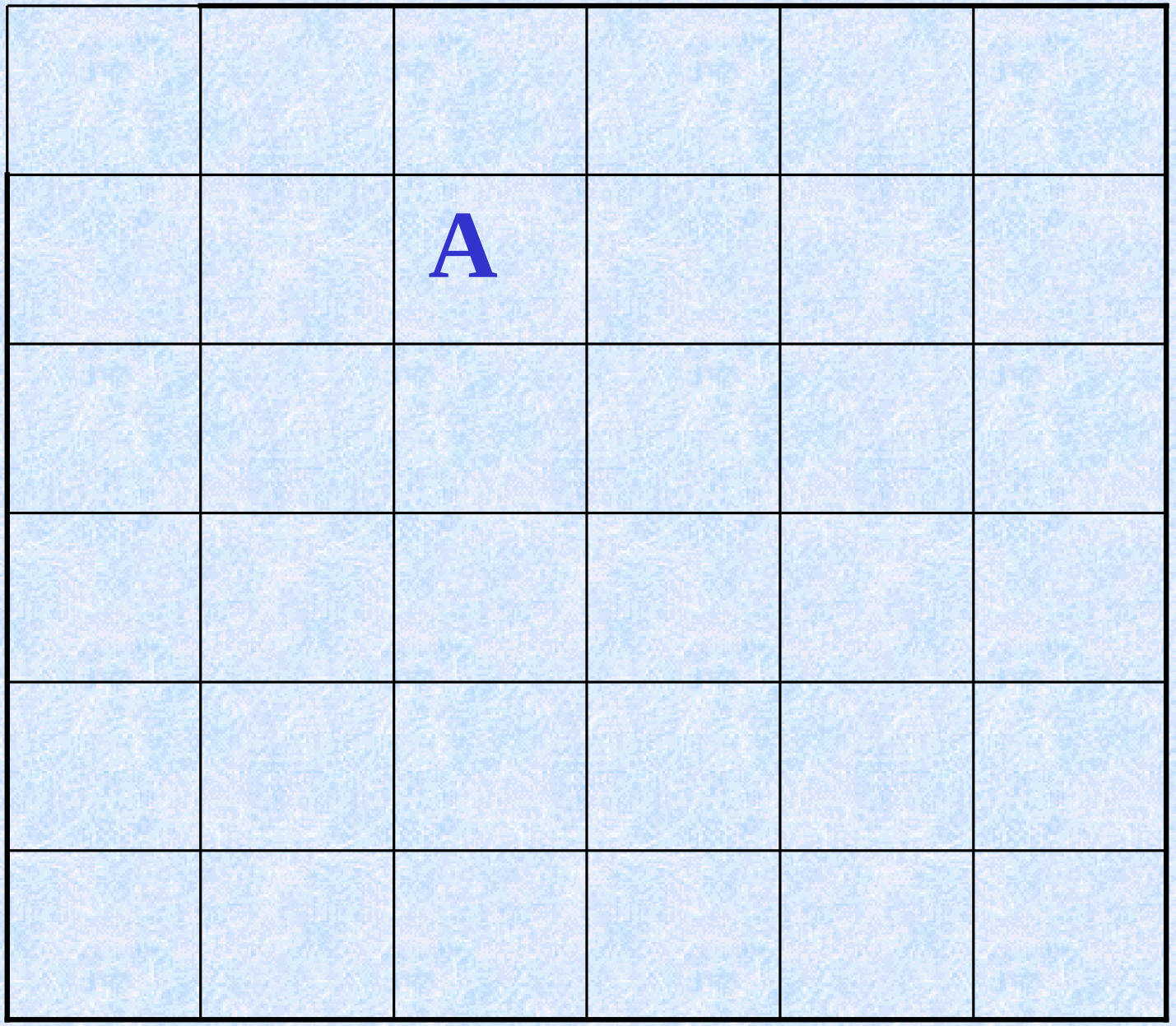
Diffusion of A at rate D_a



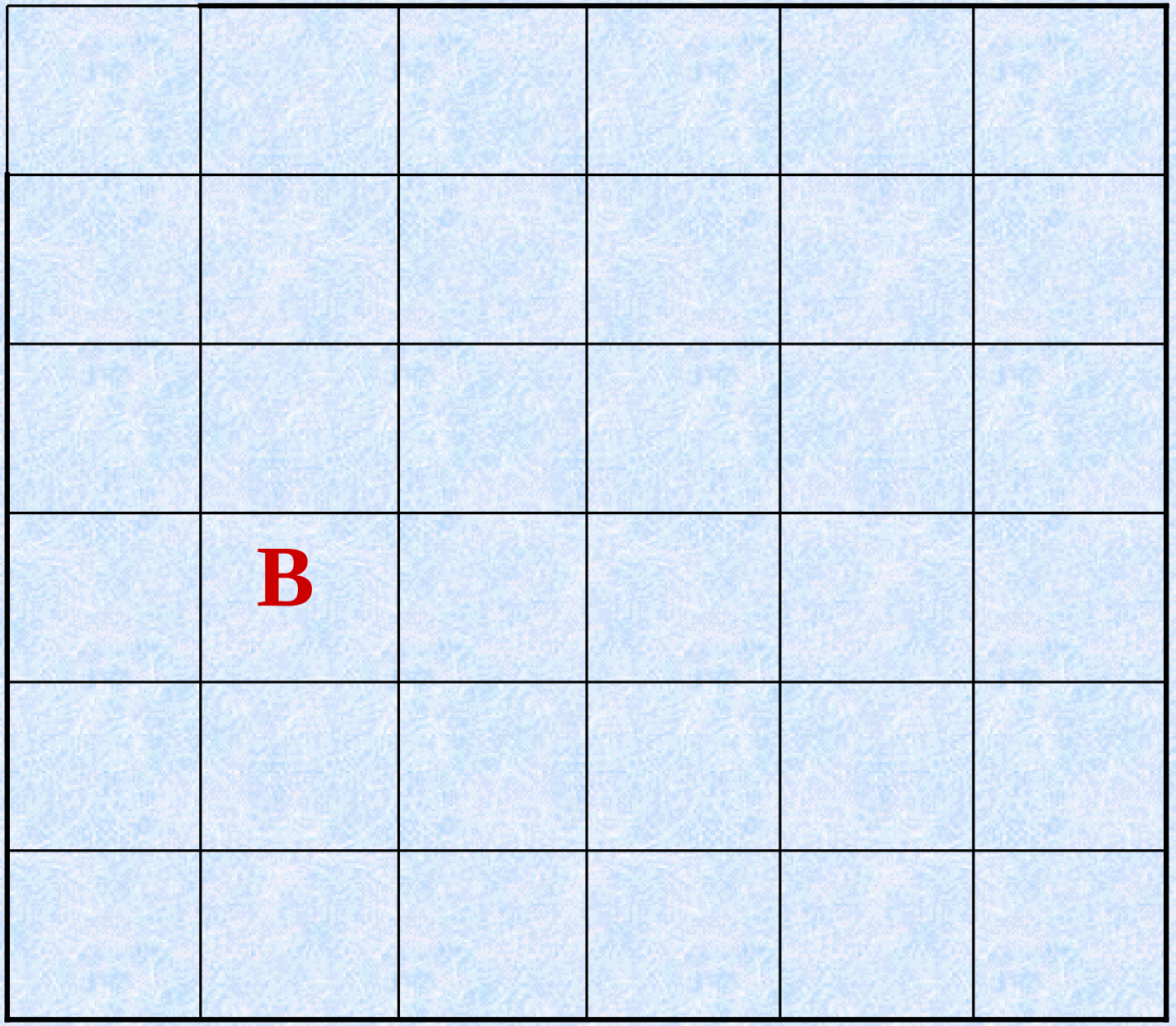
Diffusion of A at rate D_a



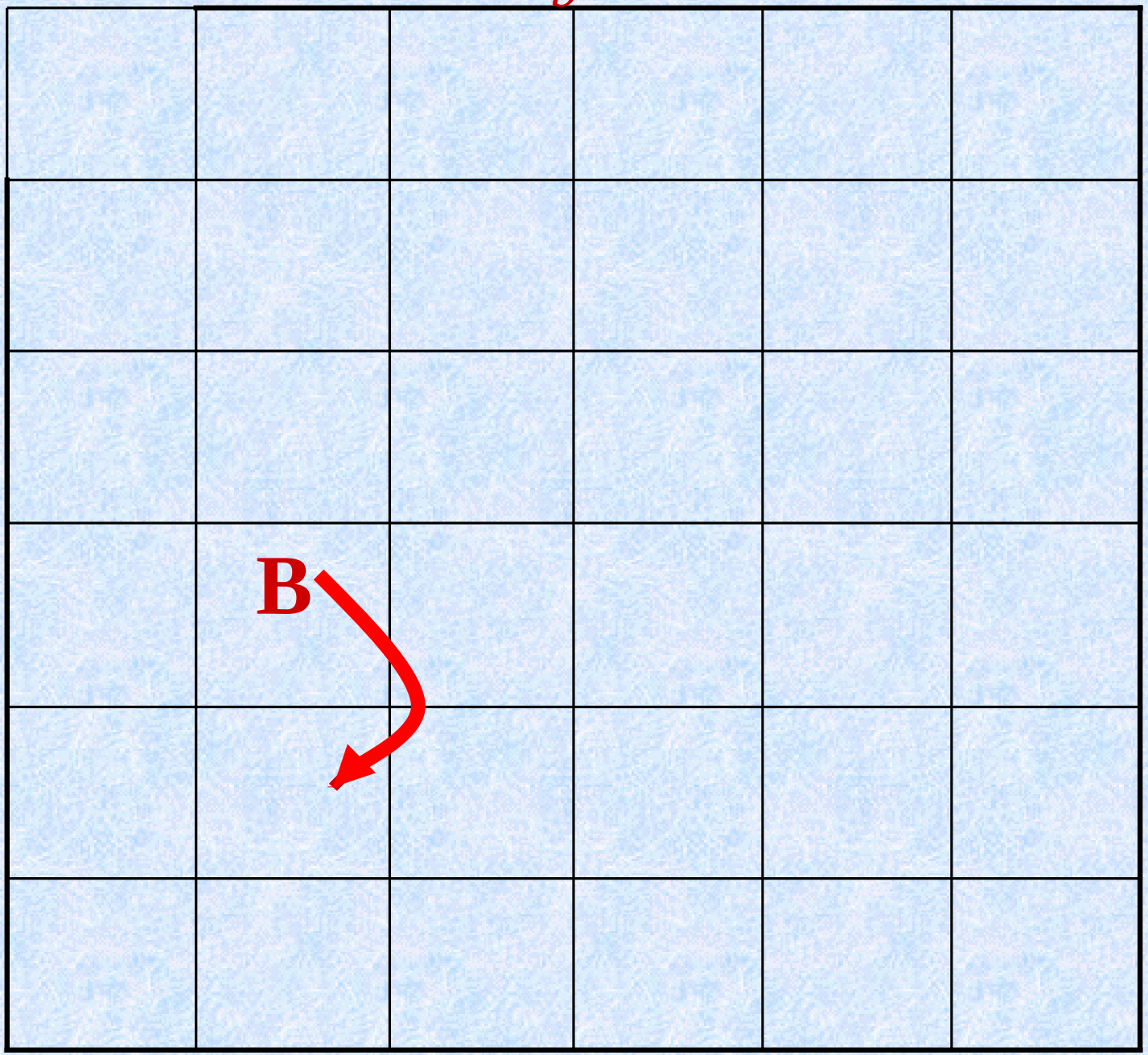
Diffusion of A at rate D_a



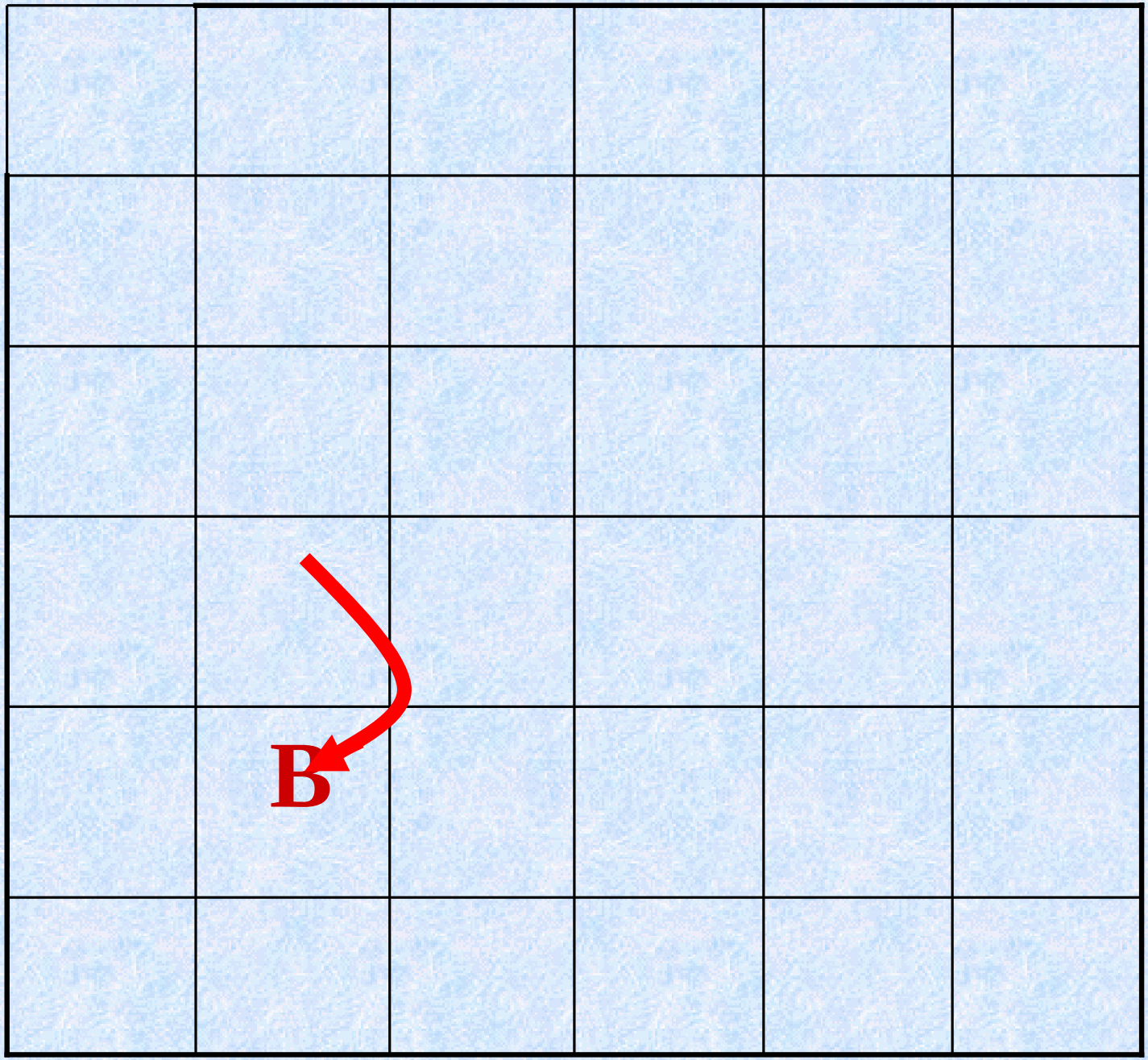
Diffusion of B at rate D_b



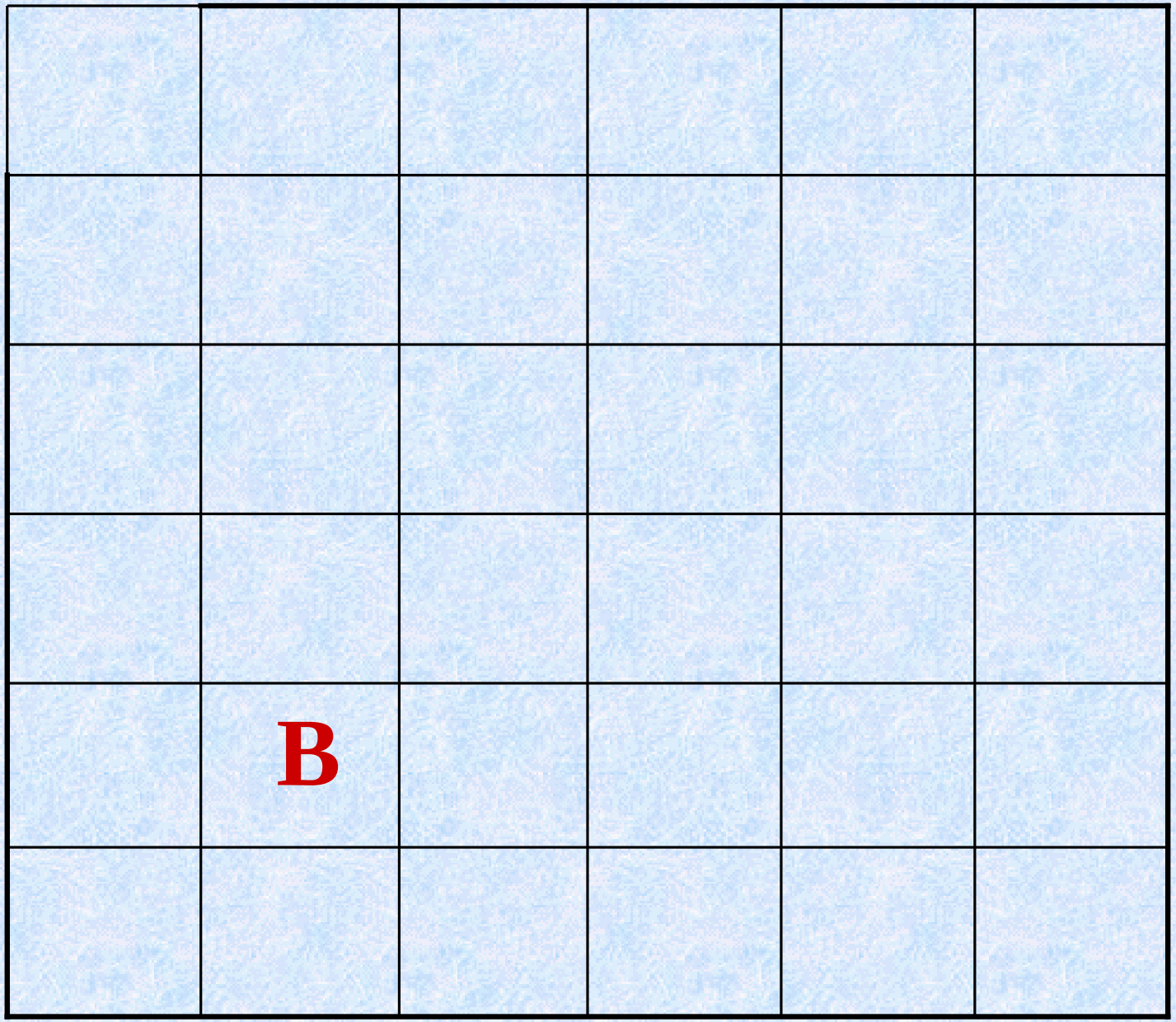
Diffusion of B at rate D_b



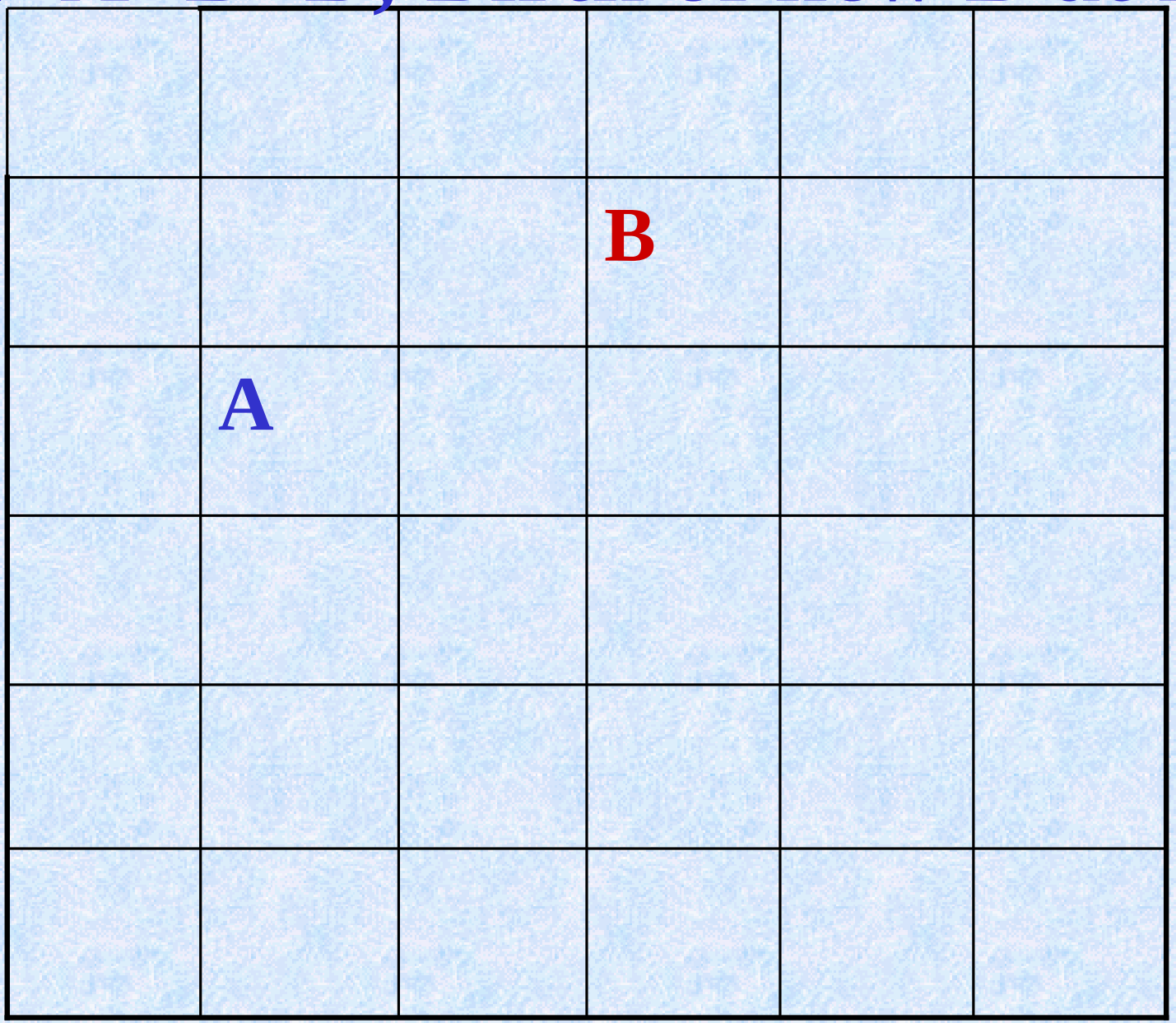
Diffusion of B at rate D_b



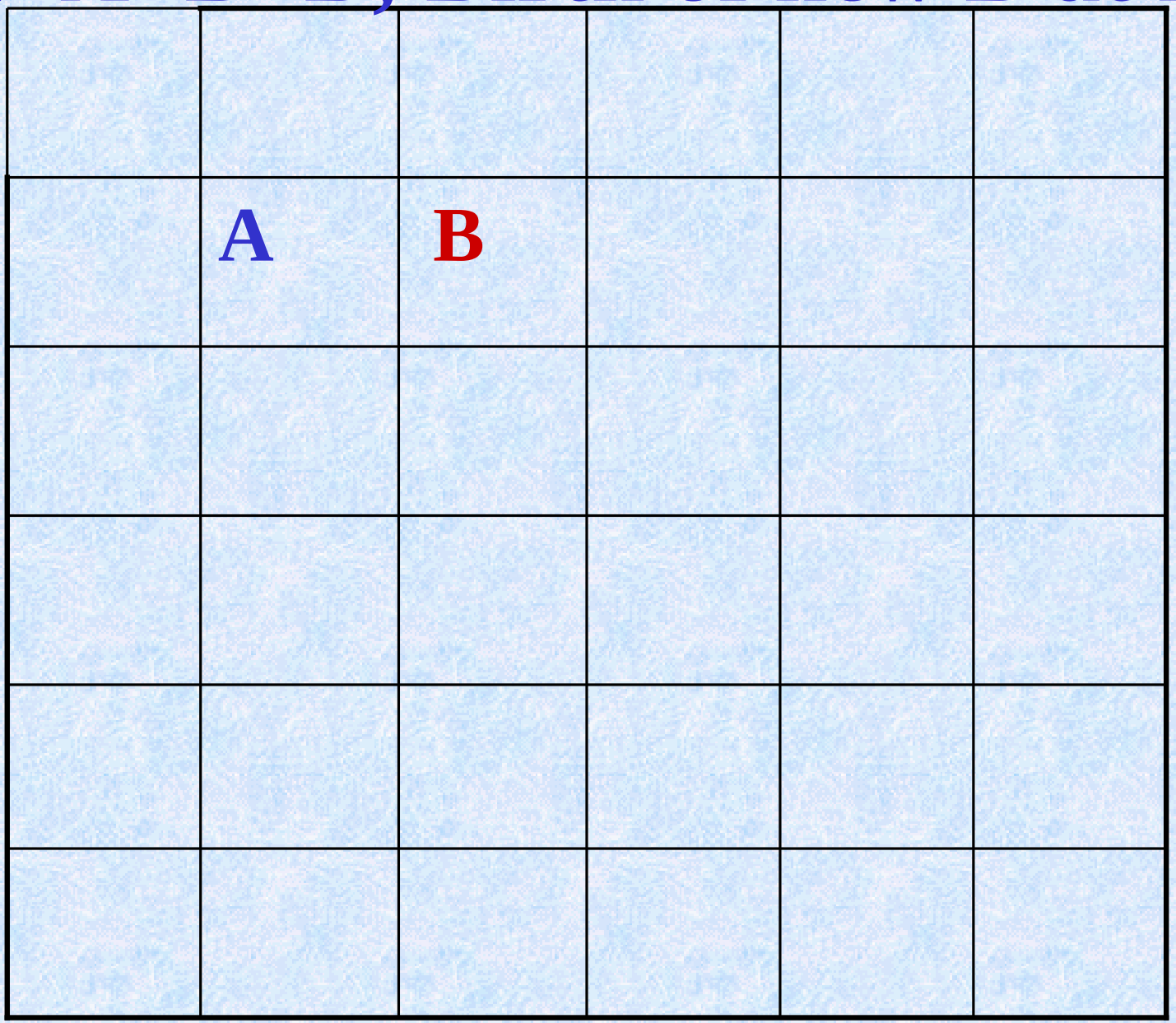
Diffusion of B at rate D_b



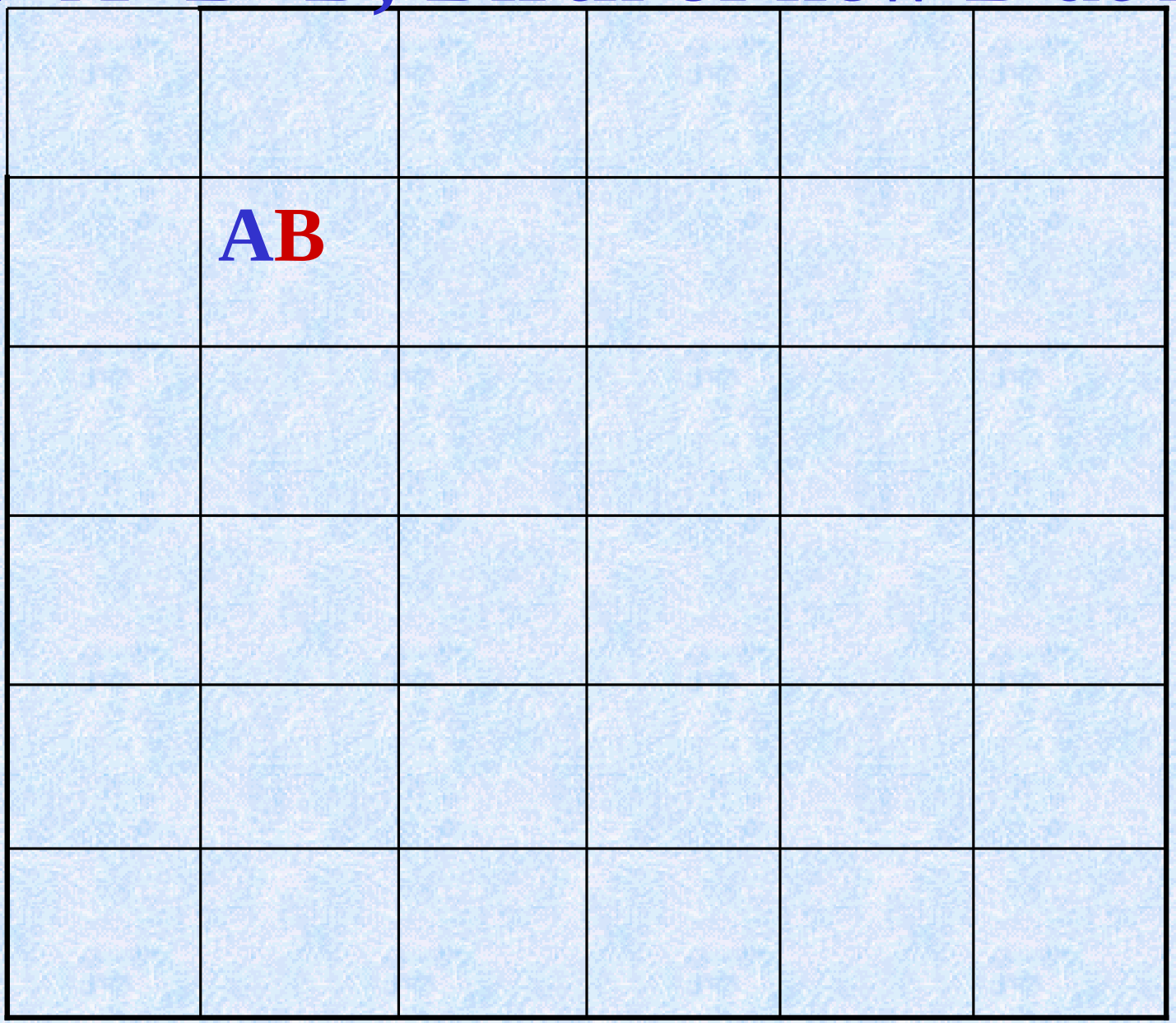
A+B → A+B+B; Birth of new B at rate λ



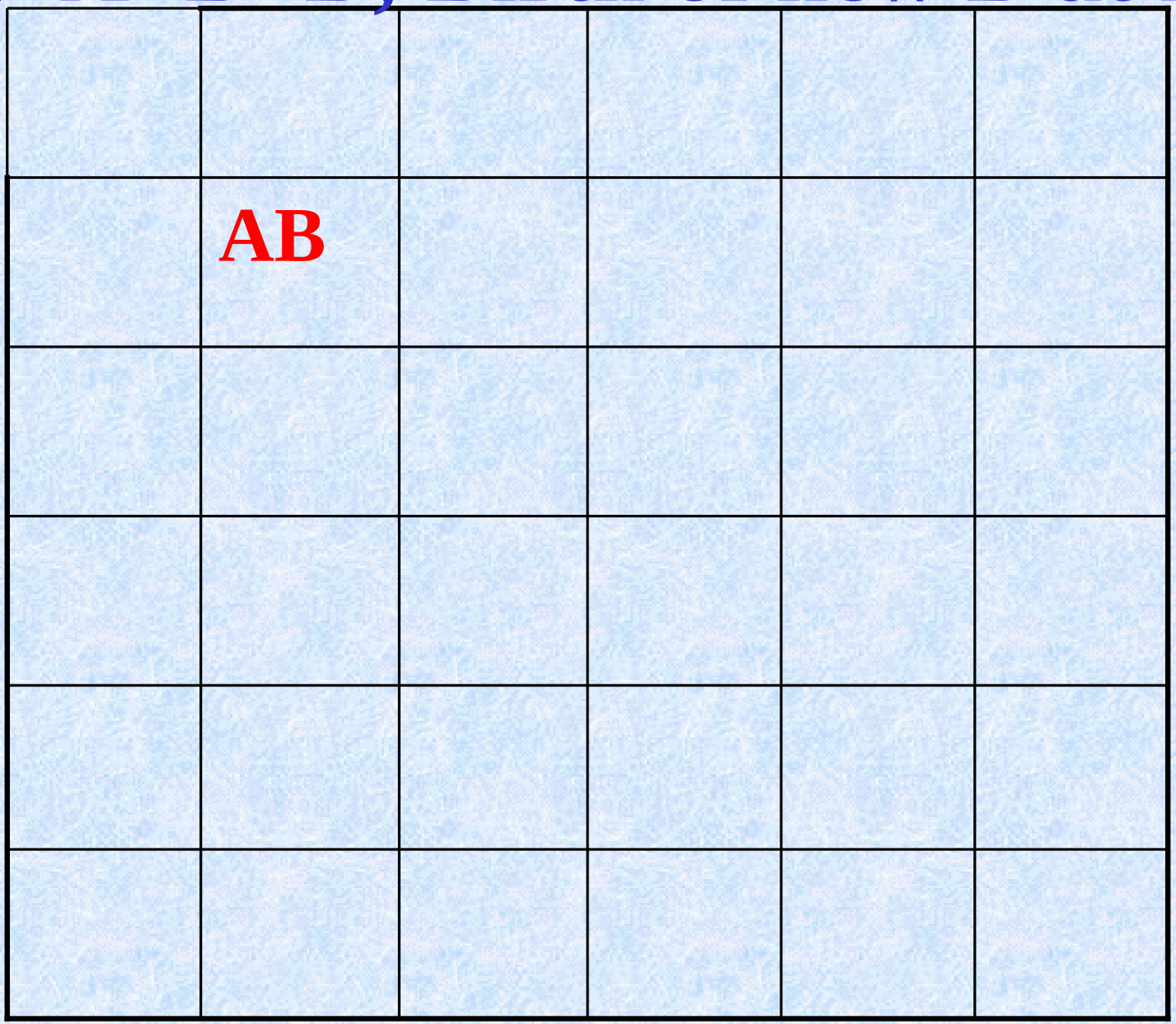
A+B → A+B+B; Birth of new B at rate λ



A+B → A+B+B; Birth of new B at rate λ



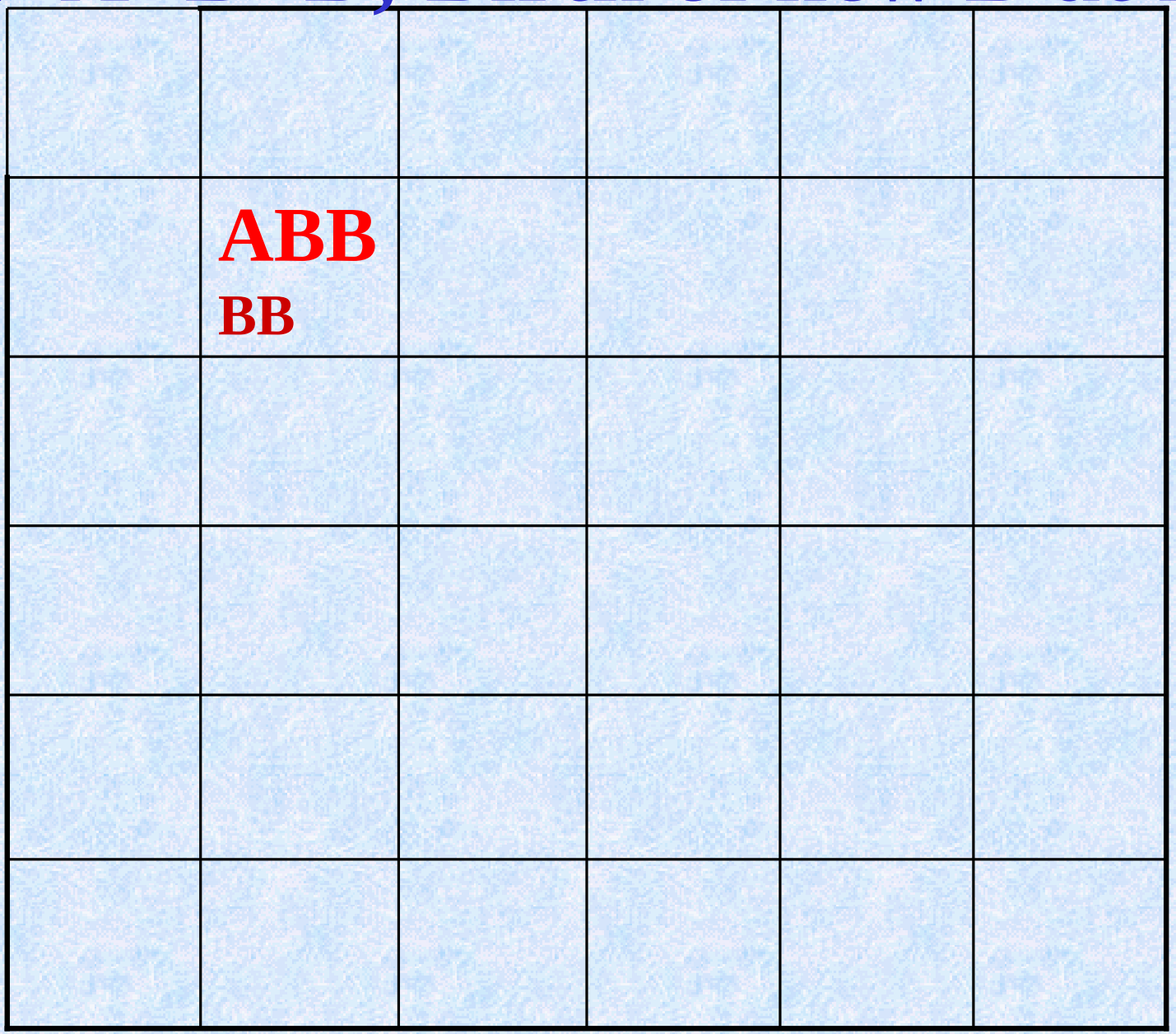
A+B → A+B+B; Birth of new B at rate λ



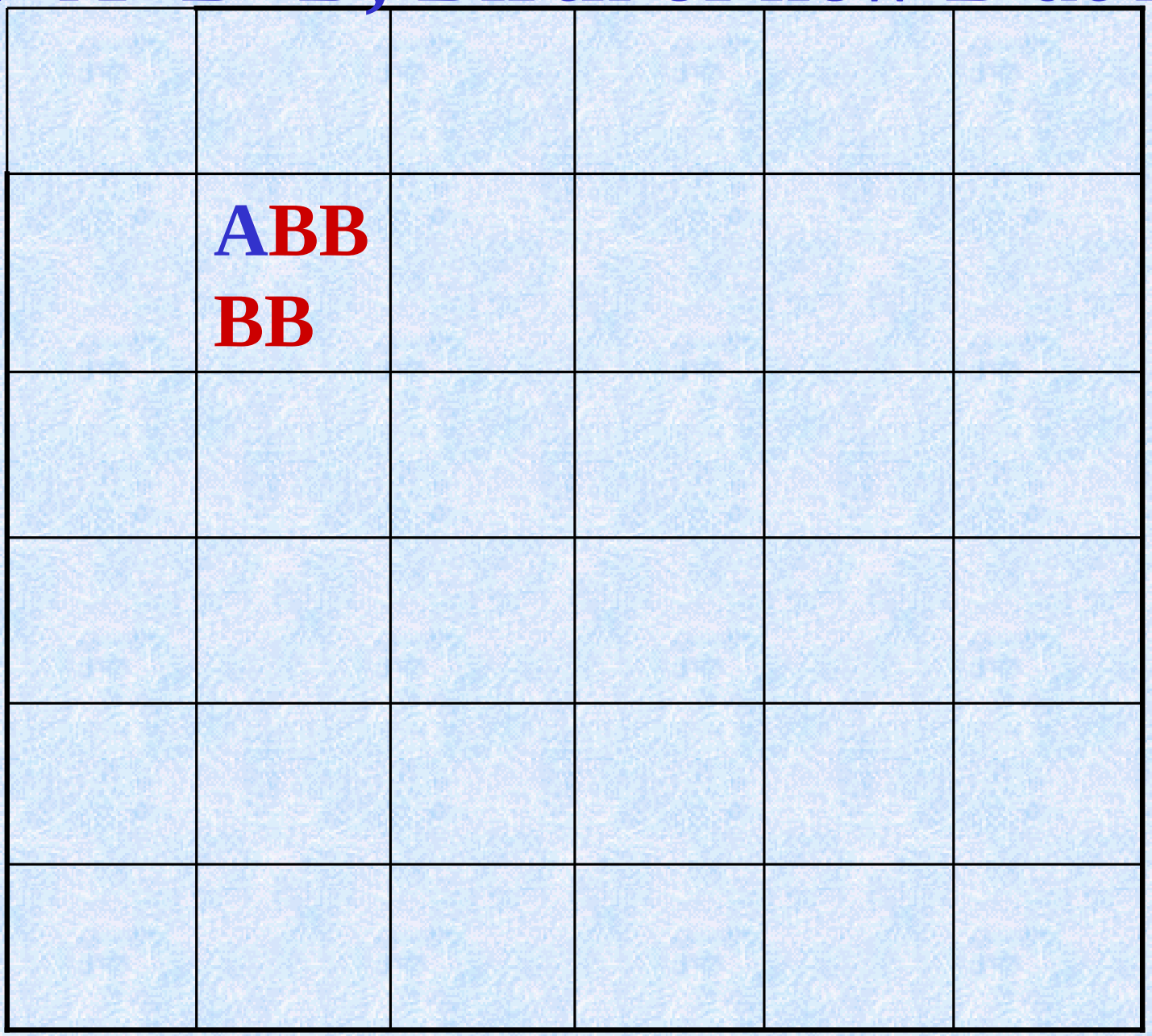
A+B → A+B+B; Birth of new B at rate λ

A+B → A+B+B; Birth of new B at rate λ

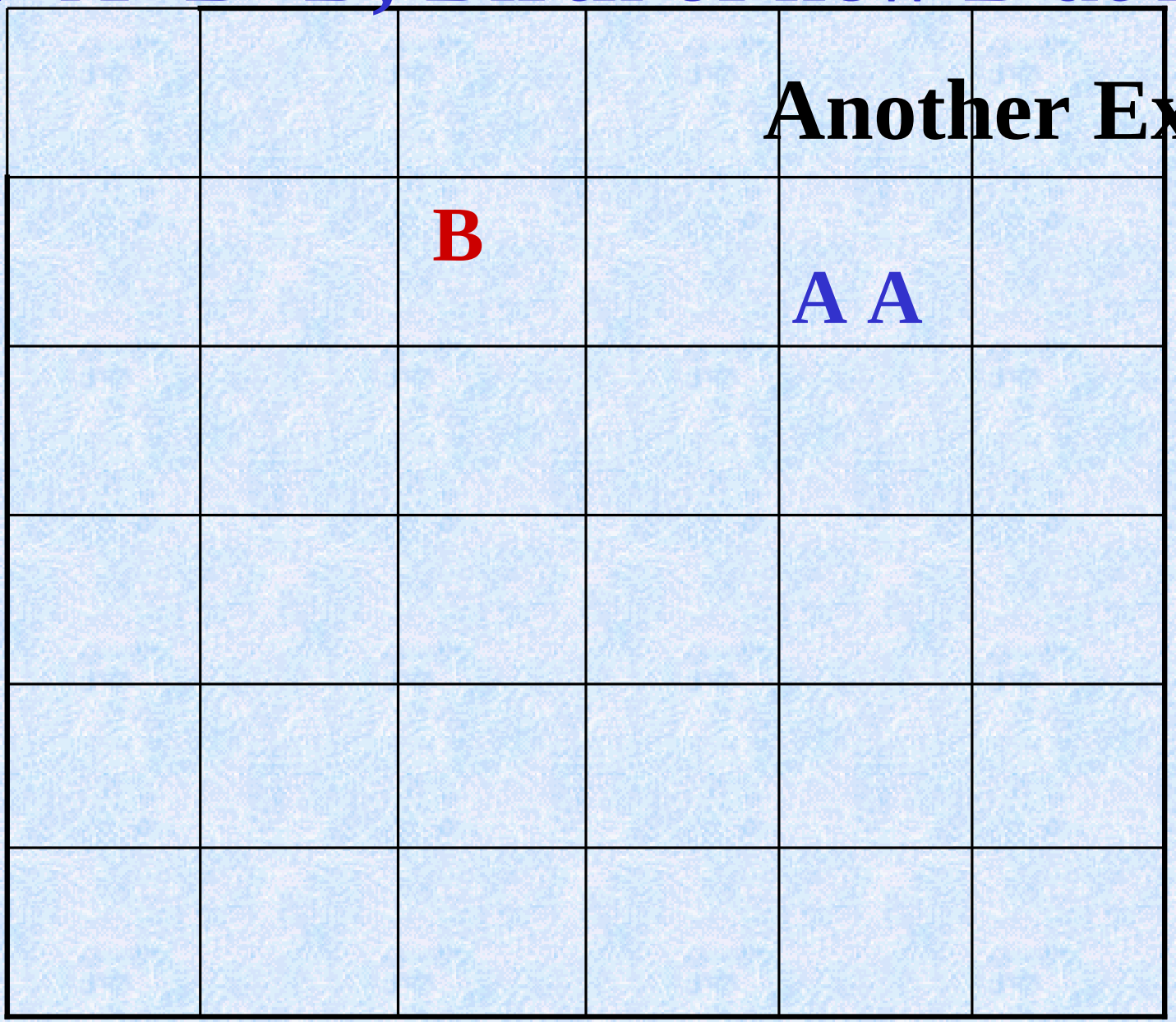
A+B → A+B+B; Birth of new B at rate λ



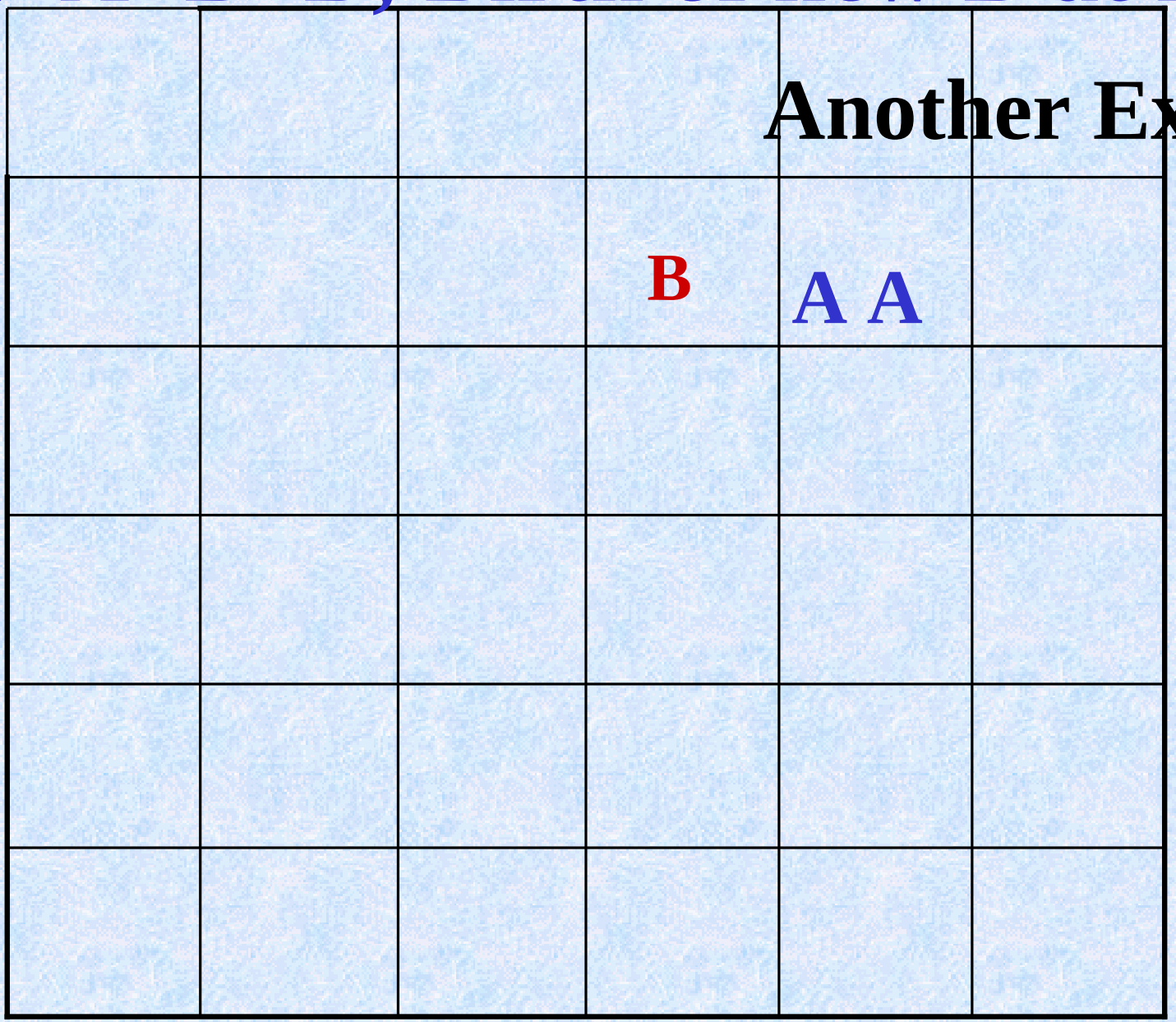
A+B → A+B+B; Birth of new B at rate λ



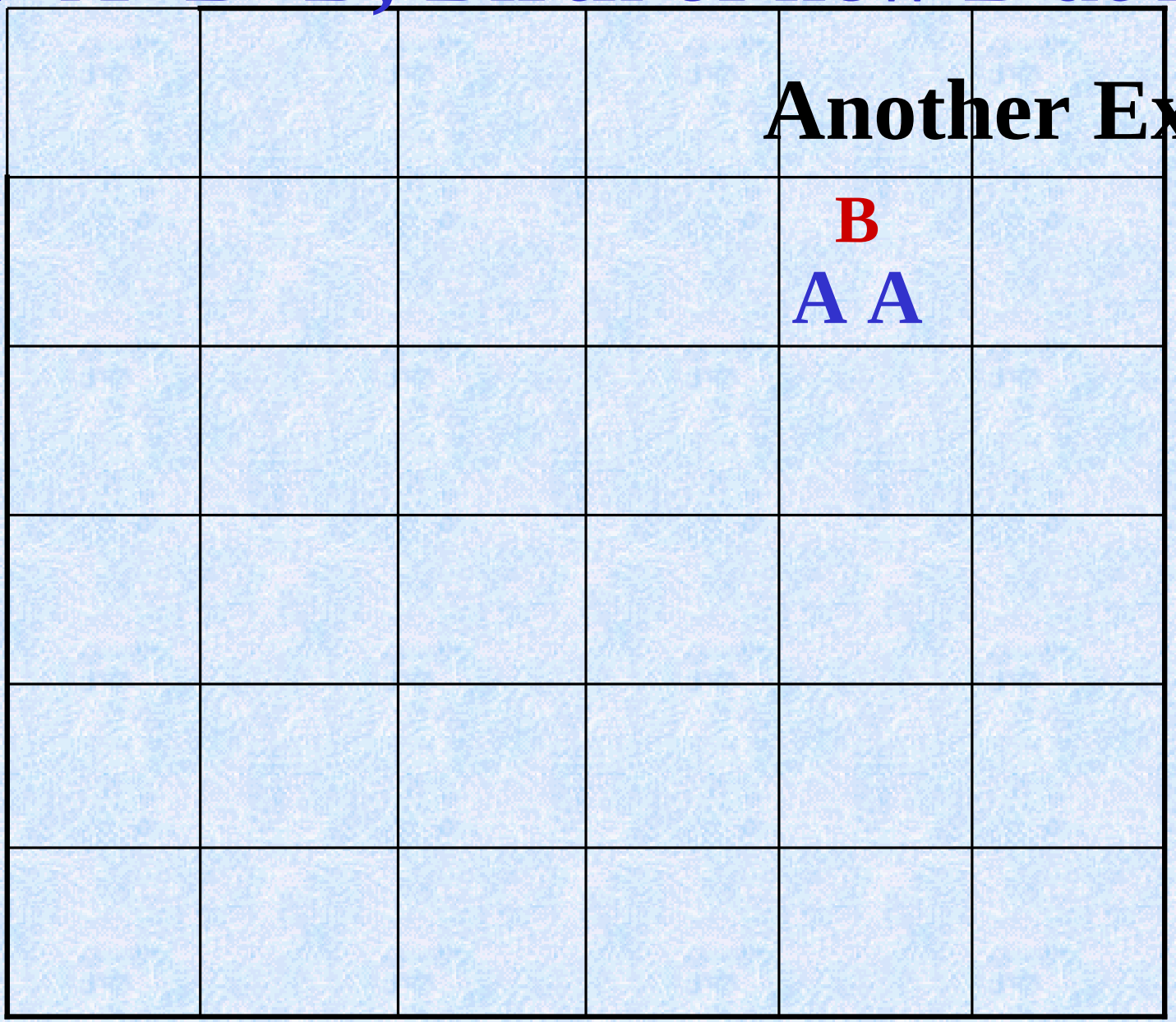
A+B → A+B+B; Birth of new B at rate λ



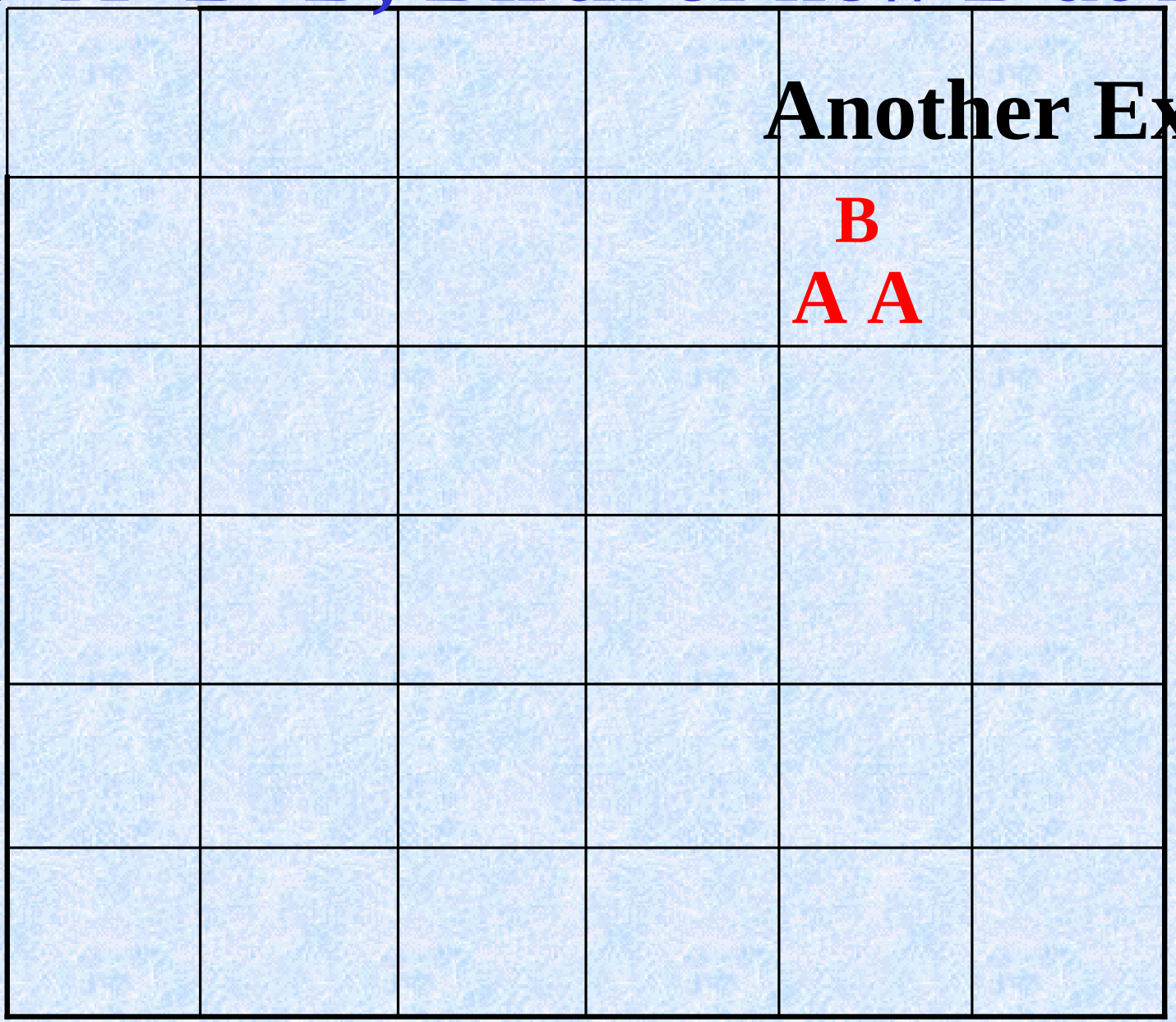
A+B → A+B+B; Birth of new B at rate λ



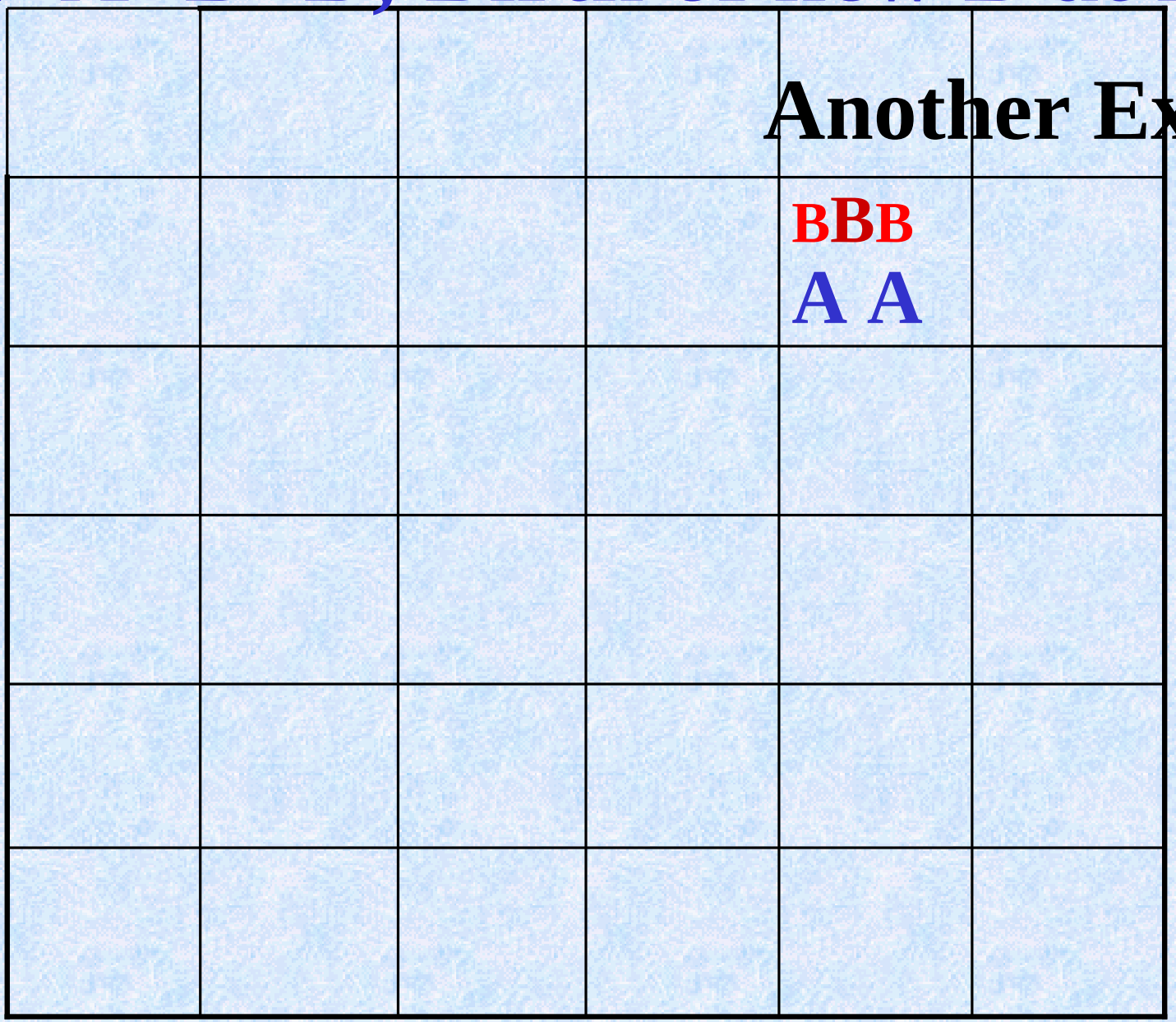
A+B → A+B+B; Birth of new B at rate λ



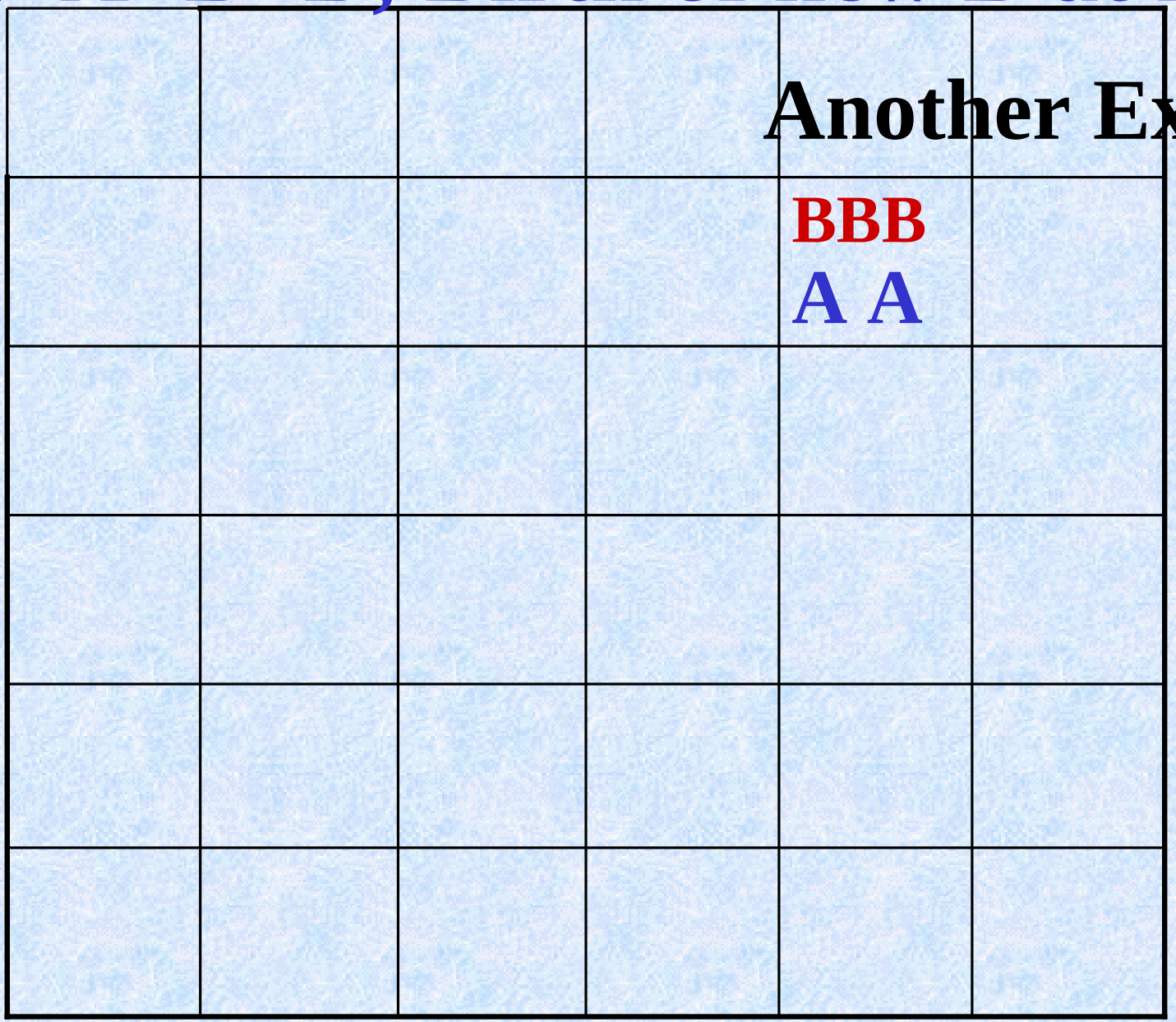
A+B → A+B+B; Birth of new B at rate λ



A+B → A+B+B; Birth of new B at rate λ

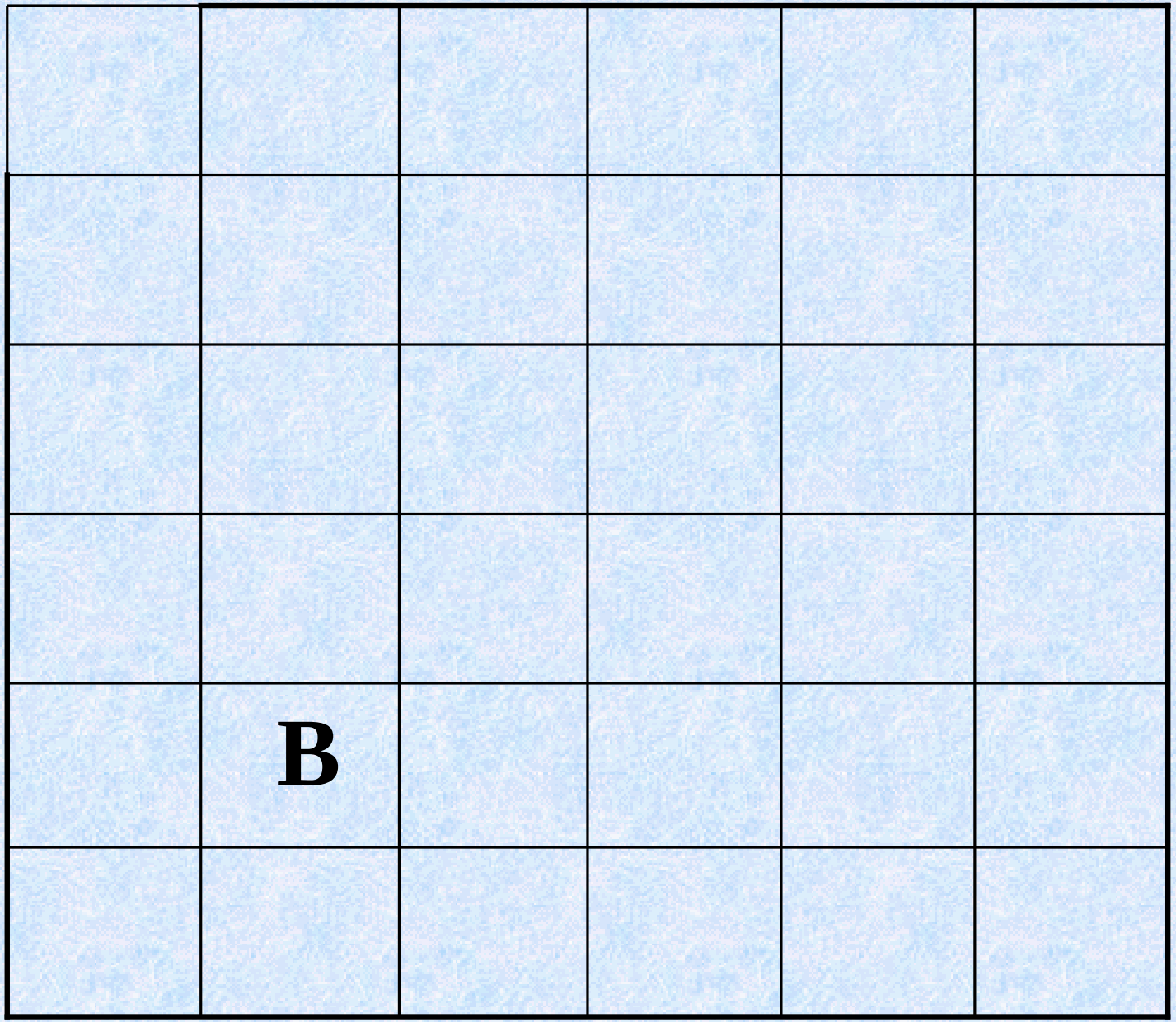


A+B → A+B+B; Birth of new B at rate λ



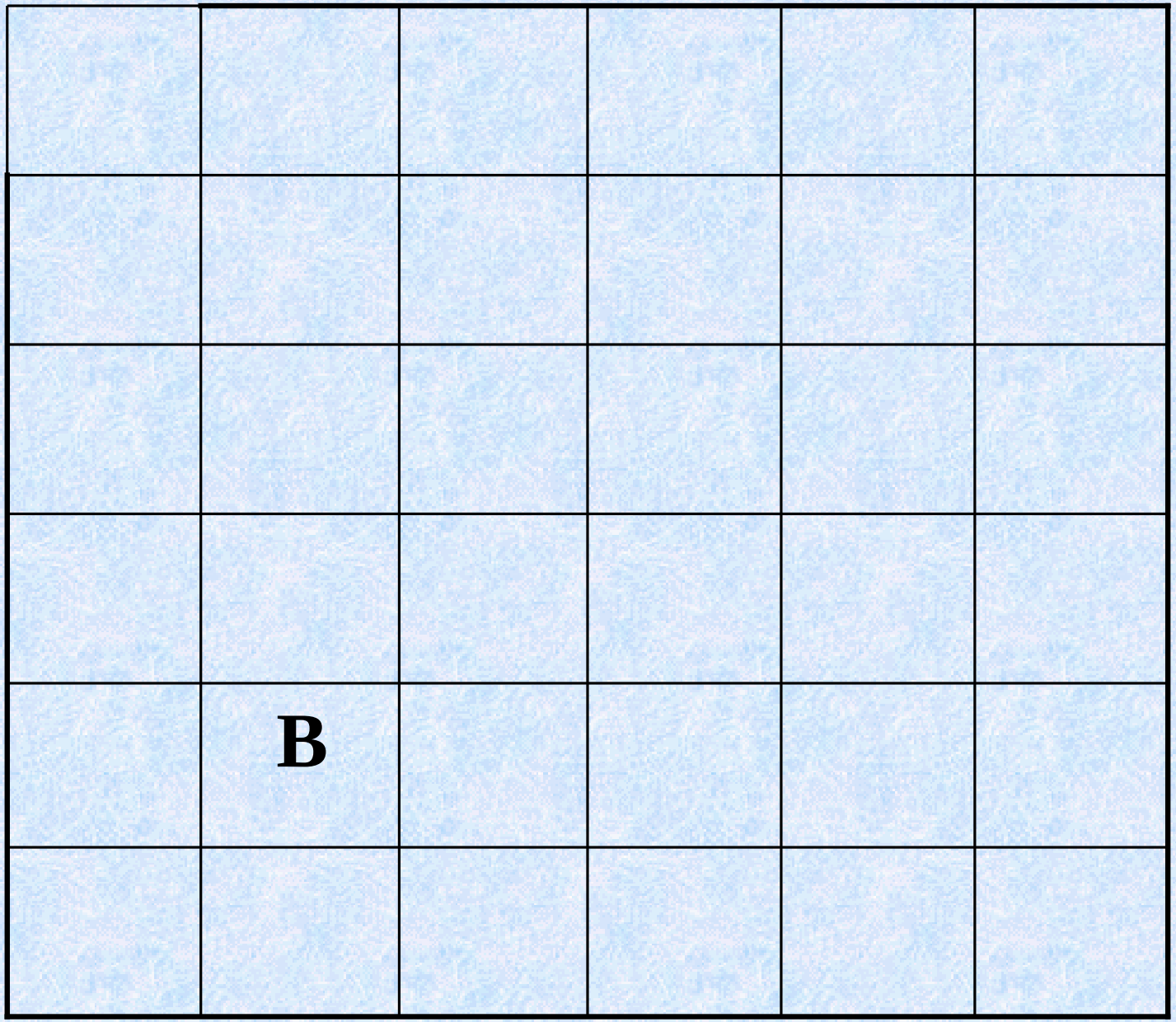
B →∅

Death of B at rate μ



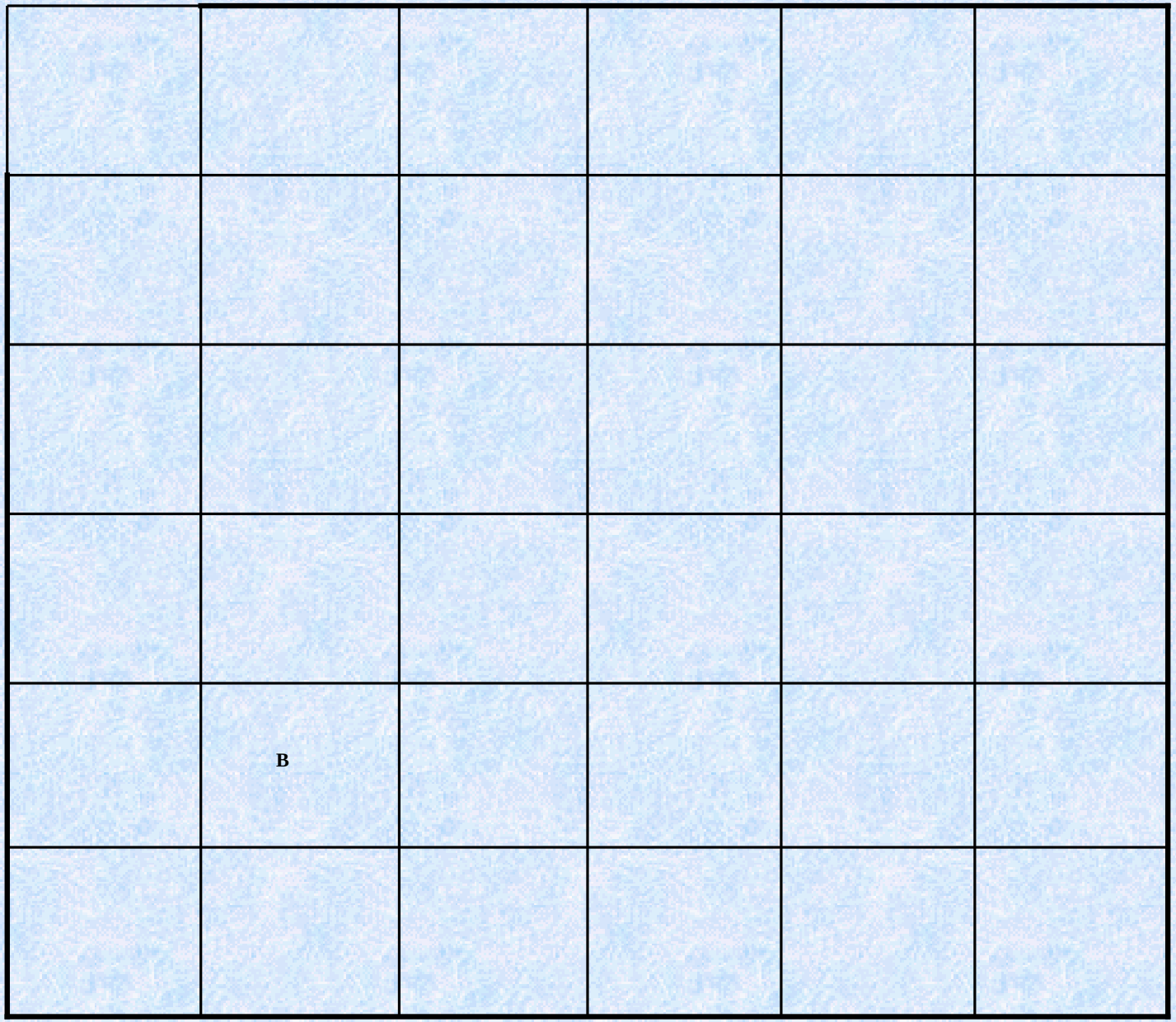
B →∅

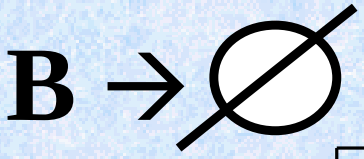
Death of B at rate μ



$B \rightarrow \emptyset$

Death of B at rate μ





Death of B at rate μ

What will happen?

The naive lore: continuum density distributions=>

- **A** maintains a homogeneous distribution density $a(x,t) = a_o$

- the number of **A**'s at any location where any **B** resides is a_0

- therefore the rate at which each **B**
produces new **B**'s is uniform $\lambda\, a_0$

- the probability rate for each **B** to die is uniform μ

Together, this gives a uniform rate of change

for the number of **B**'s : $b^{\bullet}(x,t) = (\lambda\, a_0 - \mu)\, b(x,t) < 0$

Therefore

$b(x,t)$ decays to 0 as $b_o(t) = b_o(0)\, e^{(\lambda\, a_0 - \mu)\, t}$

The continuum view: $a(x,t) = 2$.

Wherever the **B**'s are, they

- generate a new **B** at rate 2λ

and

- disappear at rate μ.

If $2\lambda - \mu < 0$, the total **B** number decreases to 0 as

$$e^{(2\lambda - \mu)t}$$

E.g $\mu = 5/2\ \lambda$

AA	AA	AA	AAB	AA	AA
AA	AA	AA	AA	AA	AA
AA	AAB	AA	AA	AA	AA
AA	AA	AA	AA	B AA	B AA
AA	AA	AA	AA	AA	AA

$$e^{(2\lambda - 5/2\ \lambda)t} = e^{-1/2\lambda t}$$

$$e^{-1/2\lambda t} + e^{-1/2\lambda t} \rightarrow 0$$

$$e^{(2\lambda - 5/2\ \lambda)t} = e^{-1/2\lambda t}$$

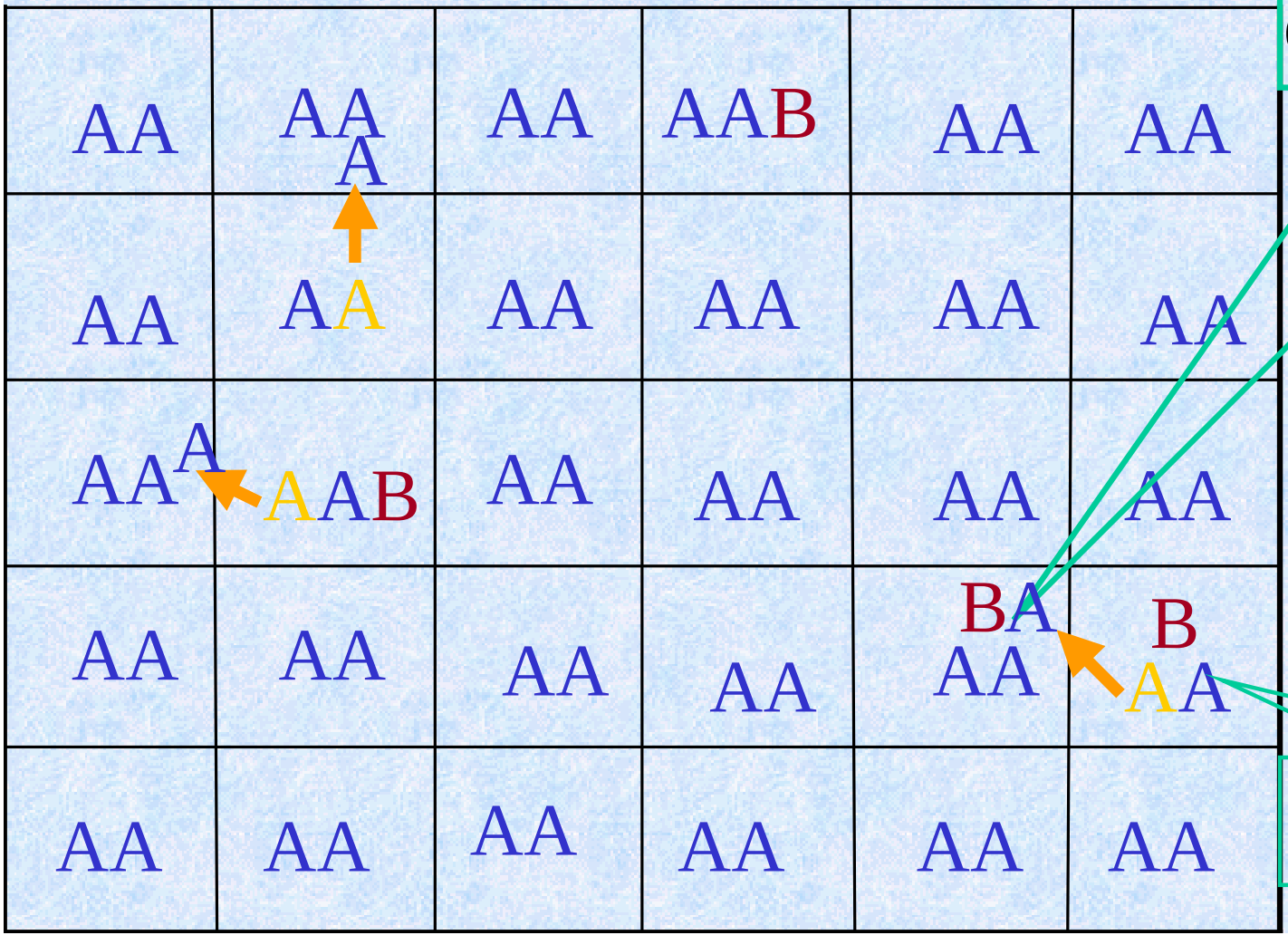
The discrete view: $a(x,t) \neq 2$.
But $< a(x,t) > = 2$. Now **B**'s

- generate new **B** at rate $a(x,t)\,\lambda$ and
- disappear at rate μ.

Even if $2\lambda - \mu < 0$, **b(x,t)** may still **increase** as

$$e^{(a(x,t)\lambda - \mu)\,t}$$

E.g $\mu = 5/2\,\lambda$



$e^{(3\lambda - 5/2\,\lambda)\,t} = e^{+1/2\lambda t}$

$e^{+1/2\lambda t} + e^{-3/2\lambda t} \to \infty$

$e^{(\lambda - 5/2\,\lambda)\,t} = e^{-3/2\lambda t}$

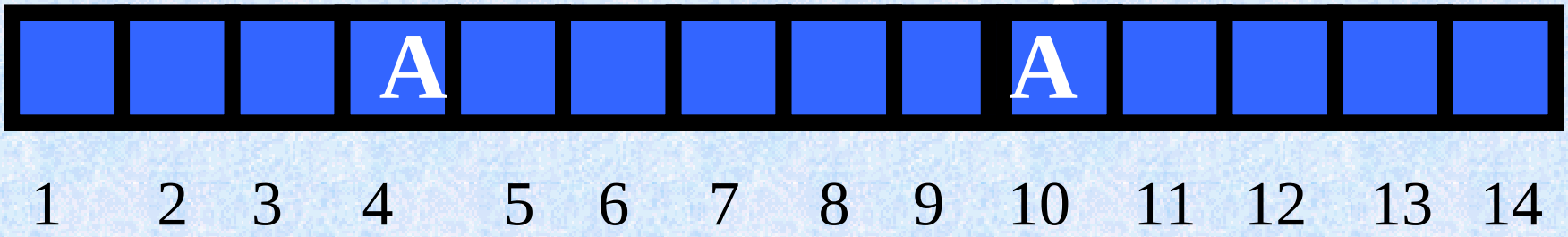
A one –dimensional example

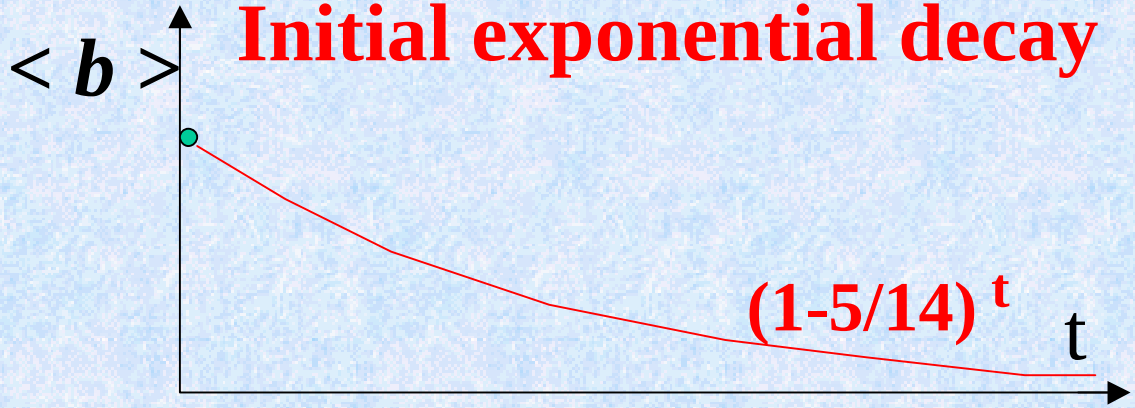
$$\lambda = 1 \quad a_0 = 1/7 \quad \mu = 1/2$$

$$1 \times 1/7 - 1/2 = -5/14 < 0$$

$$b(t+1) - b(t) = (\lambda a_0 - \mu)\, b(t) = -5/14\, b(t)$$

$$b(t+1) = 9/14\, b(t)$$



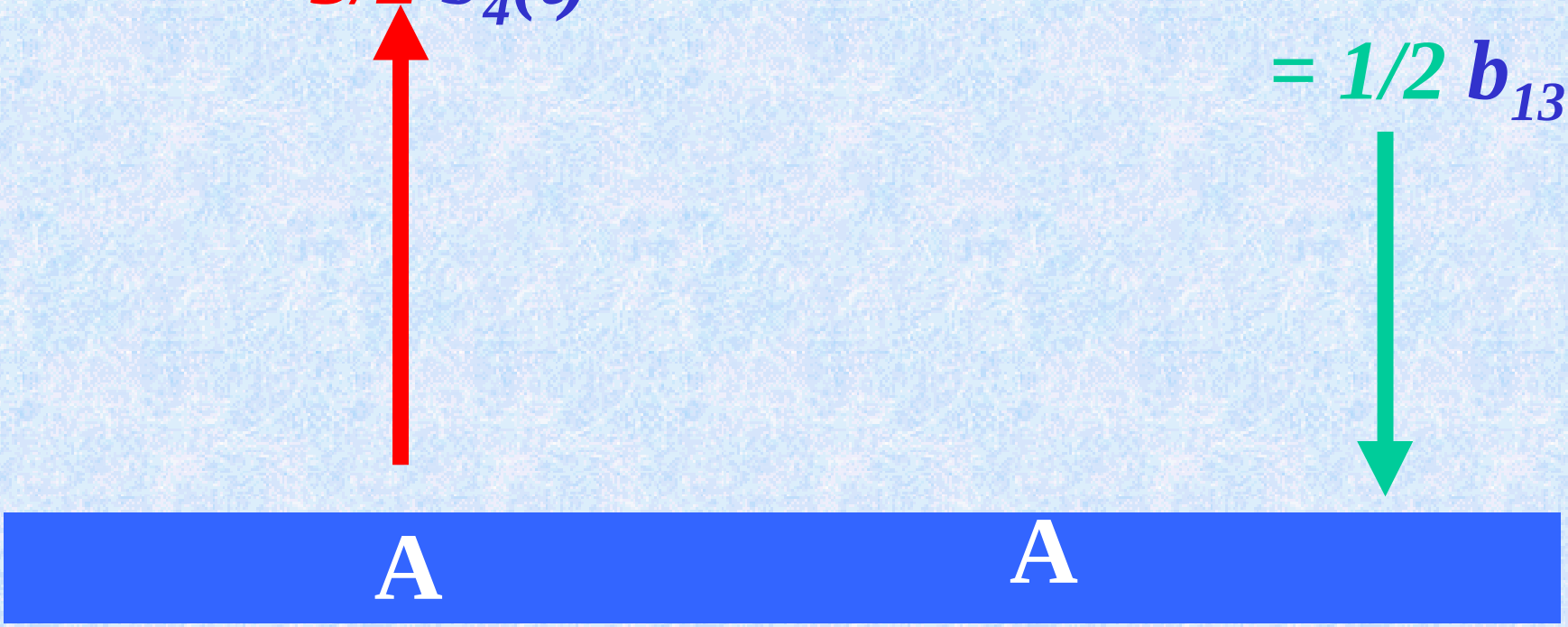


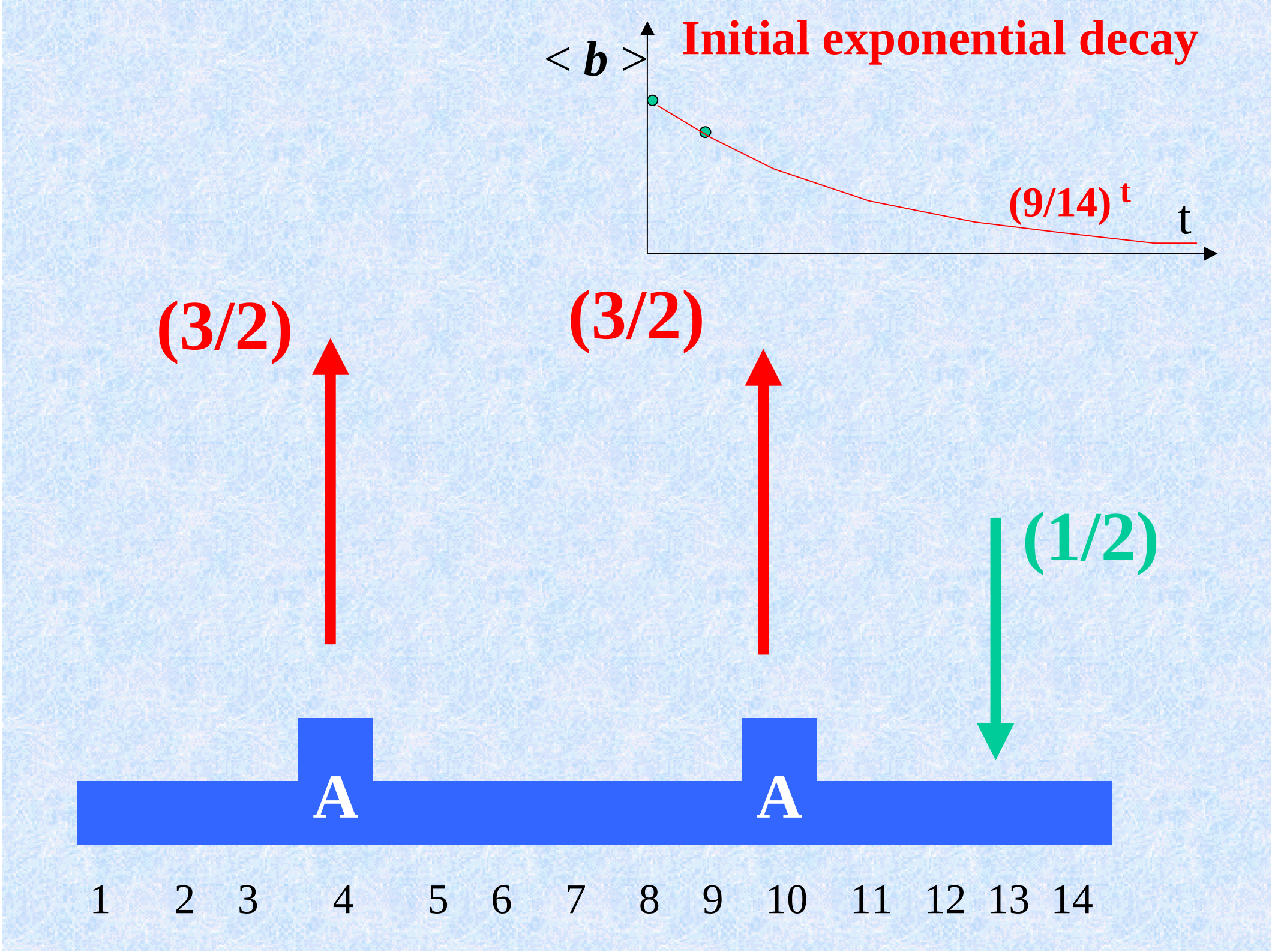
$b_4(t+1) = (1 + \lambda - \mu)\, b_4(t)$

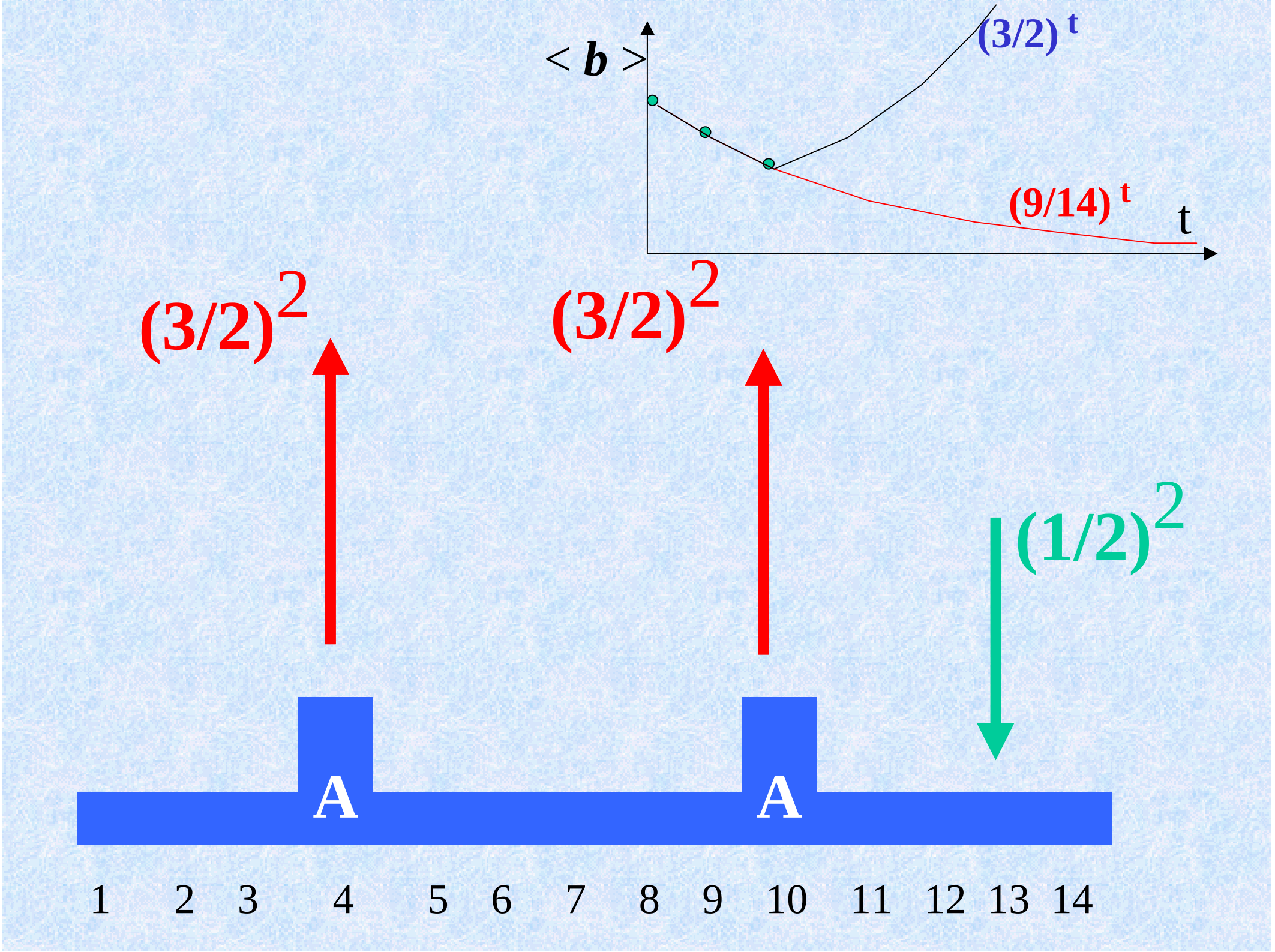
$= 3/2\, b_4(t)$

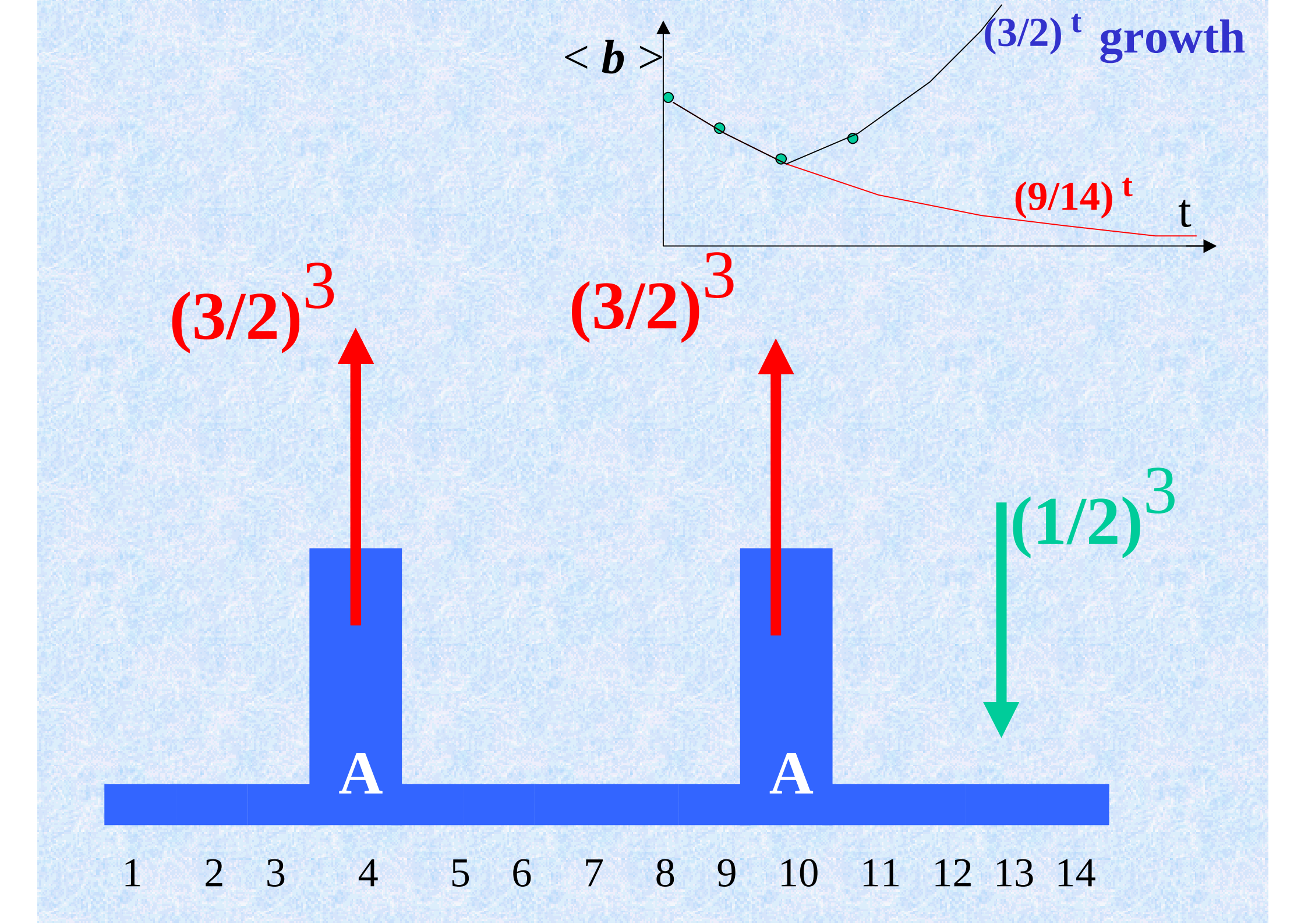
$b_{13}(t+1) = (1 - \mu)\, b_{13}(t)$

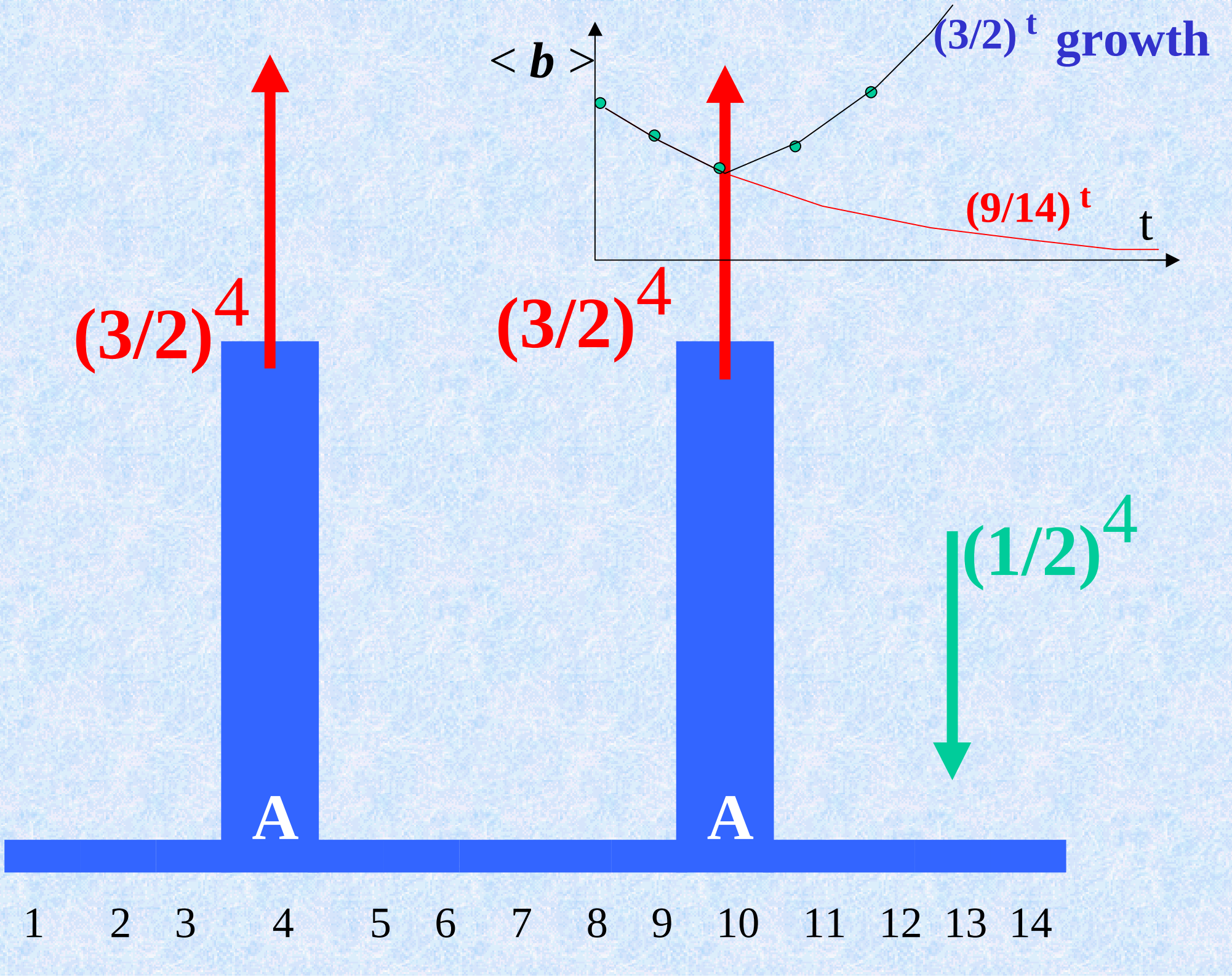
$= 1/2\, b_{13}(t)$

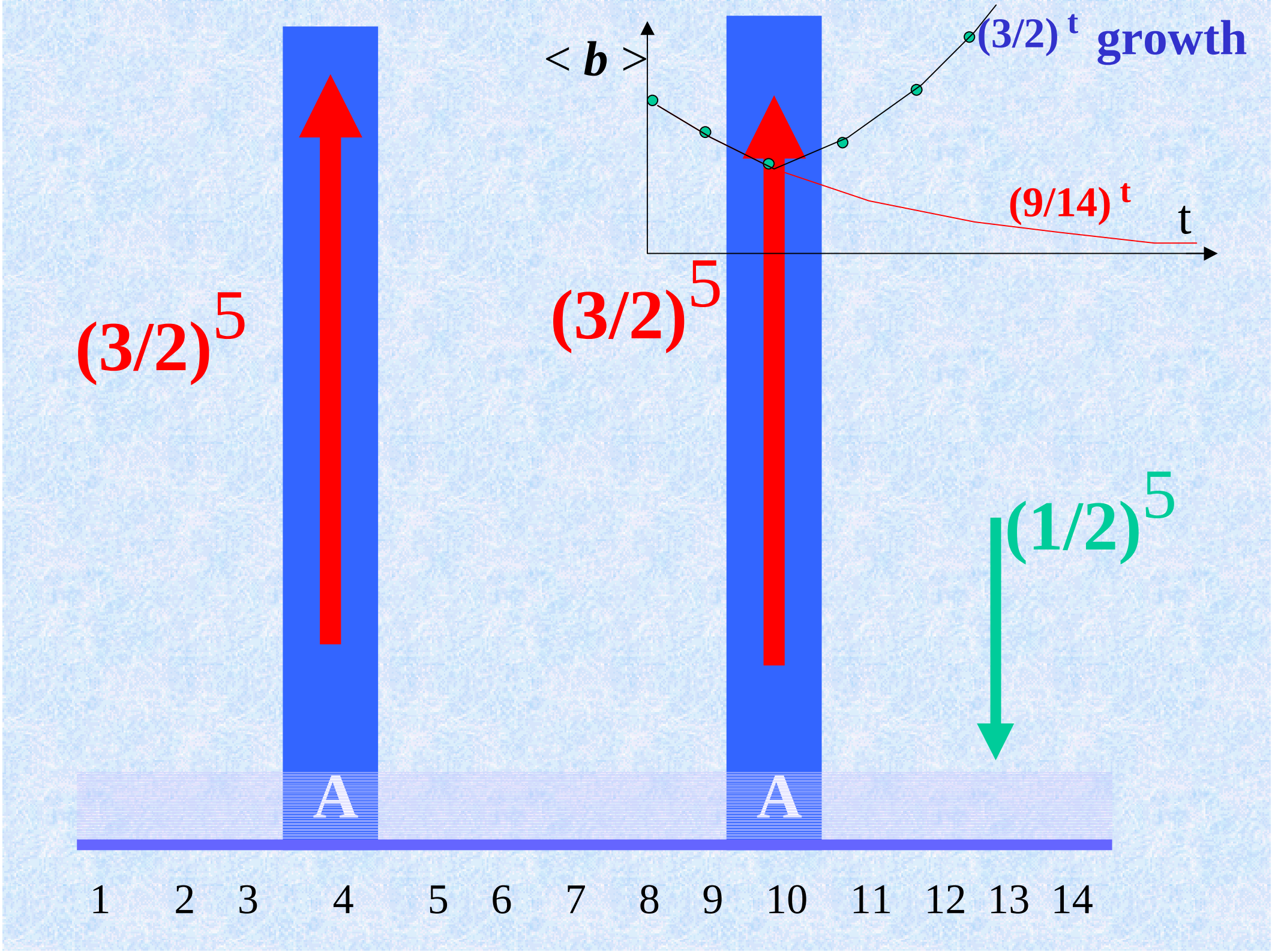



< b >
Initial exponential decay
(9/14) t
t
(3/2)
(3/2)
(1/2)
A
A
1 2 3 4 5 6 7 8 9 10 11 12 13 14


< b >
(3/2) t
(9/14) t
t
(3/2)2
(3/2)2
(1/2)2
A
A
1 2 3 4 5 6 7 8 9 10 11 12 13 14


< b >
(3/2) t growth
(9/14) t
t
(3/2)3
(3/2)3
(1/2)3
A
A
1 2 3 4 5 6 7 8 9 10 11 12 13 14


< b >
(3/2) t growth
(9/14) t
t
(3/2)4
(3/2)4
(1/2)4
A
A
1 2 3 4 5 6 7 8 9 10 11 12 13 14


< b >
(3/2) t growth
(9/14) t
t
(3/2)5
(3/2)5
(1/2)5
A
A
1 2 3 4 5 6 7 8 9 10 11 12 13 14

Diffusion could smear the effects of A discreteness

But it doesn't

one can prove rigorously

(RG flow, Branching Random Walks Theorems)

that:

- On a large enough 2 dimensional surface,

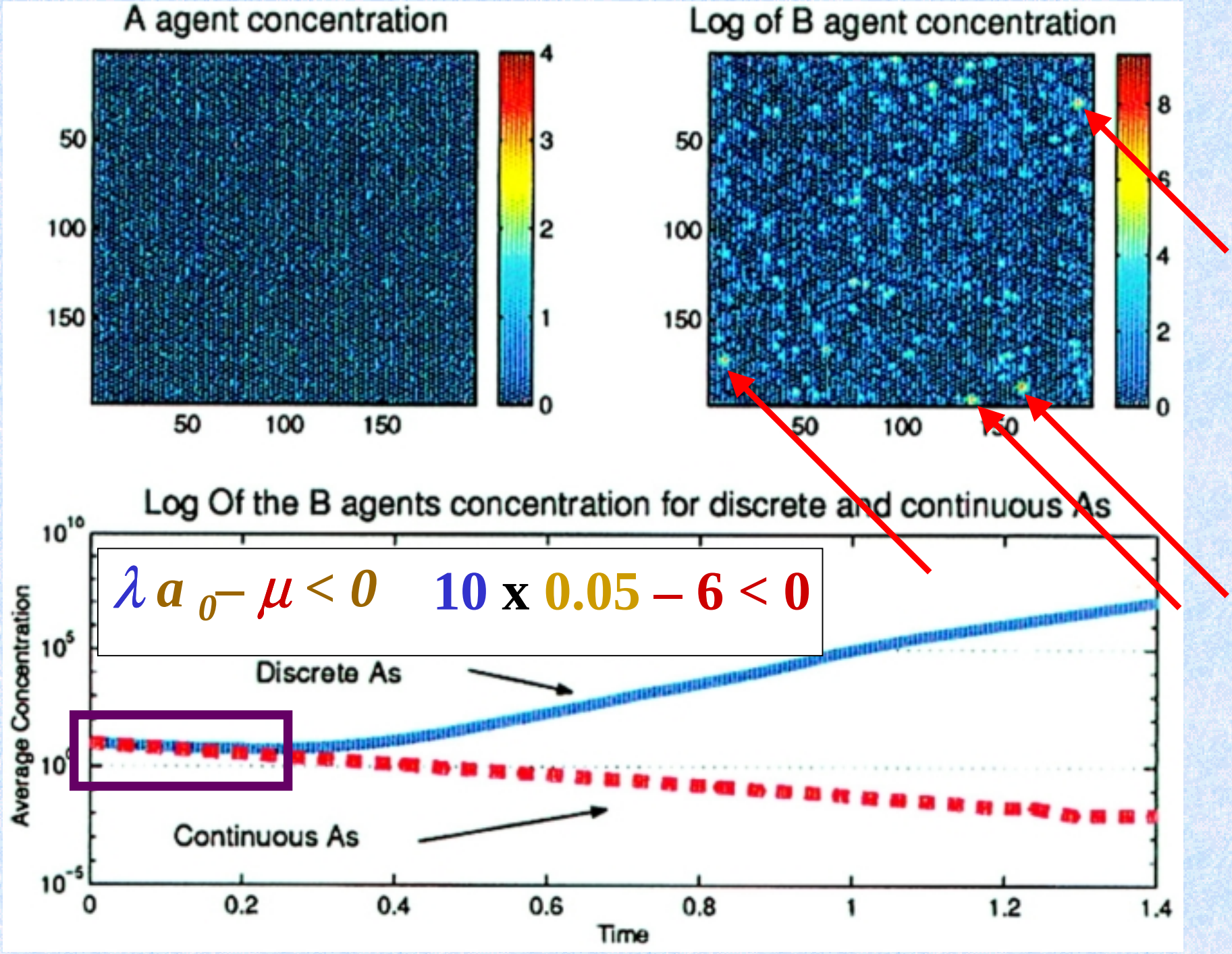
the B population *always grows!*

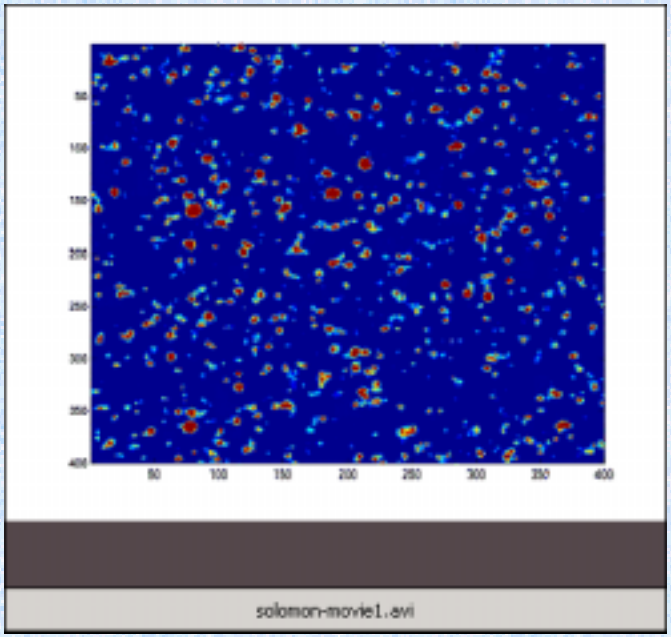
- In higher dimensions, $\lambda > D_a$

always suffices $\forall \mu, \mathbf{D}_b, < a(x,t) >!$

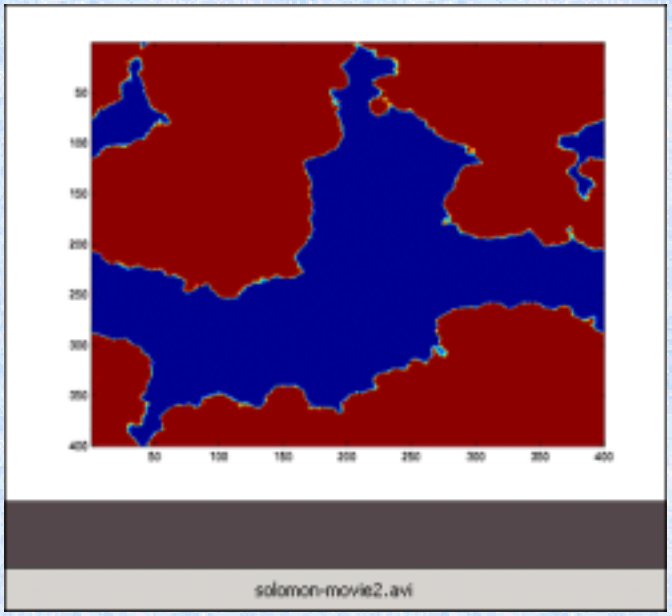
$D_a = 0.2$

$D_b = 10$

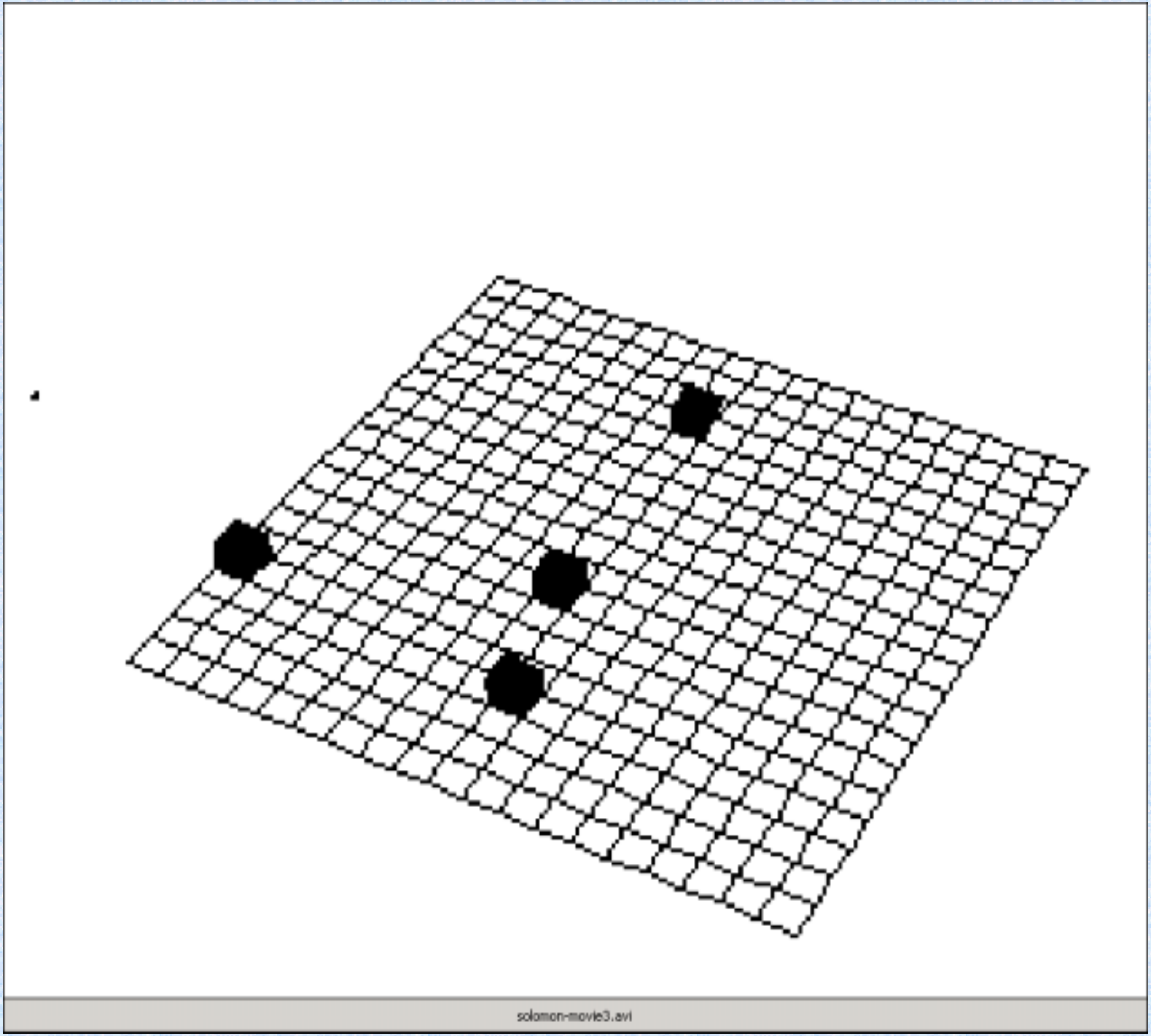

A agent concentration
Log of B agent concentration
Log Of the B agents concentration for discrete and continuous As
$\lambda a_0 - \mu < 0$
10 x 0.05 – 6 < 0
Discrete As
Continuous As
Average Concentration
Time



Angels and Mortals by my students
Eldad Bettelheim and Benny Lehmann



Angels and Mortals by my students
Eldad Bettelheim and Benny Lehmann


solomon-movie3.avi

The Role of DIFFUSION

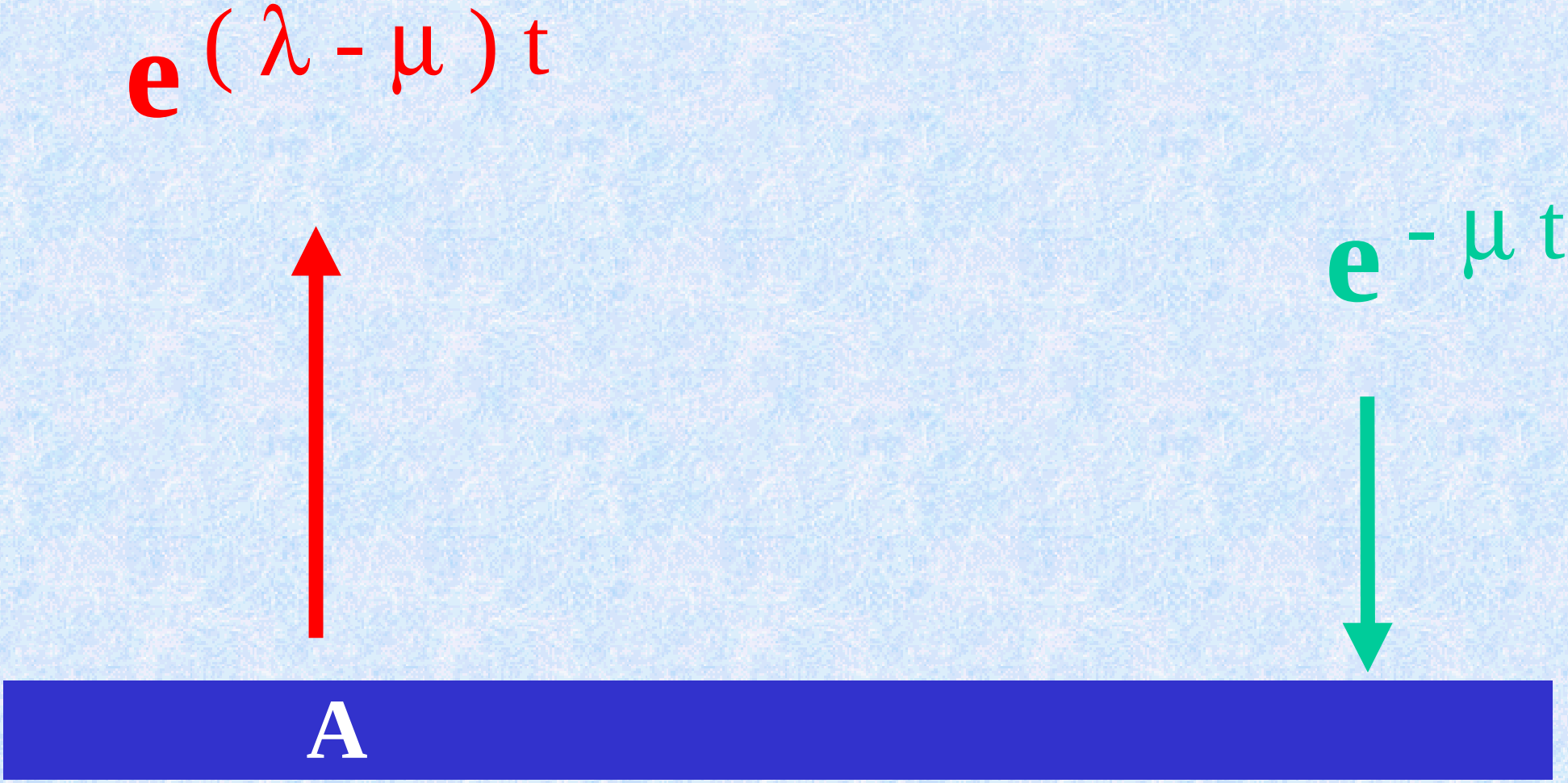
The Emergence of Adaptive B islands

Take just one A in all the lattice:

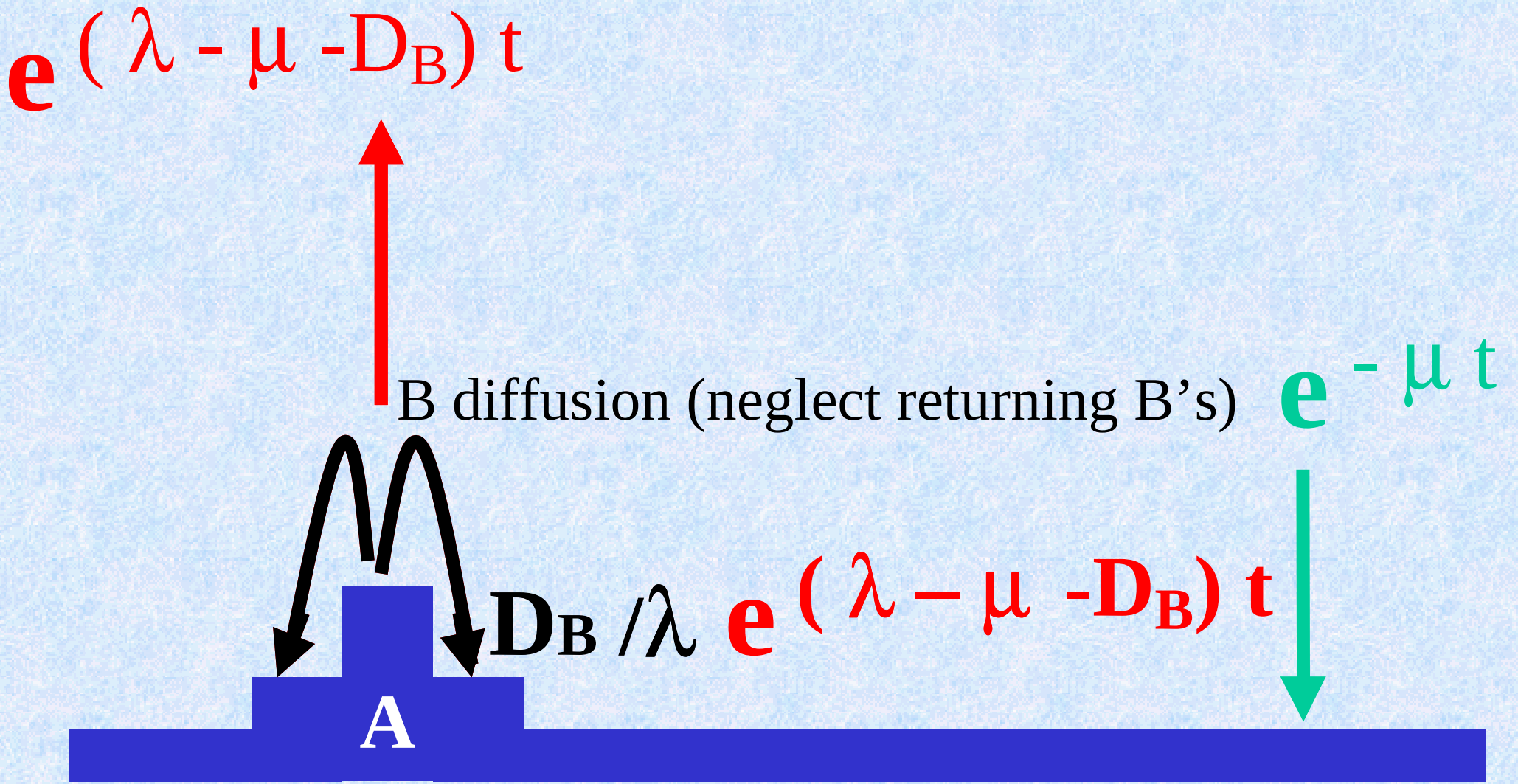
I.e. $a_0 = \langle a(x,t) \rangle = 1/Volume$

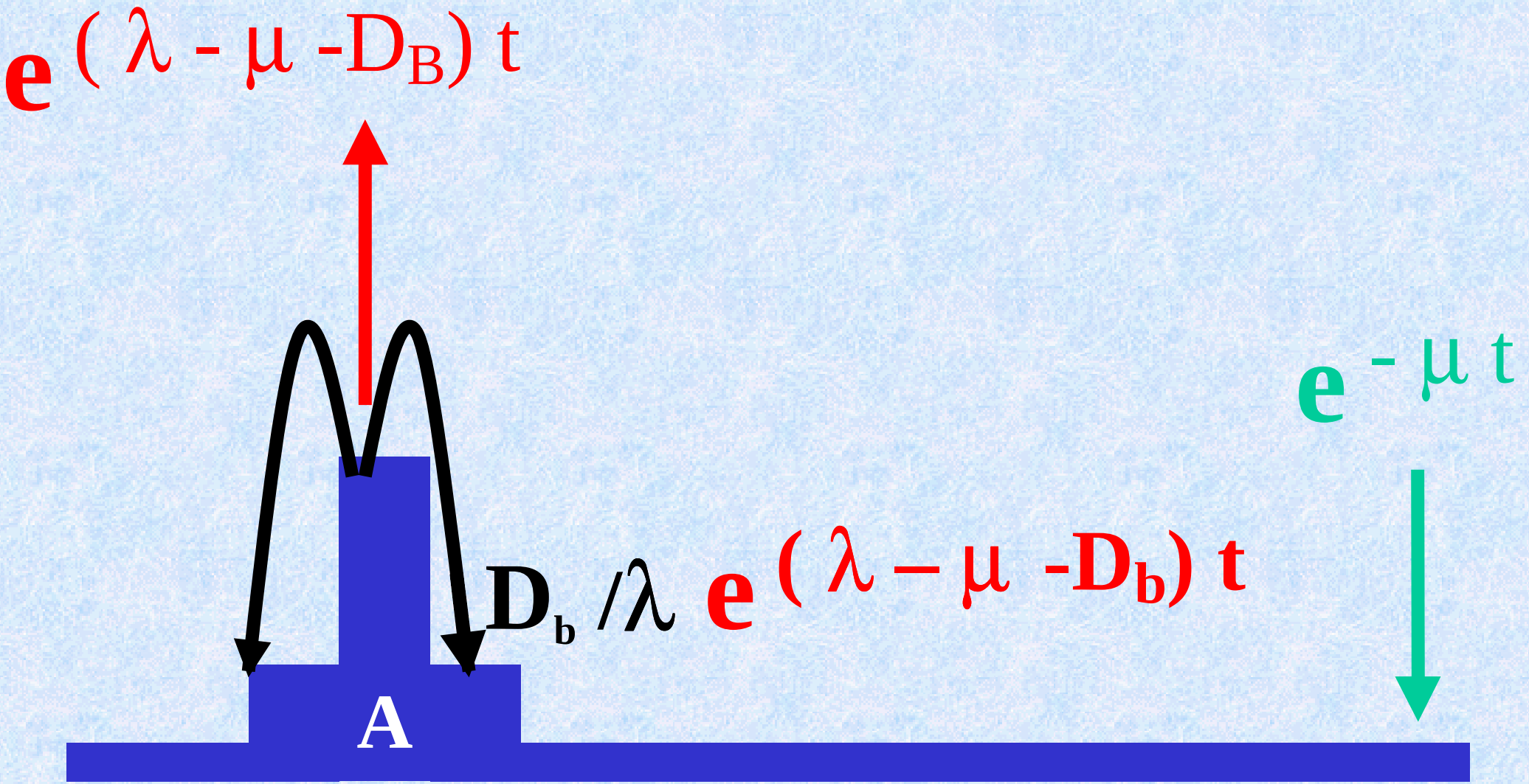
(on a 14 site 1-dim lattice $a_0 = 1/14$)

And $\lambda = 2\mu$, but $\lambda a_0 = \mu/7 << \mu$


$e^{(\lambda - \mu)t}$
$e^{-\mu t}$
A


$e^{(\lambda - \mu)t}$
$e^{-\mu t}$
A

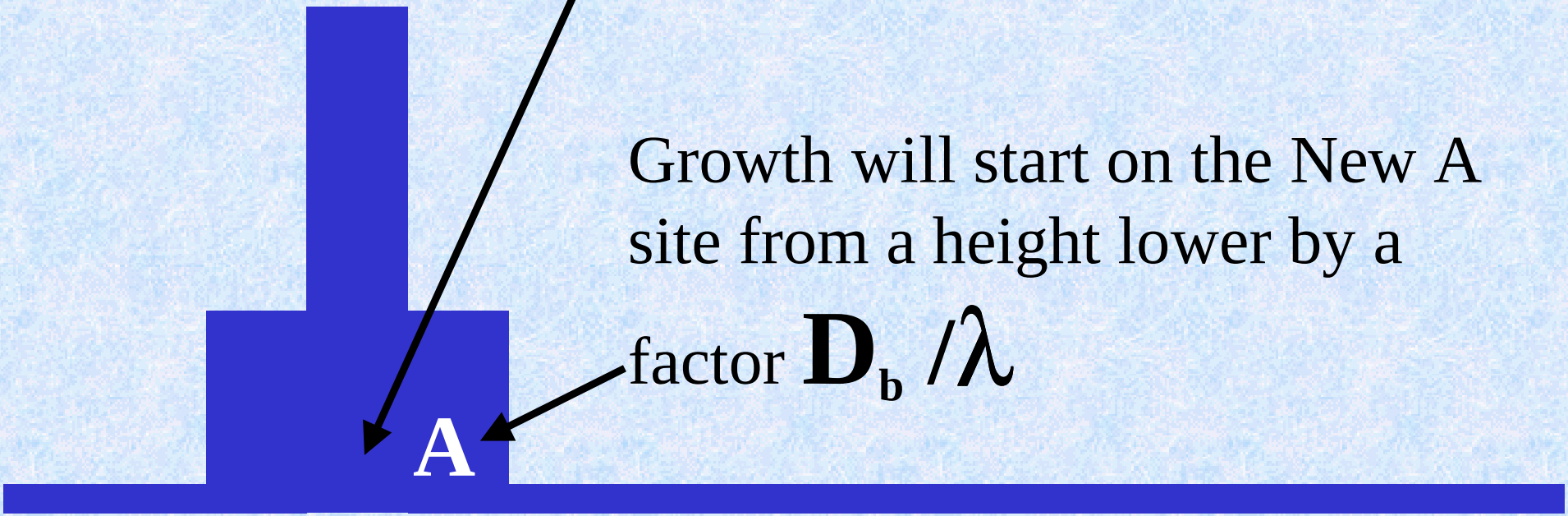

$e^{(\lambda - \mu - D_B)t}$
B diffusion (neglect returning B's)
$e^{-\mu t}$
D_B/λ
$e^{(\lambda - \mu - D_B)t}$
A


$e^{(\lambda - \mu - D_B)t}$
$D_b/\lambda\, e^{(\lambda - \mu - D_b)t}$
$e^{-\mu t}$
A

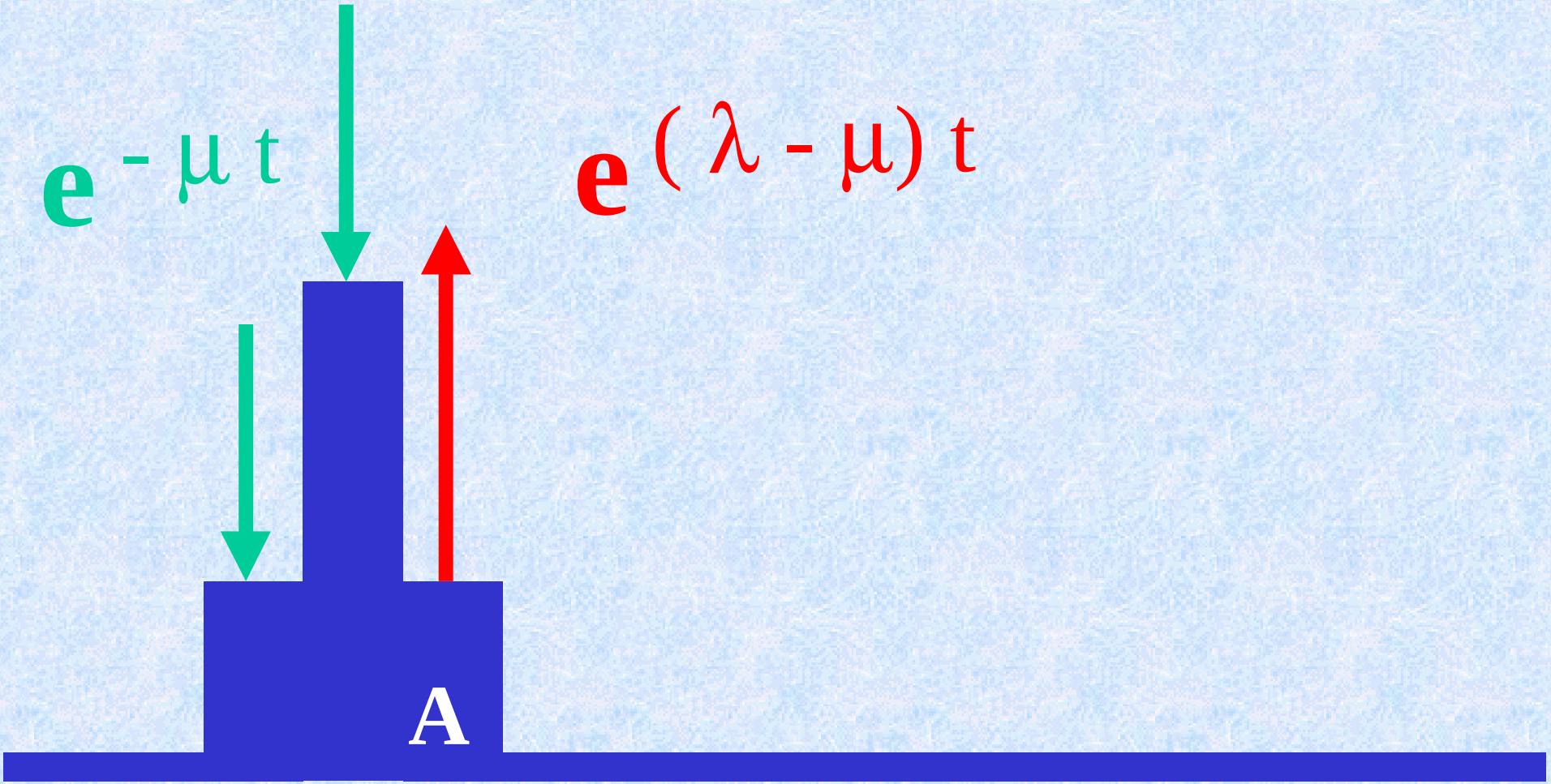
Growth stops when A jumps to a neighboring site

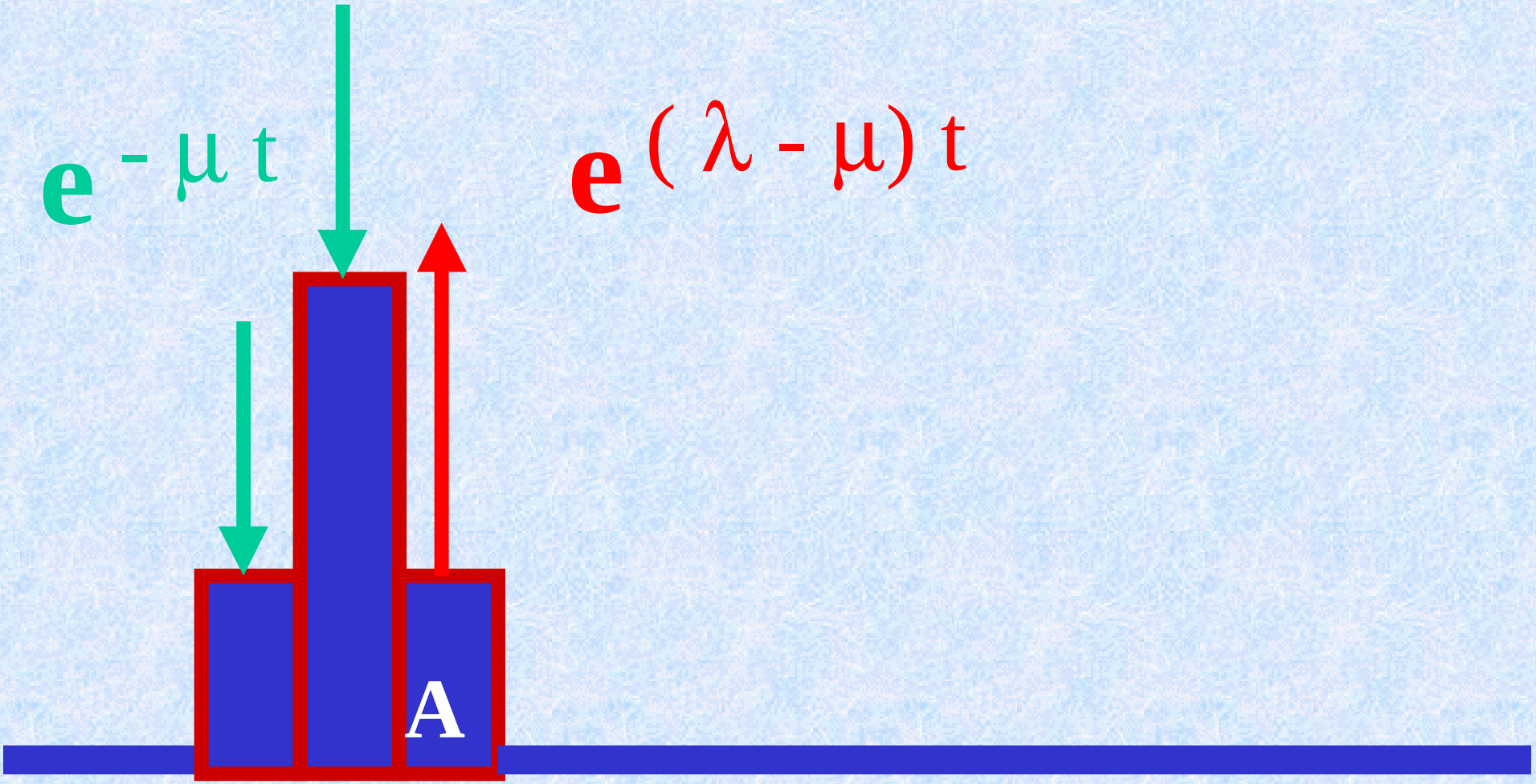

A A

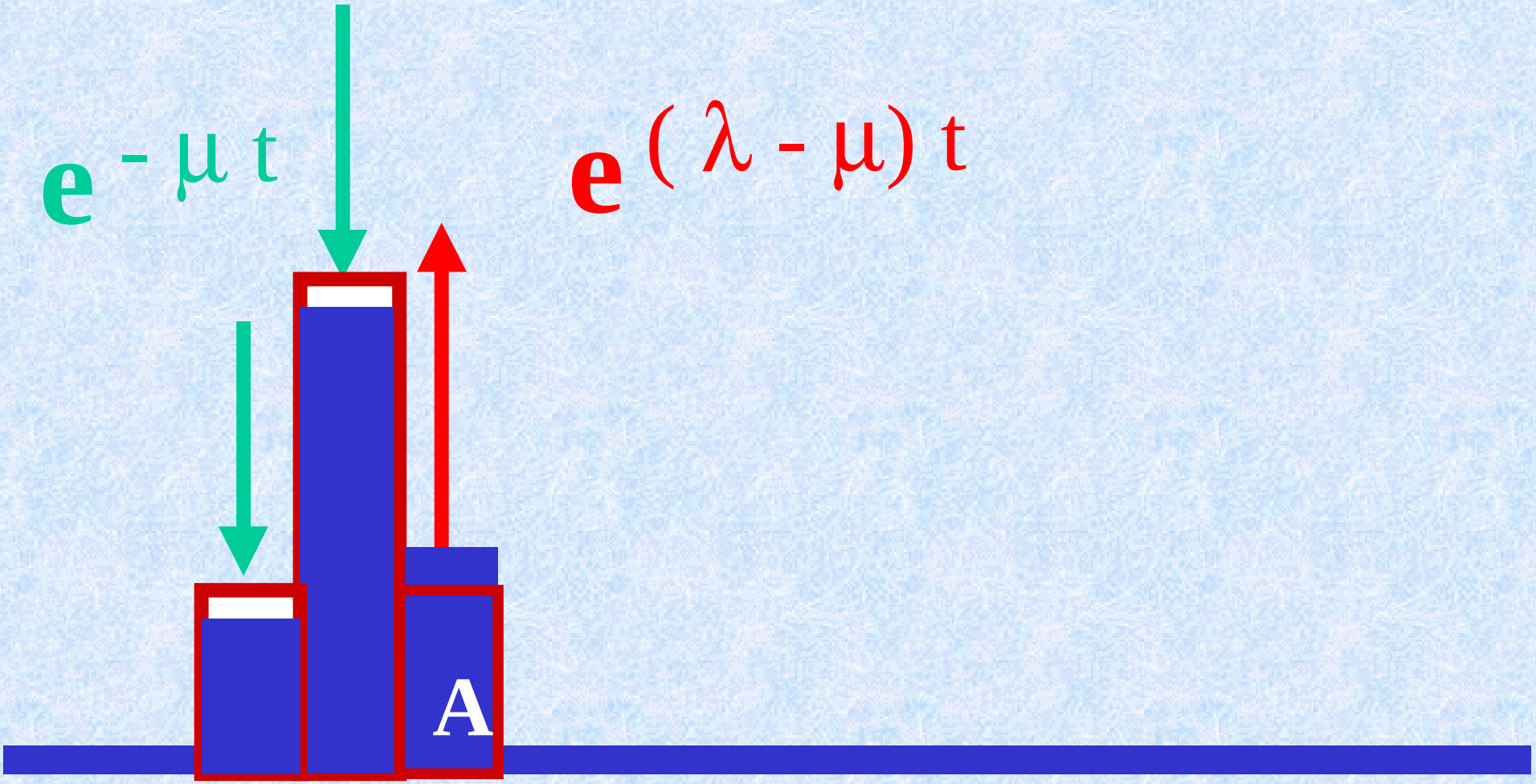
B population on the old A site will decrease

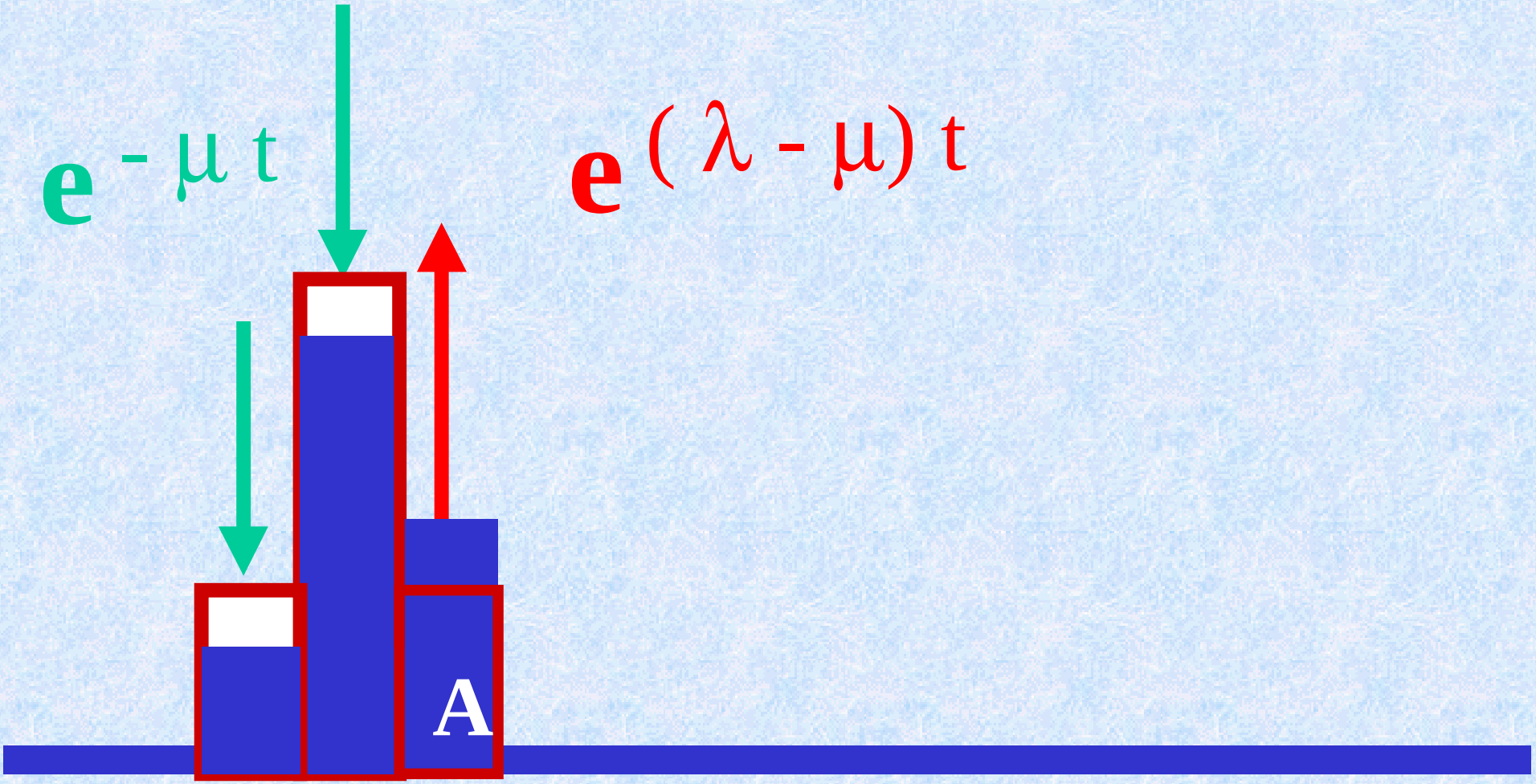


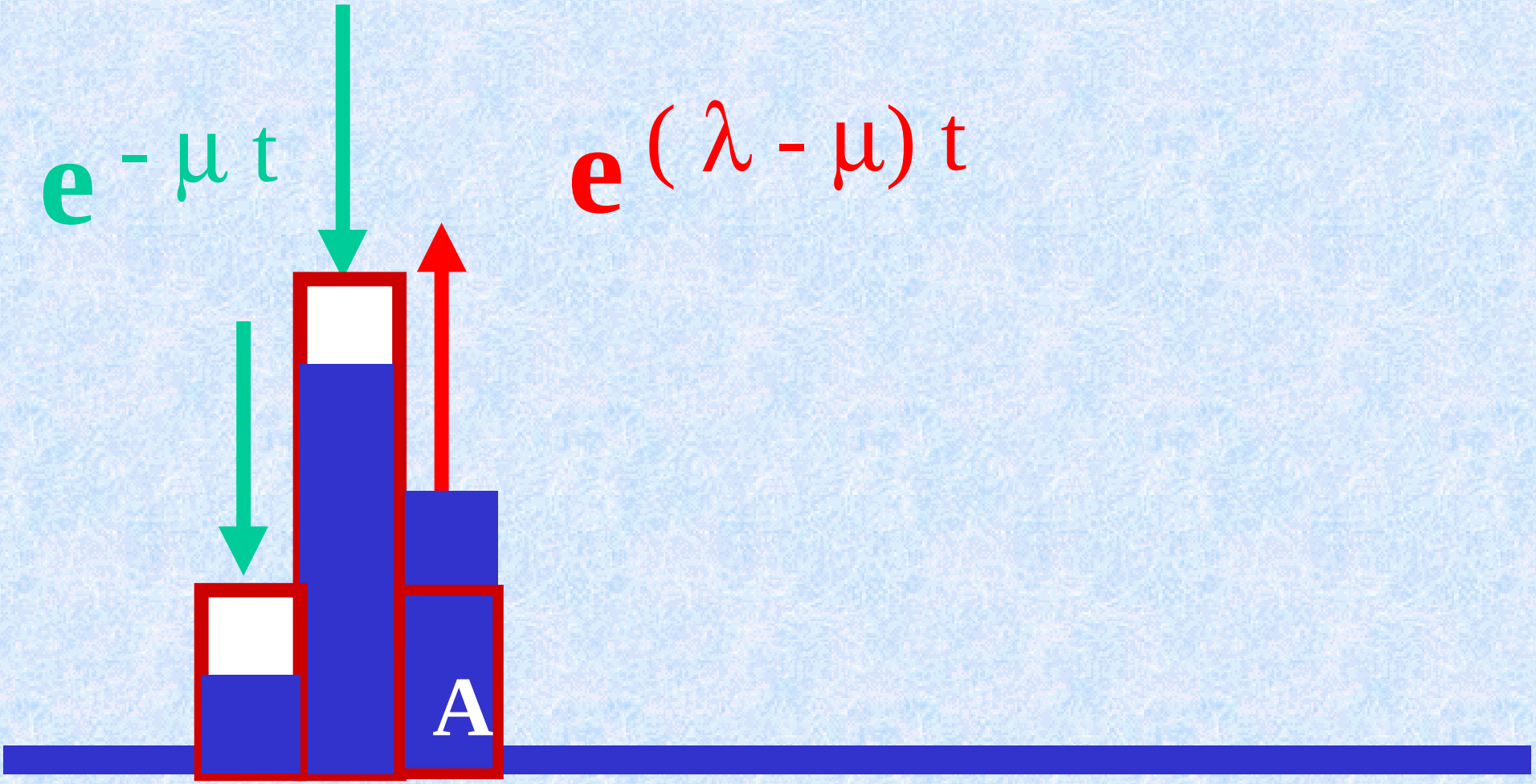
Growth will start on the New A site from a height lower by a factor $\mathbf{D_b}/\lambda$

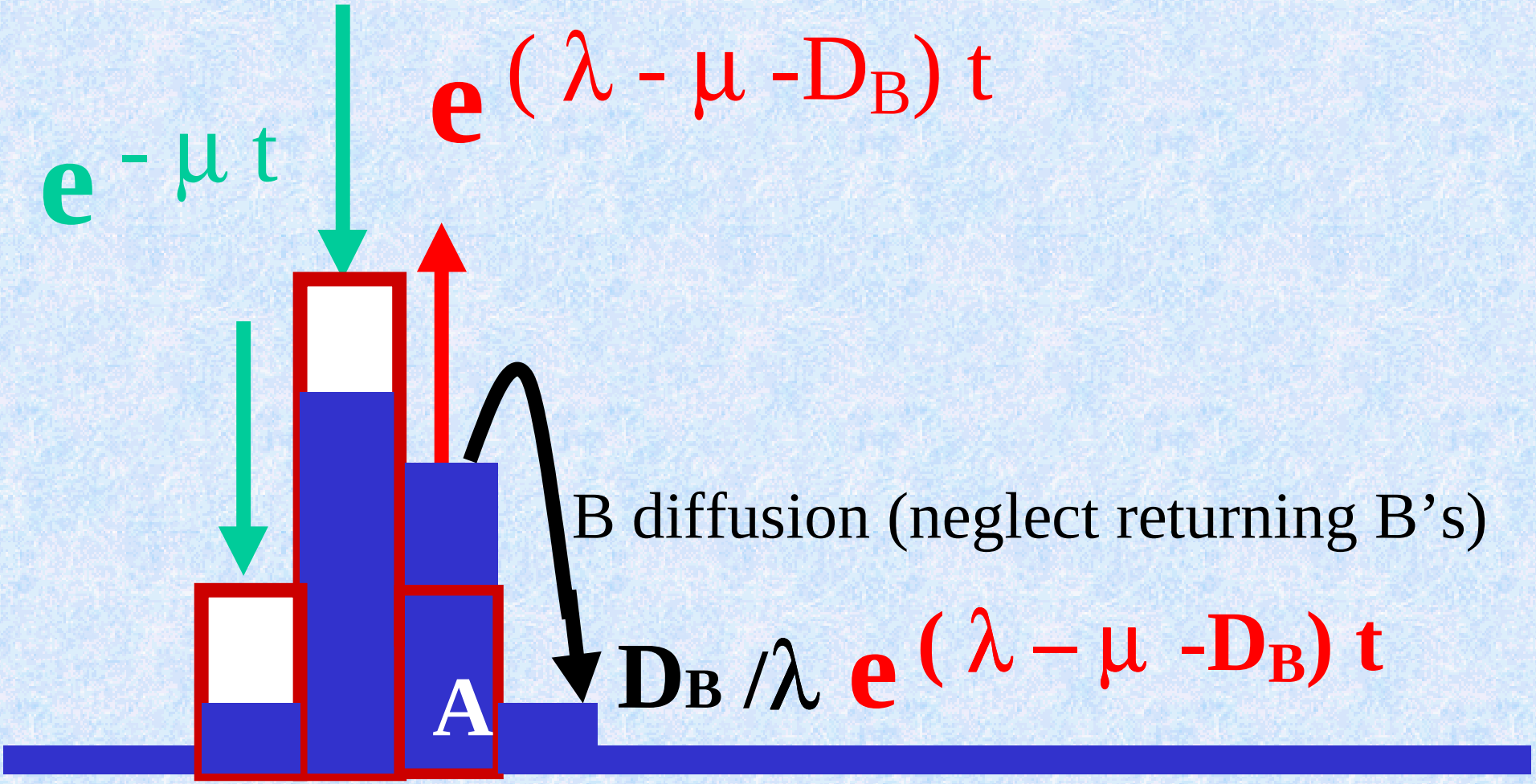

$e^{-\mu t}$
$e^{(\lambda-\mu)t}$
A

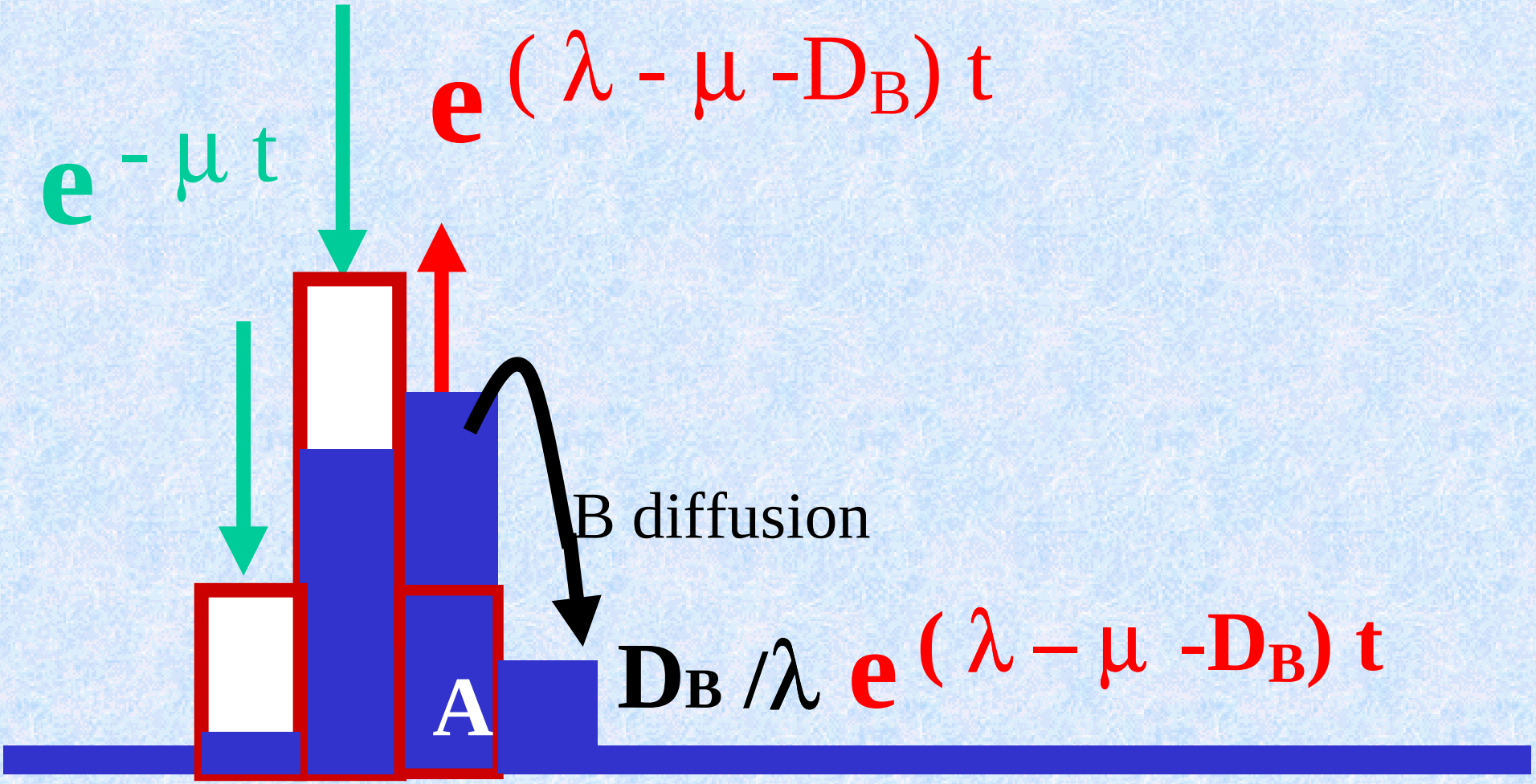

$e^{-\mu t}$
$e^{(\lambda-\mu)t}$
A


$e^{-\mu t}$
$e^{(\lambda-\mu)t}$
A

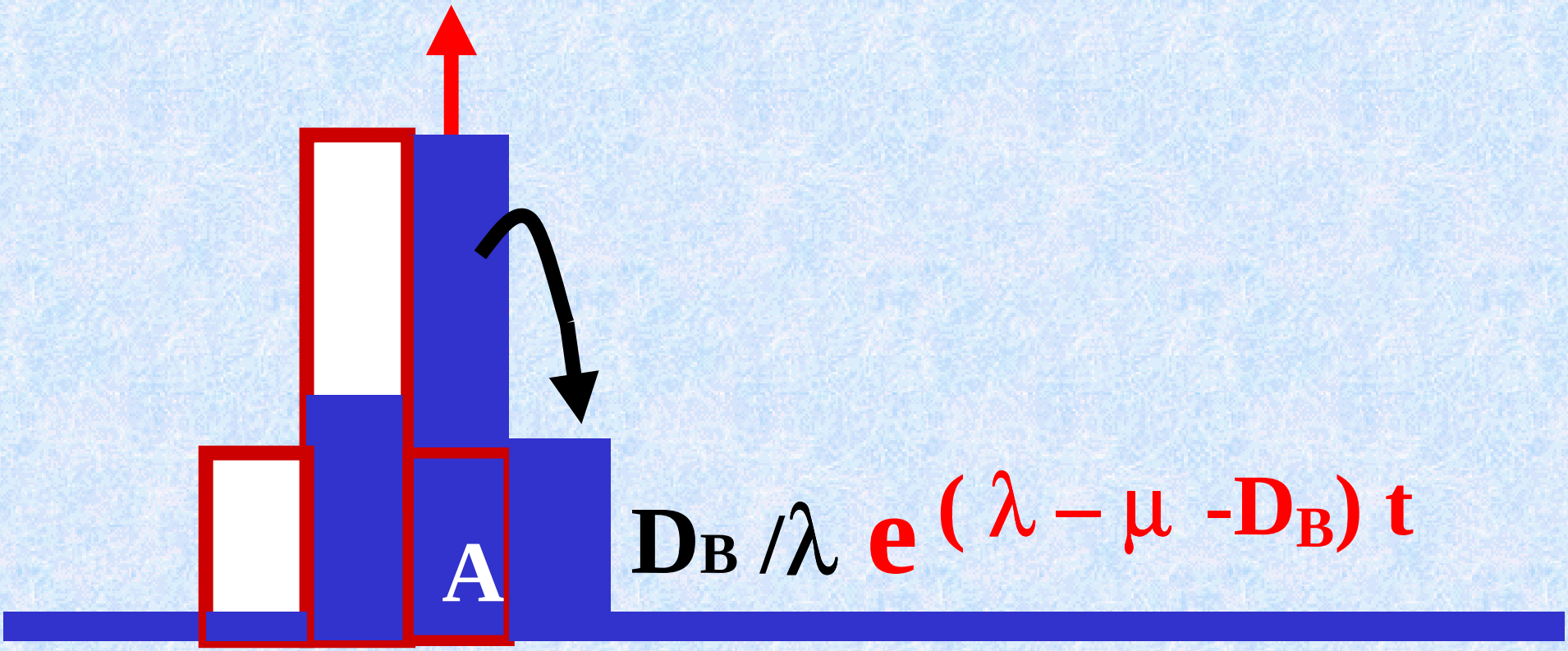

$e^{-\mu t}$
$e^{(\lambda-\mu)t}$
A


$e^{-\mu t}$
$e^{(\lambda-\mu)t}$
A


$e^{-\mu t}$
$e^{(\lambda - \mu - D_B) t}$
B diffusion (neglect returning B's)
$D_B/\lambda\ e^{(\lambda - \mu - D_B) t}$
A

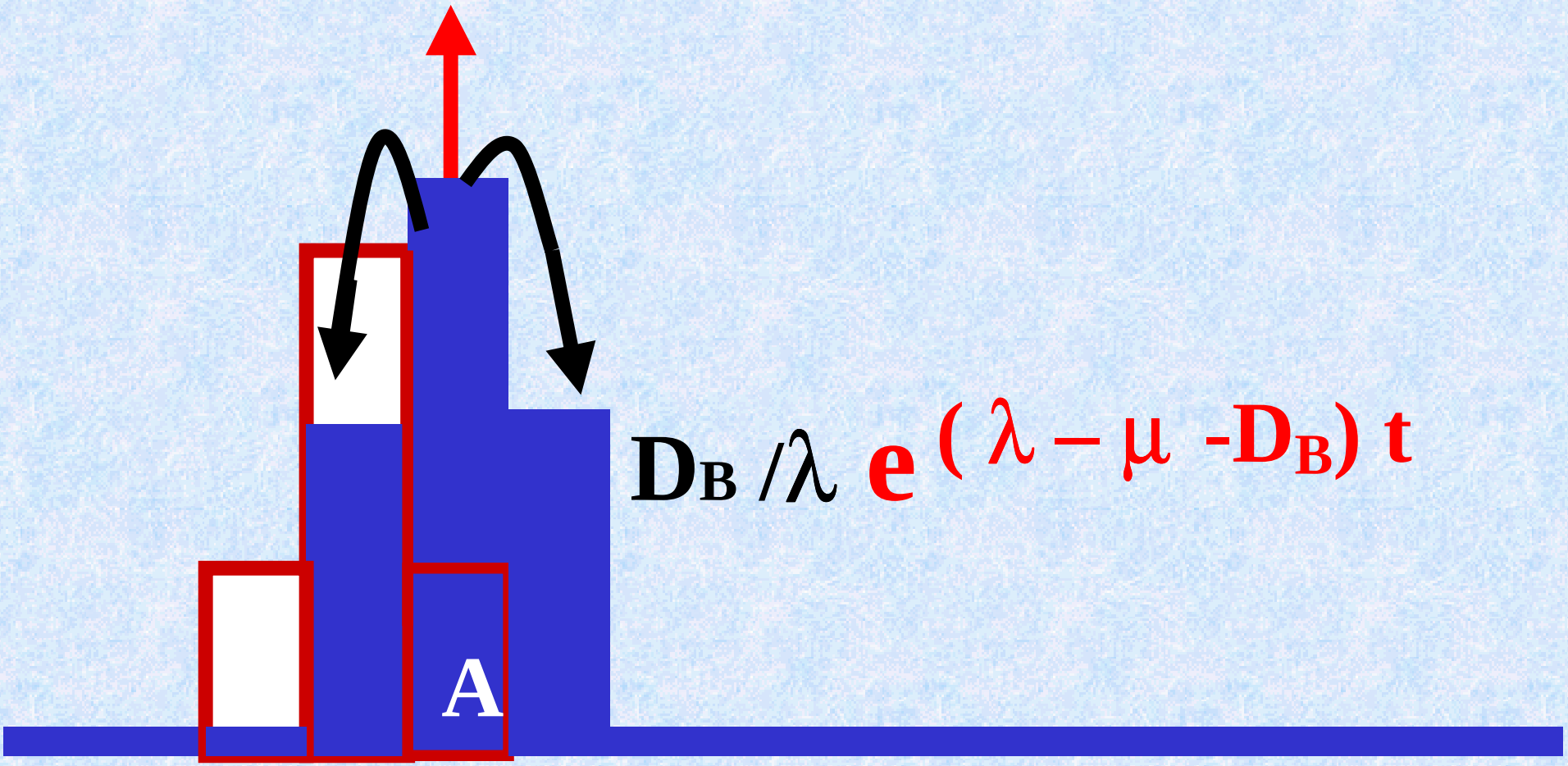

$e^{-\mu t}$
$e^{(\lambda - \mu - D_B) t}$
B diffusion
A
$D_B / \lambda \; e^{(\lambda - \mu - D_B) t}$

$$e^{(\lambda - \mu - D_B)\, t}$$



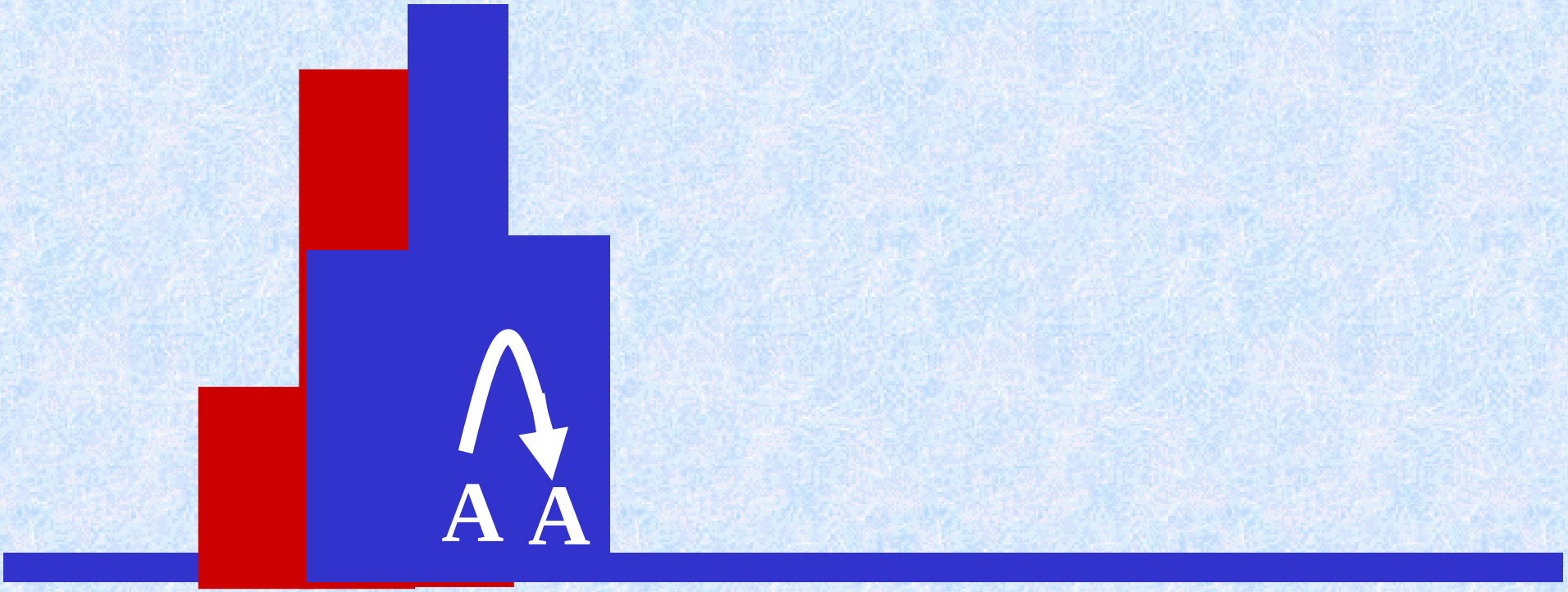
$$D_B / \lambda \; e^{(\lambda - \mu - D_B)\, t}$$

$$e^{(\lambda - \mu - D_B)\, t}$$



$$D_B / \lambda \; e^{(\lambda - \mu - D_B)\, t}$$

Growth stops again when A jumps again
(typically after each time interval $1/D_A$)



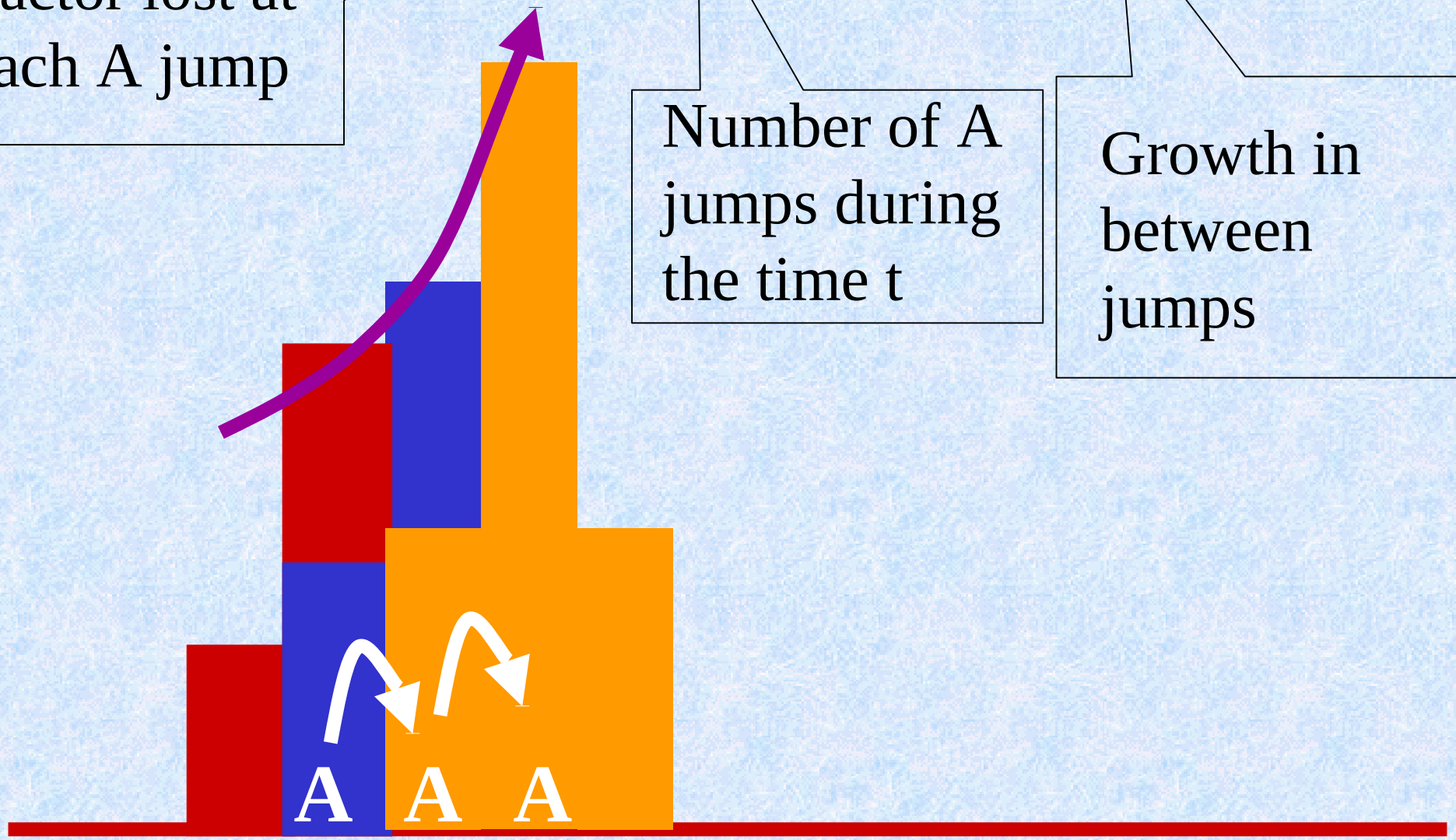

A A A

$$(D_B/\lambda)^{D_A t} e^{(\lambda - \mu - D_B) t}$$

Factor lost at each A jump

Number of A jumps during the time t

Growth in between jumps



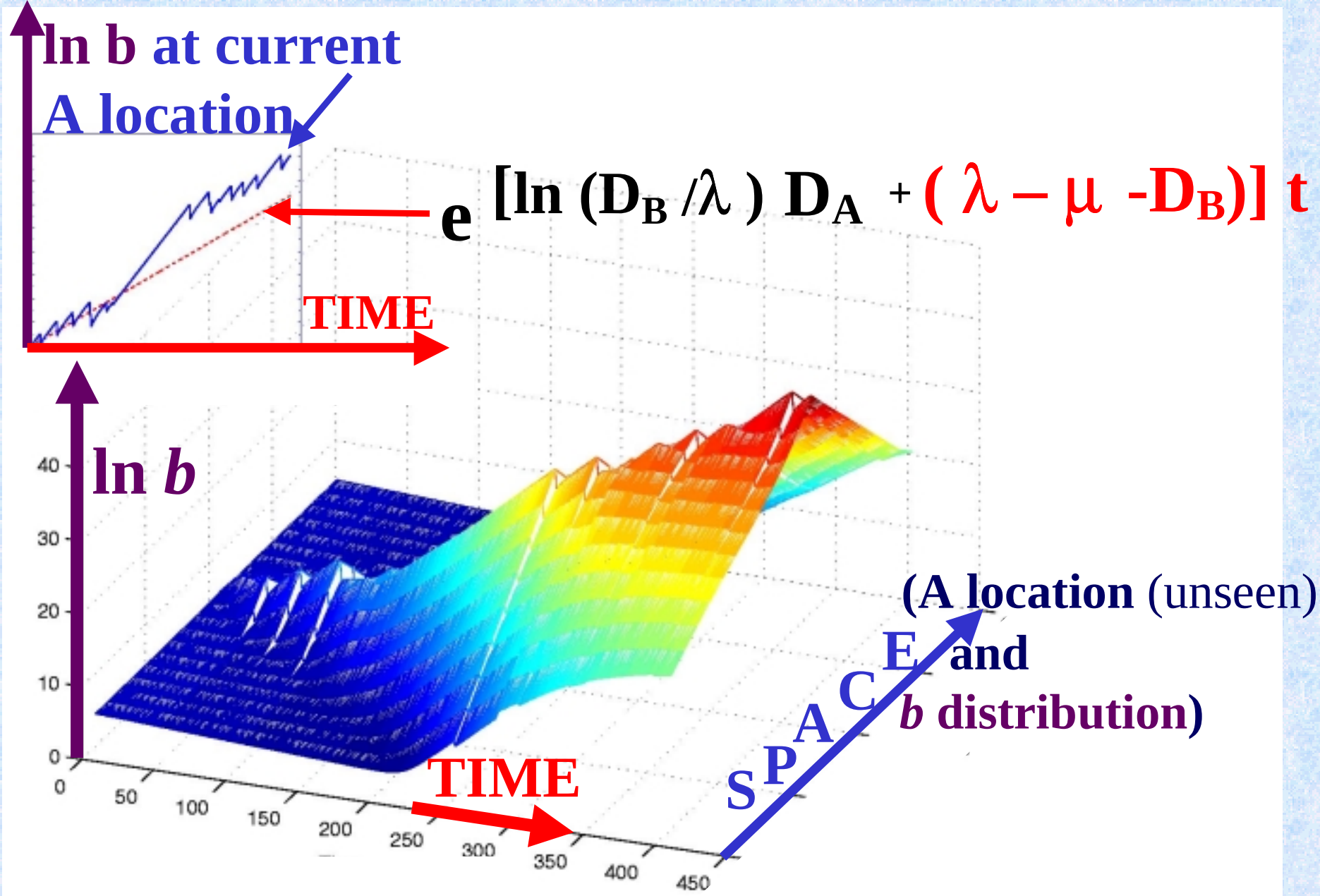


Figure 3: Single A aproximation. The profile of a B island as a function of time as it follows the random motion of an A agent. The cross-section of the island is taken through the current location of the A agent. The

Before more theoretical study,
let us list applications/ interpretations

Collaborations that identified and studied systems

in **biology, finance** and **social sciences** that

- are **naively non-viable** (decay to extinction) when viewed macroscopically
- but **perfectly viable in reality**
- (and when simulated / analyzed correctly at the microscopic individual level).

In particular, most of the **species** in nature could be in this regime: **---**

- negative naive average growth rate but
- actual survival and proliferation.

Ordinary miracles Michael Brooks **New Scientist** magazine, May 2000:

<< Jeff Kirkwood, a population dynamics researcher
at Imperial College, London, says

this close look is particularly valuable when predicting population growth in a diverse environment.

"If you looked **'on average'**, the conditions are just hopeless and **no one has any right to survive**," he says.

But if there are patches where it is possible to survive,>>

Interpretations in Various Fields:

Origins of Life:

- individuals =**chemical molecules**,
- spatial patches = first self-sustaining proto-cells.

Speciation:

- **Sites: various genomic configurations.**
- B= individuals; **Jumps of B= mutations**.
- **A= advantaged niches** (evolving fitness landscape).
- emergent adaptive patches= **species**

Immune system:

- **B cells; A antigen**

B cells that meet antigen with complementary shape **multiply.**
(later in detail the AIDS analysis)

Finance:- sites: investment instruments

- B = capital units, A= profit opportunities.

Newton (after loosing 20 K Pounds in stock market)

"I can calculate the motions of heavenly bodies, but not the madness of people."

Financial markets **don't need wise/ intelligent investors** to work:

Capital can
survive and even proliferate
simply by
being autocatalytic

Adam Smith's invisible hand…

doesn't even need investor's self-interest

What if λ, μ, etc are arbitrary ?

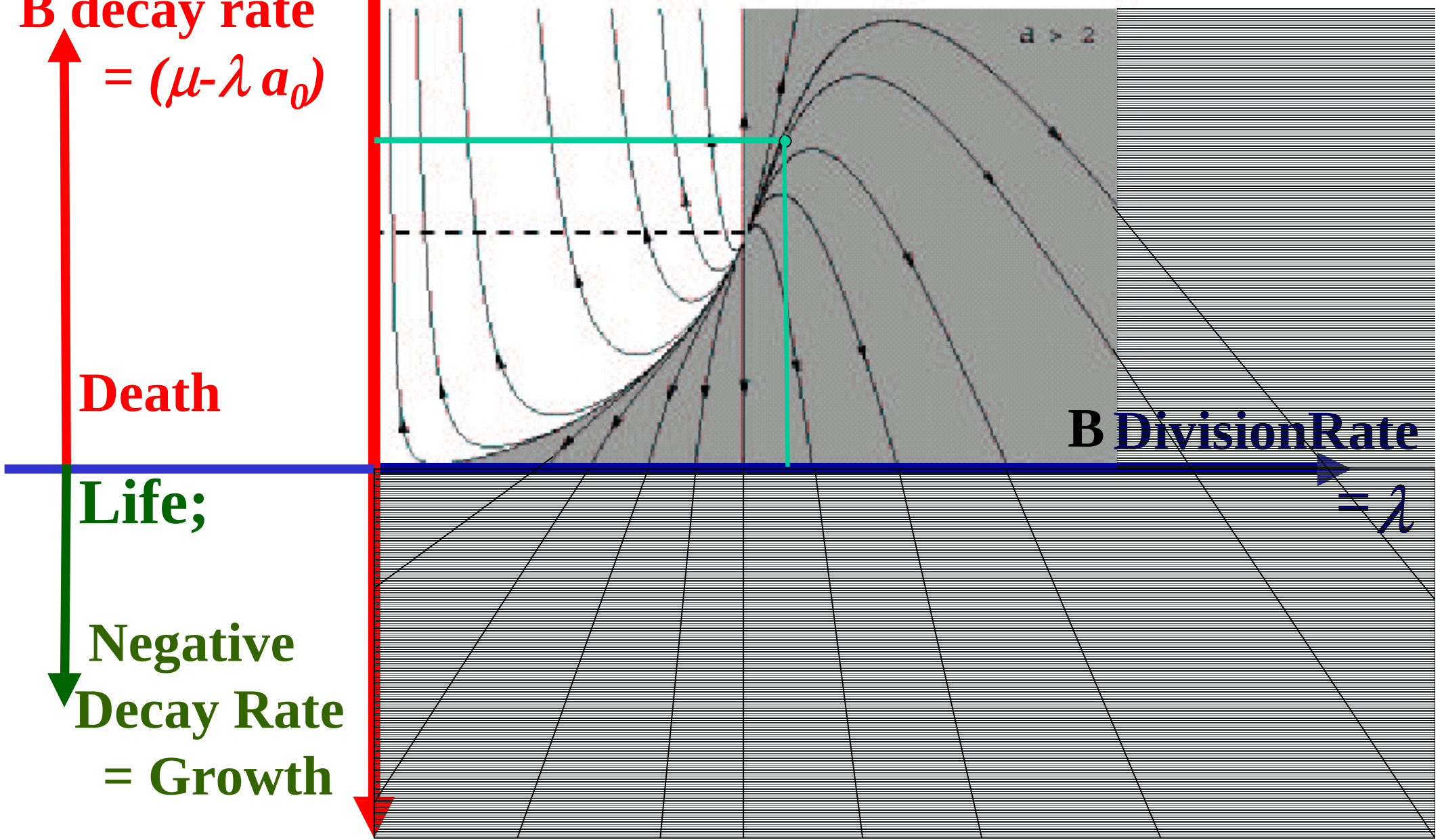
Expressing the AB system formally as a
Statistical Field Theory model and applying
Renormalization Group Analysis

to obtain its phases.

For more details see: Reaction-Diffusion Systems with Discrete Reactants ,
Eldad Bettelheim , MSc Thesis, Hebrew University of Jerusalem 2001
http://racah.fiz.huji.ac.il/~eldadb/masters/masters2.html

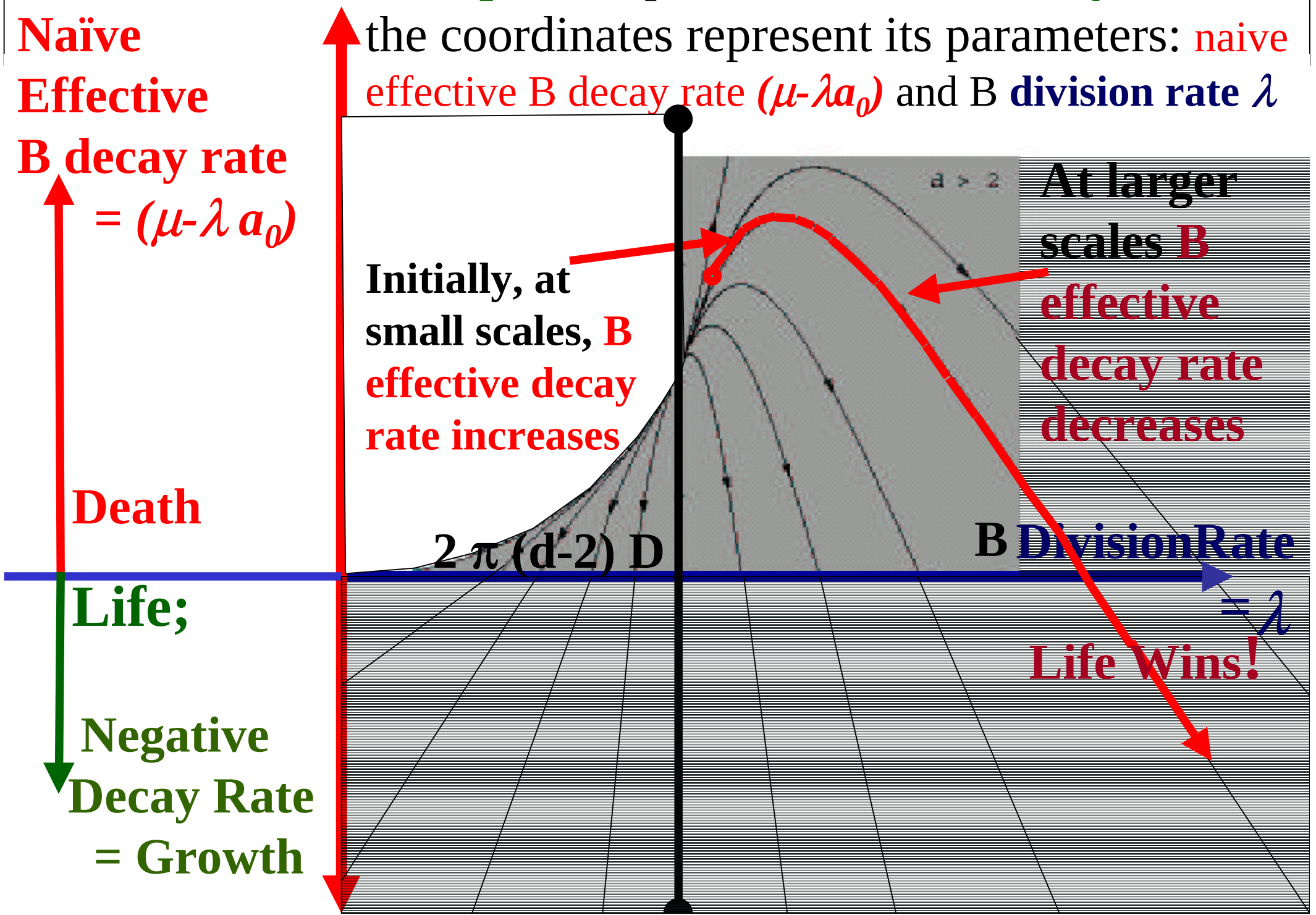
Each **point** represents another **AB system:**
the coordinates represent its parameters: naive effective B decay rate $(\mu-\lambda a_0)$ and B **division rate** λ

Positive
Naïve
Effective
B decay rate
$= (\mu-\lambda\, a_0)$



Each **point** represents another **AB system:**
the coordinates represent its parameters: naive effective B decay rate $(\mu-\lambda a_0)$ and B **division rate** λ

Positive Naïve Effective B decay rate $= (\mu-\lambda\, a_0)$



Ordinary miracles Michael Brooks, **New Scientist** magazine, May 2000

<< According to John Beringer, an expert on microbial biology at the University of Bristol:

"**Microbes** that need oxygen will be found close to the **surface** of soil, and microbes that are very fastidious about oxygen concentration will be found in **bands** at the appropriate oxygen concentration."

Microbes concentrating on a **two-dimensional** resource may have been **more successful** than their cousins who tried exploiting a three-dimensional feast.>>

Finite cut-off

What if A and B have finite size?
(I.e. finite momentum cut-off)

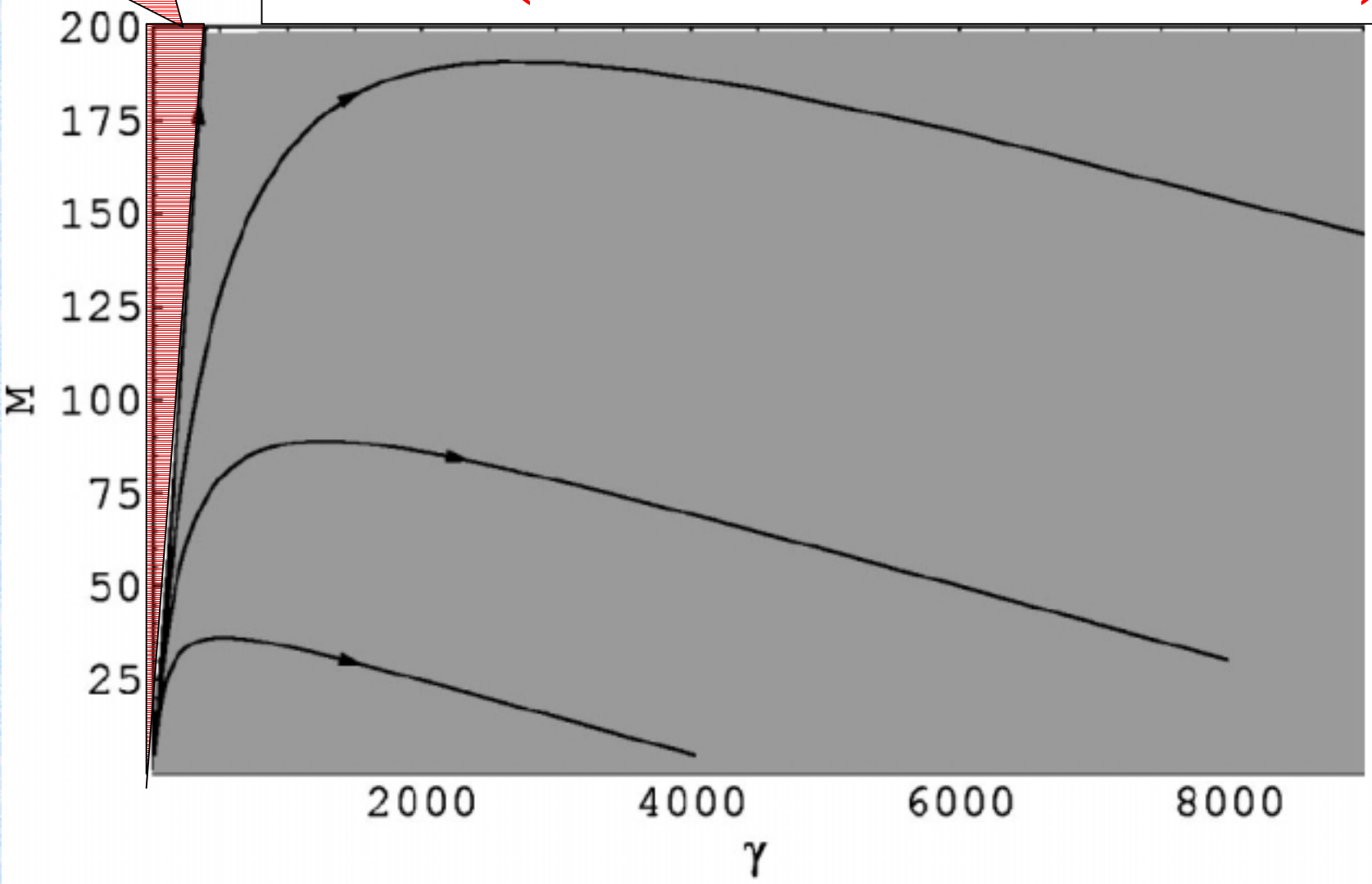


FIG. 6. Renormalization flows for $d=2$ at finite lattice spacing. Unlike Fig. 4, here there is a region in parameter space (unshaded) where the extinction phase is stable.

Pólya 's Random Walk Constant

What is the probability $P_d(\infty)$ that eventually an A returns to its site of origin?

Pólya : $P_1(\infty) = P_2(\infty) = 1$

but for d>2 $P_d(\infty) < 1$; $P_3(\infty) = 0.3405373$

Kesten and Sidoravicius studied the AB model using it (preprint 75 p):

On large enough 2 dimensional surfaces $\forall \lambda, \mu, D_b, D_a, a_0$

-Total/average B population always grows.

Study the effect of one A on *b(x,t)* on its site of origin *x*

Ignore for the moment the death and emigration and other A's

Probability of one **A return** by time ***t*** (d-dimesional grid): $P_d(t)$

Typical duration of an **A visit**: $1/D_a$

Average increase of ***b(x,t)*** per **A visit**: e^{λ / D_a}

Expected increase in ***b(x,t)*** due to 1 return events: $e^{\lambda / D_a} P_d(t)$

Study the effect of one A on $b(x,t)$ on its site of origin x

Ignore for the moment the death and emigration and other A's

Probability of one **A return** by time t (d-dimesional grid): $P_d(t)$

Typical duration of an **A visit**: $1/D_a$

Average increase of $b(x,t)$ per **A visit**: e^{λ/D_a}

Expected increase in $b(x,t)$ due to 1 return events: $e^{\lambda/D_a} P_d(t)$

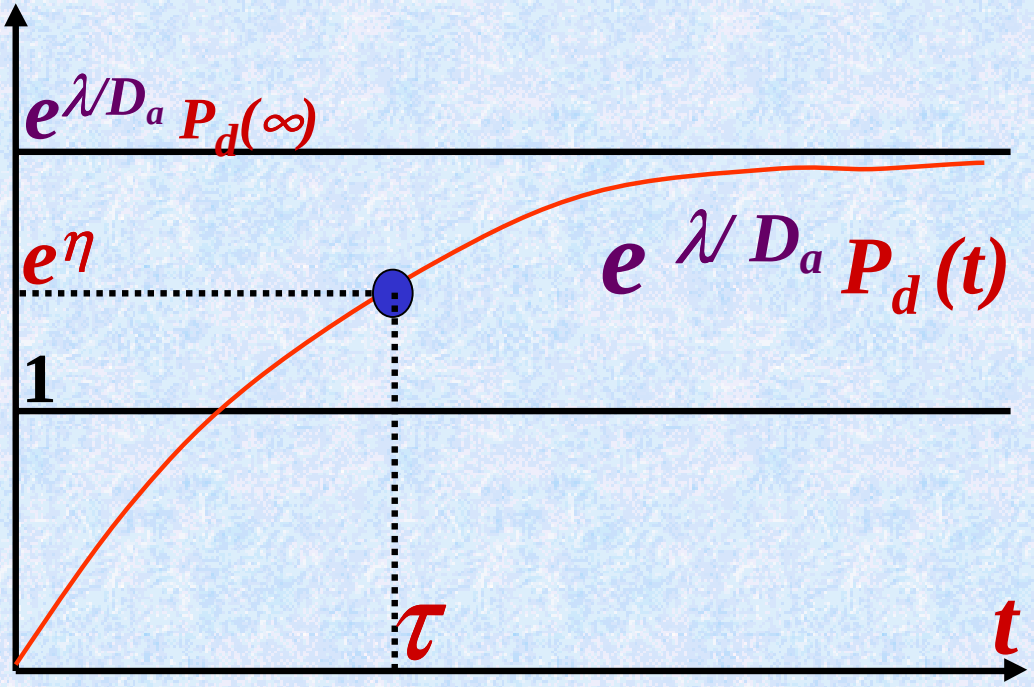
But for d= 2 $P_d(\infty) = 1$

so $e^{\lambda/D_a} P_d(\infty) > 1$

$\eta \sim \lambda/2D_a > 0$

$\tau \sim e^{D_a/2\lambda} < \infty$

finite positive growth in finite time !



{Probability of **n returns** before time **t = n** τ} > $P^{n}_{d}(\tau)$

Growth induced by such an event: $e^{n\lambda/D_a}$

Expected factor to ***b(x,t)*** due to n return events:

> $[e^{\lambda/D_a} P_d(\tau)]^n = e^{n\eta} = e^{\eta t/\tau}$ exponential time growth!

Taking in account death rate μ,

emigration rate D_b and that

there are ***a(x,0)*** such **A**'s:

$< b(x,t) > > b(x,0)\, e^{-(\mu + D_b)t}\, e^{a(x,0)\eta t/\tau}$

-increase is expected at all ***x's*** where: $a(x,0) > (\mu + D_b)\tau/\eta$

There is a finite density of such ***a(x,0)***'s => $<b(x,t)> \rightarrow \infty$

Losing All Battles and Wining the War

HIV time hierarchy:

U Hershberg, Y Louzoun, H Atlan and S Solomon

Physica A: 289 (1-2) (2001) pp.178-190 ;

http://xxx.lanl.gov/abs/nlin.AO/0006023

A = antigens (virons)

B = cells of the immune system

i = index of the particular characteristic shape of virus/immune cell

$A_i \rightarrow A_i + A_i$ **Virons multiply**

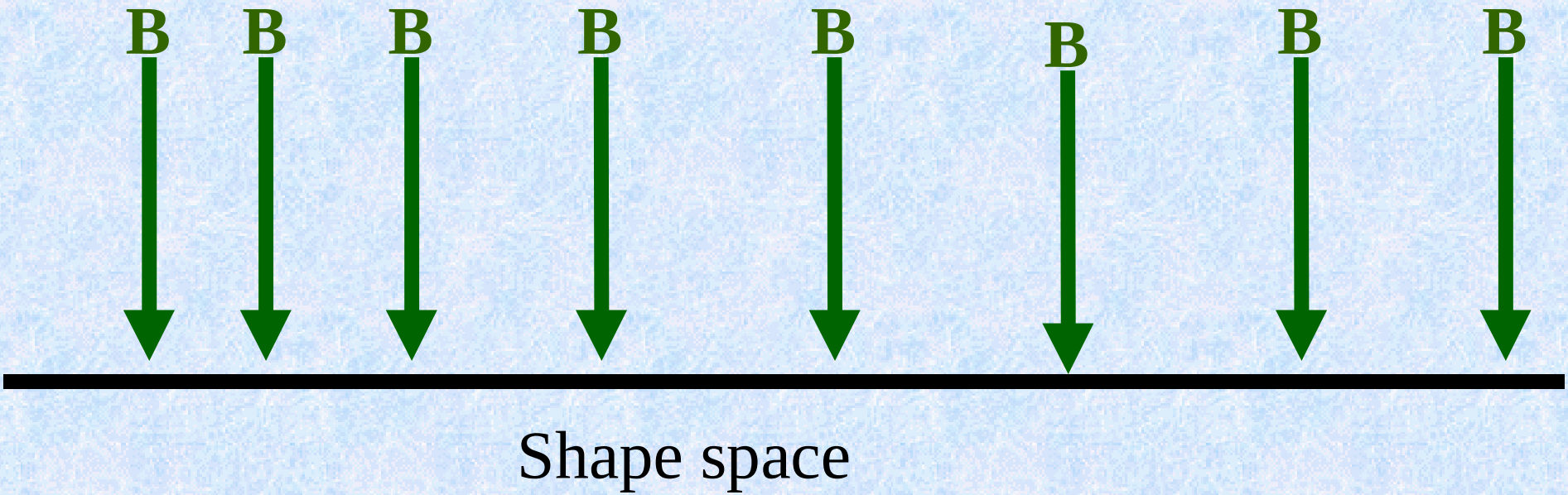
$A_i + B_i \rightarrow A_i + B_i + B_i$ **Immune cells multiply when they meet virons with complementary shape to theirs**

$B_i + A_i \rightarrow B_i$ **Virons are destroyed when detected by immune cells of complementary shape**

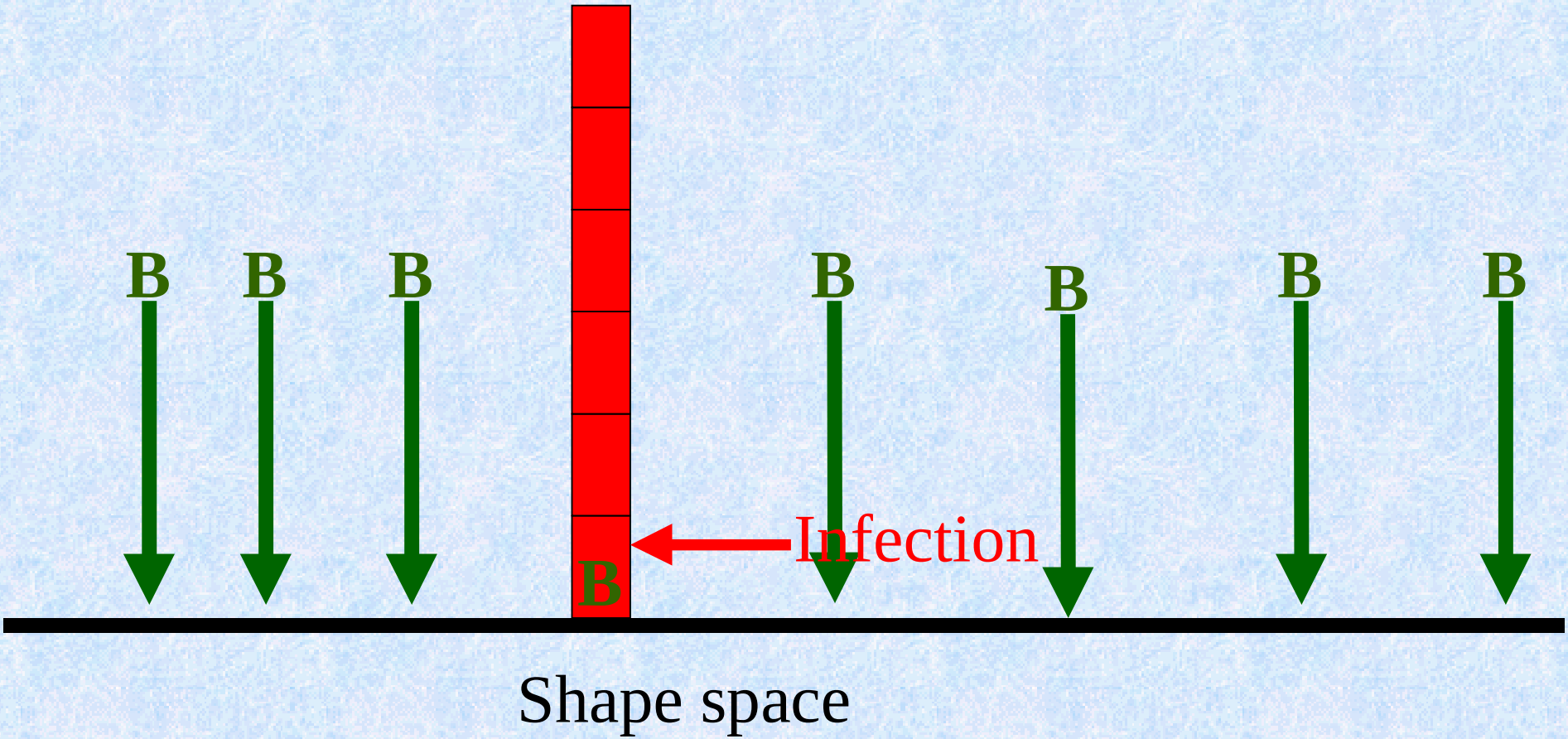
$A_i \rightarrow A_{i \pm 1}$ **Virons can mutate (actually in a n-dim space)**

$A_i + B_* \rightarrow A_i$ **Immune cells of any type are destroyed when infected by viruses of any type**

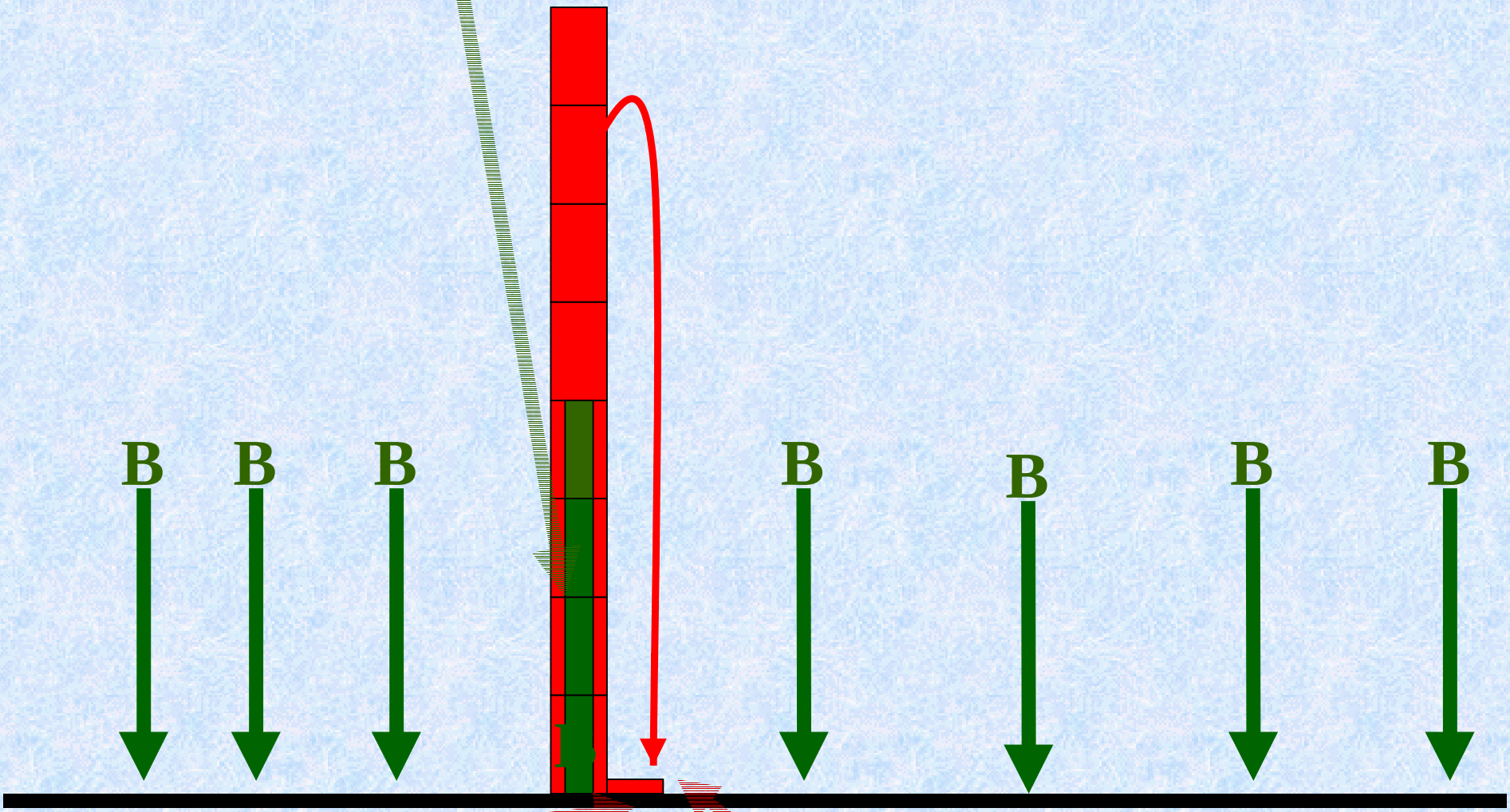
The immune system generates cells with various characteristic shapes to probe for the presence of **antigens with complementary shapes**.



Once some virons get in the system, they multiply unhindered as long as none of them meets an immune system cell with complementary shape.

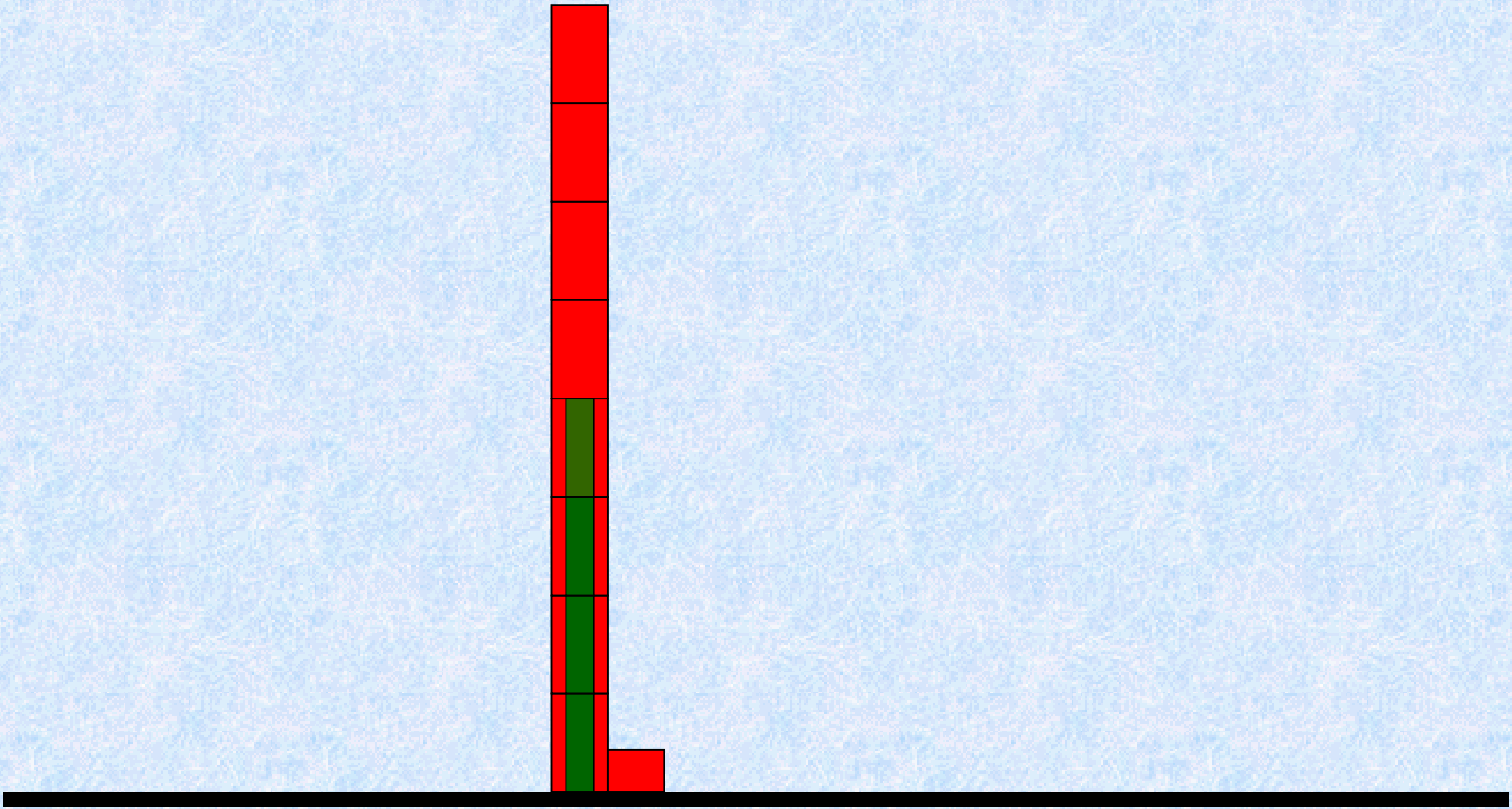


Once one viron (individual from the strain) meets an immune system cell **the cell keeps multiplying** and its descendents meet more virons and multiply too.



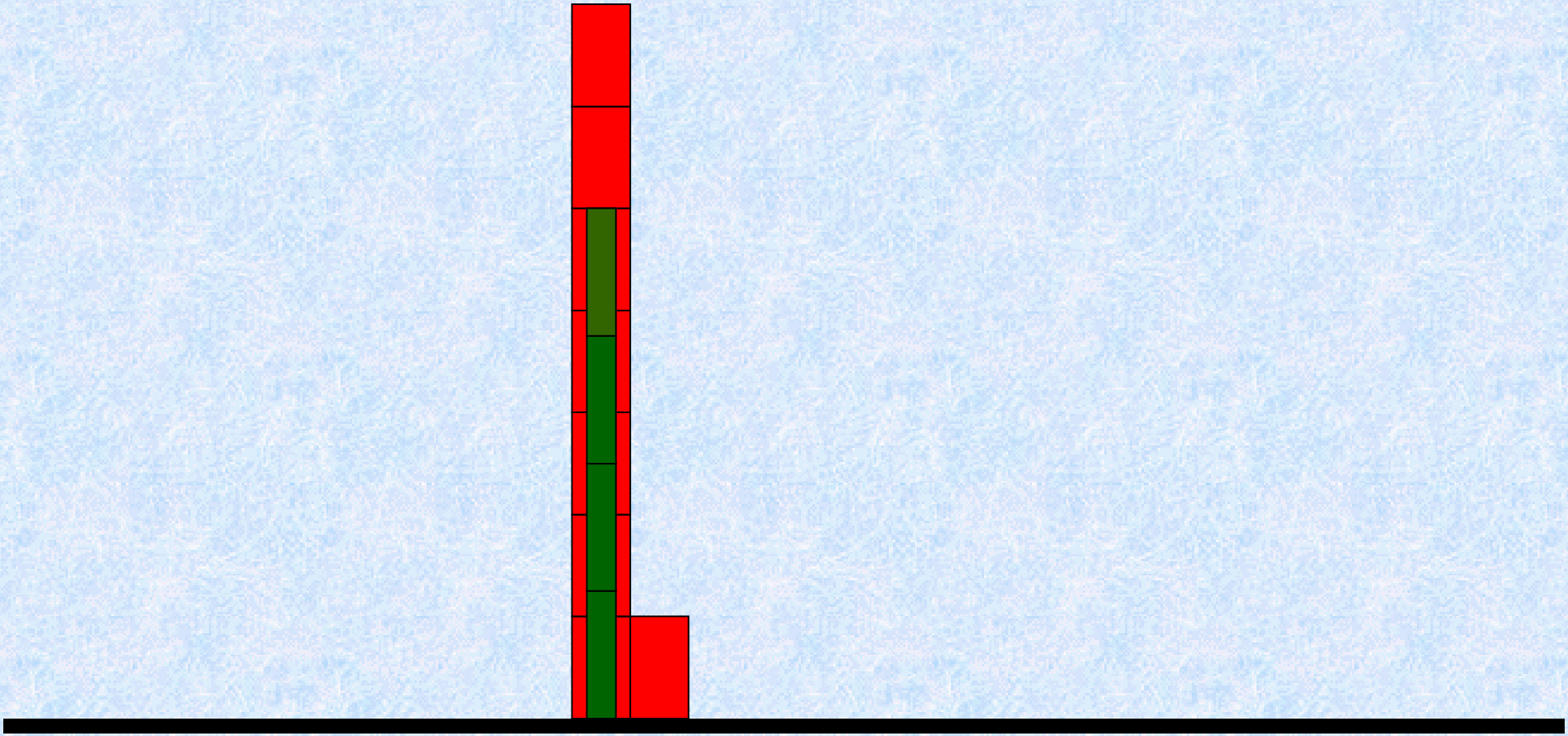
Some mutant virons with different shape (and therefore undetectable by the present strain of immune cells) are produced.

The virons from the strain detected by the cells with complementary shape are destroyed.



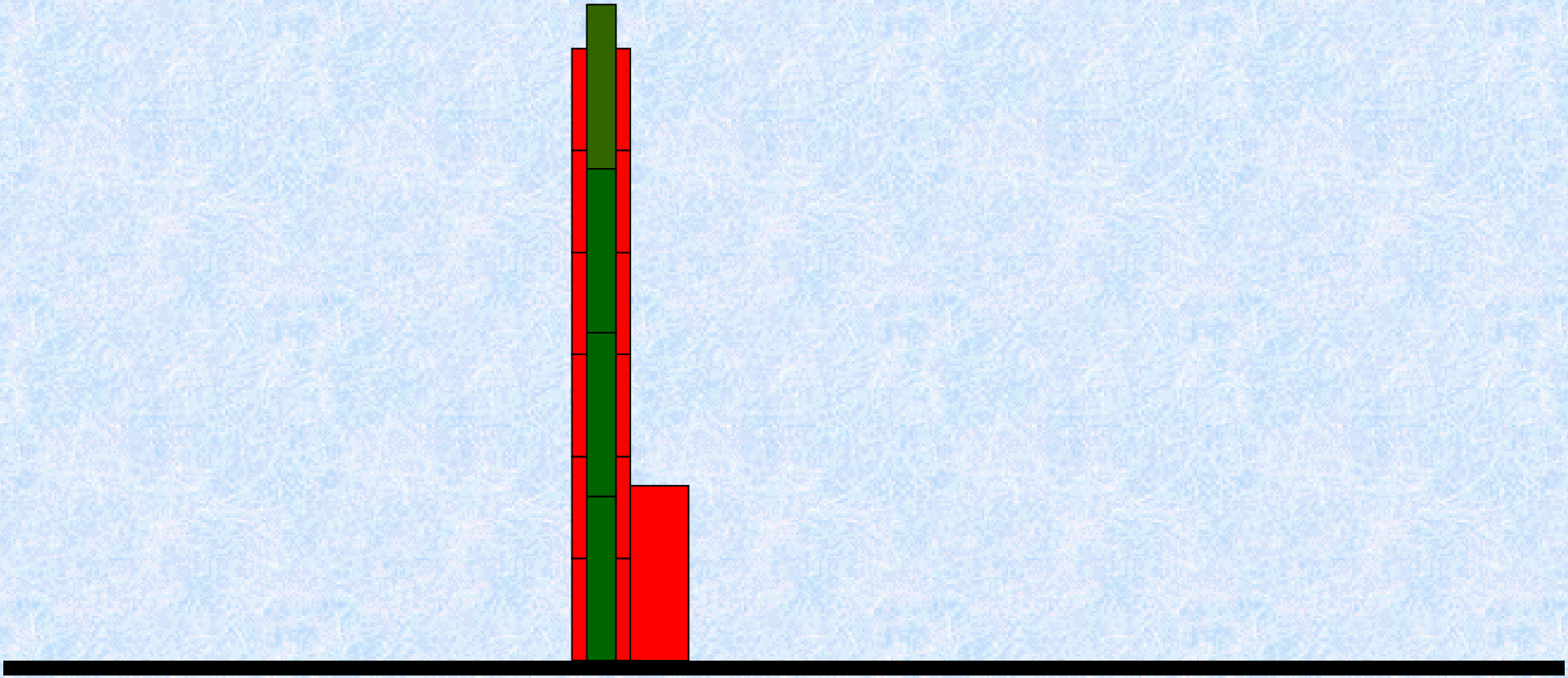
The mutant ones have different shape. They are not detected (yet) so they multiply unhindered.

The detected viron strain is destroyed by the immune system.



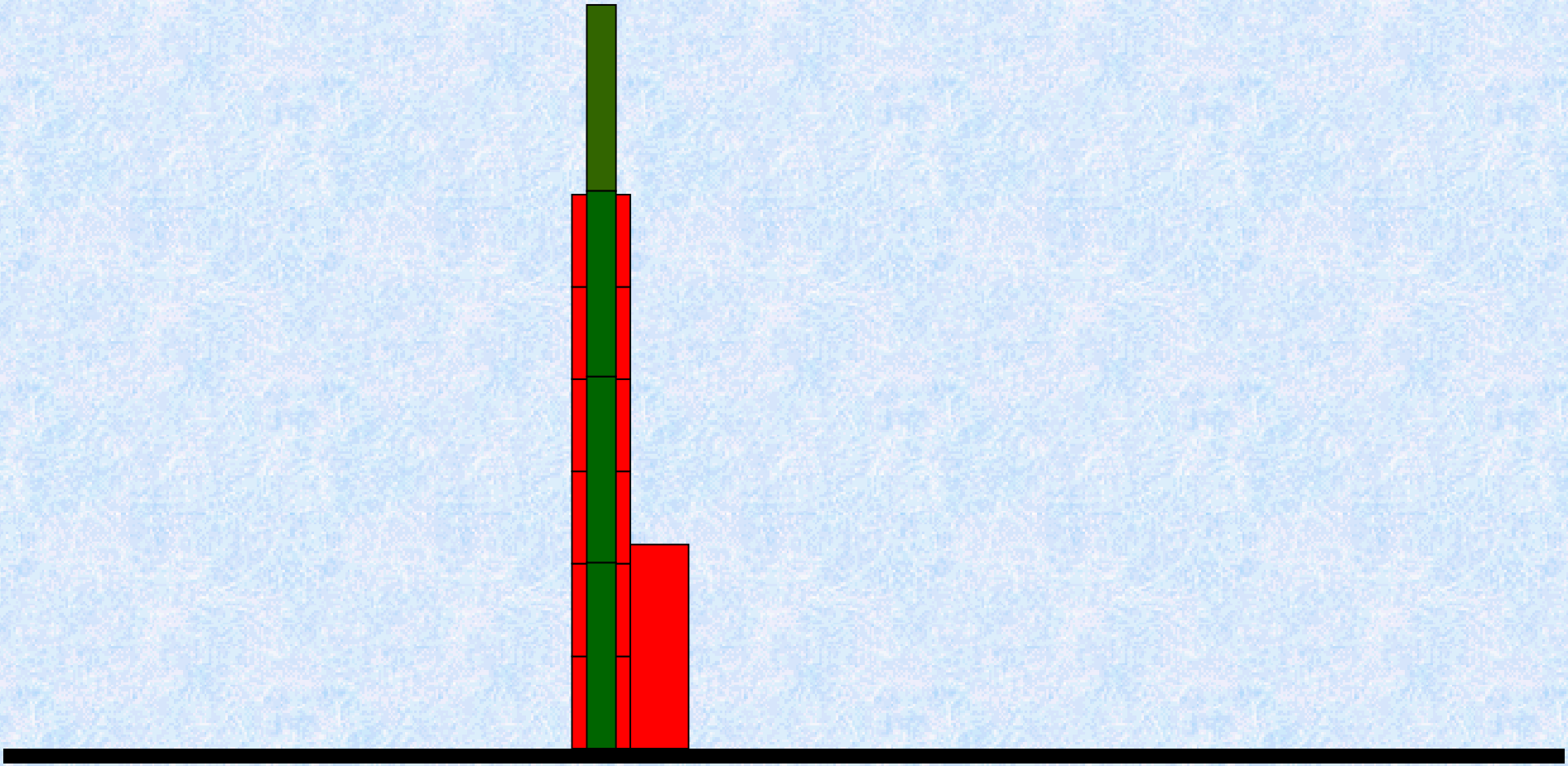
Shape space

The detected viron strain is destroyed by the immune system.



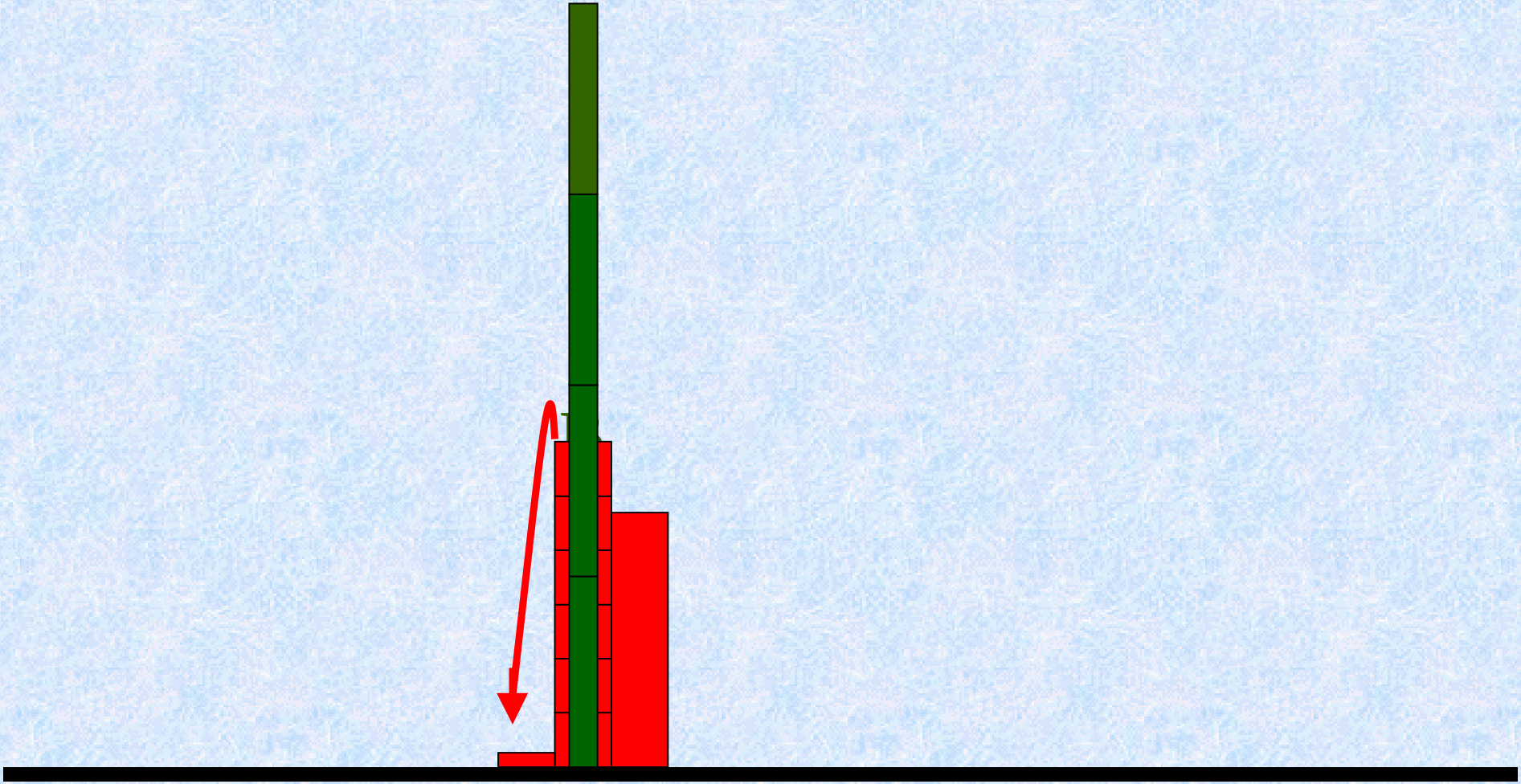
Shape space

The detected viron strain is destroyed by the immune system.



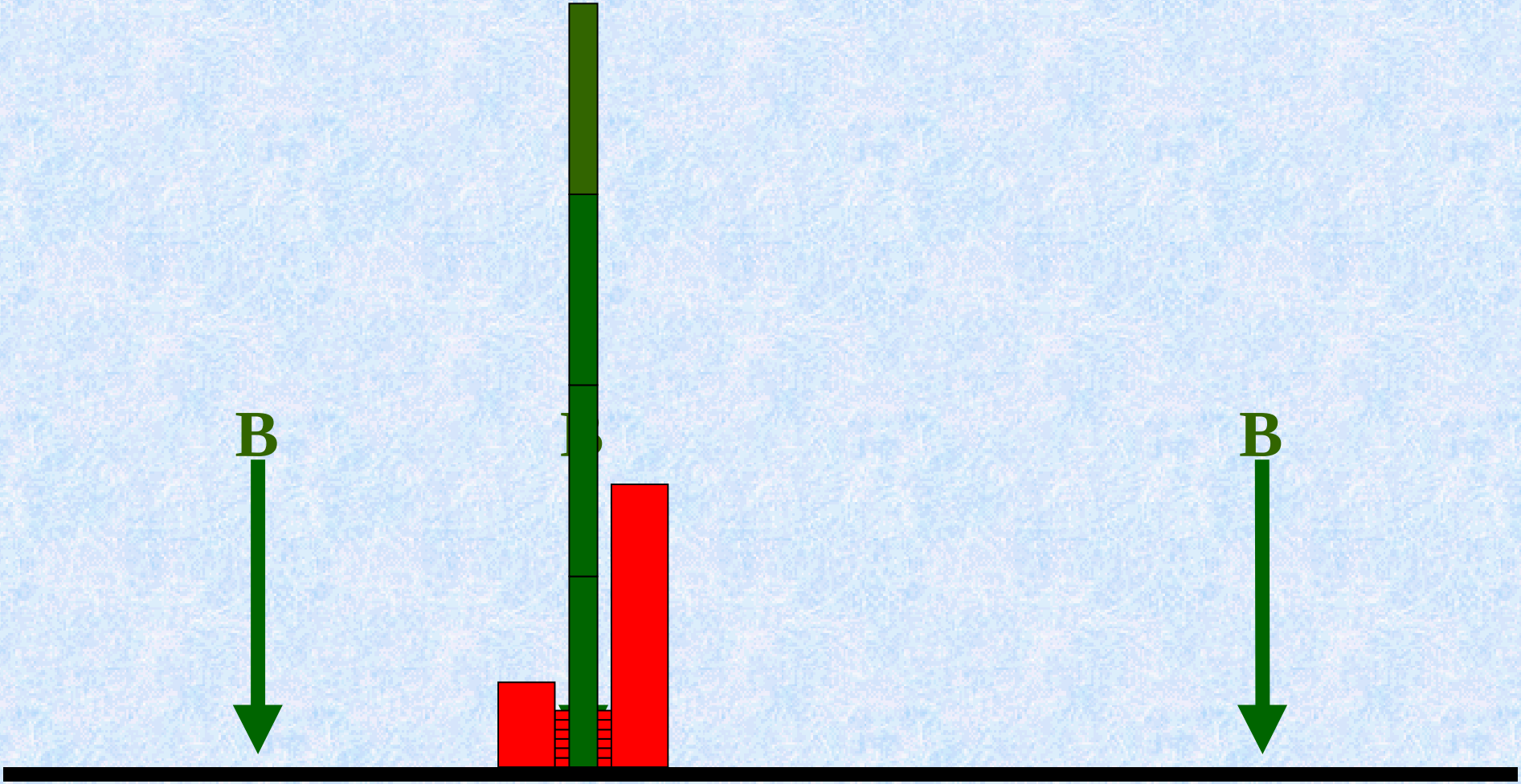
Shape space

Before being completely destroyed, the detected strain is able to generate randomly more mutants, with different characteristic shape.

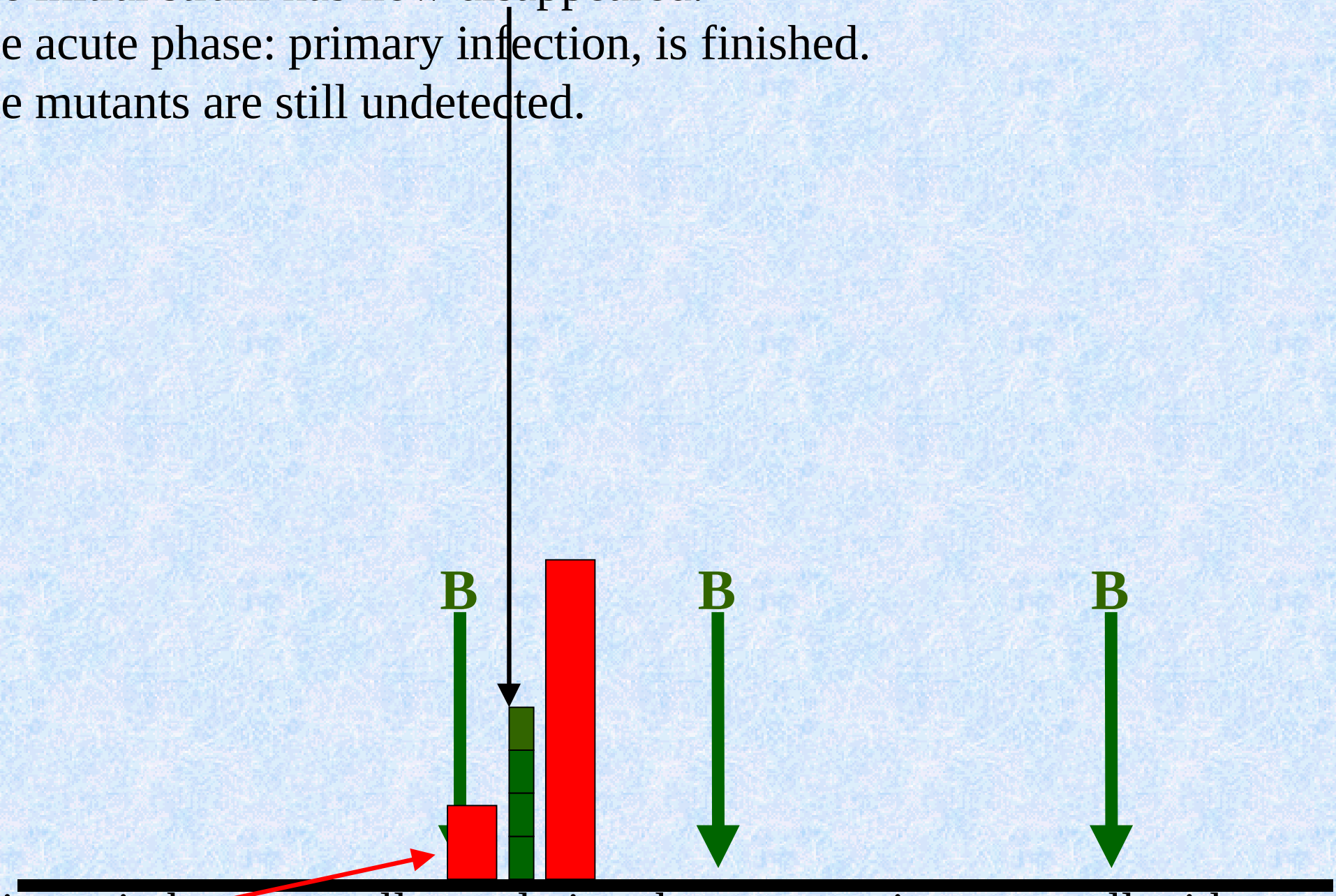


Shape space

The initial strain is decimated but the mutants are still undetected and multiply unhindered.

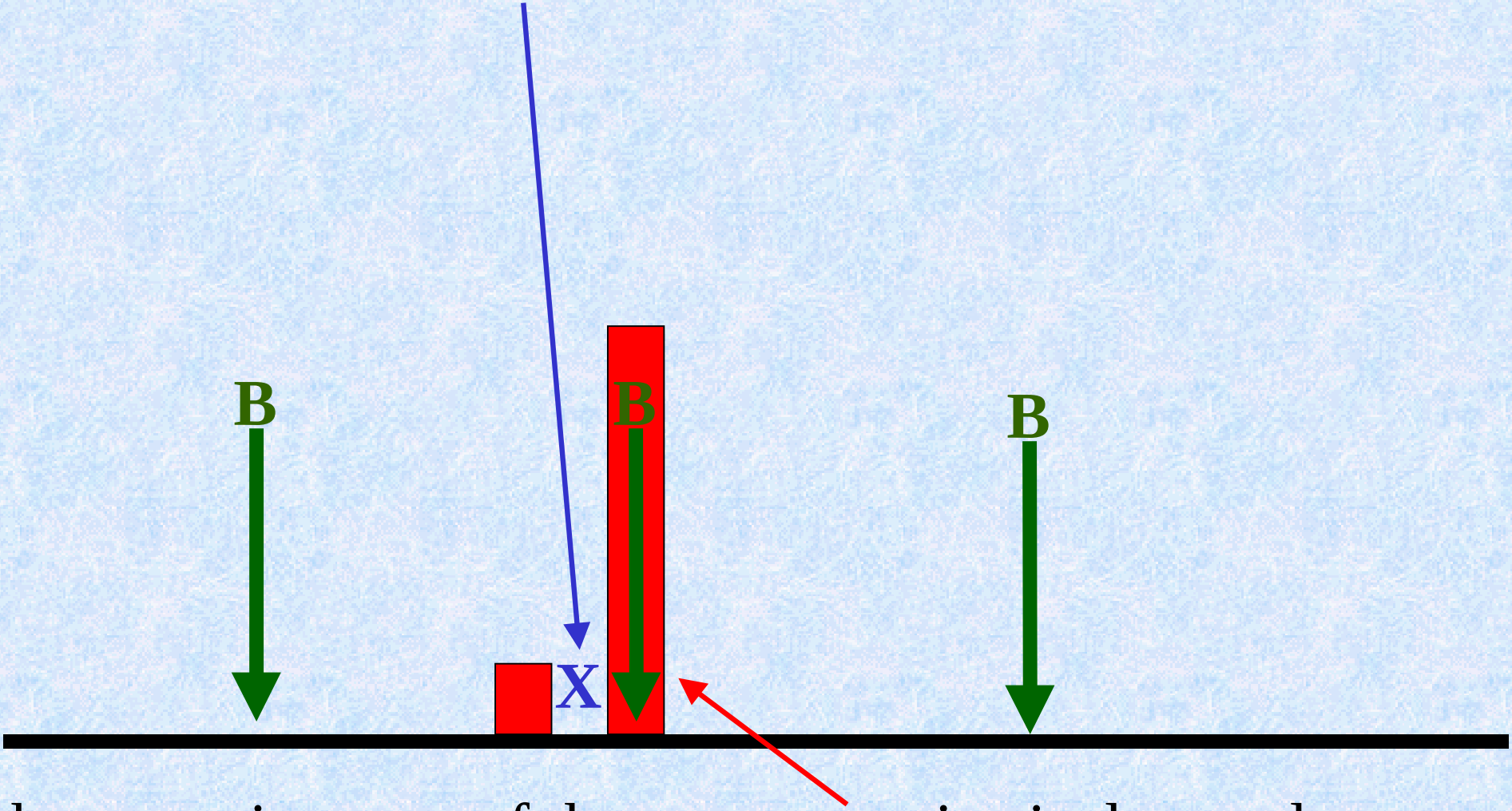


The initial strain has now disappeared.
The acute phase: primary infection, is finished.
The mutants are still undetected.

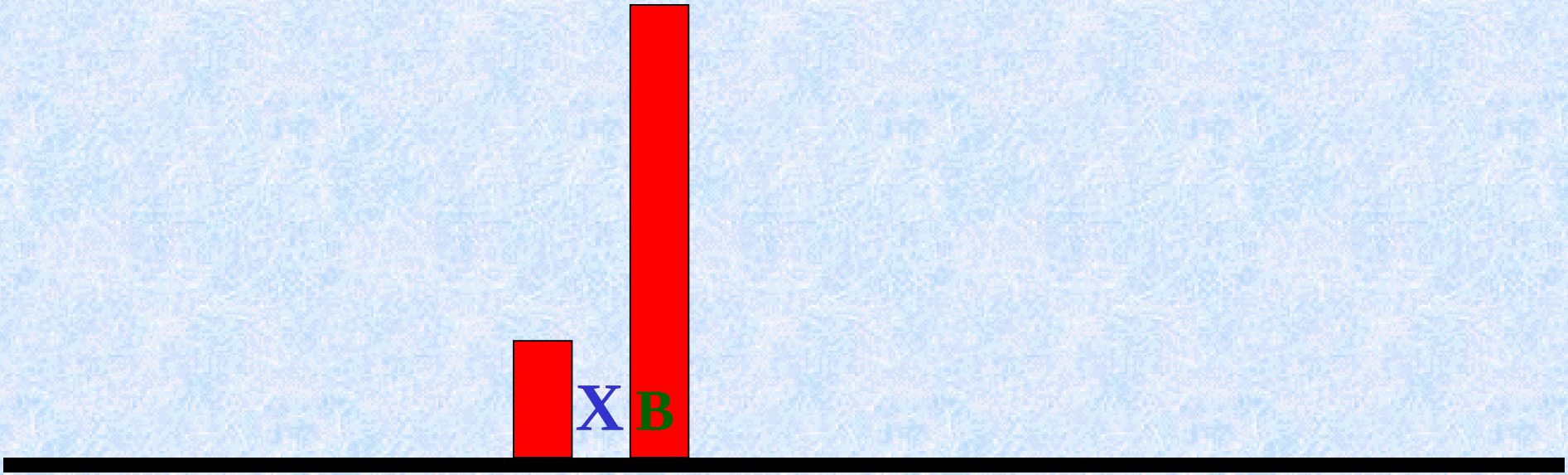


This strain has so small population that even an immune cell with complementary shape doesn’t meet/detect any of its individuals.

After the initial strain is destroyed, the immune cells with complementary shape do not meet any excitation and they die without multiplying. Some “memory cells” with the information of the initial strain shape are left (forever).

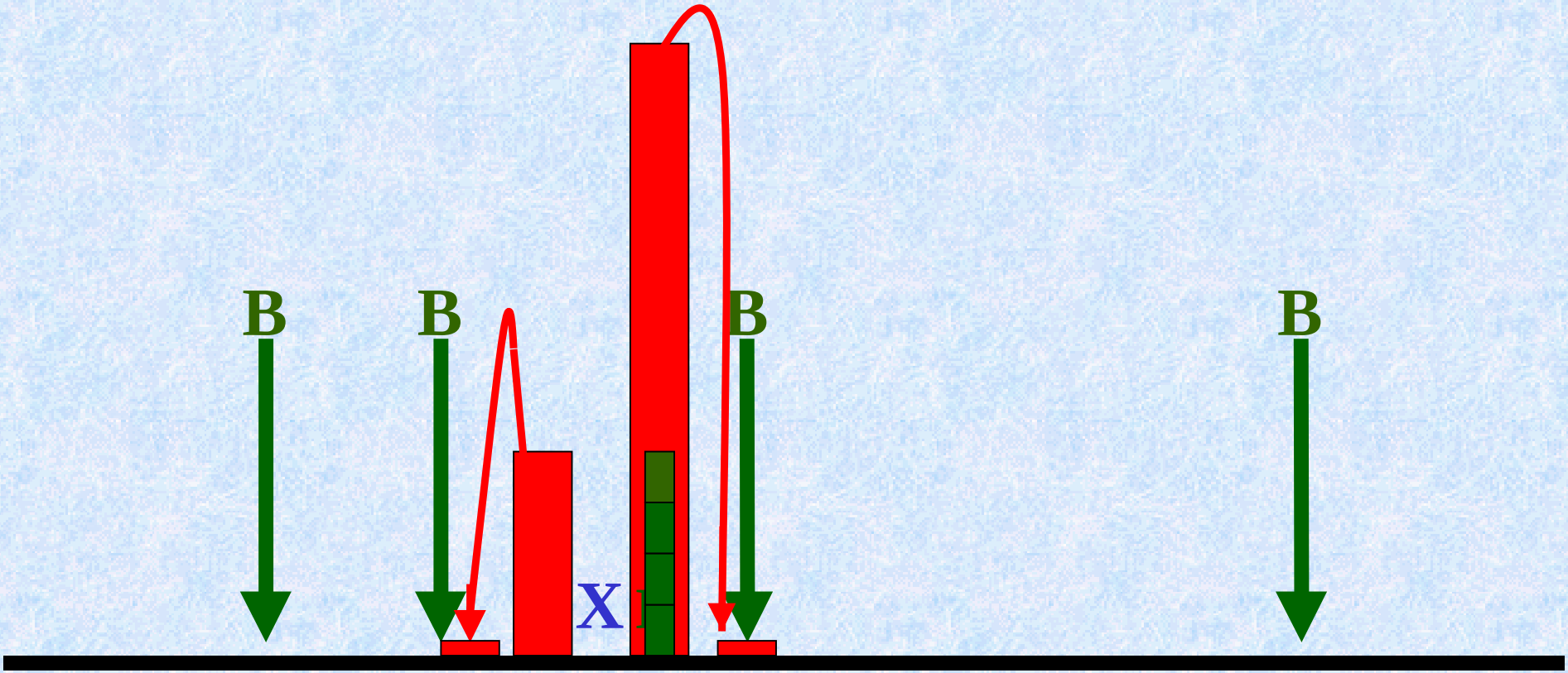


In the meantime one of the mutant strains is detected

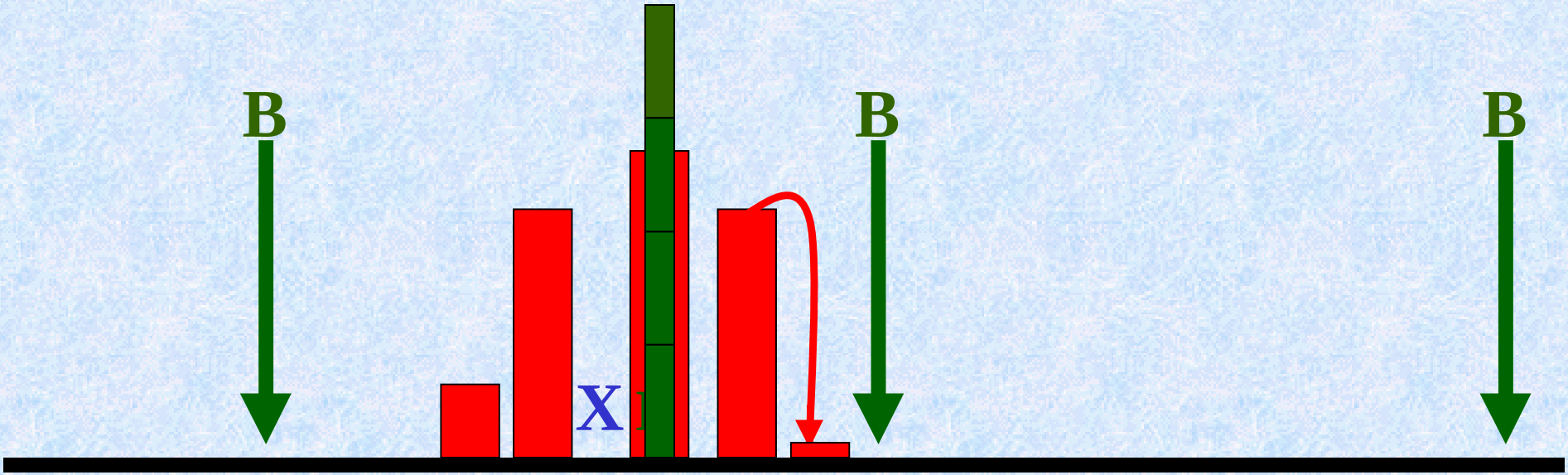


Shape space

The immune cells with the complementary shape to the detected strain multiply. They are not many enough yet to stop the multiplication of the strain and in particular the generation of some mutants.

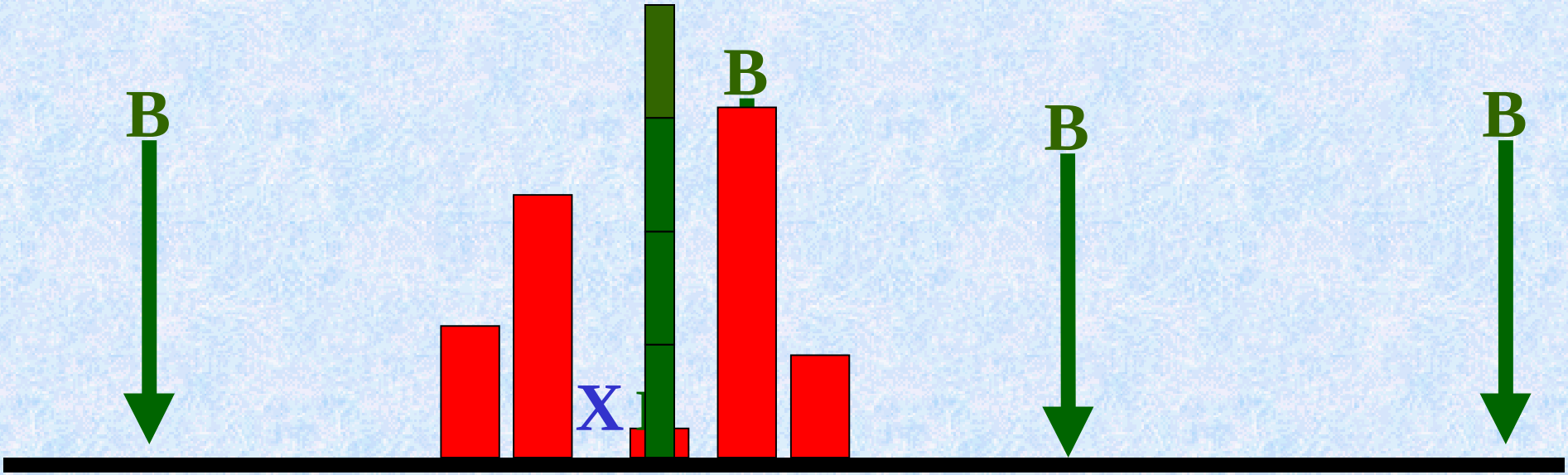


The detected strain is being decimated but its mutants do well and in fact produce mutants of themselves.



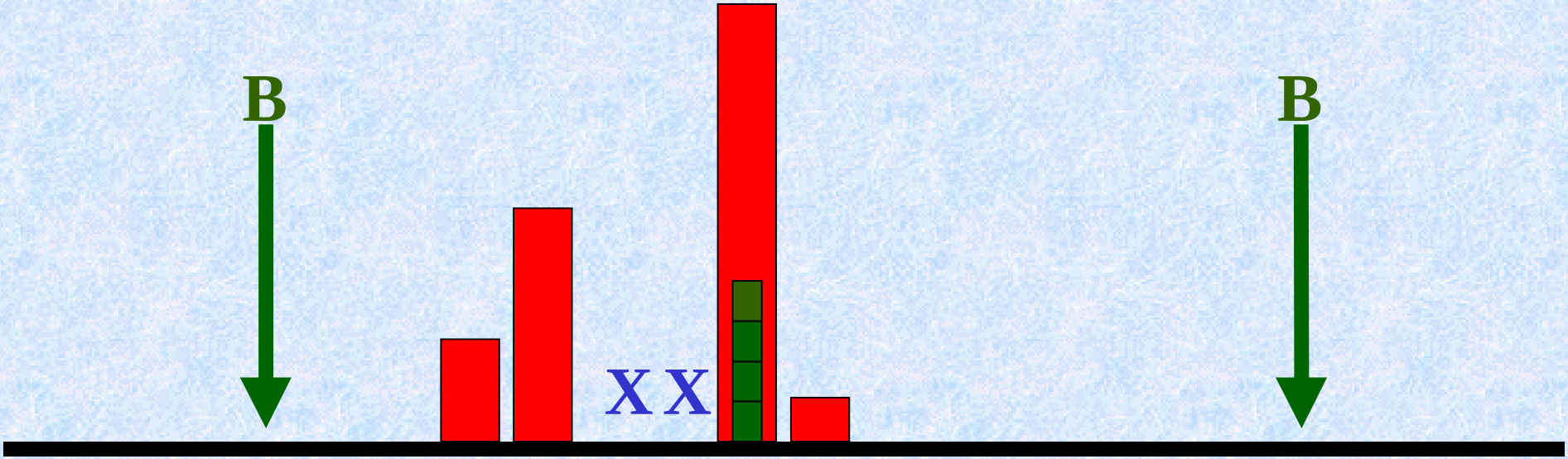
Shape space

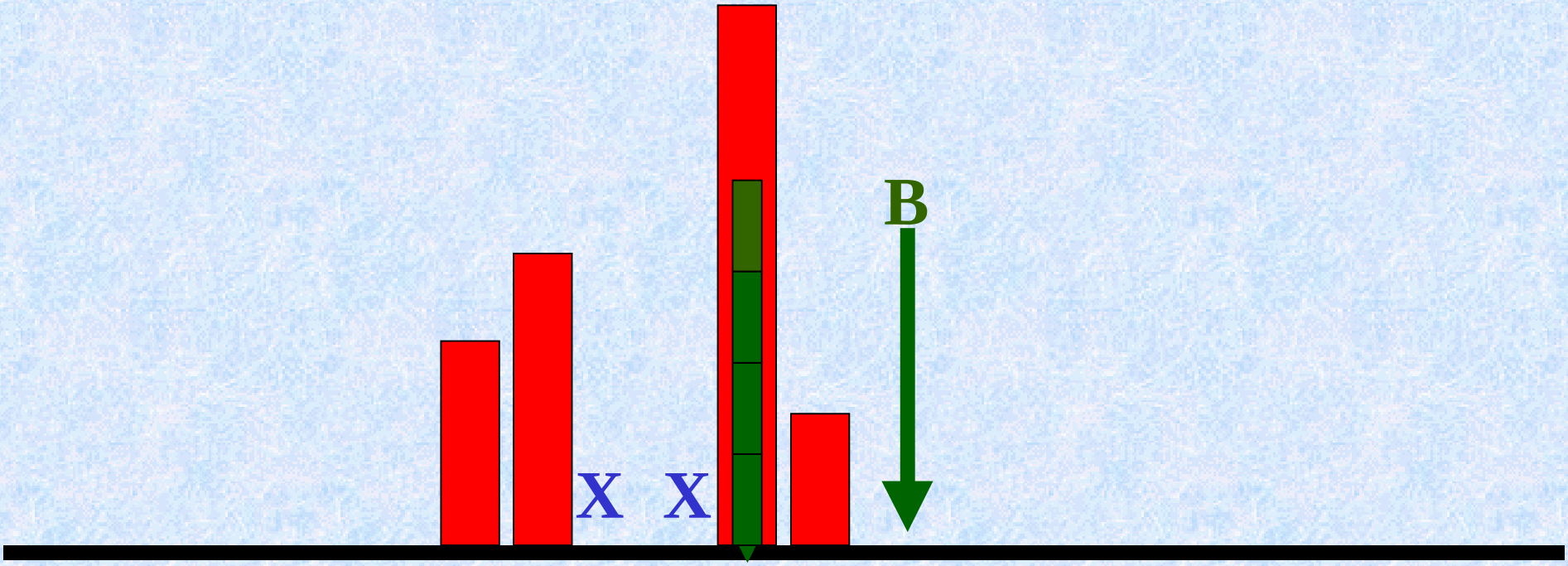
The detected strain is about to disappear and another strain is just being detected.

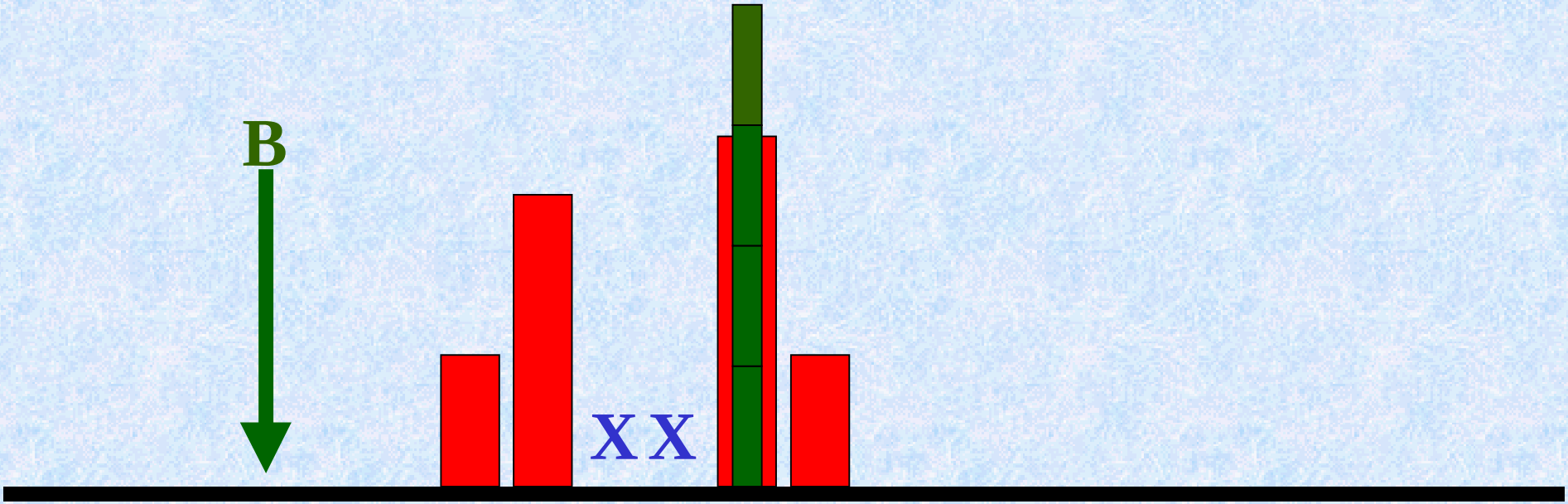


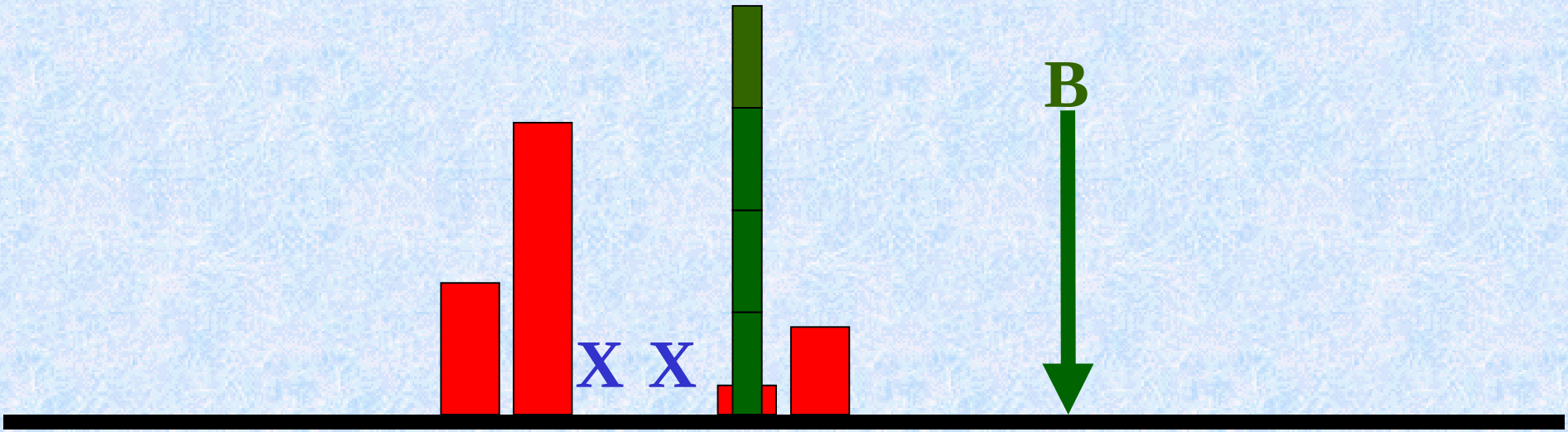
The antibodies corresponding to the destroyed strain disappear. Only memory cells are left.

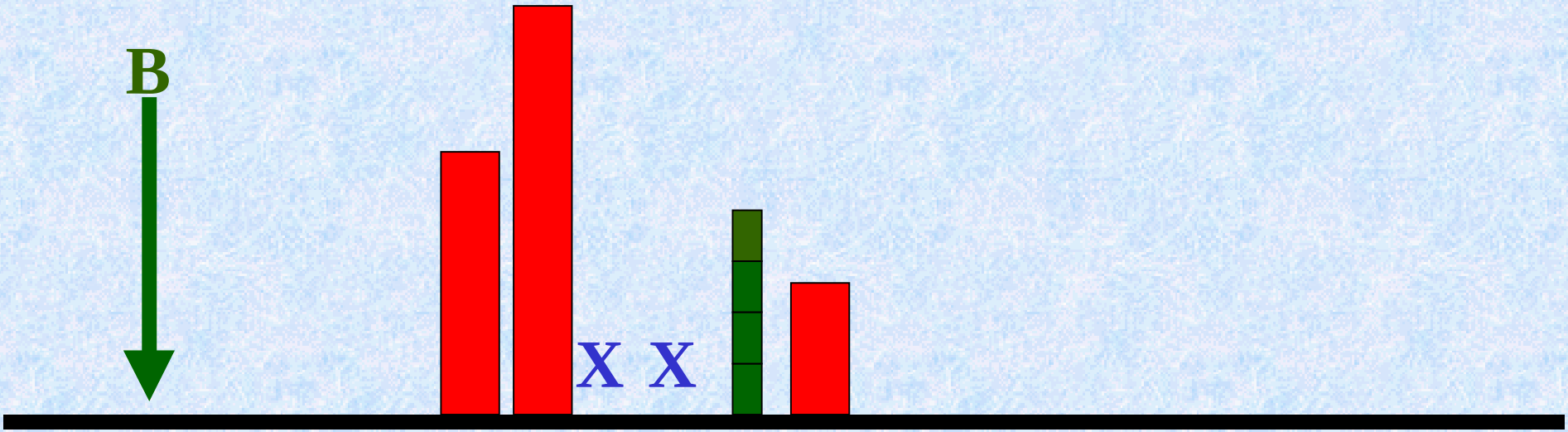
Antibodies corresponding to the newly detected strain are being produced.



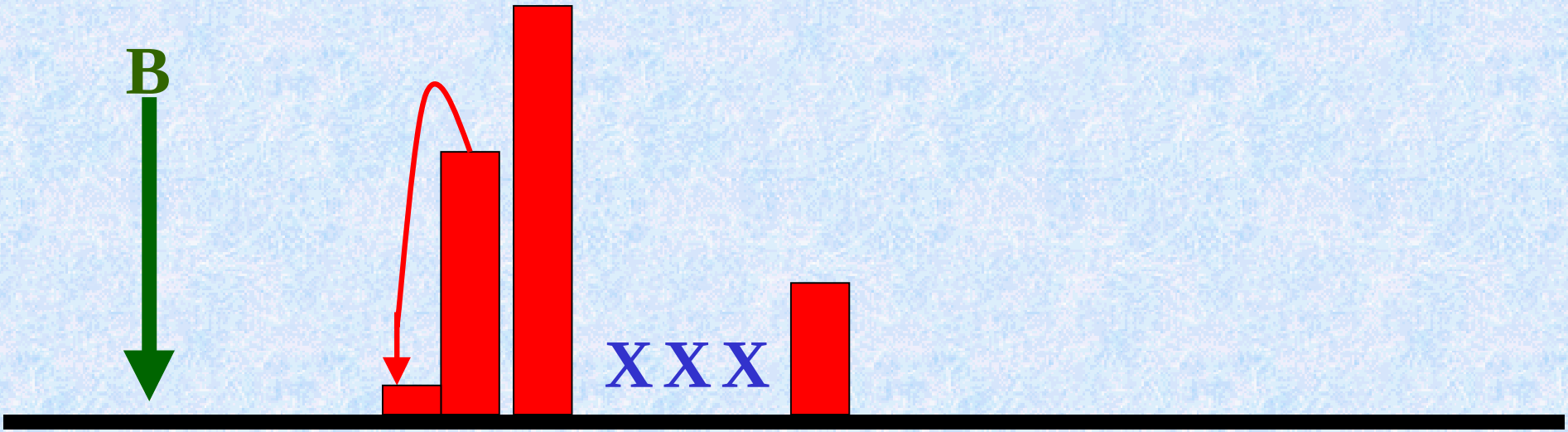

B
X
X


B
X X

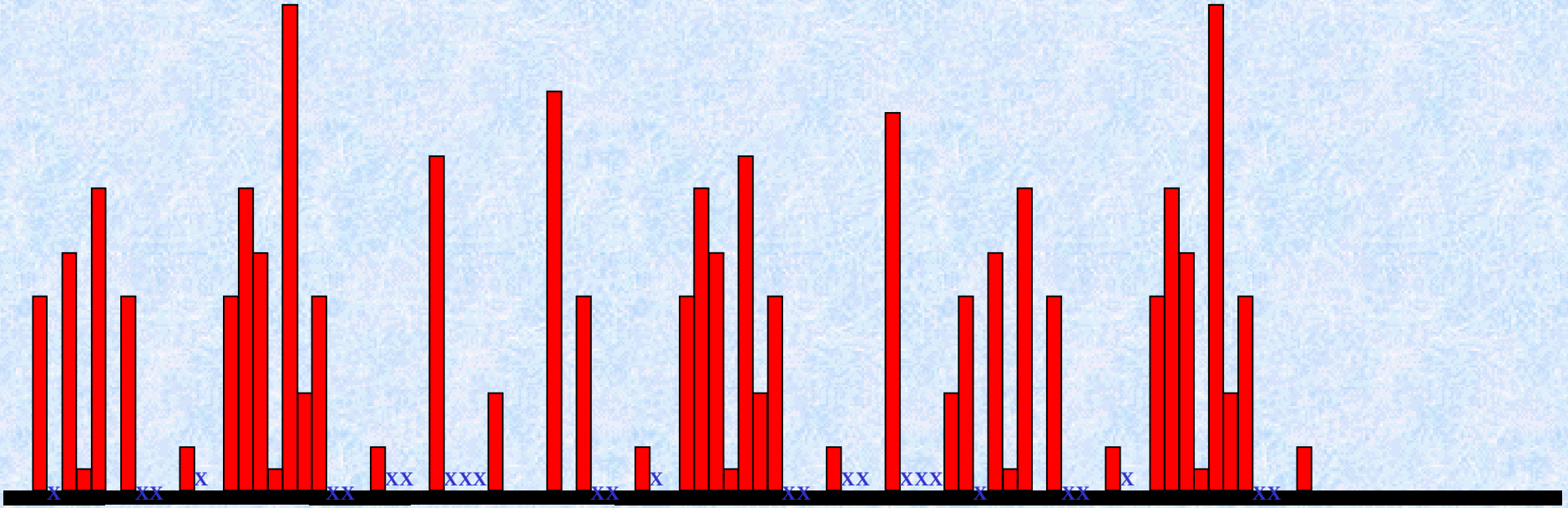

B
X X


B
X X

The virus looses another battle but the number of strains keeps increasing. Copy at the beginning



The virus looses another battle but the number of strains keeps increasing until it overcomes the immune system.

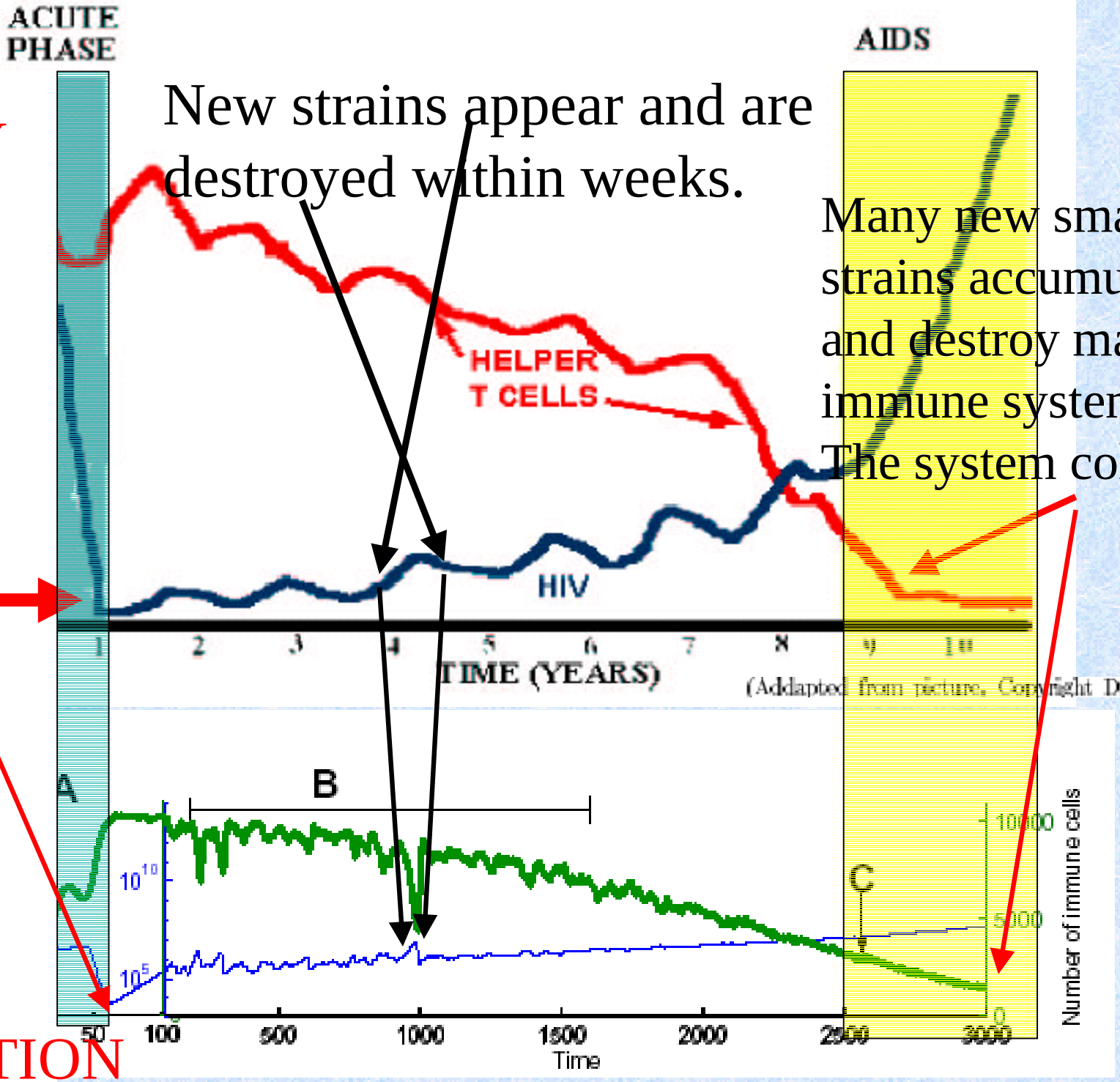


REALITY

New strains appear and are destroyed within weeks.

Many new small strains accumulate and destroy many immune system cells. The system collapses

The strains of the first invasion are completely wiped out



SIMULATION

CONCLUSION

Discretization=> **micro-inhomogeneity**

Auto-catalysis ($\dot{b} \sim b$)=> **amplification**

Complex Collective objects with **emergent properties**

Identity
Spatio-temporal localization

Adaptation
Increase chances of survival
Sustainability

Cognition
Searching, Finding and Exploiting **A** fluctuations

Applies to many real life systems

Slides Left out of the Talk

Desertification and Reclaim

Measurements of organic matter distribution across a region ranging

from total (uniform) desert

to (uniform) mediteranean vegetation

In between: semi-arid regions: adaptive patches

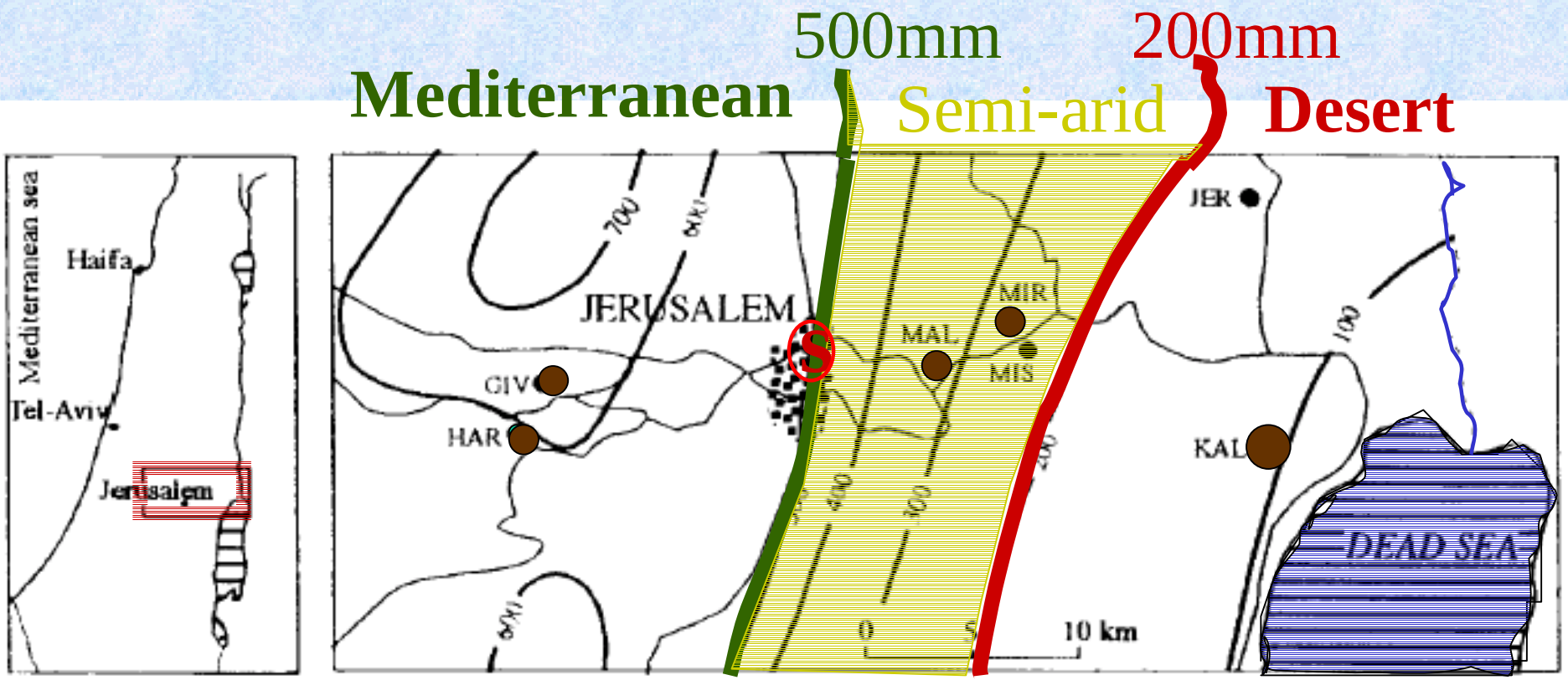


Figure 1. Location of research sites along the climatological transect (mean annual rainfall, in millimetres, is indicated by isohyetes).

LAND DEGRADATION & DEVELOPMENT
Land Degrad. Develop. **9**, 407–422 (1998)

THE IMPACT OF CLIMATE CHANGE ON GEOMORPHOLOGY AND DESERTIFICATION ALONG A MEDITERRANEAN-ARID TRANSECT

H. LAVEE,[1]* A. C. IMESON[2] AND P. SARAH[1]
[1]*Department of Geography, Bar Ilan University, Ramat Gan, Israel*
[2]*Landscape and Environmental Research Group, University of Amsterdam, Amsterdam, The Netherlands*

Desert

uniform

Semi-arid

patchy

Mediterranean

uniform

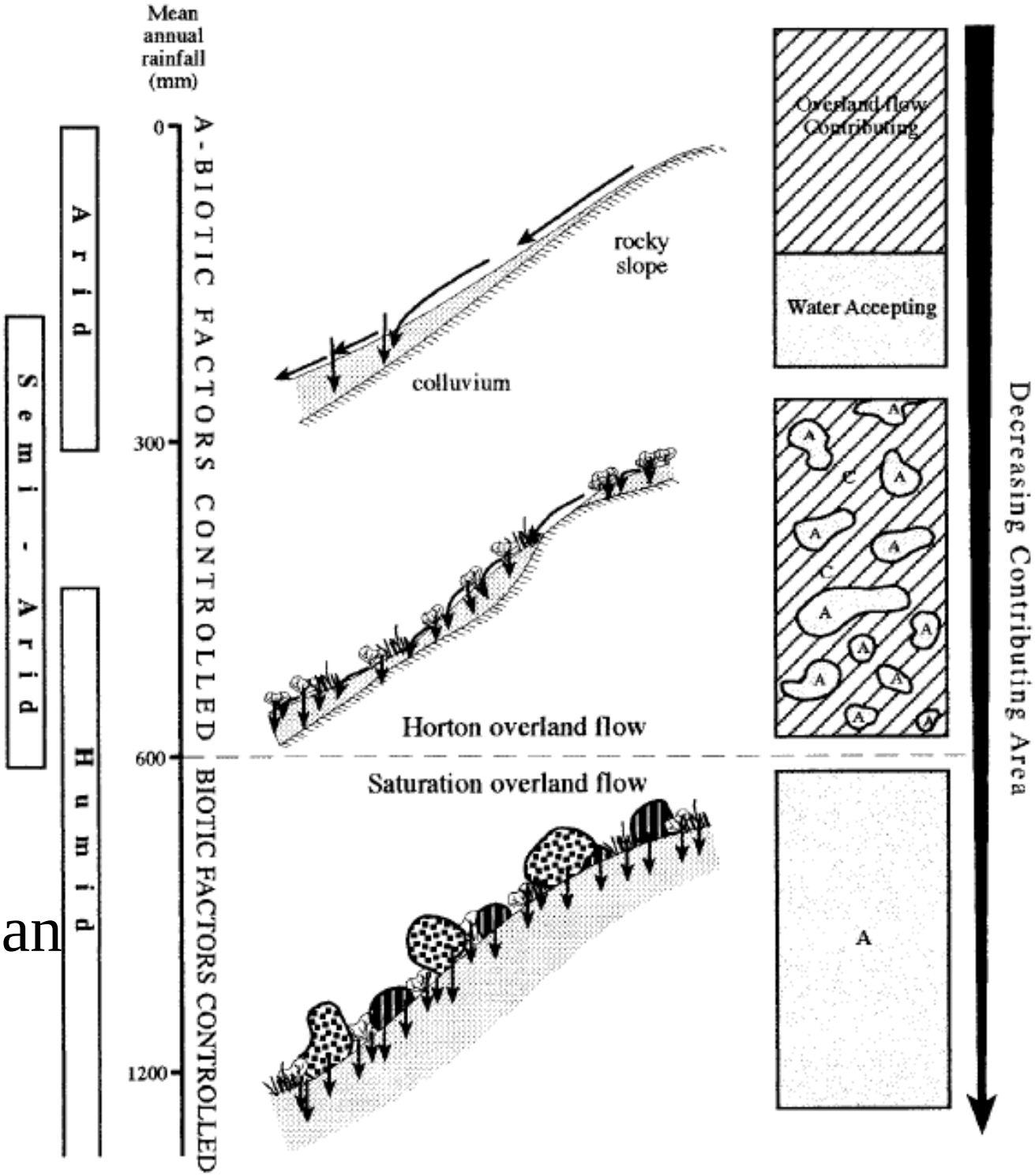


Figure 16. Rainfall redistribution under different climatic conditions (A = water accepting area; C = overland flow contributing area).

Saturation Effects;
Globalization of Competition

leads to

Localization of Growth

Malthus→ exponential growth when <birth> > <death >

$b^{\bullet}(t) = \alpha\, b(t)$ => exponential increase for $\alpha > 0$

Here → Super-Malthus: always exponential growth:

Natural death $\mu\, b(x,t)$ does not lead to saturation.

For very large $b(x,t)$'s competition necessary (**Verhuulst** 1838):

Logistic equation $b^{\bullet}(t) = \alpha\, b(t) - \gamma\, b(t)^2$

Montroll: "almost all the **social phenomena**, except in their relatively brief abnormal times obey the logistic growth".

Tasmania Sheep

Lotka and Volterra:

$b(t)$ = the size of an animal/plant population,

α = the aggregated effects of birth and natural death,

$-\gamma\, b(t)^2$ = competition for limited resources.

Economics, **Aoki**

- $b(t)$ = the total product demand in a market.
- $\alpha b(t)$ = emergence of new products ~ size of the market.
- $\gamma b(t)^2$ the products have to compete with one another within a finite total potential market.

Finance, Solomon and Levy have suggested that

- $b(t)$ = the total capital within a financial system.
- $\alpha b(t)$ = the average returns that the system offers,
- $\gamma b(t)^2$ the effects of competition and

Local Competition

One spatially extended generalization of the Lotka Voltera system:

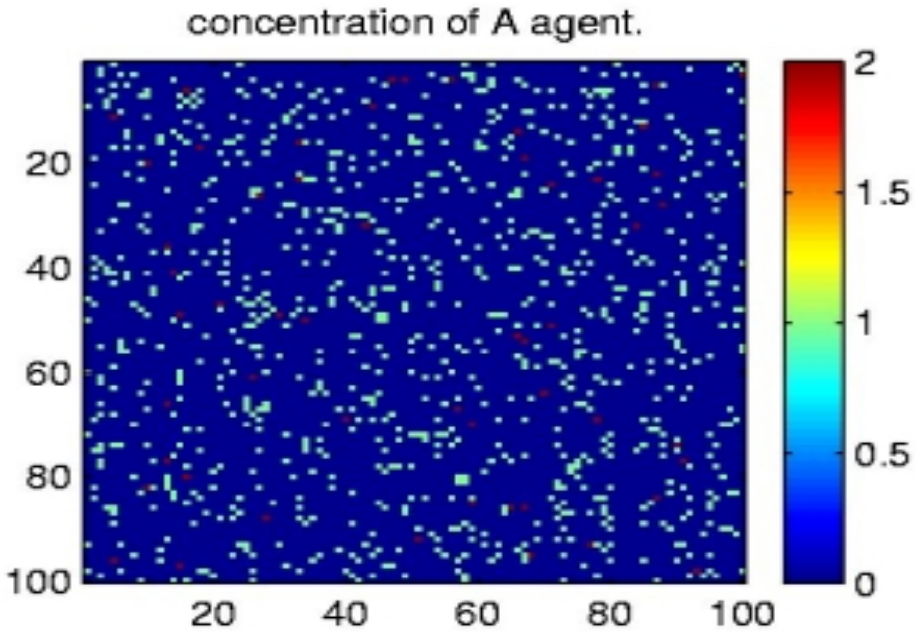
$$\dot{b}(x,t) = (\lambda\, a(x,t) - \mu)\, b(x,t) - \gamma b(x,t)^2 + D_b\, \Delta\, b(x,t)$$

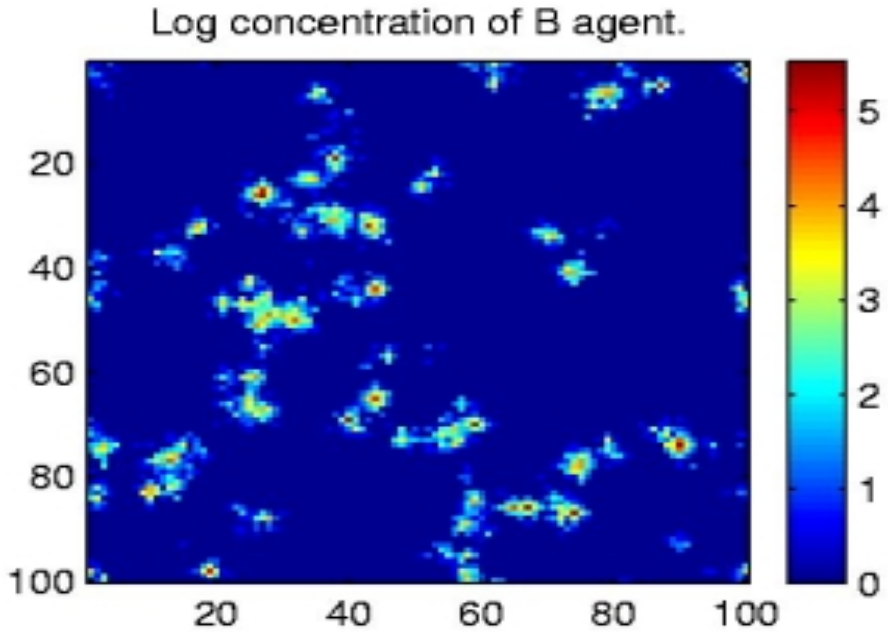
$a(x,t)$ is not a continuum function:

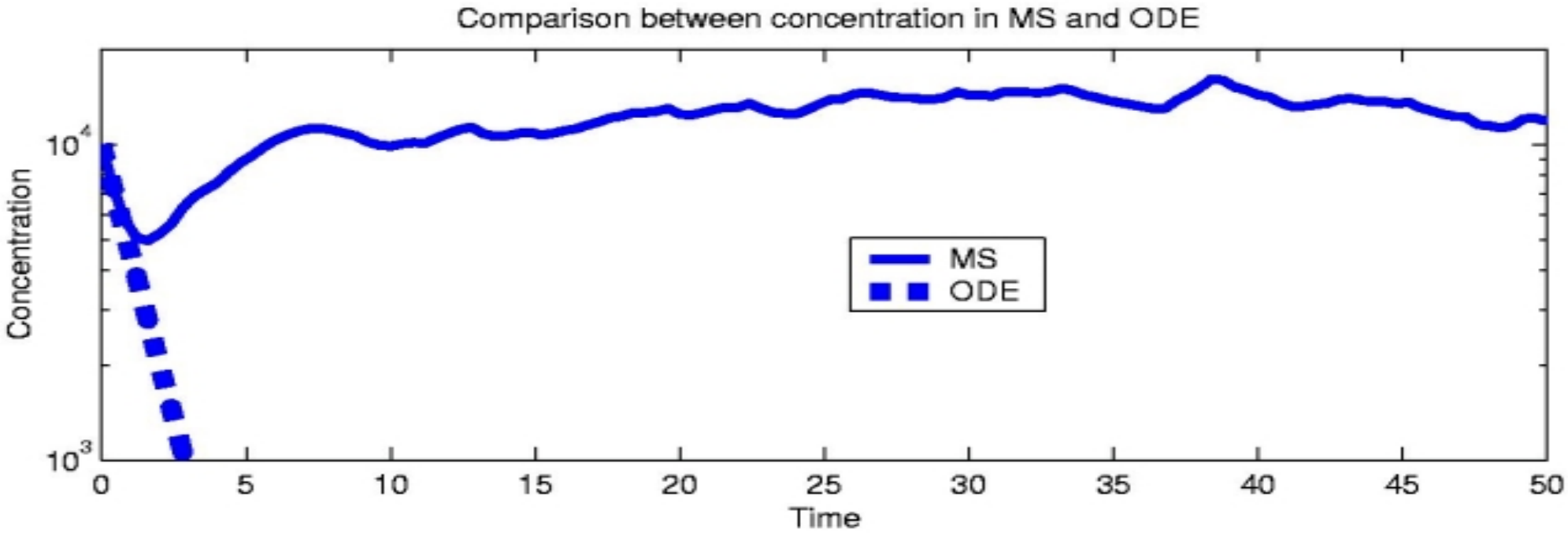
it is a stochastic integer at each site x and it changes in time according to the jumping diffusion rules discussed before.

If $(\lambda\, a(x,t) - \mu) < 0 \Rightarrow$ localized adaptive islands of finite hight.

- Improved life expectation but $b=0$ fixed point for discrete **B**.


concentration of A agent.
20
40
60
80
100
20
40
60
80
100
2
1.5
1
0.5
0


Log concentration of B agent.
20
40
60
80
100
20
40
60
80
100
5
4
3
2
1
0


Comparison between concentration in MS and ODE
Concentration
10^4
10^3
0
5
10
15
20
25
30
35
40
45
50
Time
MS
ODE

The Local Competition equation

$$\dot{b}(x,t) = (\lambda\, a(x,t) - \mu)\, b(x,t) - \gamma b(x,t)^2 + D_b \Delta b(x,t)$$

is not the natural space extended version of

$$\dot{b}(t) = \alpha\, b(t) - \gamma\ \ b(t)^2 \ .$$

Indeed averaging $\quad <\gamma\ \ b(x,t)^2>$

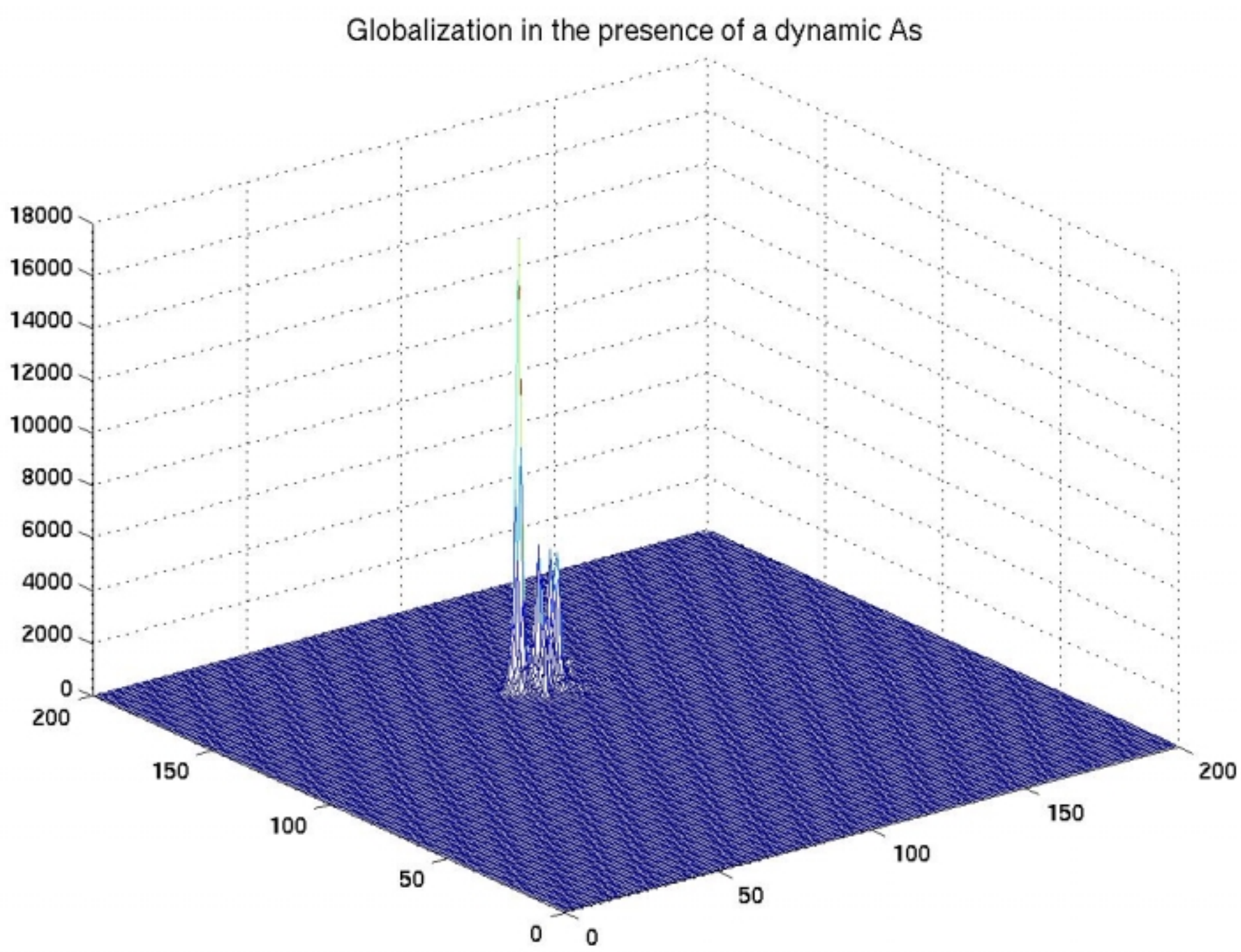
does not give $\quad \gamma\ \ b(t)^2 \equiv \gamma <\ b(x,t)\ >^2$

average over the nonlocal (infinite range) term

$$<\gamma b(x,t)\ \ <b(x,t)>_x>_x = \gamma <\ \ b(x,t)\ \ >_x^2$$ does.

One is lead to the global competition equation:

$$\dot{b}(x,t) = (\lambda\, a(x,t) - \mu)\, b(x,t) - \gamma b(x,t) <b(x,t)>_x + D_b \Delta b(x,t)$$

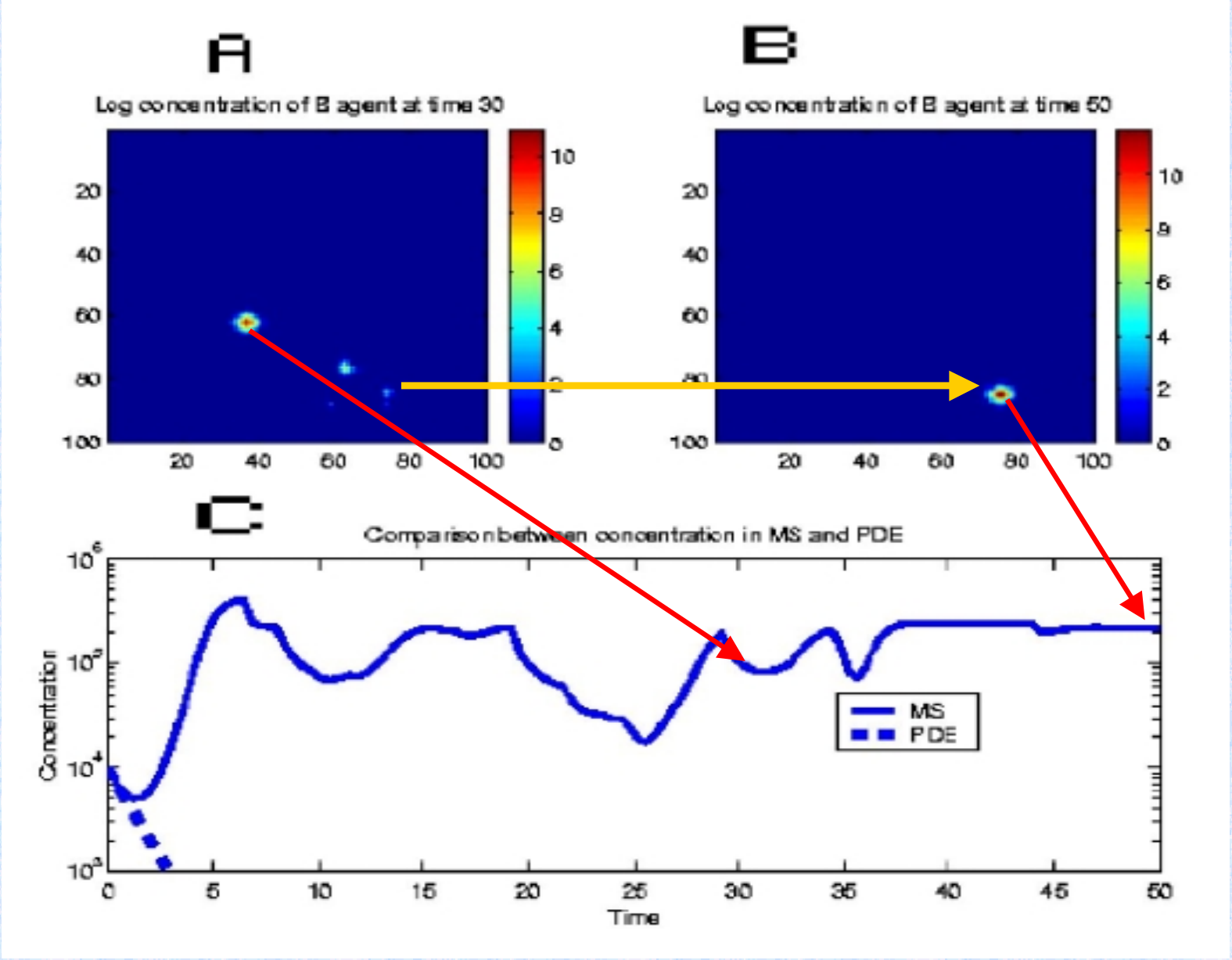

Globalization in the presence of a dynamic As
18000
16000
14000
12000
10000
8000
6000
4000
2000
0
200
150
100
50
0
0
50
100
150
200

Of course, if the location of x_{max} changes in time, one expects very large fluctuations in the total population:

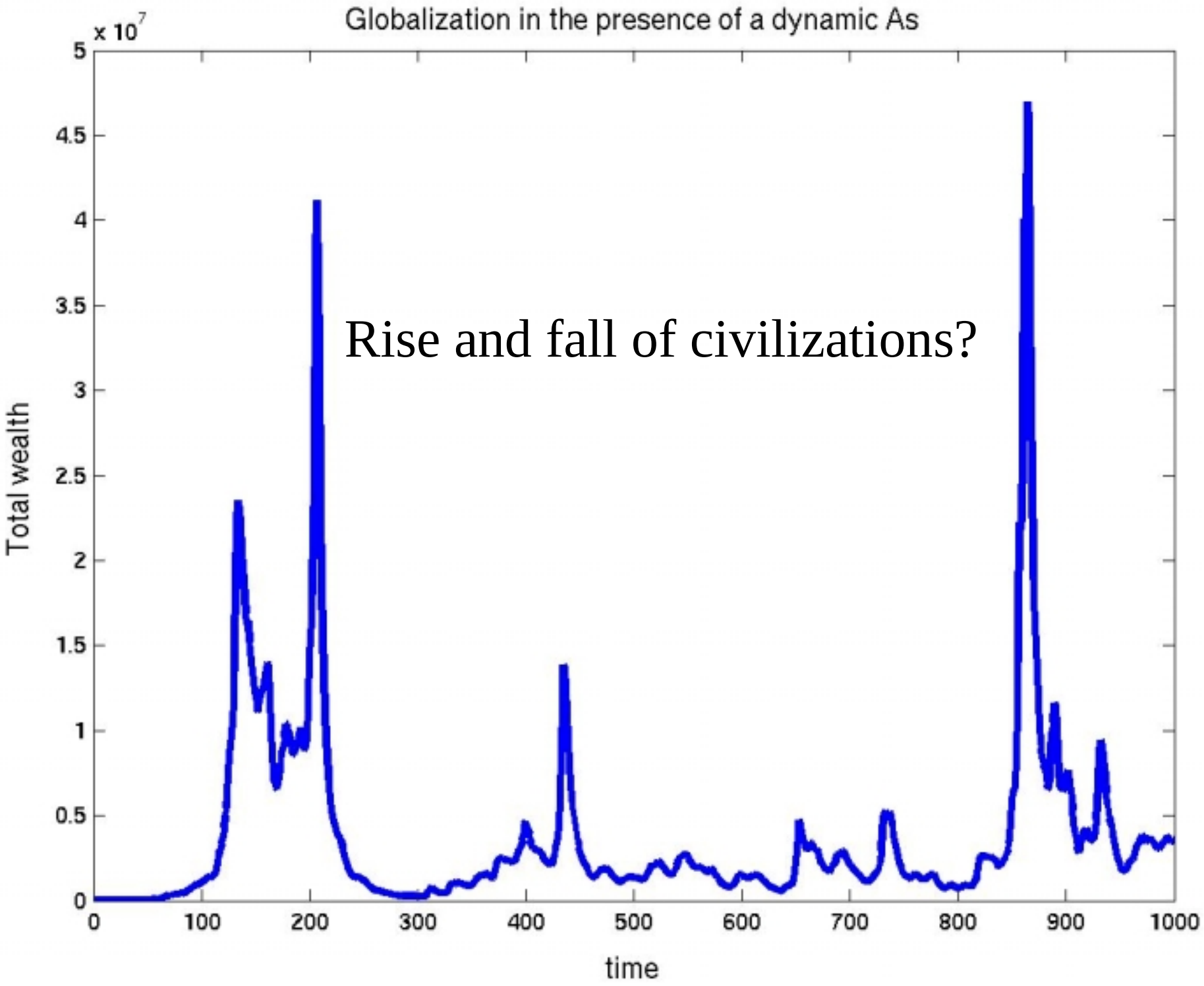
all the population of **B**'s at the old location has to disappear

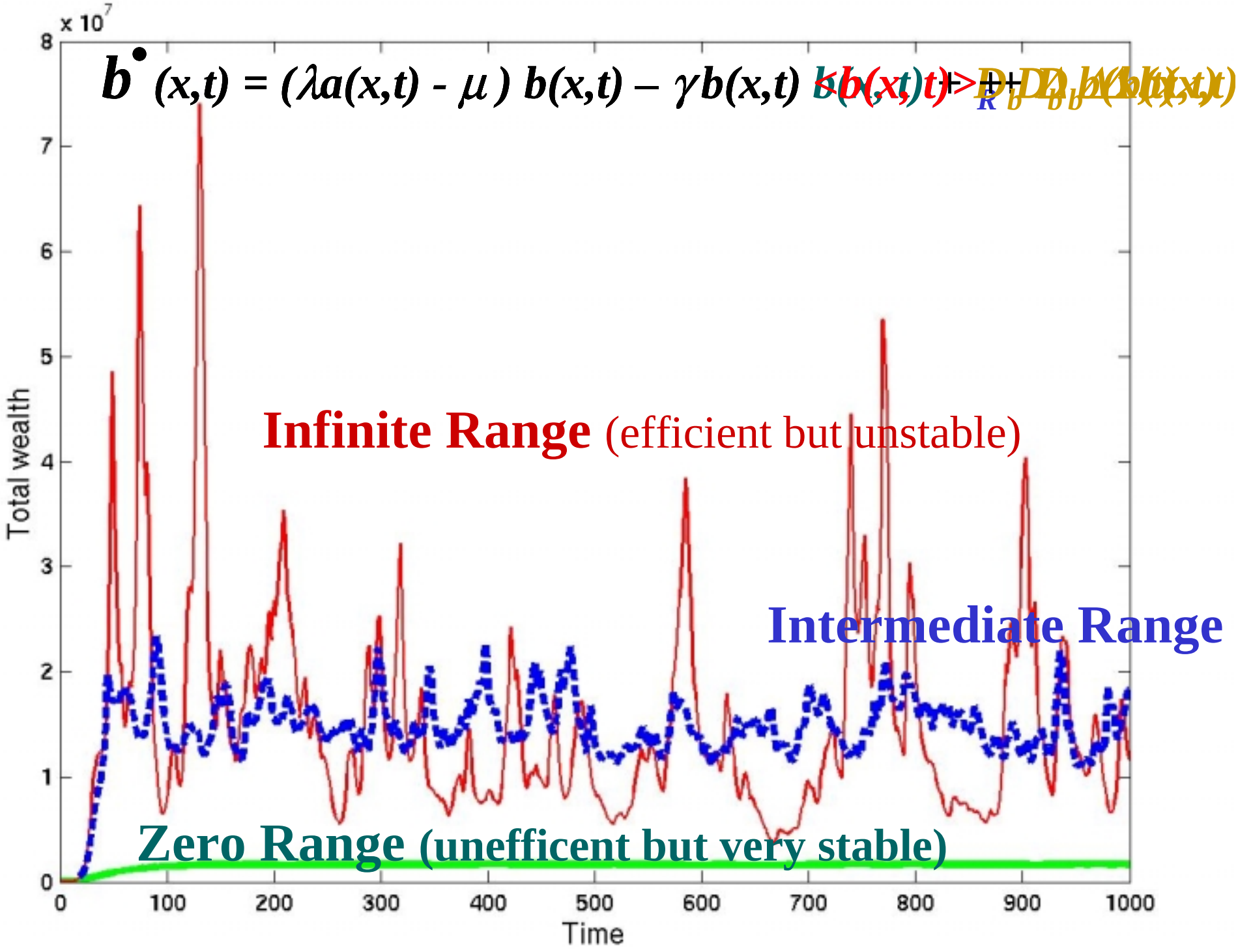
and all the population at the new location has to somehow arise.

The global competition is not only leading to localized wealth but also to significant instability.



With global competition the highest island destroys all its competitors;
price: large fluctuations

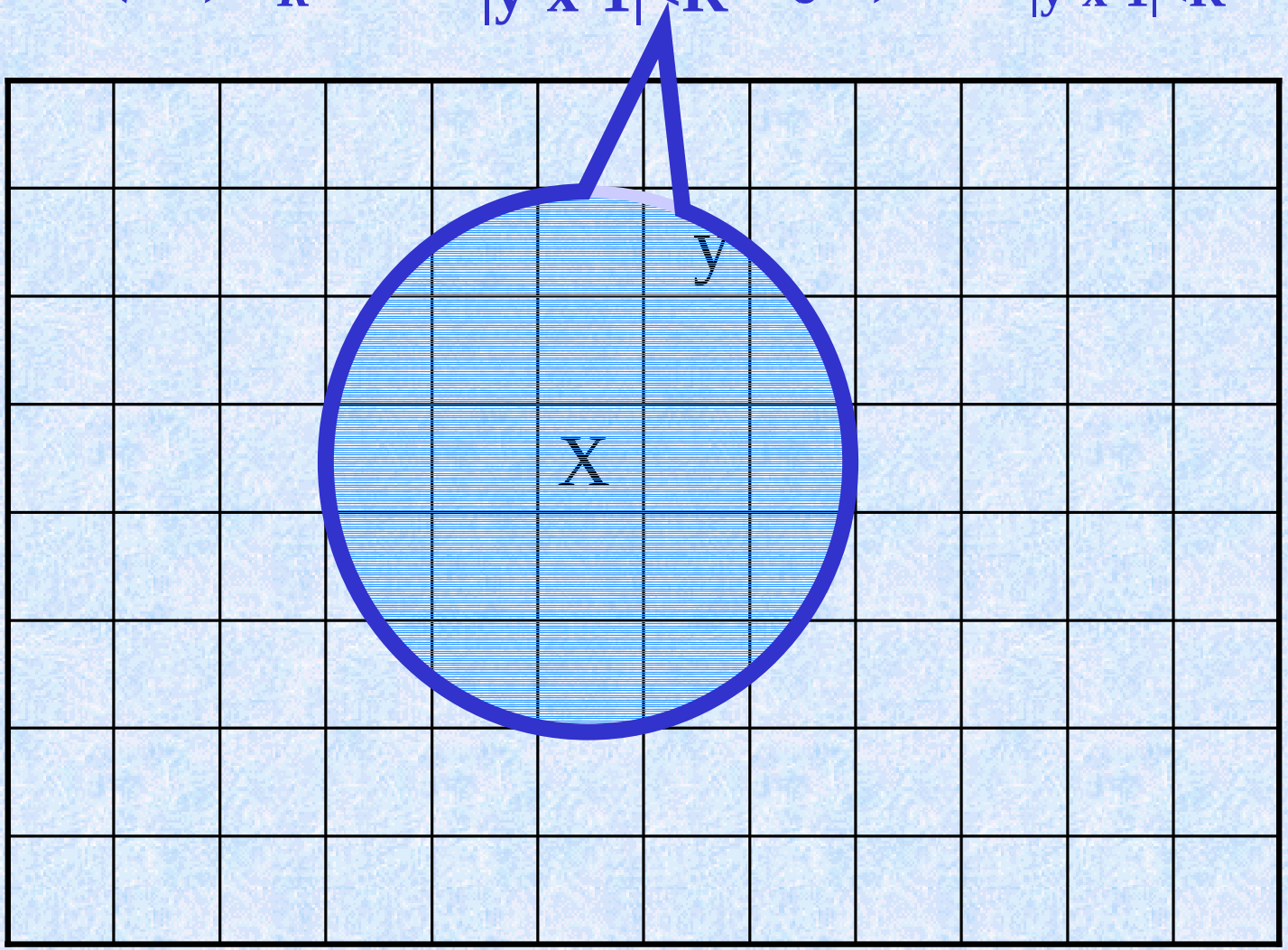

Globalization in the presence of a dynamic As
x 10^7
Total wealth
time
Rise and fall of civilizations?
0
0.5
1
1.5
2
2.5
3
3.5
4
4.5
5
0
100
200
300
400
500
600
700
800
900
1000

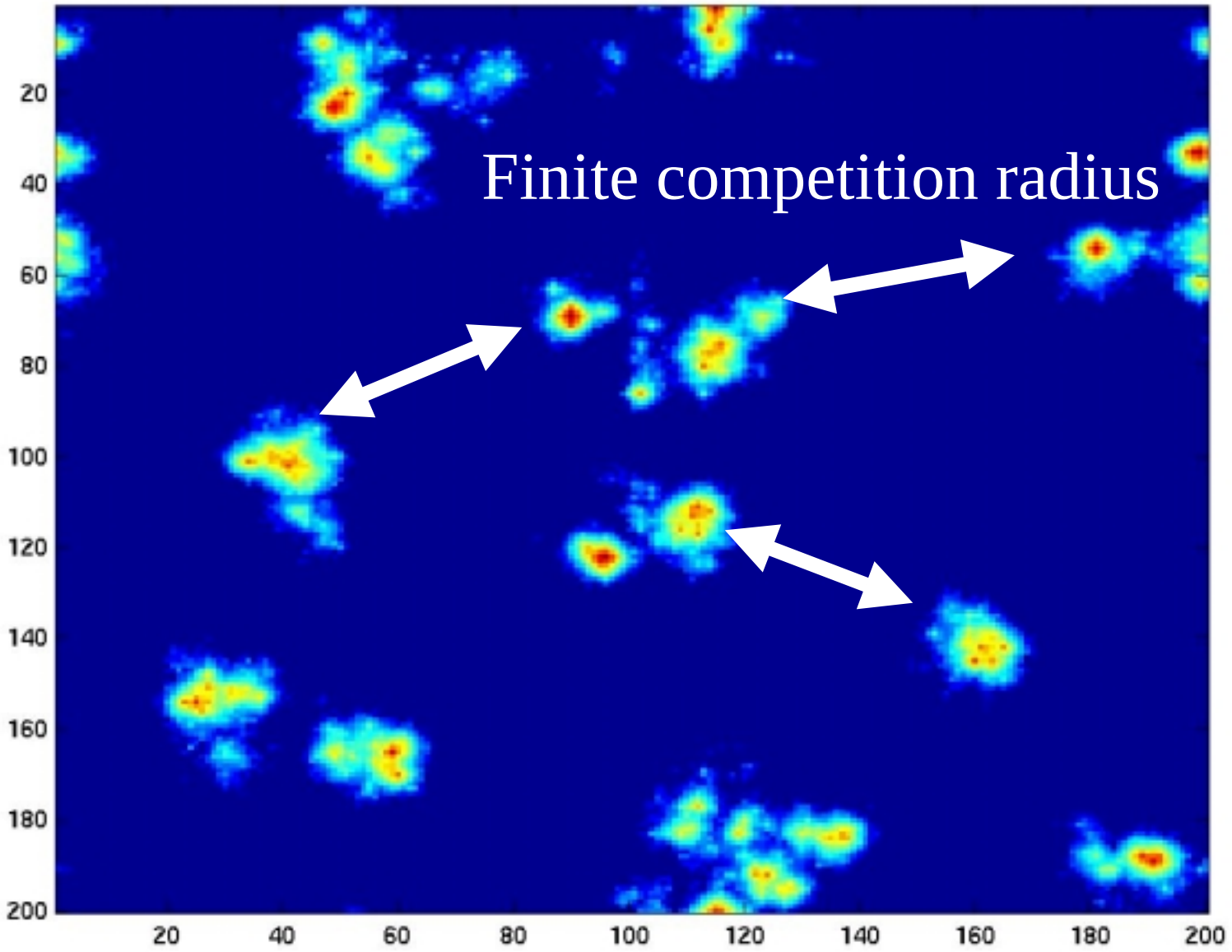

$\dot{b}(x,t) = (\lambda a(x,t) - \mu)\, b(x,t) - \gamma b(x,t)$
x 10^7
Total wealth
Time
Infinite Range (efficient but unstable)
Intermediate Range
Zero Range (unefficent but very stable)

Finite Competition Range R

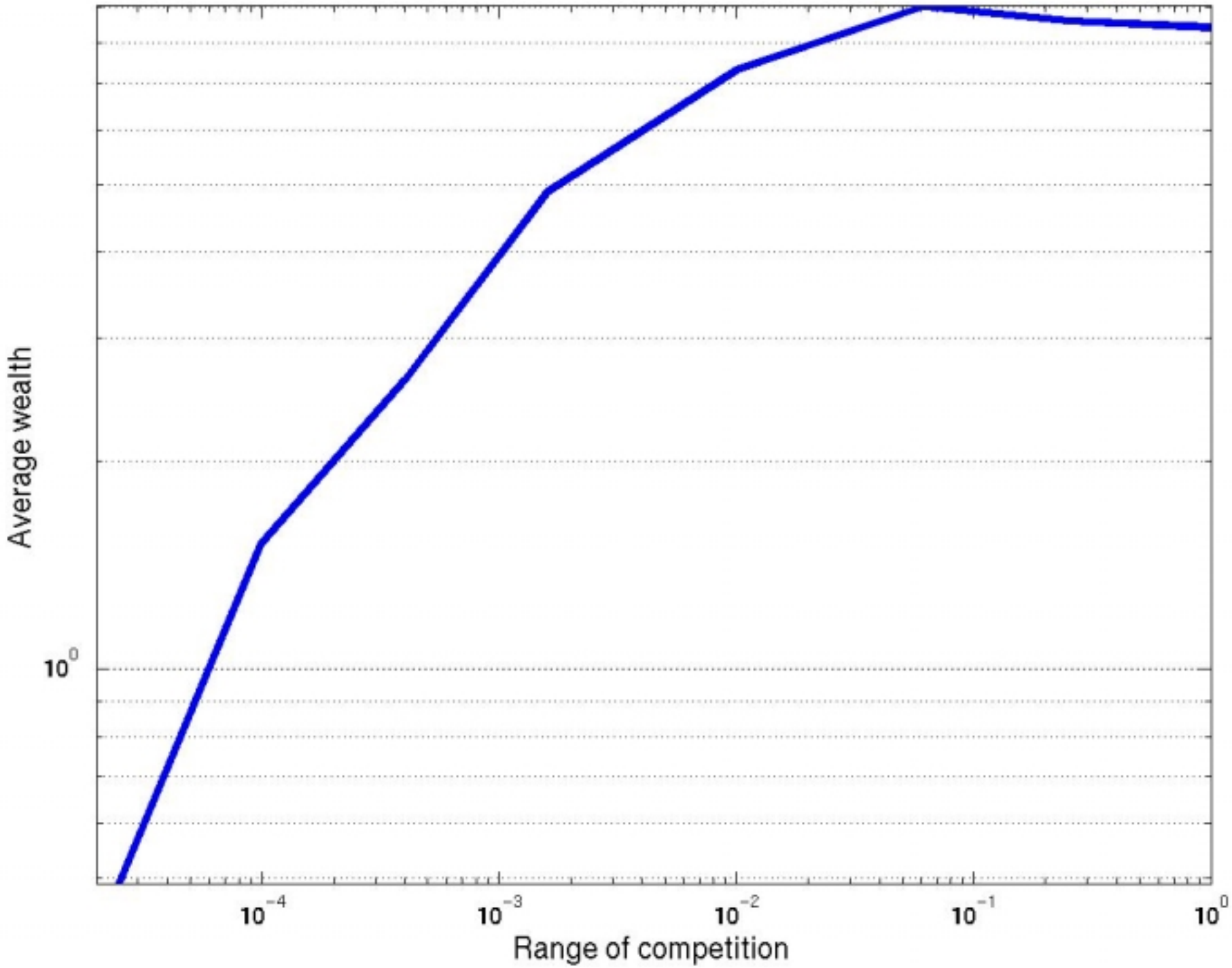
$$\dot{b}(x,t)=(\lambda a(x,t)-\mu)\, b(x,t) - \gamma b(x,t)\langle b(x,t)\rangle_R + D_b \Delta b(x,t)$$

$$\langle b(x,t)\rangle_R = \sum_{|y-x-1|<R} b(y,t) \,/\, \sum_{|y-x-1|<R} 1$$





In each region of radius R there is only one active center.
For equal γ's finite R gives greatly more B 's than R=0.
Competition increases efficiency !


Average wealth
10^0
10^{-4}
10^{-3}
10^{-2}
10^{-1}
10^0
Range of competition

One describes the dynamics of the system in terms of the probabilities of the various configurations:

$P_{nm}(x)$ **= the probability that there are m *B*'s and n *A*'s at the site x .**

The death of B's and the birth of B's in the presence of A's are represented by the first and respectively second term in the Master Equation:

$$d P_{nm} / dt = -\mu [m P_{nm} - (m+1) P_{n,m+1}]$$

$$- \lambda [mn P_{nm} - n (m-1) P_{n,m-1}]$$

Following [21] we define a set of creation-annihilation operators,

$$a^{+}|n,m\rangle = |n+1,m\rangle \quad b^{+}|n,m\rangle = |n,m+1\rangle,$$

$$a|n,m\rangle = n|n-1,m\rangle \quad b|n,m\rangle = m|n,m-1\rangle,$$

and a wave function

$$\Psi = \sum_{n,m} P_{n,m}|n,m\rangle.$$

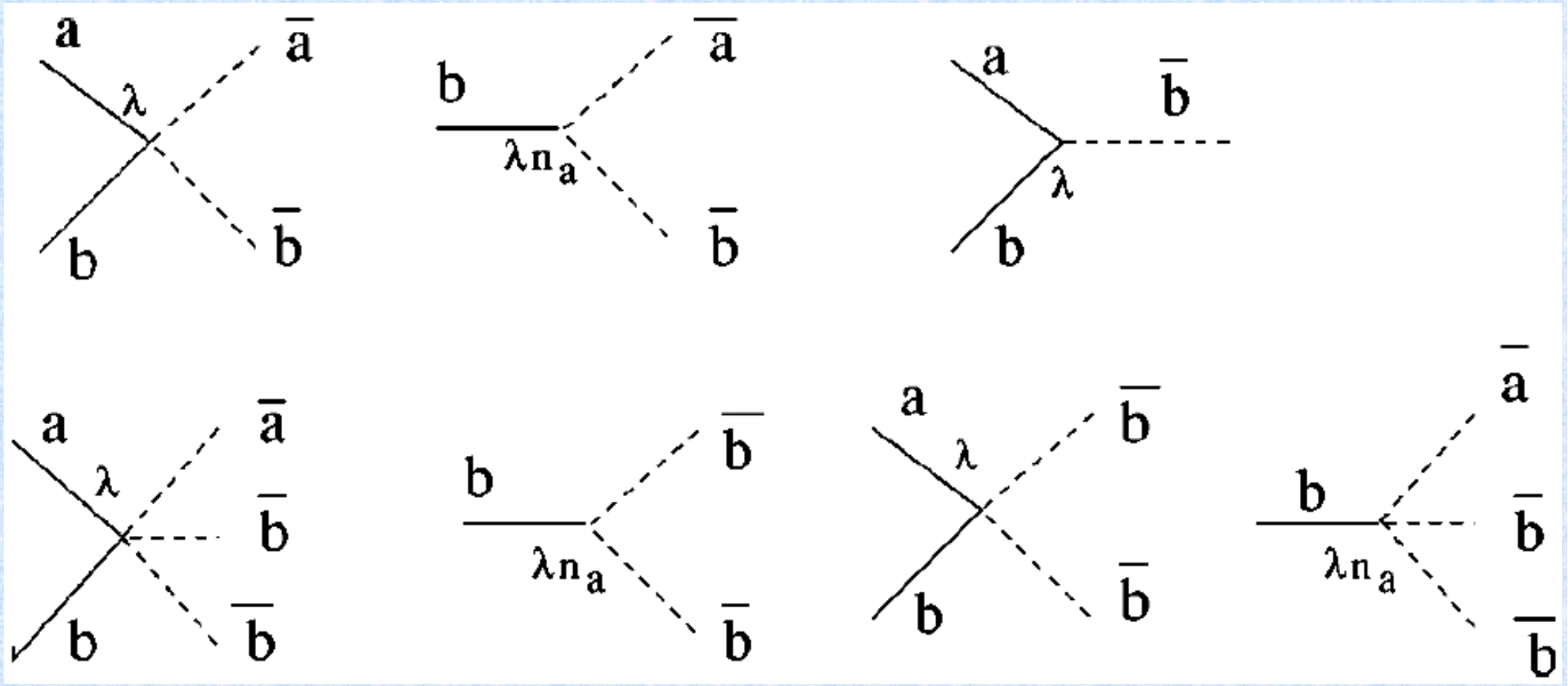
The master equation then takes the Hamiltonian form

$$\frac{\partial \Psi}{\partial t} = -H\Psi,$$

with

$$H = \sum_i \left[\frac{D_a}{l^2} \sum_{\langle e-i \rangle} a_i^+ (a_i - a_e) + \frac{D_b}{l^2} \sum_{\langle e-i \rangle} b_i^+ (b_i - b_e) \right.$$

$$\left. + \mu [b_i^+ b_i - b_i] + \frac{\lambda}{l^d} [a_i^+ a_i b_i^+ b_i - a_i^+ a_i b_i^+ b_i^+ b_i] \right]$$


a
λ
ā
b
b̄
b
λn_a
ā
b̄
a
b̄
λ
b
a
λ
ā
b
b̄
b
λn_a
b̄
b̄
a
λ
b̄
b
b̄
b
λn_a
ā
b̄
b̄

$$x \to s x$$

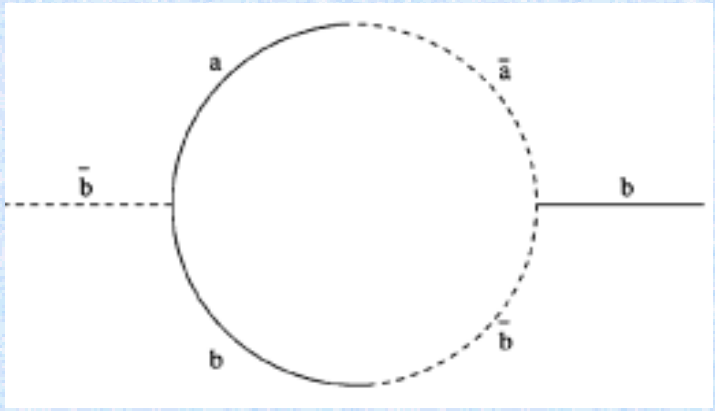
$$t \to s^z t$$

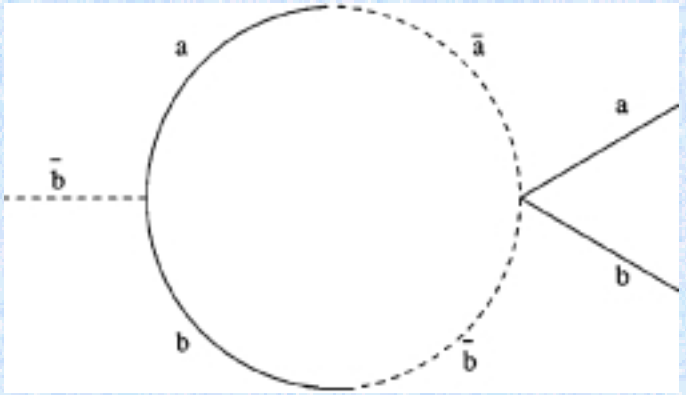
$$a \to s^{-d-\eta} a$$

$$\Lambda \to \Lambda / s$$

$m = \mu - n_a \lambda$

λ





$$\frac{dm}{d\ln(s)} = 2m - \frac{\lambda^2 n_a}{2\pi D} \frac{\Lambda^{d-2}}{1 + \frac{m}{D\Lambda^2}}$$

$$\frac{d\lambda}{d\ln(s)} = \epsilon\lambda + \frac{\lambda^2}{2\pi D} \frac{\Lambda^{d-2}}{1 + \frac{m}{D\Lambda^2}}$$

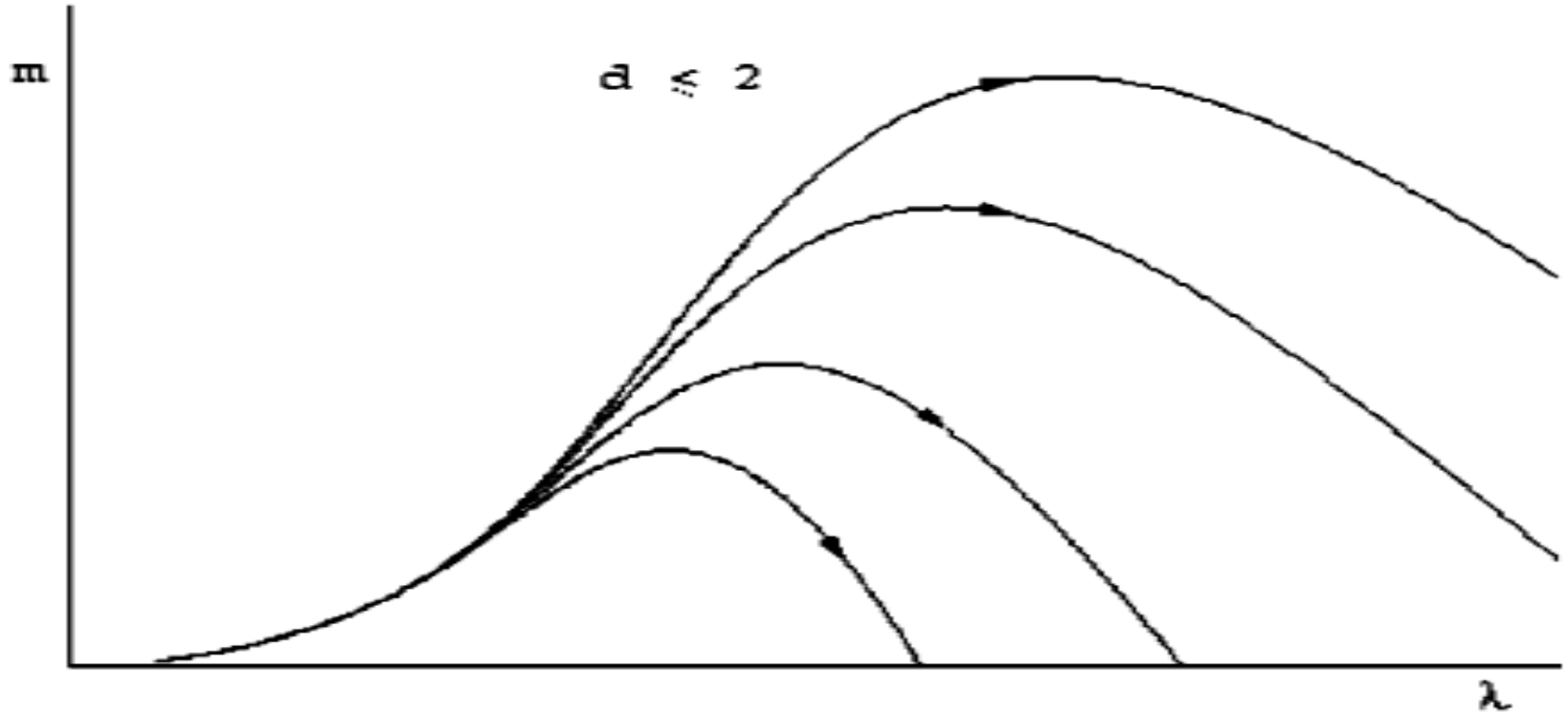


FIG. 4. RG flow lines at the continuum limit for $d \leqslant 2$ (in arbitrary units). While at short times m grows, the system flows into its active phase $m < 0$ on large time scales.

$$\frac{d\lambda}{d\ \ln(s)} = \lambda\left(\epsilon + \frac{\lambda}{2\pi D}\right)$$

$$\frac{dm}{d\ \ln(s)} = 2m - \frac{\lambda^2 n_0}{2\pi D}$$

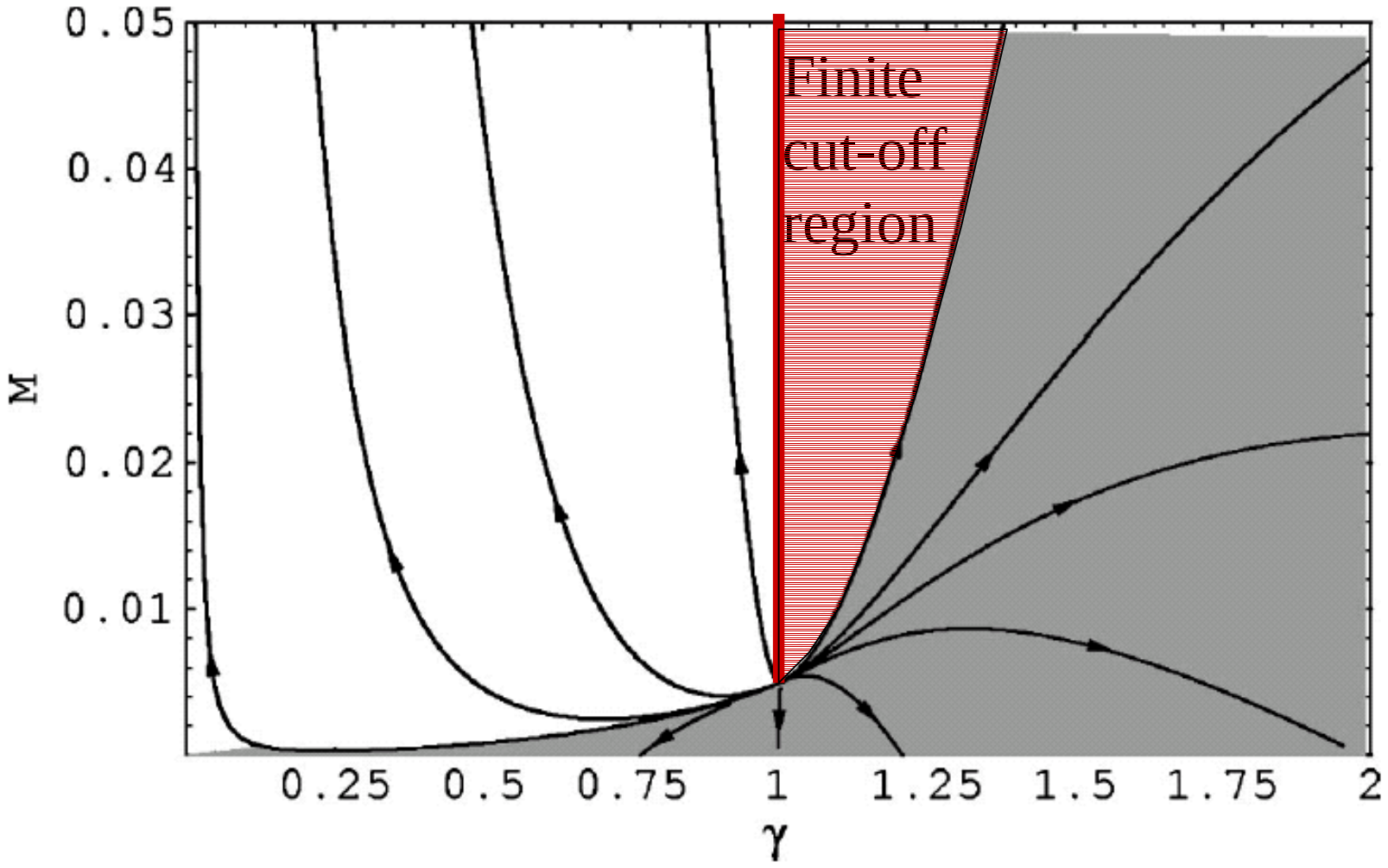


FIG. 5. Renormalization flows for $d>2$. In the shaded region, the system flows into the active phase, while in the unshaded region, the system flows to the inactive phase ($m\rightarrow\infty$).

In conclusion, our results suggest that the dimensionality of the system and its size are crucial features for its capability to emerge and sustain life. This may explain the fact that most ecological systems are two-dimensional.

Reinterpreting in the genome space, the present results provide the conceptual link between the atomized structure of the life building blocks and the explosive Darwinian tandem, noise + proliferation

John Beringer (microbial biology, Bristol U):

"Microbes that need oxygen will be found

close to the **surface** of soil, and

microbes that are very fastidious about oxygen concentration

will be found in **bands**

at the appropriate oxygen concentration."

Microbes concentrating on a **two-dimensional resource**

may have been more successful than

their cousins who tried exploiting a **three-dimensional** feast.

Finite cut-off

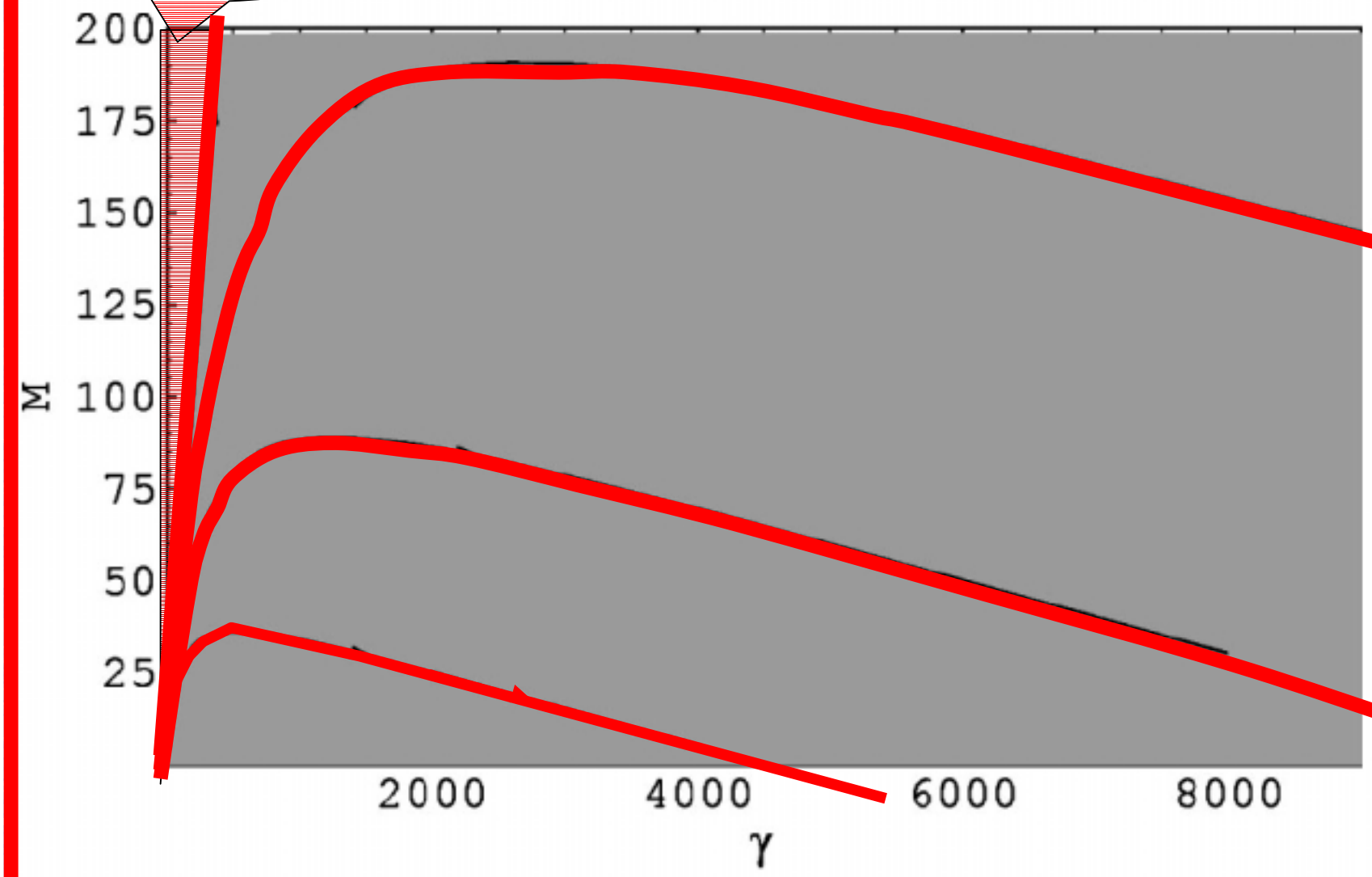
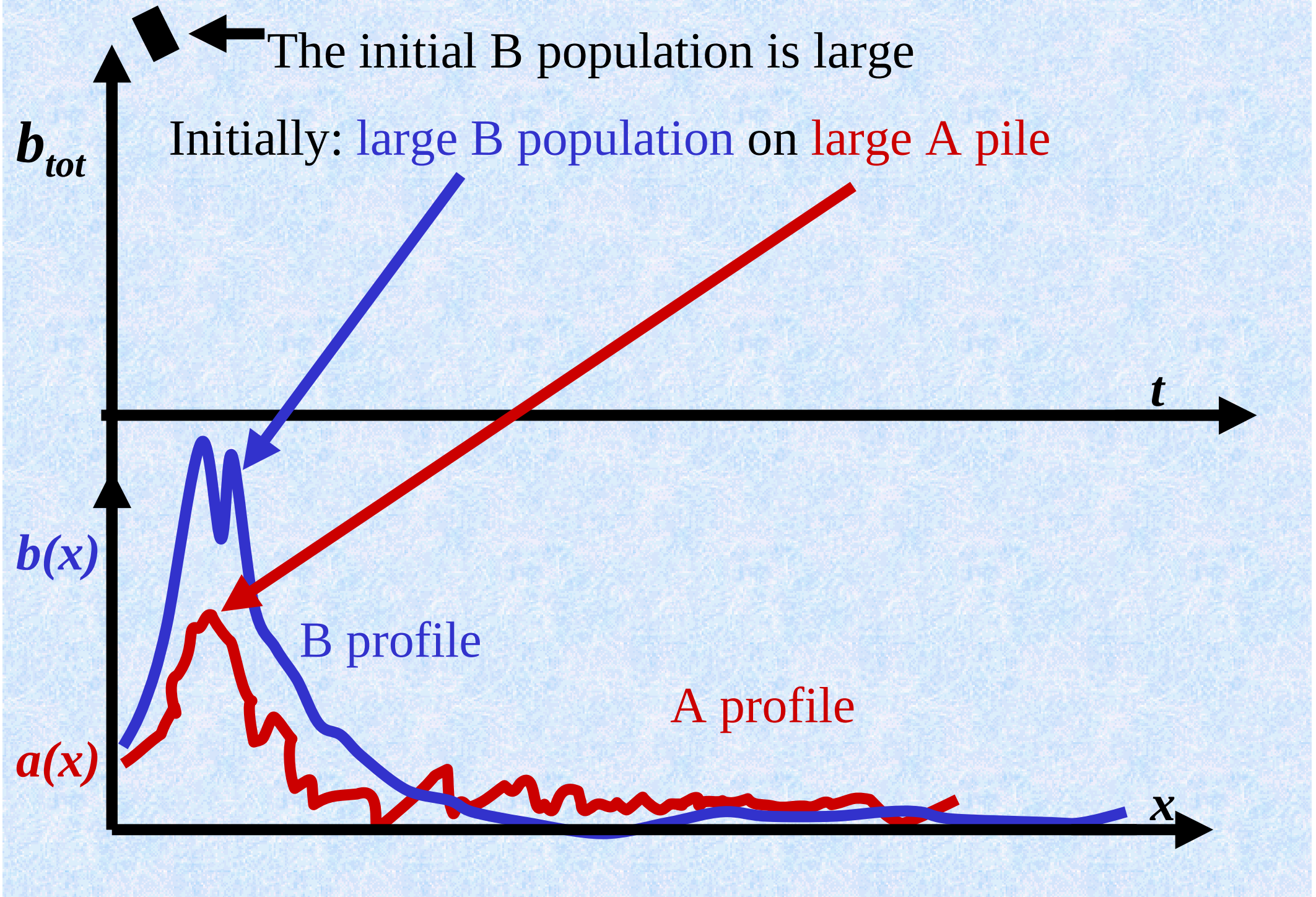
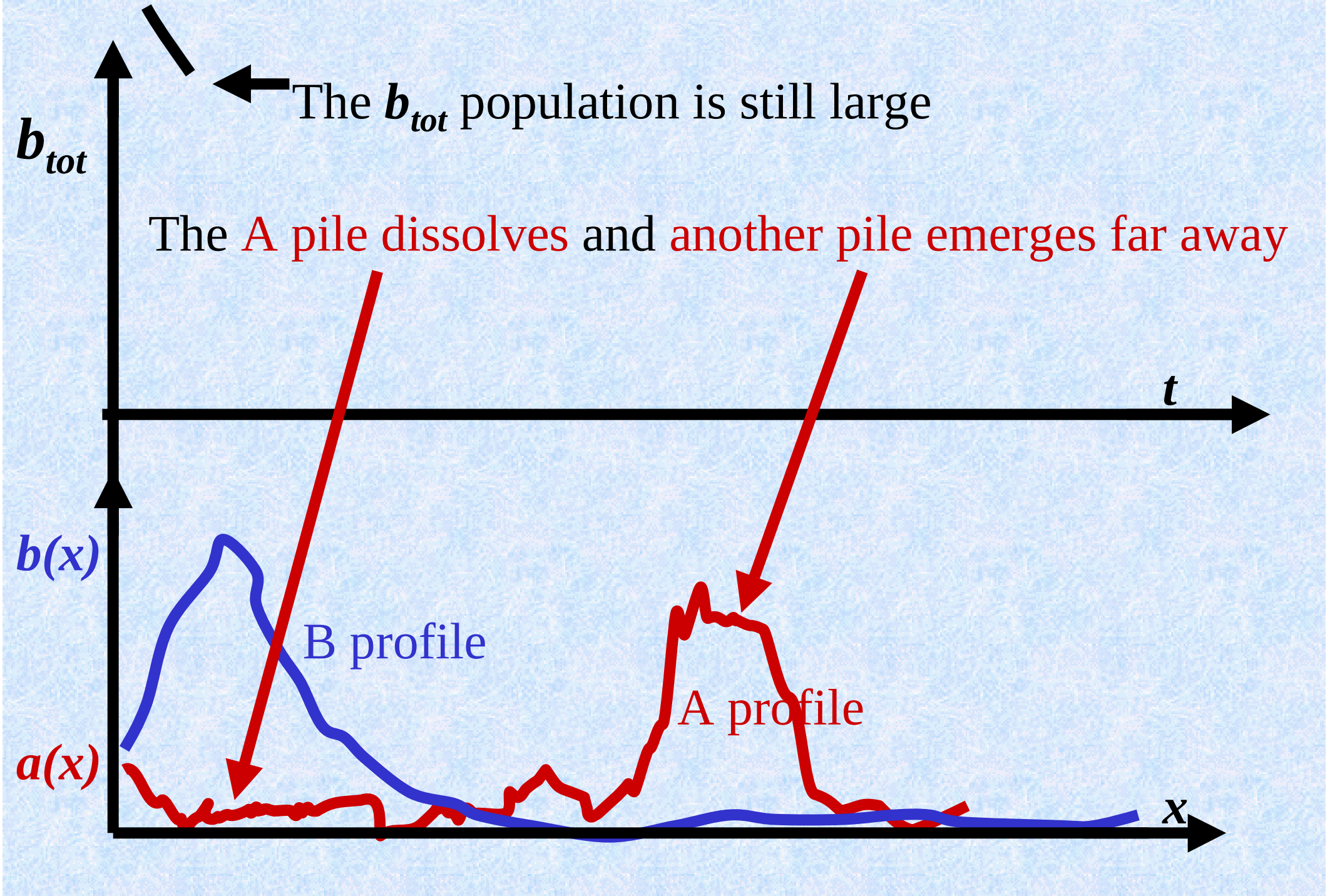
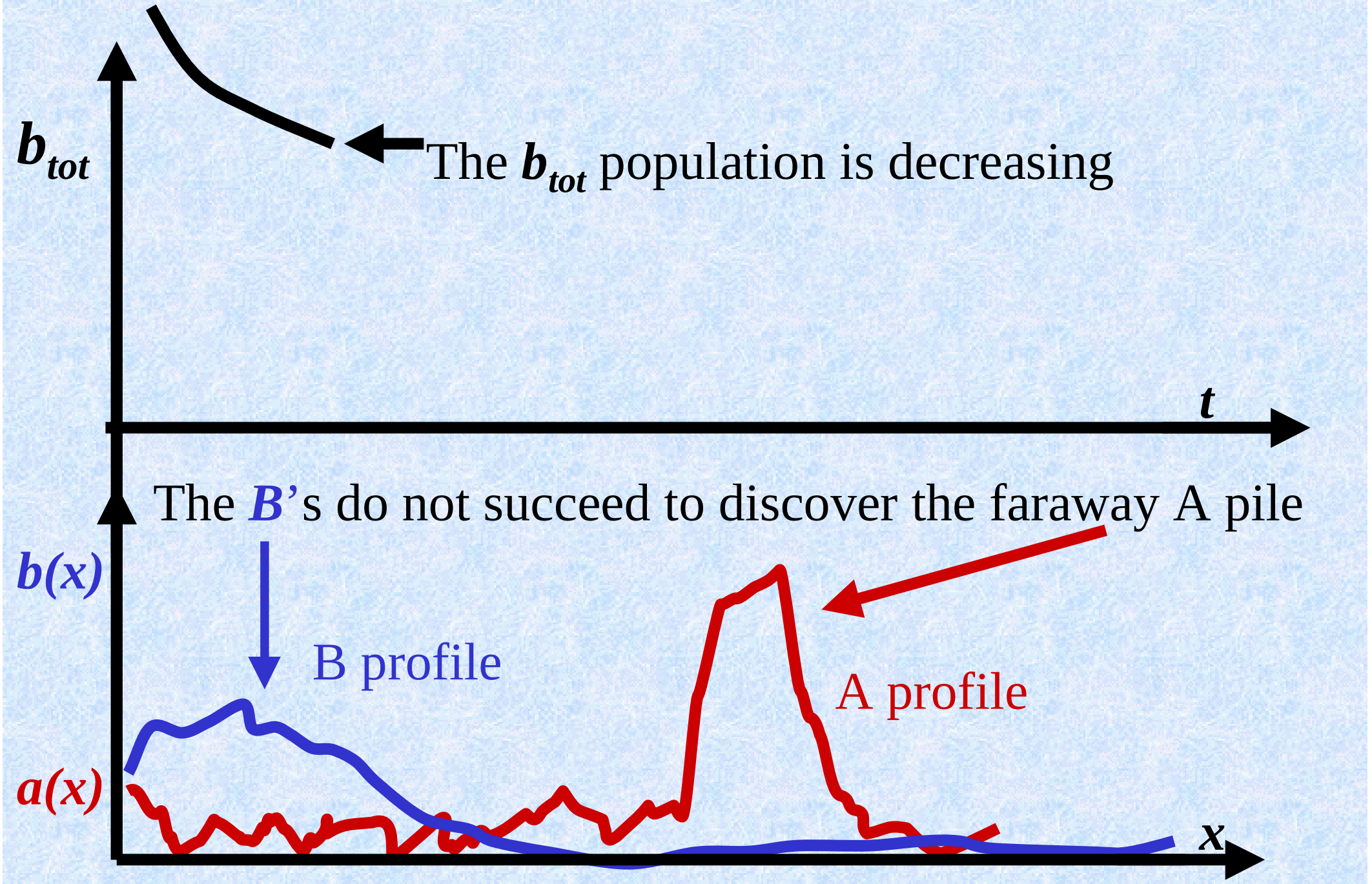
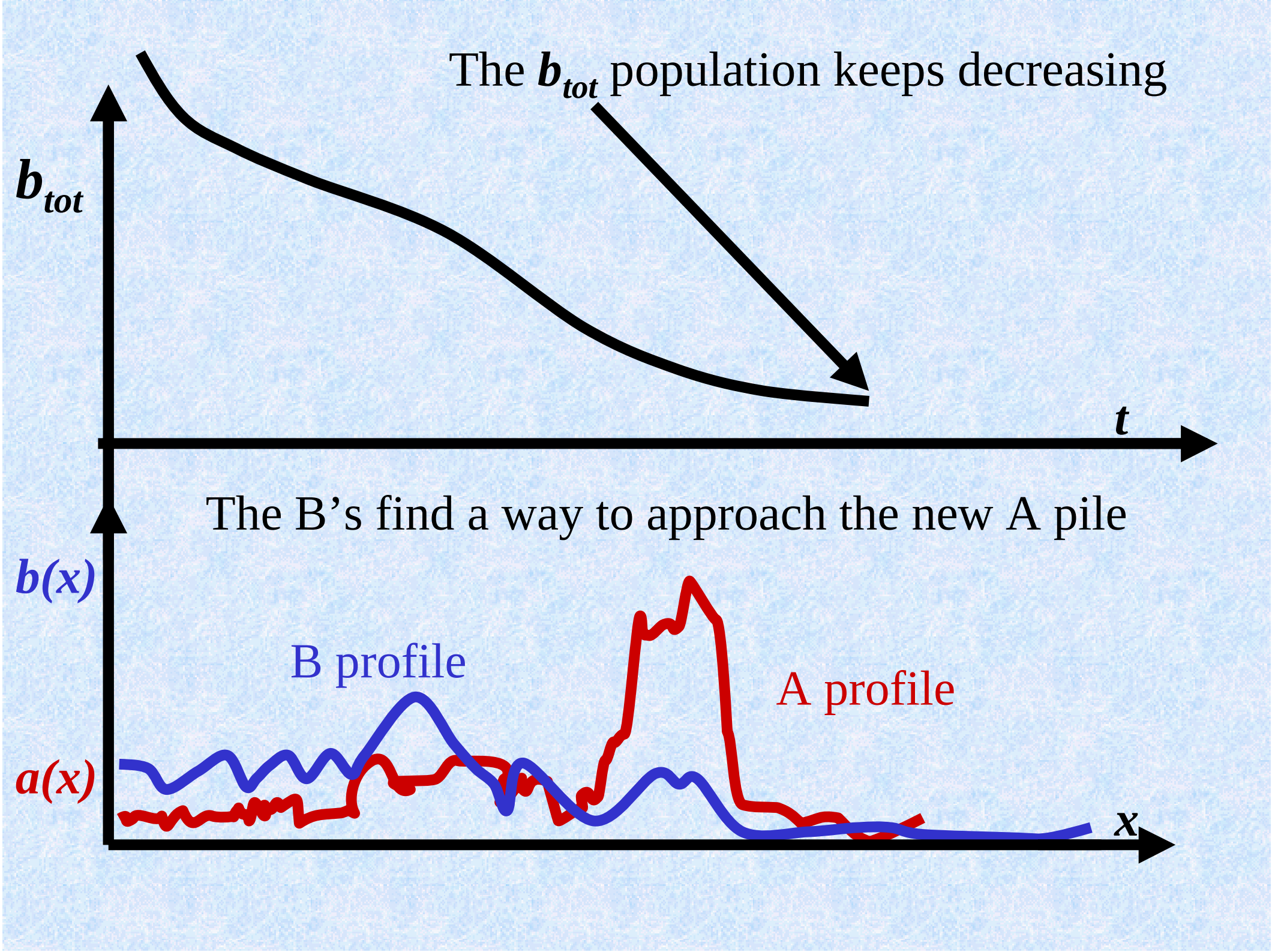


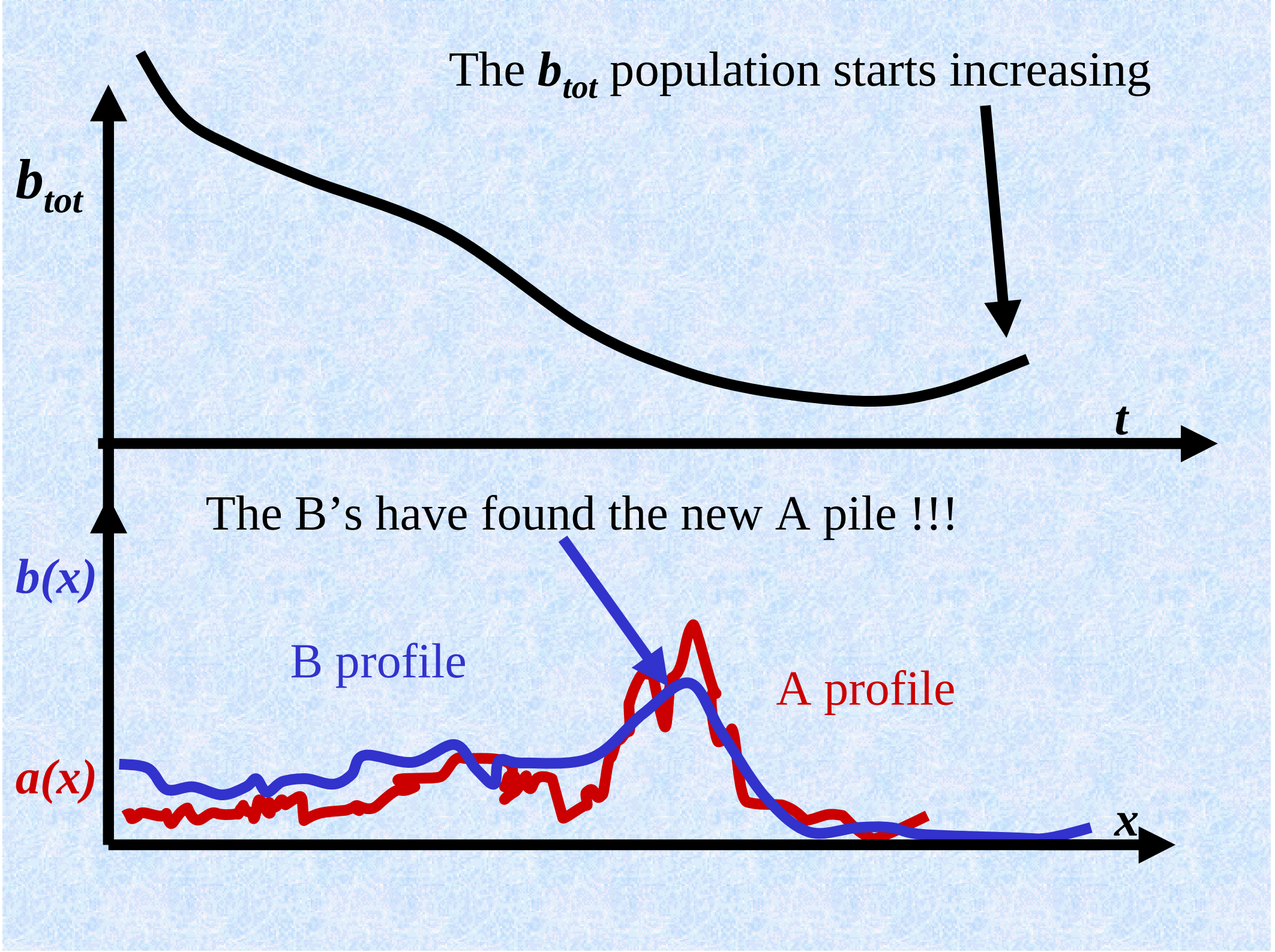
FIG. 6. Renormalization flows for $d=2$ at finite lattice spacing. Unlike Fig. 4, here there is a region in parameter space (unshaded) where the extinction phase is stable.

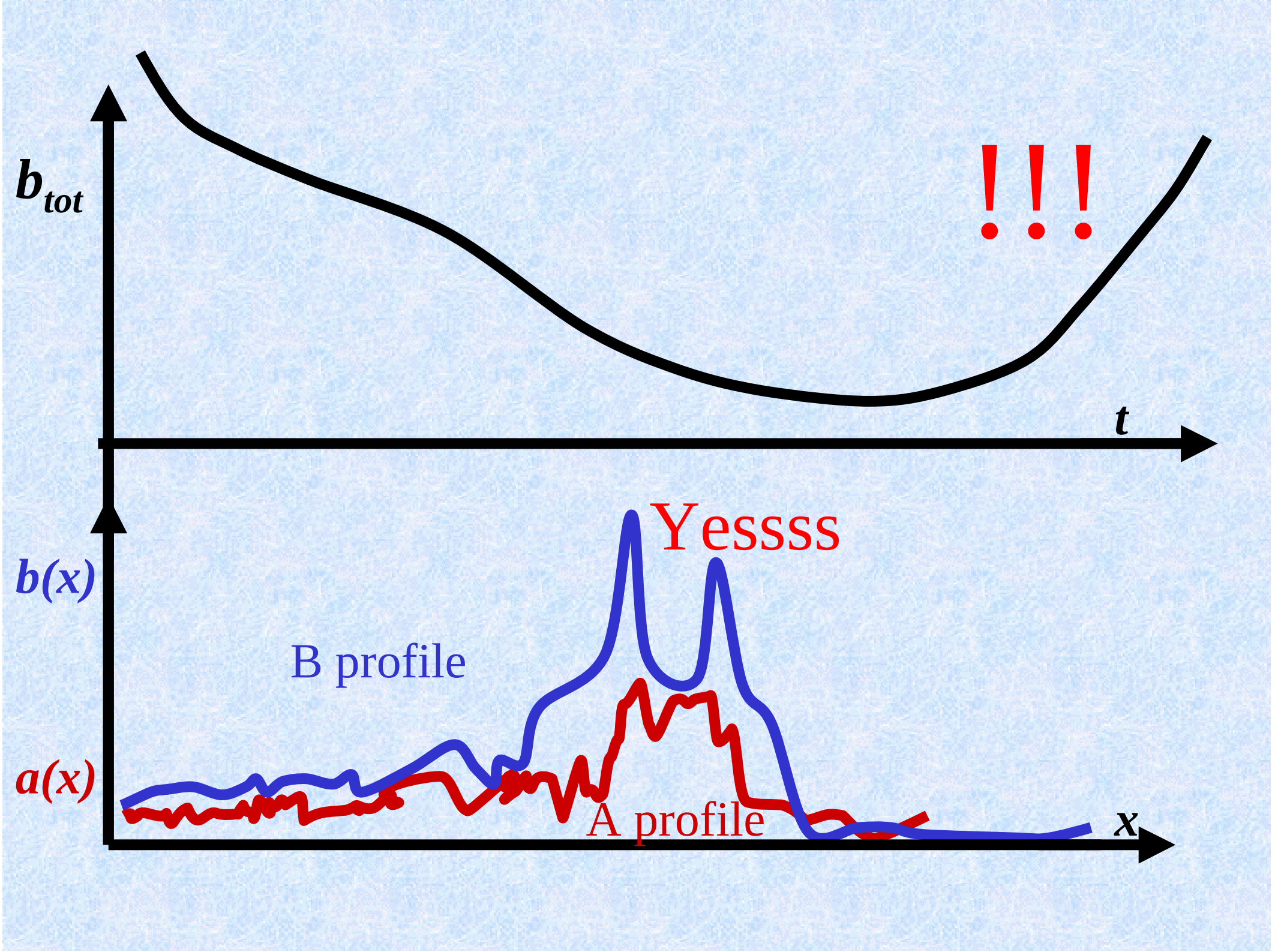

The initial B population is large
Initially: large B population on large A pile
b_{tot}
t
b(x)
B profile
A profile
a(x)
x

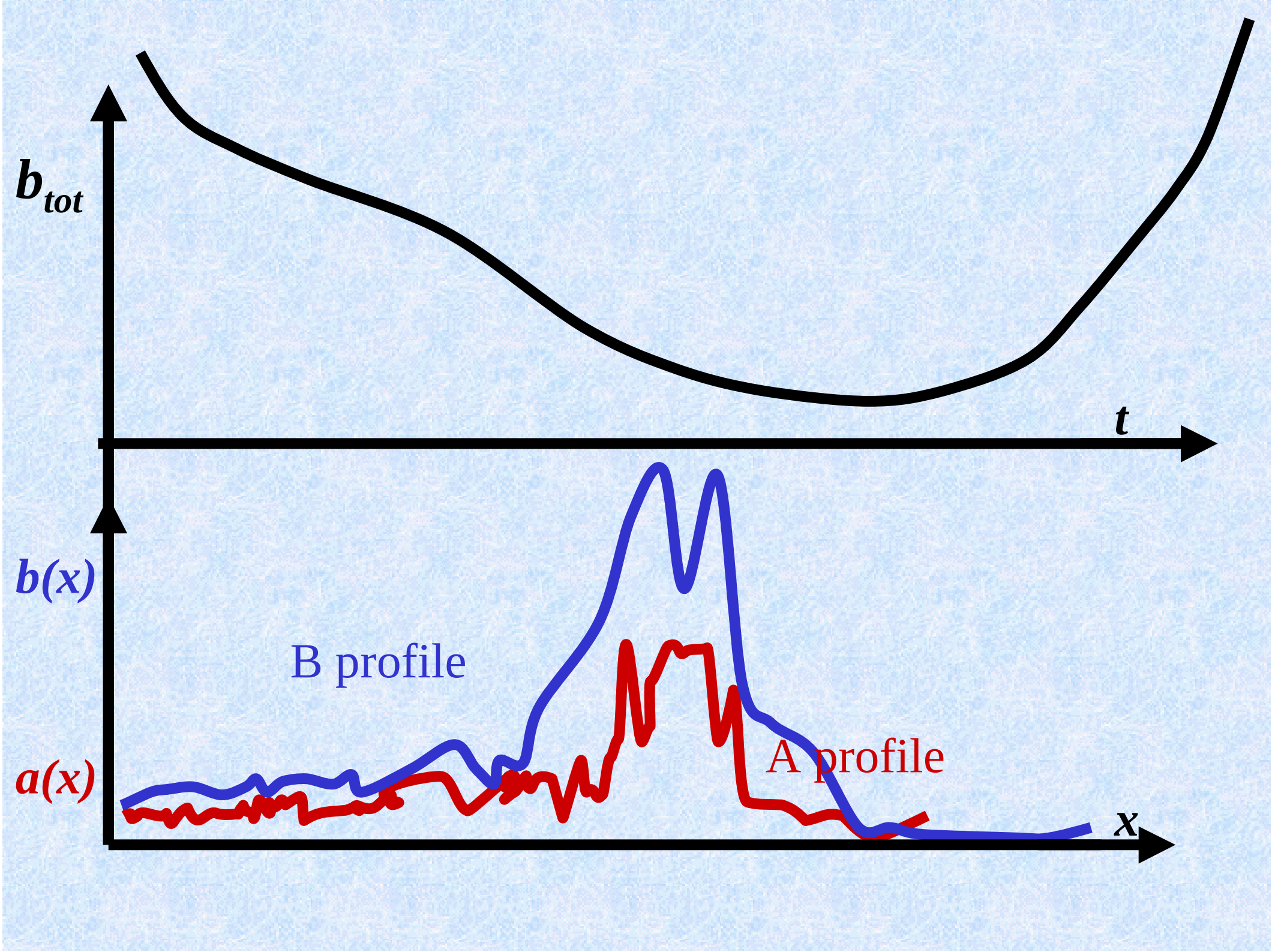

The b_{tot} population is still large
b_{tot}
The A pile dissolves and another pile emerges far away
t
b(x)
B profile
A profile
a(x)
x


b_{tot}
The b_{tot} population is decreasing
t
The B's do not succeed to discover the faraway A pile
$b(x)$
B profile
A profile
$a(x)$
x


The b_{tot} population keeps decreasing
b_{tot}
t
The B's find a way to approach the new A pile
$b(x)$
B profile
A profile
$a(x)$
x


The b_{tot} population starts increasing
b_{tot}
t
The B's have found the new A pile !!!
$b(x)$
B profile
A profile
$a(x)$
x


b_{tot}
!!!
t
b(x)
Yessss
B profile
a(x)
A profile
x


b_{tot}
t
b(x)
B profile
A profile
a(x)
x

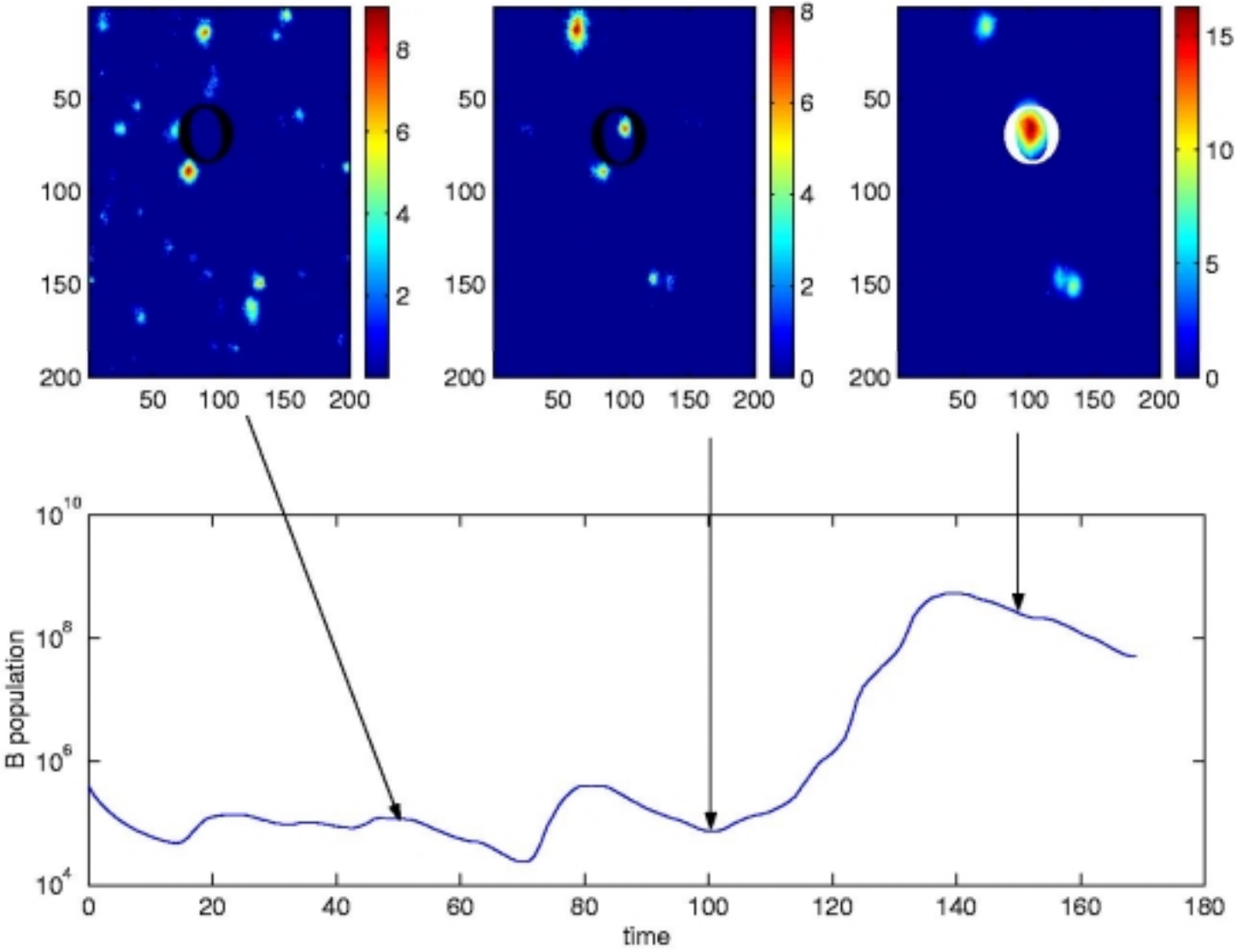


Figure 5: B dynamics with in the regime of high A density, mid values of A and B diffusion, $\lambda < \mu$ and $\lambda A < \mu$. The upper plots represents snapshots of the log of the B population at the times pointed by the arrows.

The necessity for **B to find a way to the new A pile** emphasizes the difference between

- random (space time-**uncorrelated) noise** – where **A**'s would disappear from one site and would reappear in another **randomly uncorrelated site**.

and

-diffusive (space-time **correlated) noise** – where **A** just jumps between **neighboring sites** and allows (descendents of) the B's to follow them.

Continuity of the random hostile environment in time is a crucial feature for adaptability and life emergence.

For d=3 : $\lambda > D_a \ln 1/0.34$;

d → ∞: $\lambda > D_a \ln 2d$

Returning to 2D note that the probability that the conditions leading to growth are fulfilled

[all $a(x,t)$ particles **A** keep returning to x at least after each interval τ]

decays as:

$$[P_d(\tau)]^n \, a(x,0) \sim e^{(-\lambda / D_a + \eta)\, t/\tau \, \tau(\mu + D_b)/\eta}$$

Taking $\eta \sim \lambda / 2D_a$**: this is**

$$P_d(\tau)^n \, a(x,0) \sim e^{-t \, \lambda (\mu + D_b) / (\eta D_a)} \sim e^{-t (\mu + D_b)}$$

Less and less locations create more and more population!!!

One may be a bit confused of using differential equations notation to obtain results that are incompatible with the equations.

The point is that for continuous uniform $a(x,t)$ the solution for b is uniform too (and vanishes uniformly for $\lambda a_0 - \mu < 0$).

For discrete uniformly (Poisson) distributed A's, there are always sites with larger number of A's and in particular with $a(x,t) > \mu / \lambda$.

What we found is that in the case of the global competition, if there is a site with the largest A population:

$$a(x_{max},t) > a(x,t) \quad \forall x \neq x_{max}$$

Then all the other neighbourhoods are depleted of B's.

The continuum equations are NEVER correct for the AB model with global competition (even for $(\lambda a_0 - \mu > 0)$.

For Experts (in such a big room I have to ask the usual questions) Don't look for cheap escapes:

Q - slow $a(x,t) \rightarrow a_0$ convergence:

A - **enough** $a(x,t) < \mu/\lambda$, $\forall x$ for **decay ;**

- $a(x,t)$ **starts** uniform.

Q- non-linear features in PDE

$$b^{\bullet} = (a\,\lambda - \mu)\,b + D_b\,\Delta\,b$$

A- the equation **is linear** in ***b!; b*** does not react on ***a!***

Q - instability of the homogenous $b(x,t) = b(0,t)$ solution:

A- The homogenous solution **is stable** for $\lambda\,a_0 - \mu < 0$

Once a continuous $a(x,t)$ is accepted,

the death sentence for $\lambda\,a_0 - \mu < 0$ is **unavoidable**

Continuous functions ***a(x,t)*** and ***b(x,t)*** just do not represent faithfully the **system of particles A and B** **even for infinite number of particles per unit area (i.e. in the "continuum limit").**

The discreteness effects **do not fade away** (in fact increases) when one has more and more particles per site : $\langle a \rangle \rightarrow \infty$.

Even (especially**!**)

the rarest and most spatially restricted (singular) microscopic fluctuations in **A**
are amplified by the proliferation in **B.**

Again for experts

Q- Discreteness / microscopic fluctuations were known to

-influence the **approach** to the equilibrium state
(e.g. Fisher waves; Cardy annihilation)

-shift the exact value of a phase transition point.
So what is the novelty?

A- Here the very **character of the final state is affected:**

Discreteness makes the difference between life and death.

Behavioral Finance

B= financial traders,

Patches= "**herding**" behavior

(despite the fact that we do not introduce communication between B's)..

May explain some paradoxes in **finance E.g**

-between the efficient market hypothesis (absence of systematic profit opportunities in equilibrium markets) and

-the actual profits that investors extract daily from the market.

Proof of the Stronger Result:

Probability for **A to stay** in one visit at x exactly a time *t* is:

$$e^{-t\,D_a}\,D_a\,dt$$

The growth factor to B that such a stay implies is

$$e^{t\,\lambda}$$

The expected factor is then

$$\int e^{(\lambda - D_a)\,t}\,D_a\,dt = 1/(1-\lambda/D_a)$$

Taking into account the possibility of multiple visits like in the main proof, this gives the condition of life:

$$P_d(\infty)/(1-\lambda/D_a) > 1$$

i.e. $\lambda > D_a(1-P_d(\infty))$

When Physics Meets Biology *By David Bradley* (Physics Direct)

Antibodies (**B**) attach themselves to virus particles, which allows immune cells to mop them up.

An antibody has to have just the right sequence to grab hold of a particular strain of the virus, so the immune system generates antibodies at random until one fits.

Then a flood of similar antibodies are produced (B+A→ B+B+A) obliterating that viral strain.

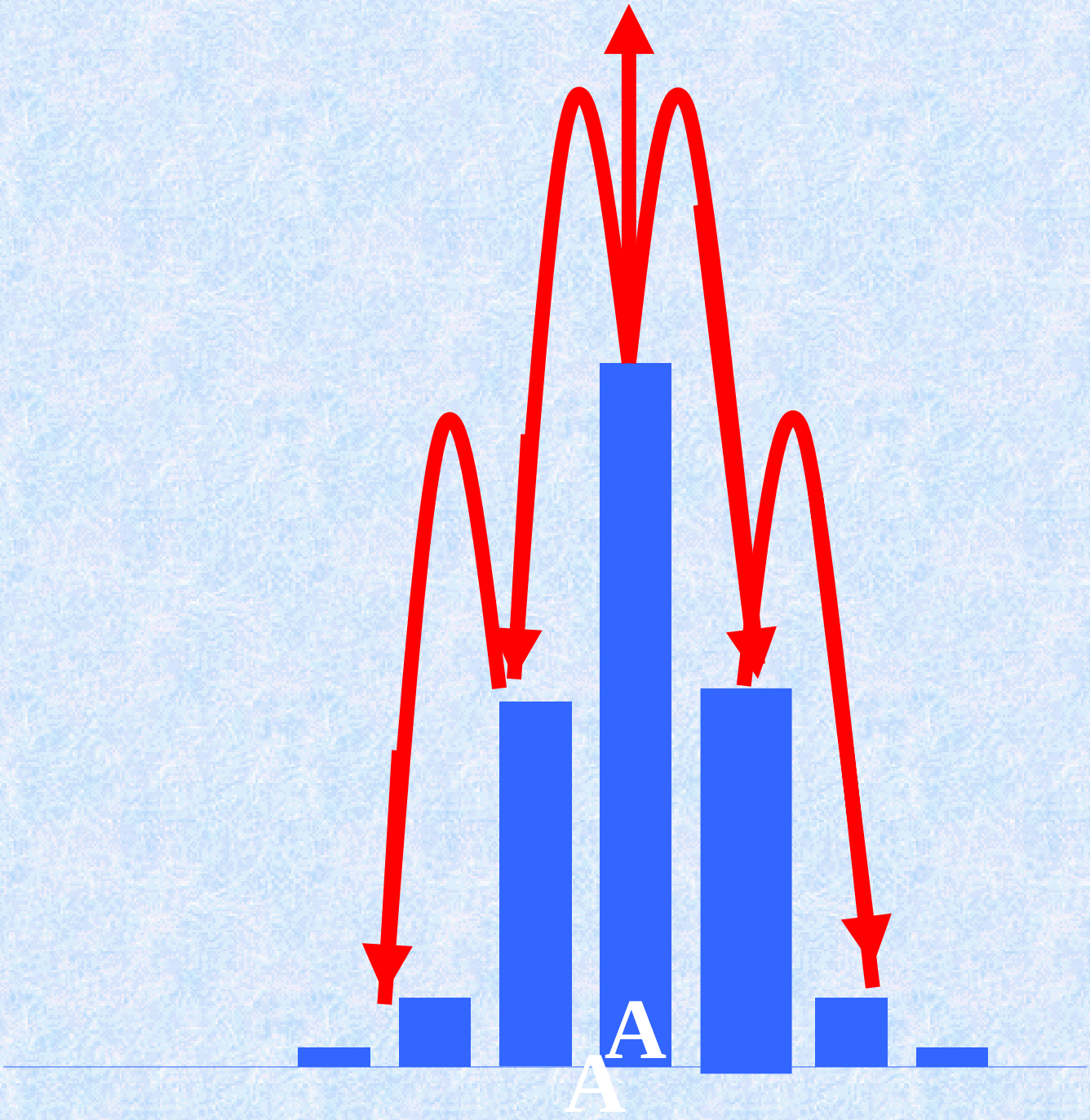
But HIV mutates rapidly. You can imagine strains of virus wandering around in an abstract genetic space as they mutate.

Every strain will eventually encounter a deadly antibody, and then the game's up for that strain. If just more than one virion mutates appropriately from each population, the islands of virus proliferate.

The immune system wins in every confrontation with any particular HIV strain, but as the mutant strains become more numerous, the immune system eventually collapses under their collective pressure."

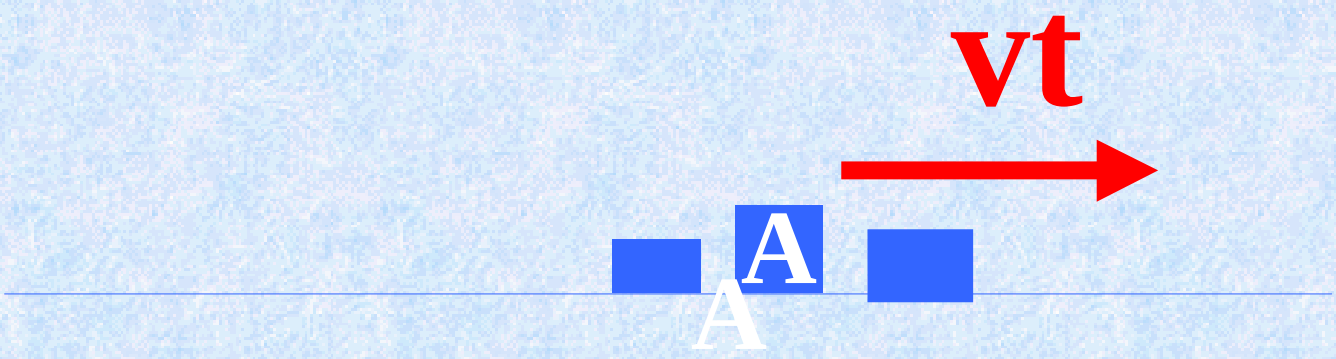
-After several hundred such battles, the number of small 'undetectable' strains become very numerous. Although each is too small to be detected, they are numerous enough in total to destroy a large number of immune cells. At this point one enters the third phase of the disease: HIV infection becomes AIDS.

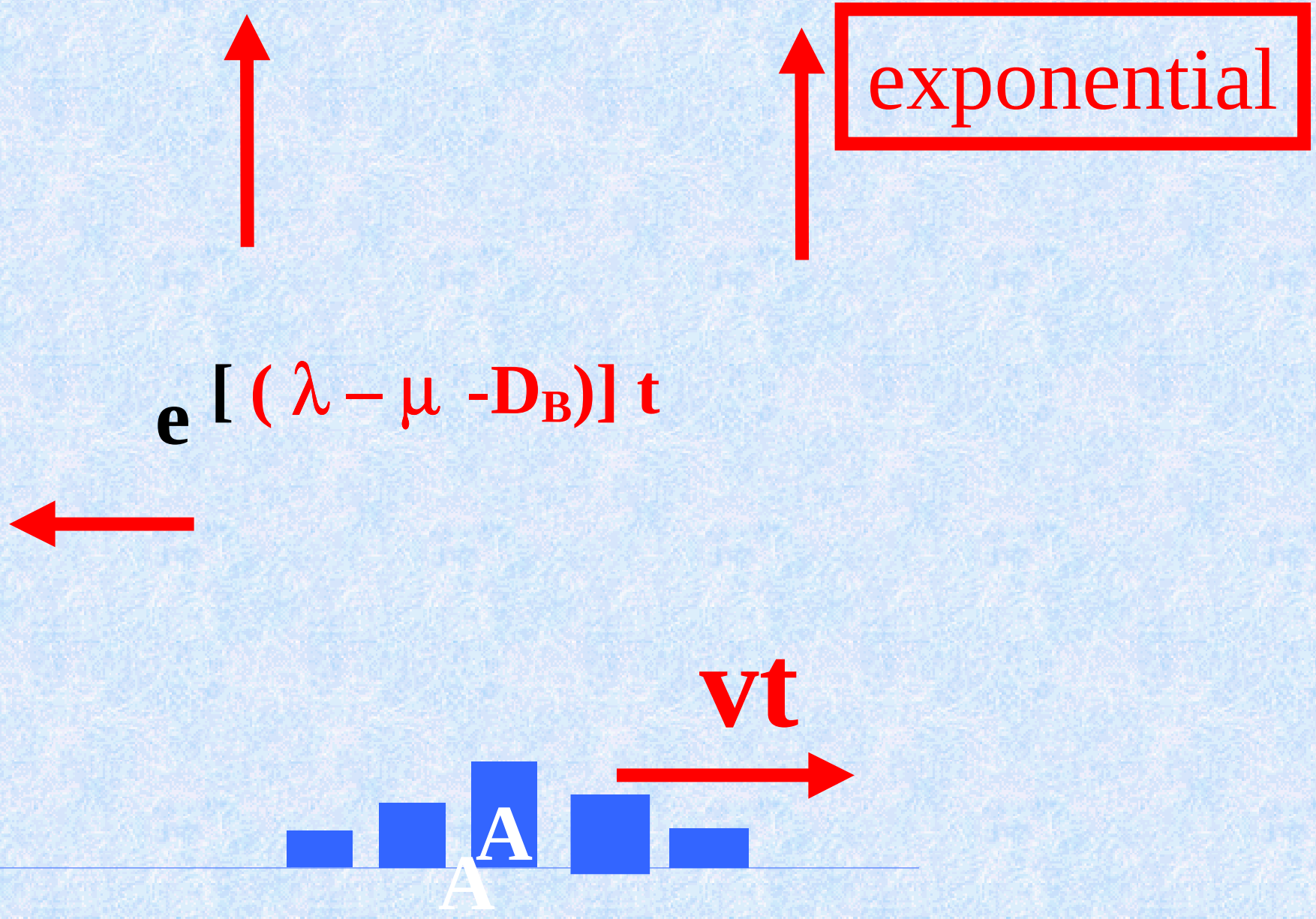
- Our study is already being confirmed experimentally by independent research.


A
A

exponential

$$e^{[\ln (D_B/\lambda)\, D_A + (\lambda - \mu - D_B)]\, t}$$

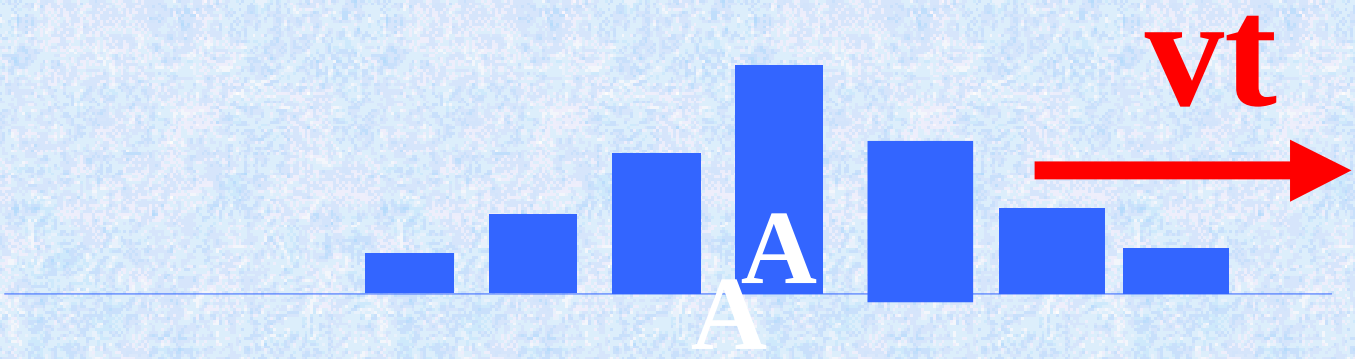



exponential
e [(λ – μ -DB)] t
vt
A
A

$$e^{[\ln (D_B/\lambda)]\, t}$$

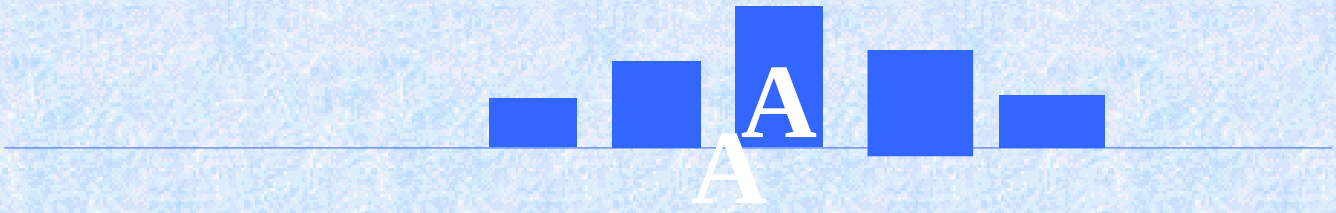
exponential

$e^{[(\lambda - \mu - D_B)]t}$



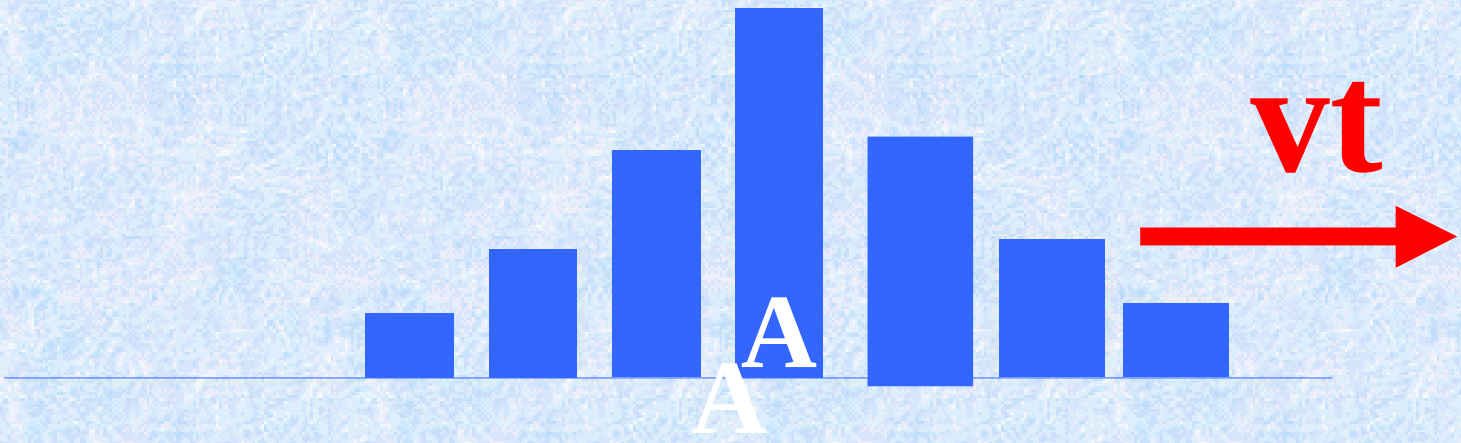
exponential

$$e^{[\ln (D_B/\lambda)\, D_A]\, t}$$



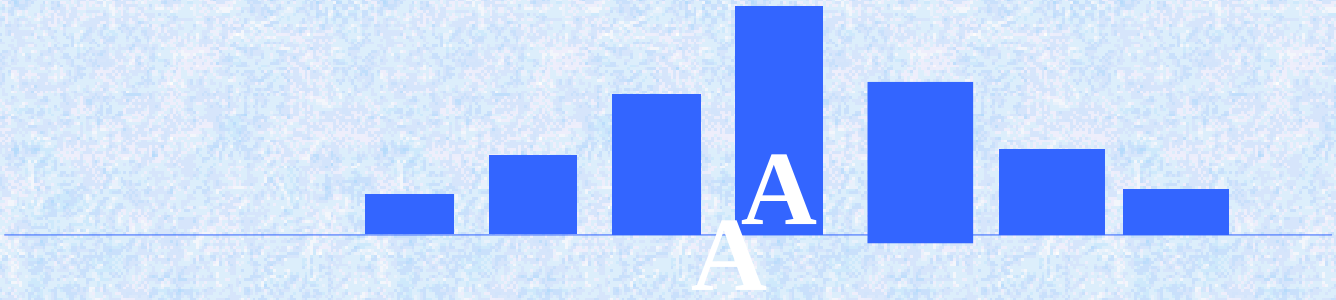
exponential

$e^{[(\lambda - \mu - D_B)]t}$



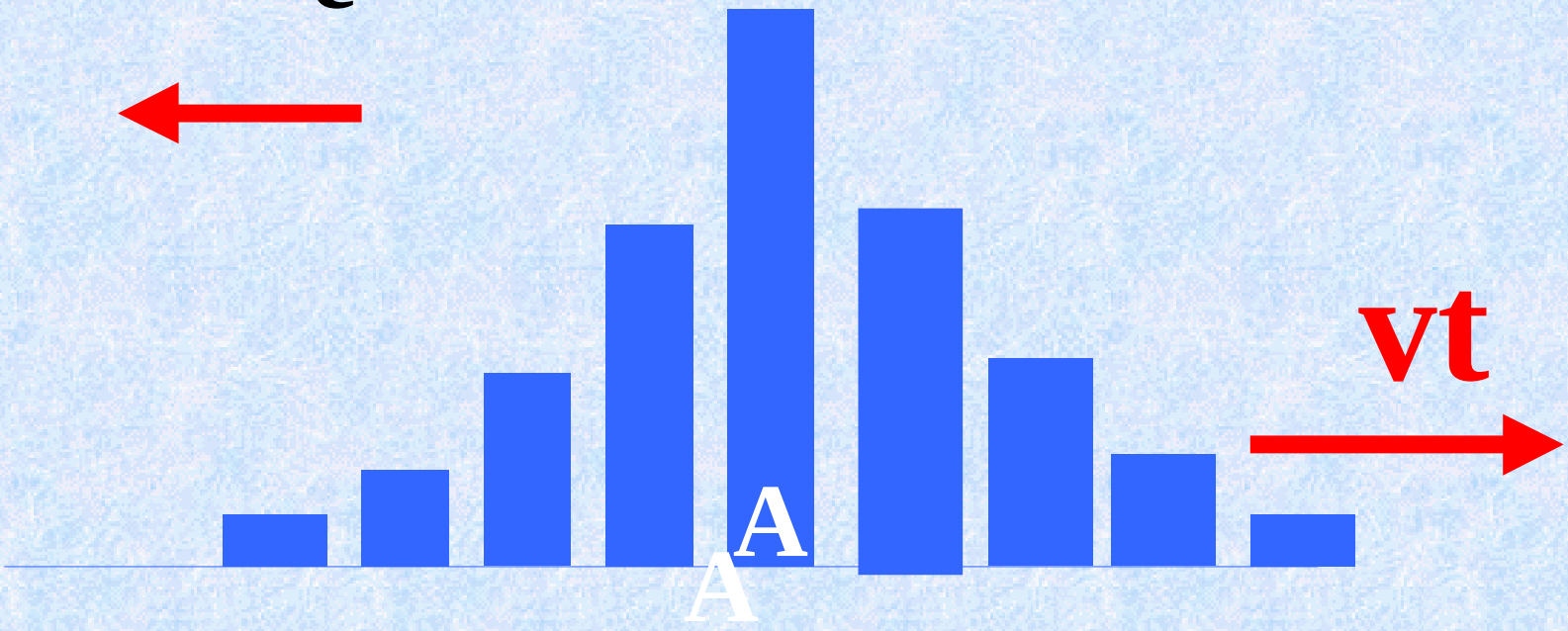
exponential

$$e^{[\ln (D_B/\lambda)\, D_A]\, t}$$



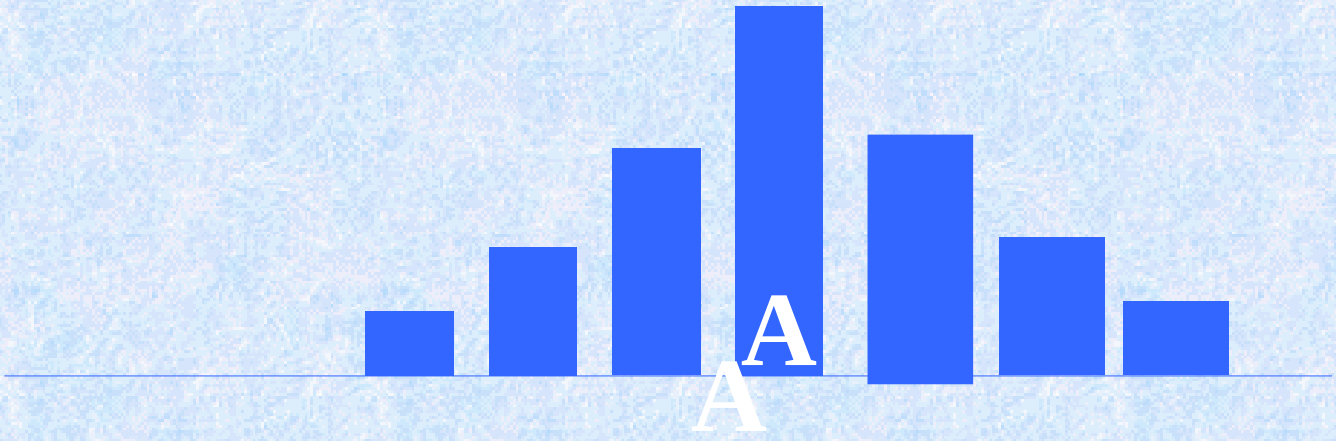
exponential

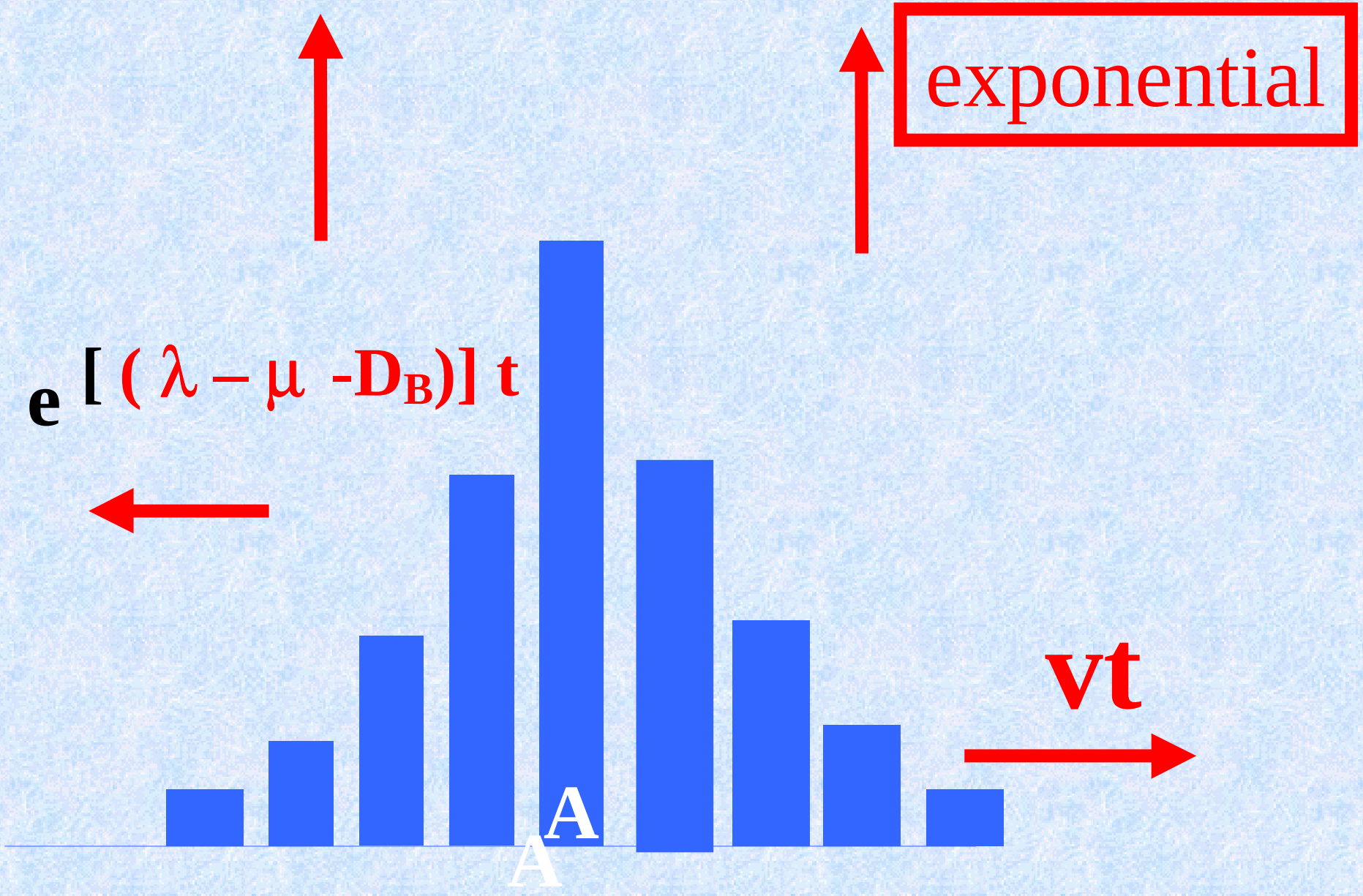
$e^{[(\lambda - \mu - D_B)]t}$



exponential

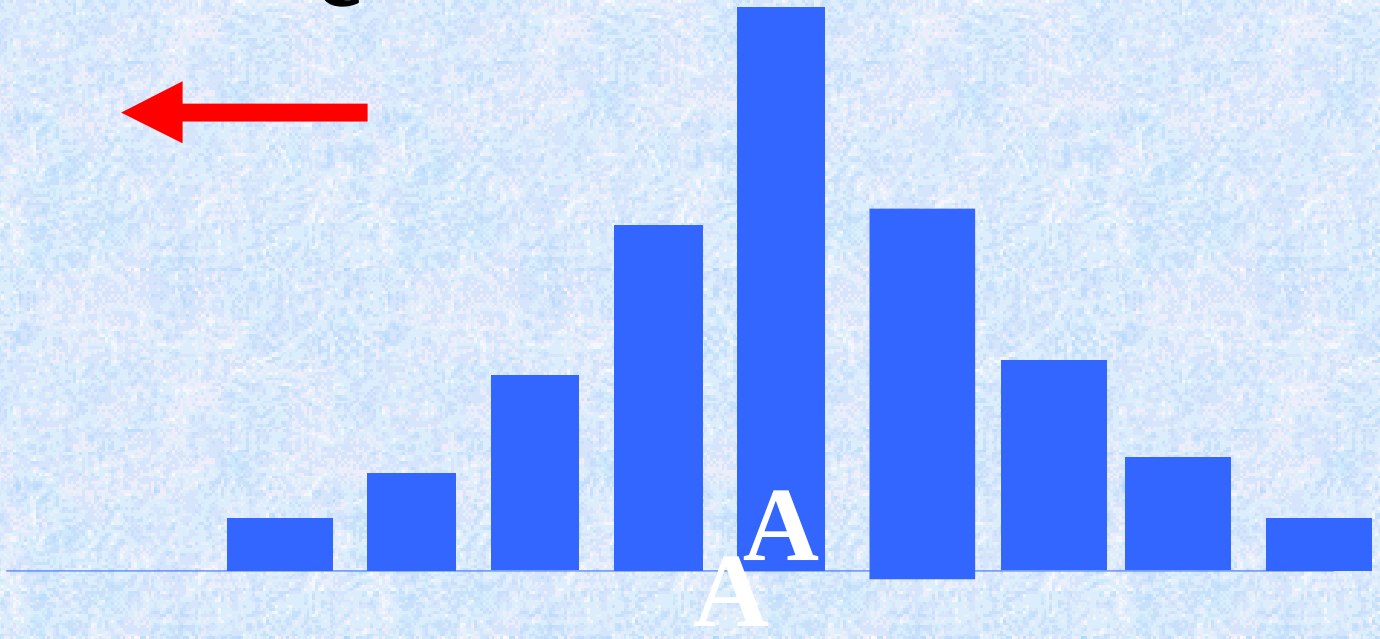
$$e^{[\ln (D_B/\lambda)\, D_A]\, t}$$

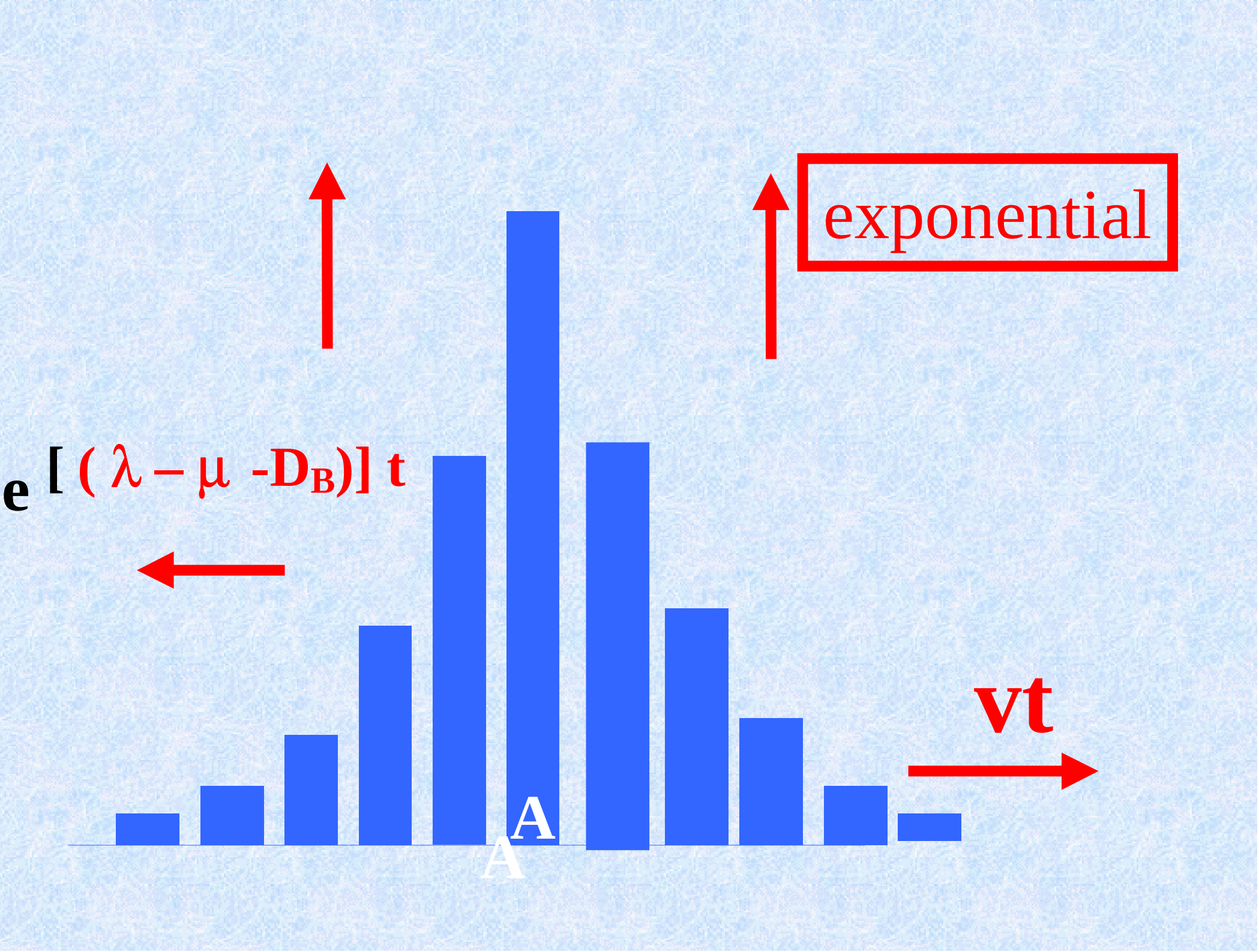


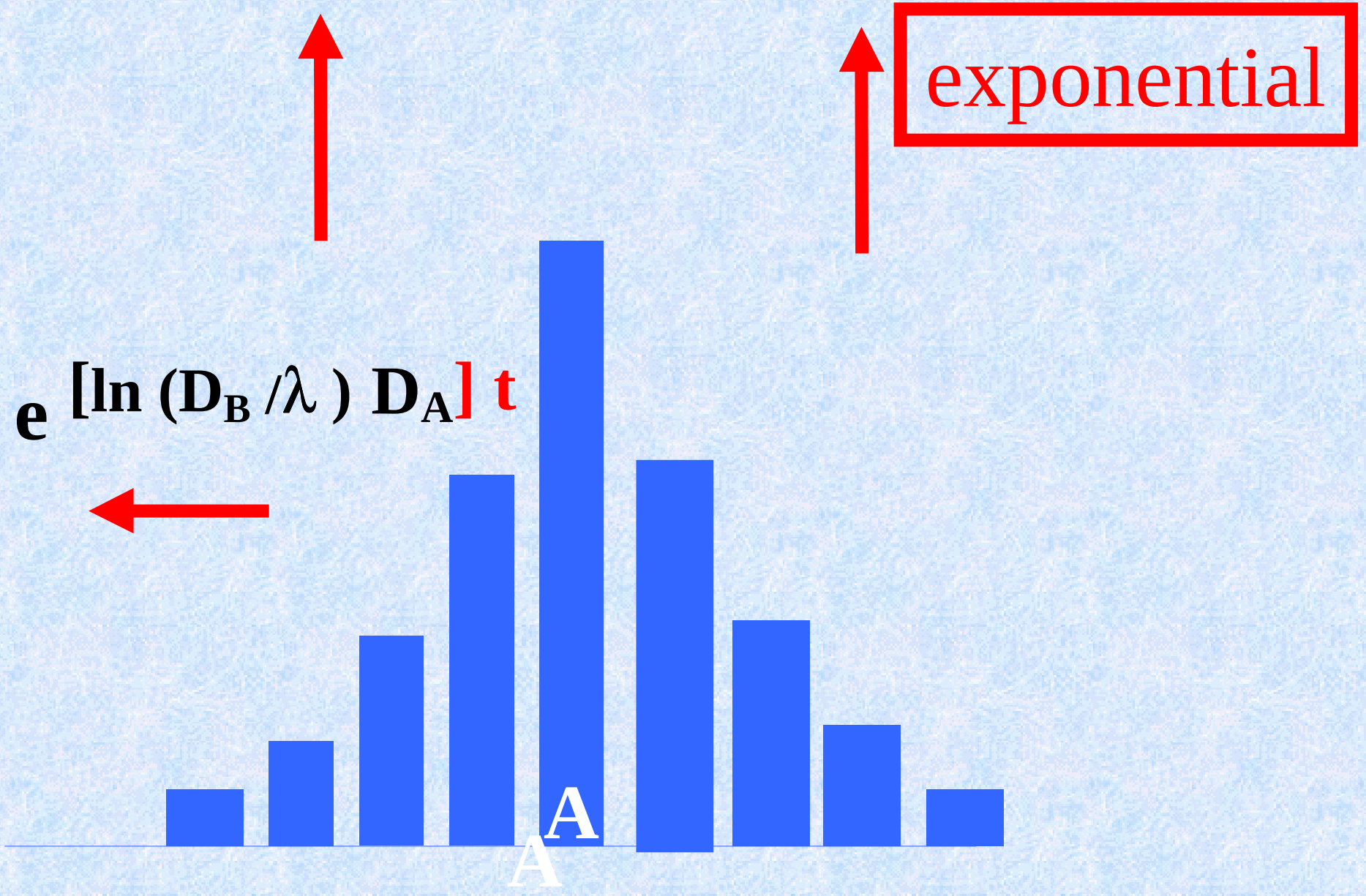

exponential
$e^{[(\lambda - \mu - D_B)]t}$
vt
A
A

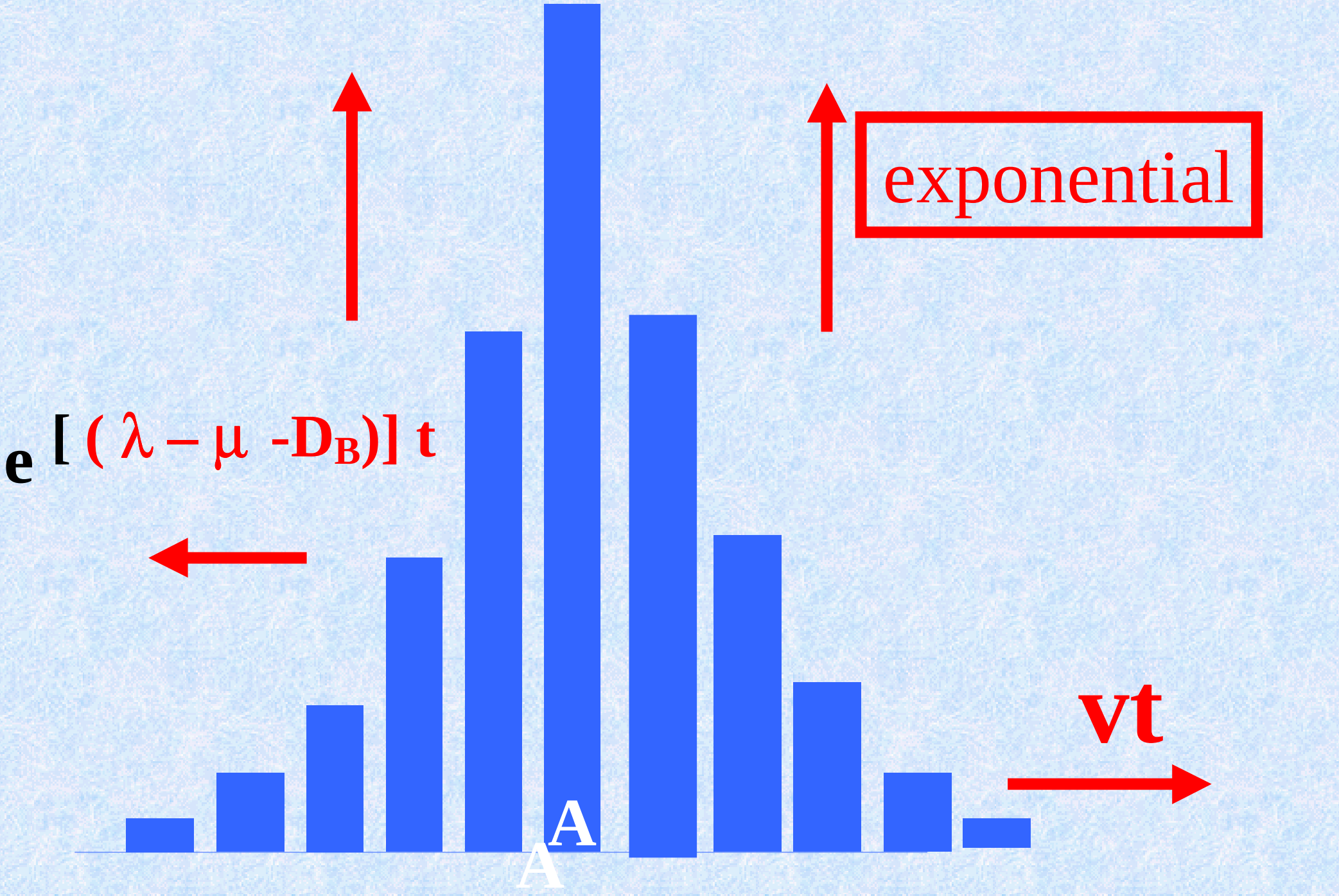
exponential

$e^{[\ln (D_B/\lambda)\, D_A]\, t}$

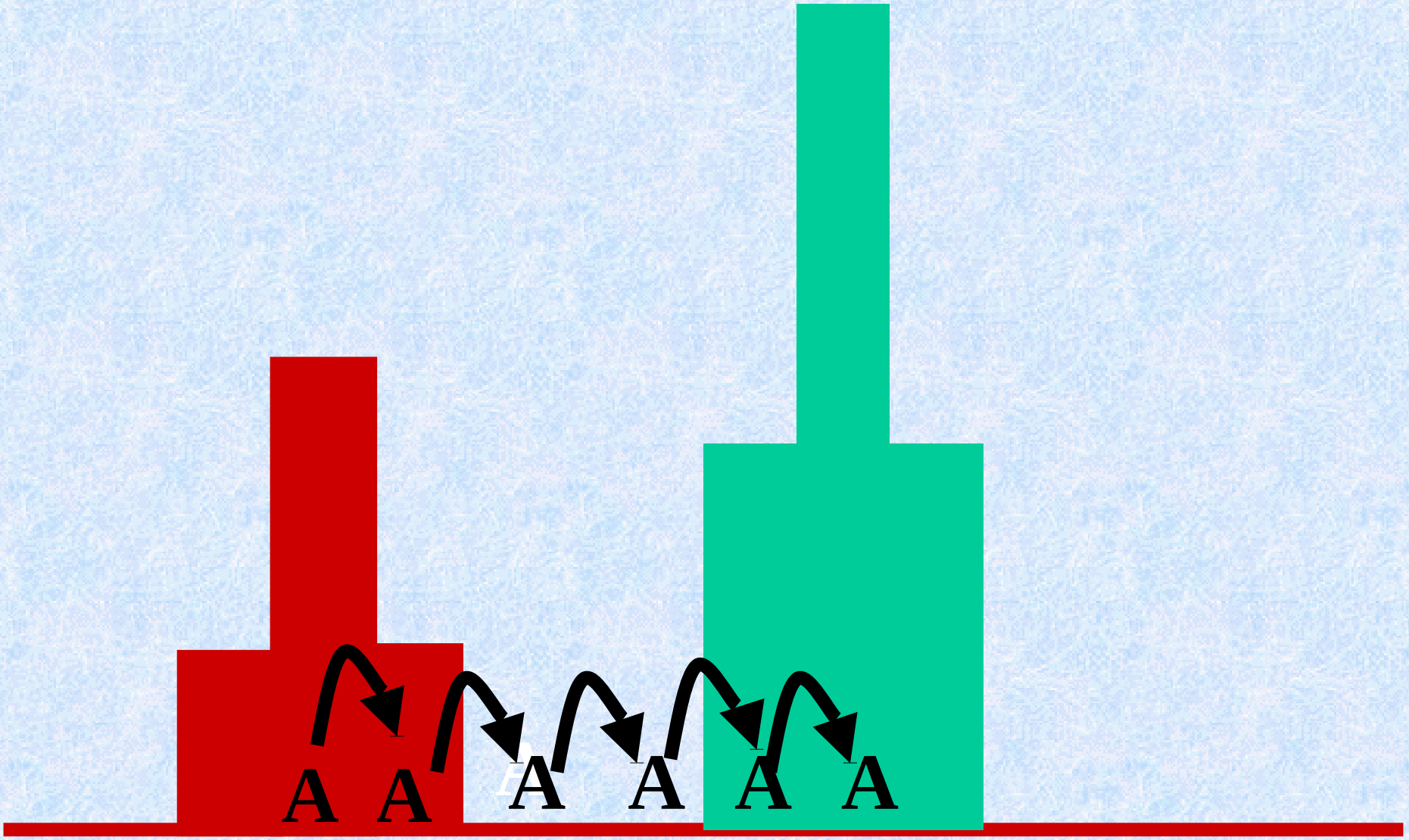


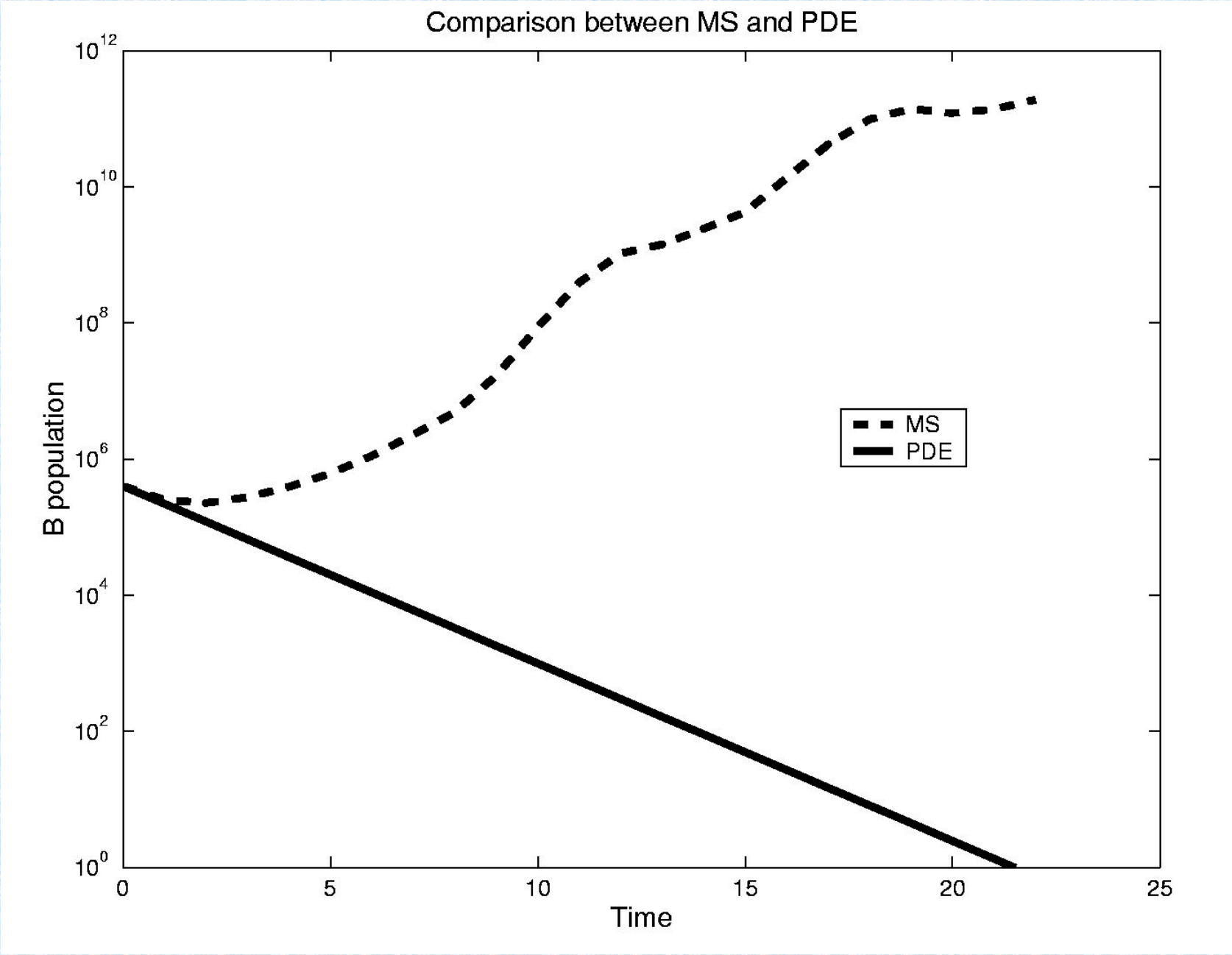

exponential
$e^{[(\lambda - \mu - D_B)]t}$
vt
A
A


exponential
e [ln (D_B /λ) D_A] t
A
A


exponential
$e^{[(\lambda - \mu - D_B)]t}$
vt
A
A

$$e^{[\ln (D_B/\lambda)\, D_A + (\lambda - \mu - D_B)]\, t}$$





$\lambda = 0.4$, $\mu = 1$, $D_B = 0.1 = D_A$, $a_0 = 0.5$, $\lambda a_0 - \mu = -0.8$

$0.4 < 1 + 0.1$

Black circle

White circle

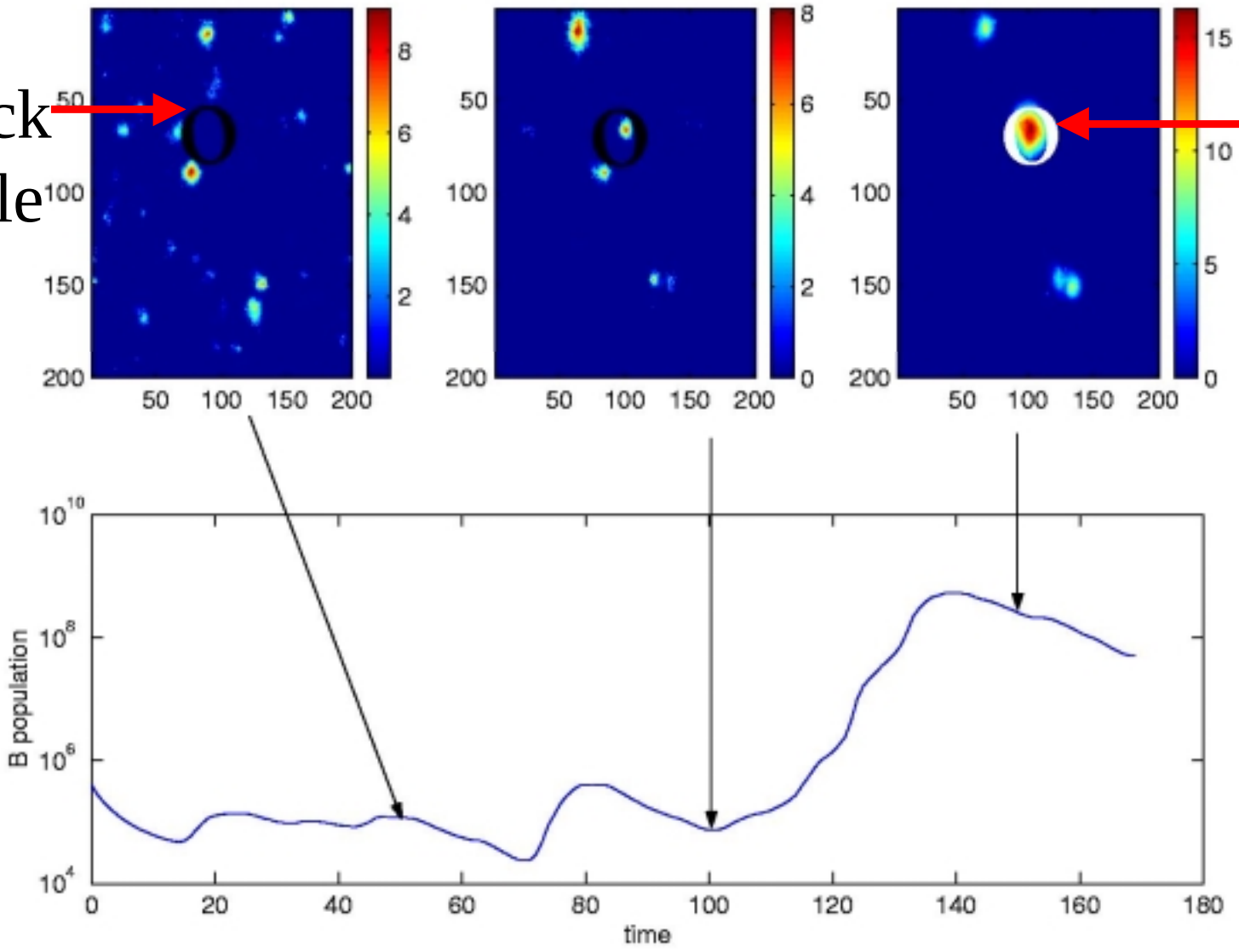


Figure 5: B dynamics with in the regime of high A density, mid values of A and B diffusion, $\lambda < \mu$ and $\lambda A < \mu$. The upper plots represents snapshots of the log of the B population at the times pointed by the arrows.

$$\dot{b}(x,t)=(\lambda a(x,t)-\mu)\, b(x,t) - \gamma\, b(x,t) \langle b(x,t)\rangle_x + D_b\, \Delta\, b(x,t)$$

Stable state (neglecting diffusion) is given by **:**

$$\dot{b}(x,t)=0=[(\lambda\, a(x,t)-\mu) - \gamma\langle b(x,t)\rangle_x]\, b(x,t) \qquad (1)$$

Concentrate on the site x_{max} with the property:

$$a(x_{max},t) > a(x,t):\ \forall\, x \neq x_{max}$$

according (1) its population keeps increasing until

$$[(\lambda\, a(x_{max},t) -\mu) - \gamma\langle b(x,t)\rangle_x] = 0 \qquad (2)$$

This fixes $\langle b(x,t)\rangle_x$. But this means that for $\forall\, x \neq x_{max}$

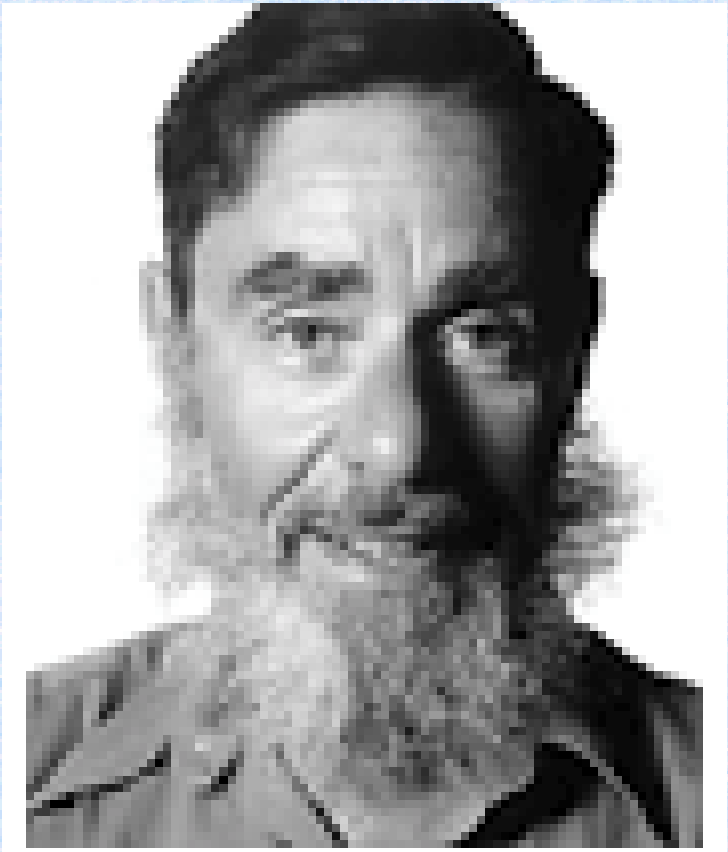
$$[(\lambda\, a(x,t) -\mu) - \gamma\langle b(x,t)\rangle_x] < 0$$

And according (1) $b(x,t)$ will decrease until $b(x,t) = 0$ $\forall\, x \neq x_{max}$

The entire **B** population is in an island around x_{max} with population

$$b(x_{max},t) \sim (\lambda\, a(x_{max},t) - \mu)\ \text{x Volume}$$

George Pólya (1887-1985)



Harry Kesten

No B's

Almost all B's

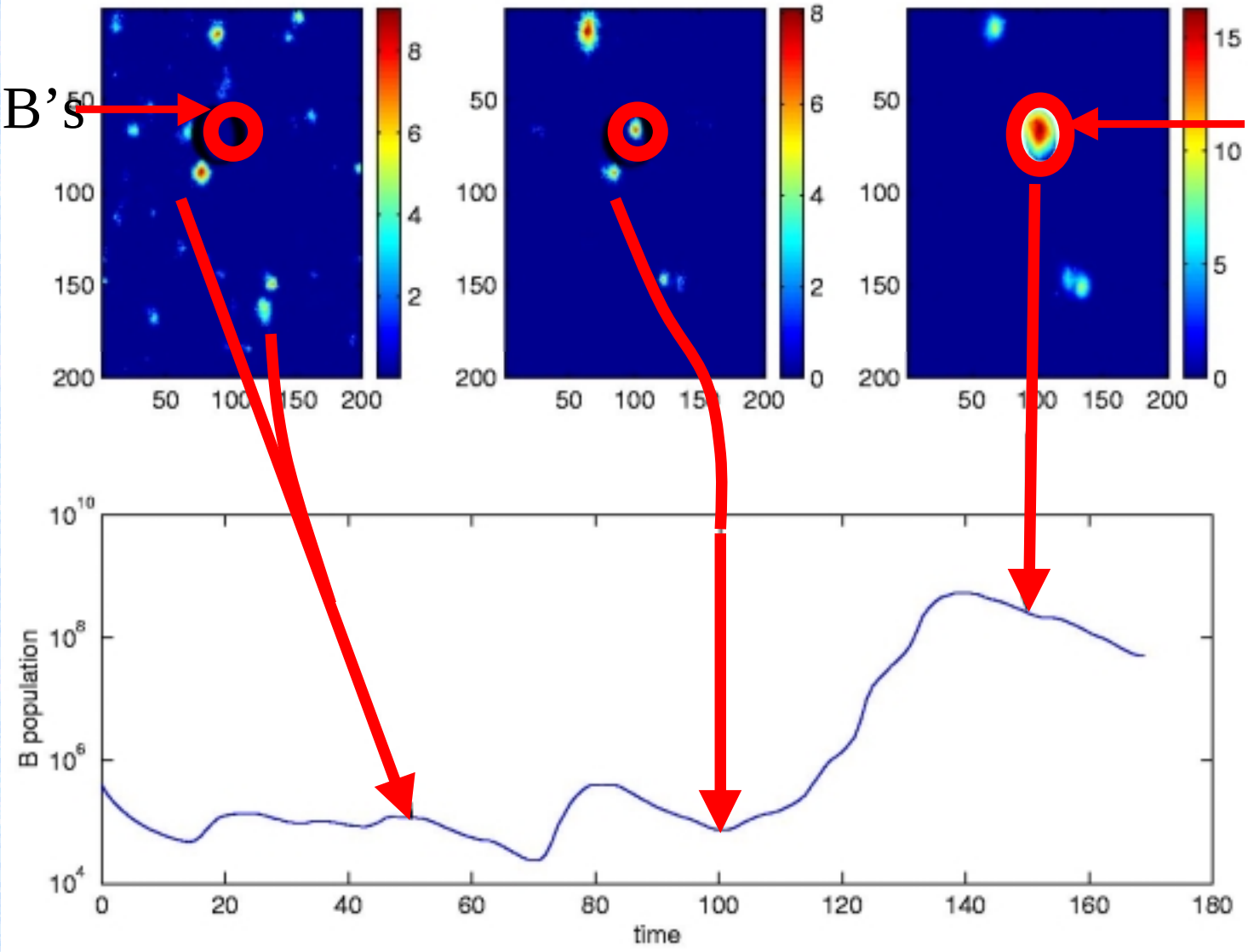
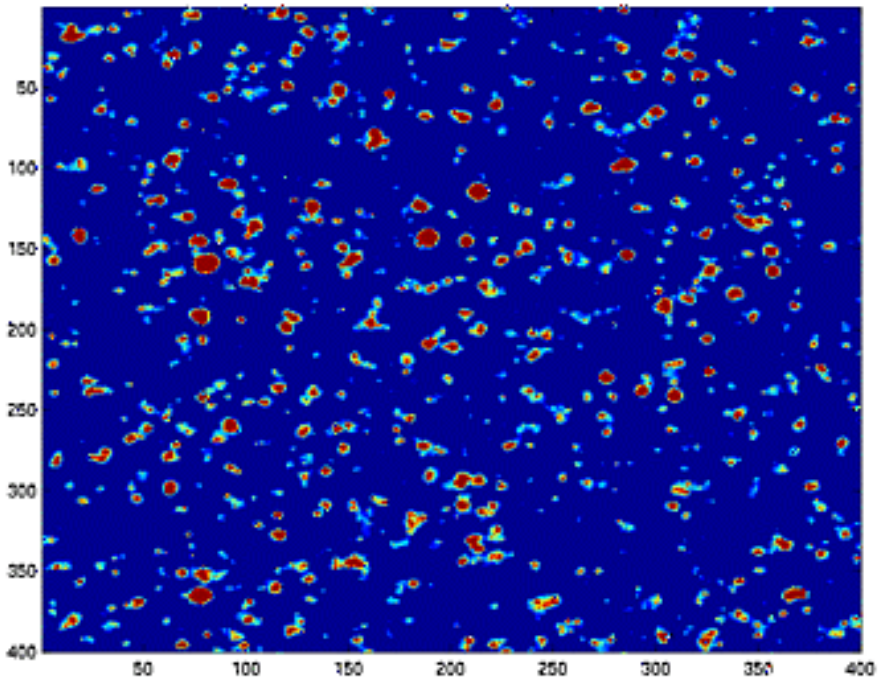


Figure 5: B dynamics with in the regime of high A density, mid values of A and B diffusion, $\lambda < \mu$ and $\lambda A < \mu$. The upper plots represents snapshots of the log of the B population at the times pointed by the arrows.

movie

sorin.html



continuum treatment:

underestimates resilience ;

misses emergence of
complex adaptive collective objects

One can study the **discrete** system by using:

- Microscopic representation techniques (agent simulations)

- Field theory techniques (renormalization group),

in 2D life wins if ultraviolet cut-off → infinity (RG misses singular)

- Stochastic processes theorems (branching random walks)

- isolated adaptive B islands
- 2D survival even with finite cut-off

The Importance of an A on $b(x,t)$ on its site of origin x

One return before $t = \tau$	Two returns before $t = 2\tau$		n returns before $t = n\tau$
prob: $P_d(\tau; 1) = P_d(\tau)$	$P_d(2\tau; 2) > P_d^2(\tau)$	.....	$P_d(n\tau; n) > P_d^n(\tau)$
growth: e^{λ / D_a}	$e^{2\lambda / D_a}$	.....	$e^{n\lambda / D_a}$
Expct. growth $P_d(\tau) e^{\lambda / D_a}$	$P_d(2\tau; 2) e^{2\lambda / D_a}$	.....	$P_d(n\tau; n)\, e^{n\lambda / D_a}$
$P_d(\tau) e^{\lambda / D_a}$	$> P^2{}_d(\tau) e^{2\lambda / D_a}$	.....	$> P^n{}_d(\tau) e^{n\lambda / D_a}$
$P_d(\tau) e^{\lambda / D_a} = e^{\eta}$ by def τ	$> [P_d(\tau) e^{\lambda / D_a}]^2 = e^{2\eta}$	.....	$> [P_d(\tau) e^{\lambda / D_a}]^n = e^{n\eta} = e^{\eta t/\tau}$

So: If $P_d(\infty) e^{\lambda D_a} > 1$ then $\exists \tau < \infty$, $\eta > 0$ s.t.

the expected contribution over time t of **one A** initially at x to $b(x,t)$ is at least a factor $e^{\eta t/\tau}$.

$\sim (\mu + D_b) e^{D_a/\lambda} D_a/\lambda$

There is a finite density of such $a(x,0)$'s =>

=>average population increases (at least) exponentially

So: If $P_d(\infty) e^{\lambda / D_a} > 1$ then $\exists \tau < \infty$, $\eta > 0$ s.t.

the expected contribution over time t of **one A** initially at x to $b(x,t)$ is at least a factor $e^{\eta t/\tau}$.

Taking in account death rate μ,

emigration rate D_b and that

there are $a(x,0)$ such A's:

$$\langle b(x,t) \rangle > b(x,0)\, e^{-(\mu + D_b)\, t}\, e^{a(x,0)\, \eta\, t/\tau}$$

-increase is expected at all x's where: $a(x,0) > (\mu + D_b)\, \tau/\eta$

$$\sim (\mu + D_b)\, e^{D_a/\lambda}\, D_a/\lambda$$

There is a finite density of such $a(x,0)$'s =>

=>average population increases (at least) exponentially

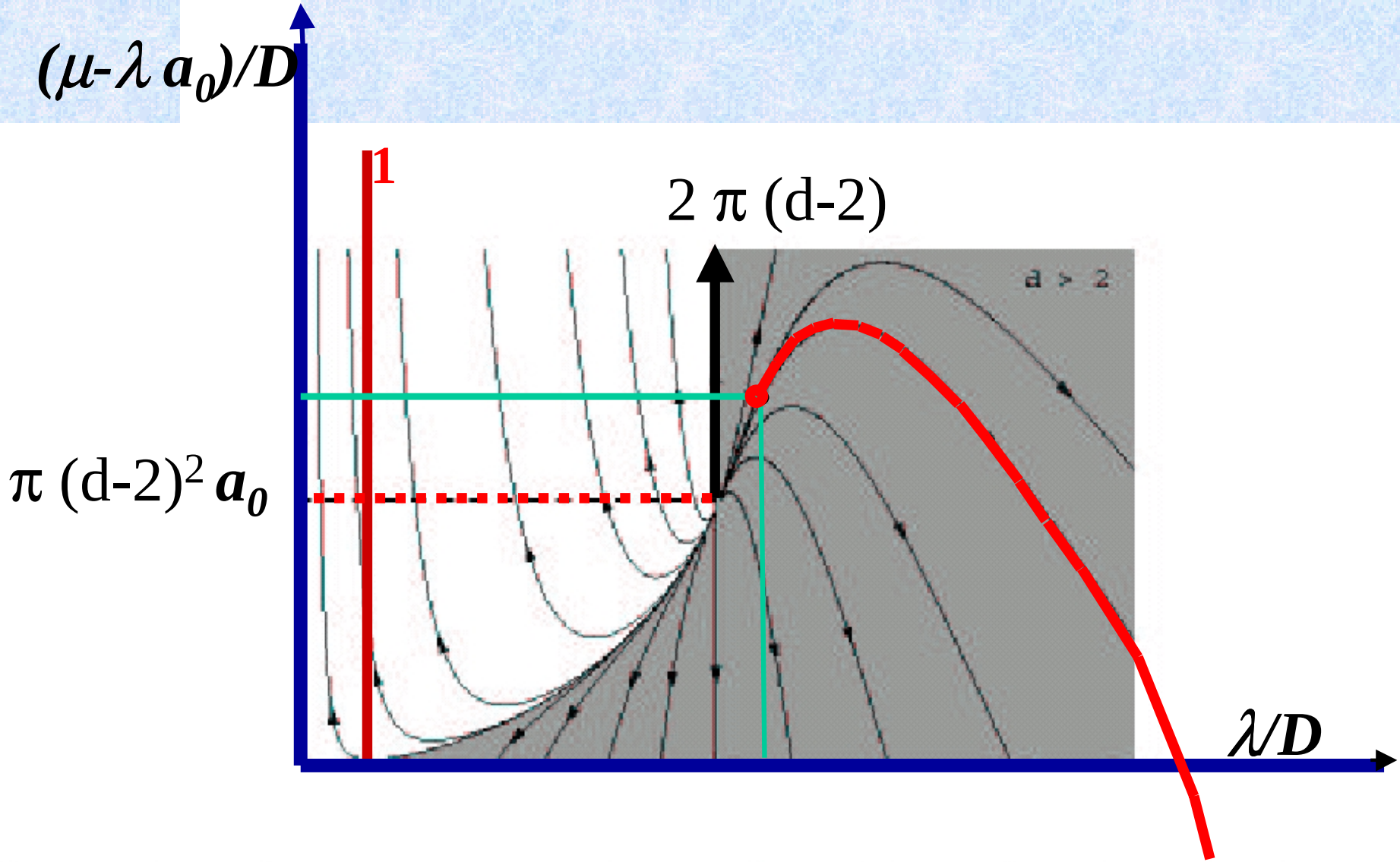


FIG. 4. Lower panel shows flow lines for $d > 2$. Shaded region flows to negative mass ("life"). Upper panel shows flow lines for $d \leq 2$, the whole parameter space flows to negative mass.

Pólya 's Random Walk Constant

What is the probability $P_d(\infty)$ that eventually an A returns to its site of origin?

Pólya : $P_1(\infty) = P_2(\infty) = 1$

but for d>2 $P_d(\infty) < 1$; $P_3(\infty) = 0.3405373$

Result based on Kesten and Sidoravicius preprint (75 p):

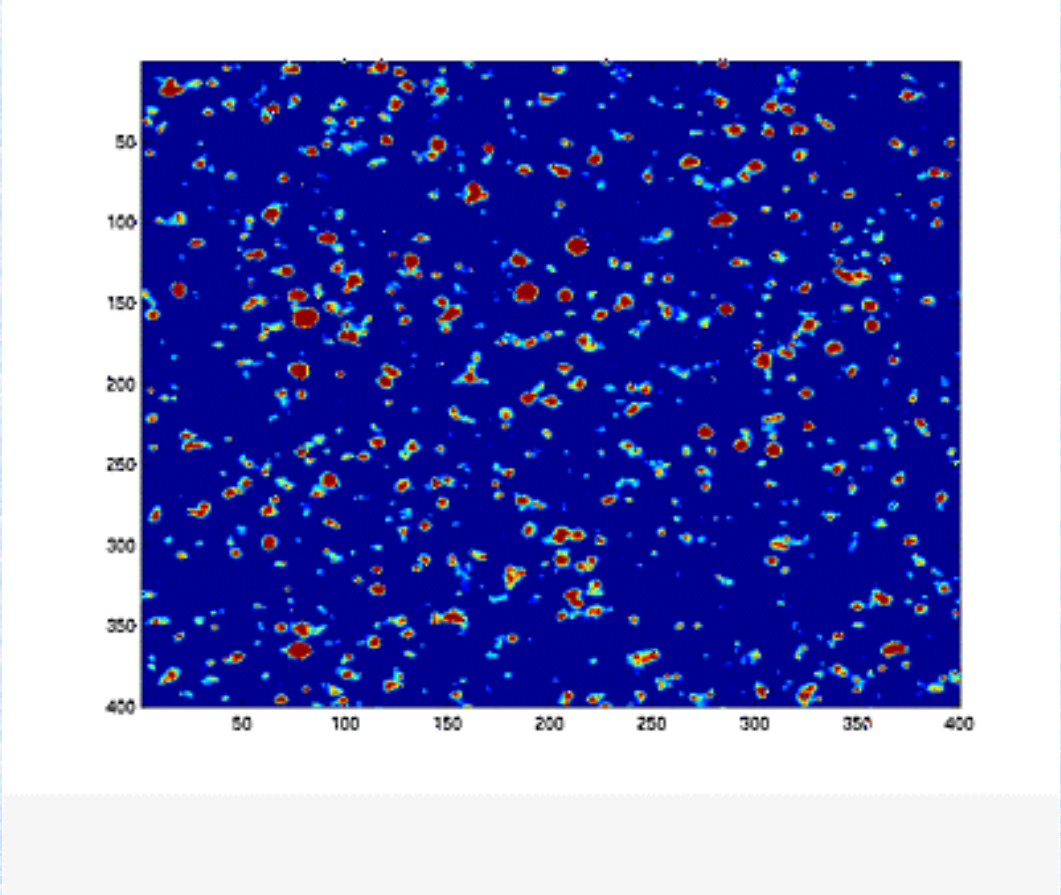
On large enough 2 dimensional surfaces $\forall \lambda, \mu, D_b, D_a, a_0$

-Total/average B population always grows:

for $t \to \infty$:

$$\lim_{R \to \infty} \left(\left[\sum_{x<R} b(x,t) \right] / R^2 \right) > e^{\alpha t}$$

Click here for the Angels and Mortals movie by Eldad Bettelheim and Benny Lehmann

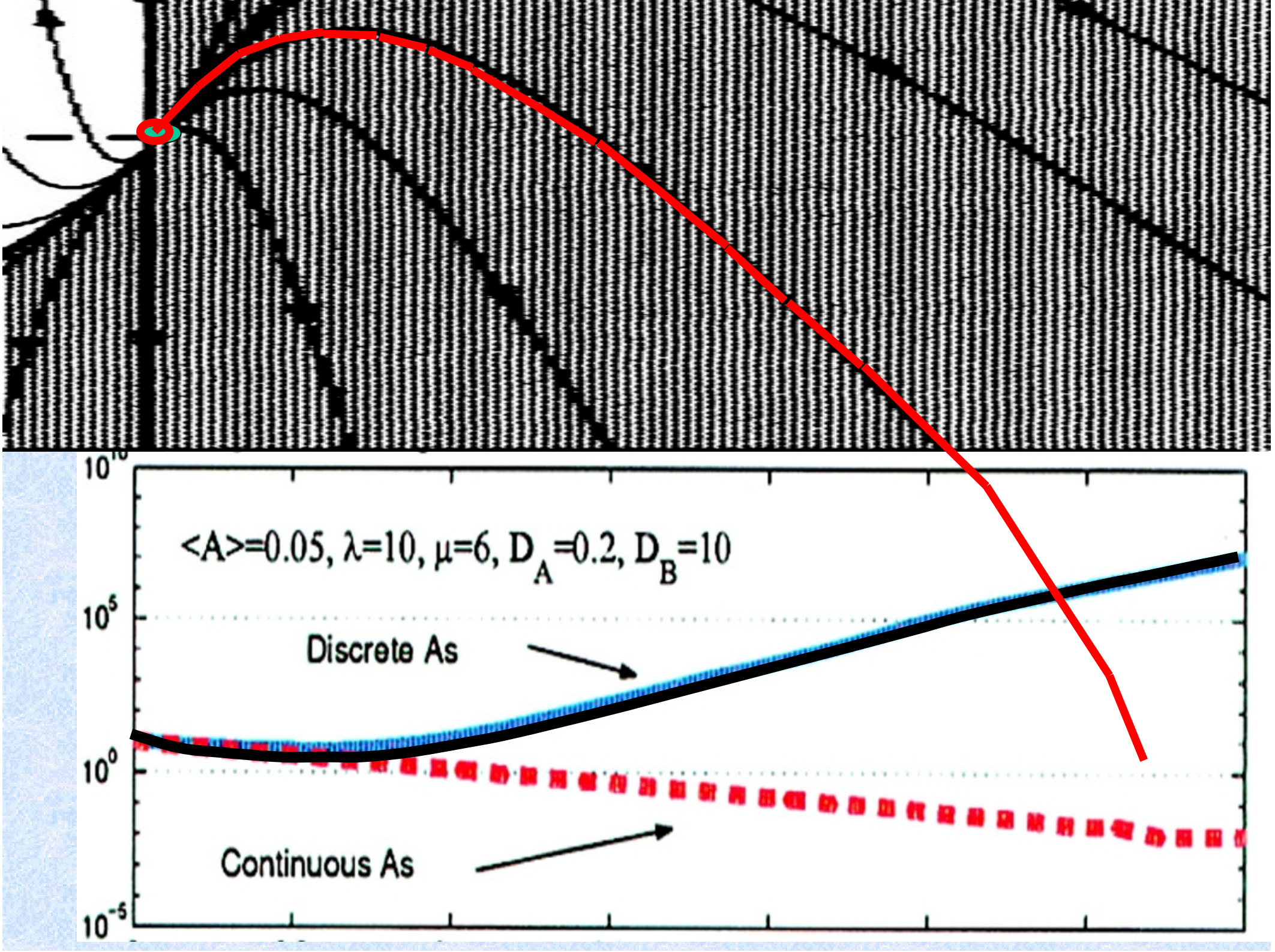


Click here for the movie

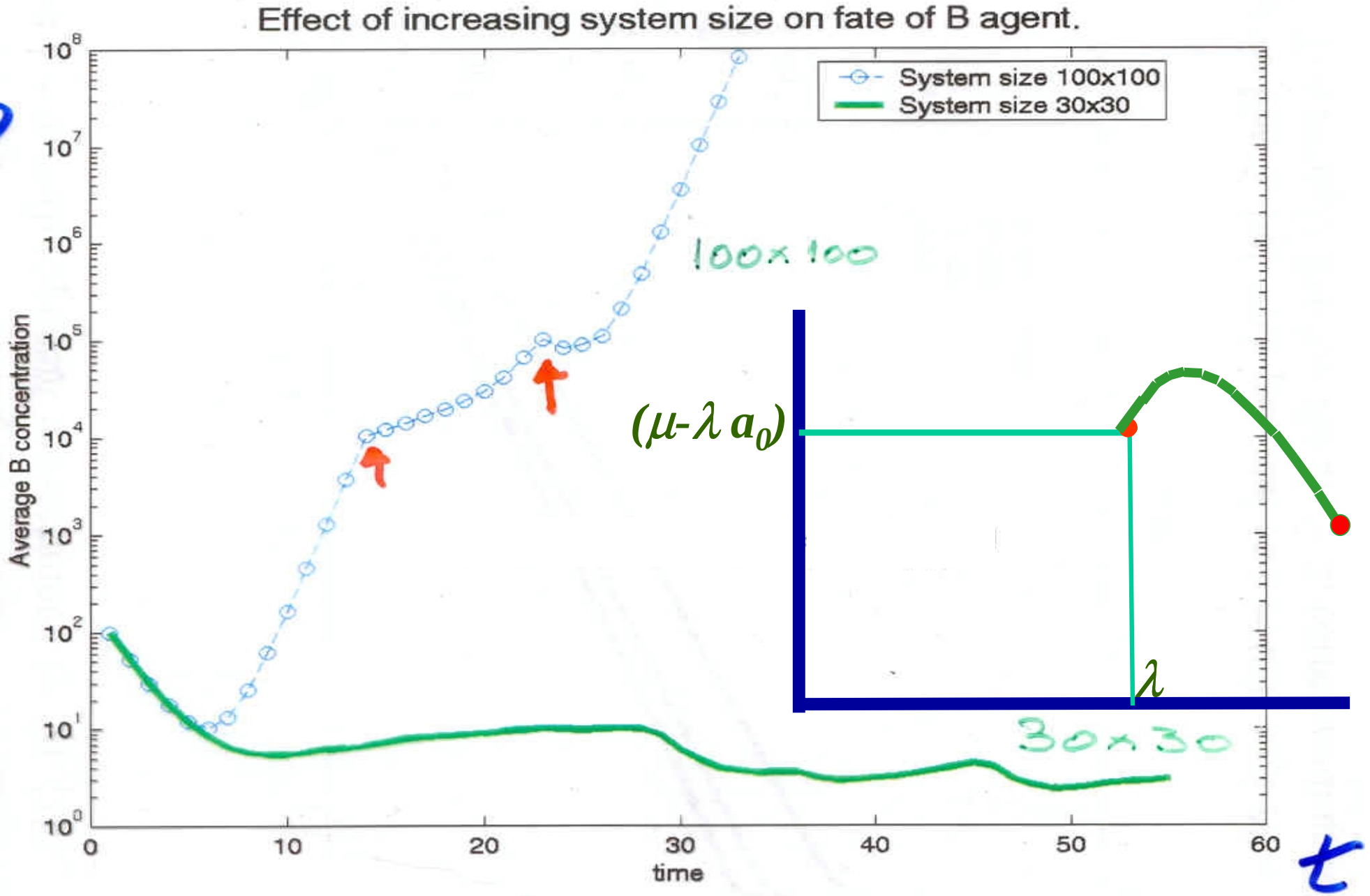
Angels and Mortals

By my students

Eldad Bettelheim and Benny Lehmann


<A>=0.05, λ=10, μ=6, D_A=0.2, D_B=10
10^{10}
10^{5}
10^{0}
10^{-5}
Discrete As
Continuous As

System Size


Effect of increasing system size on fate of B agent.
System size 100x100
System size 30x30
Average B concentration
time
100x100
30x30
$(\mu-\lambda a_0)$
λ
t

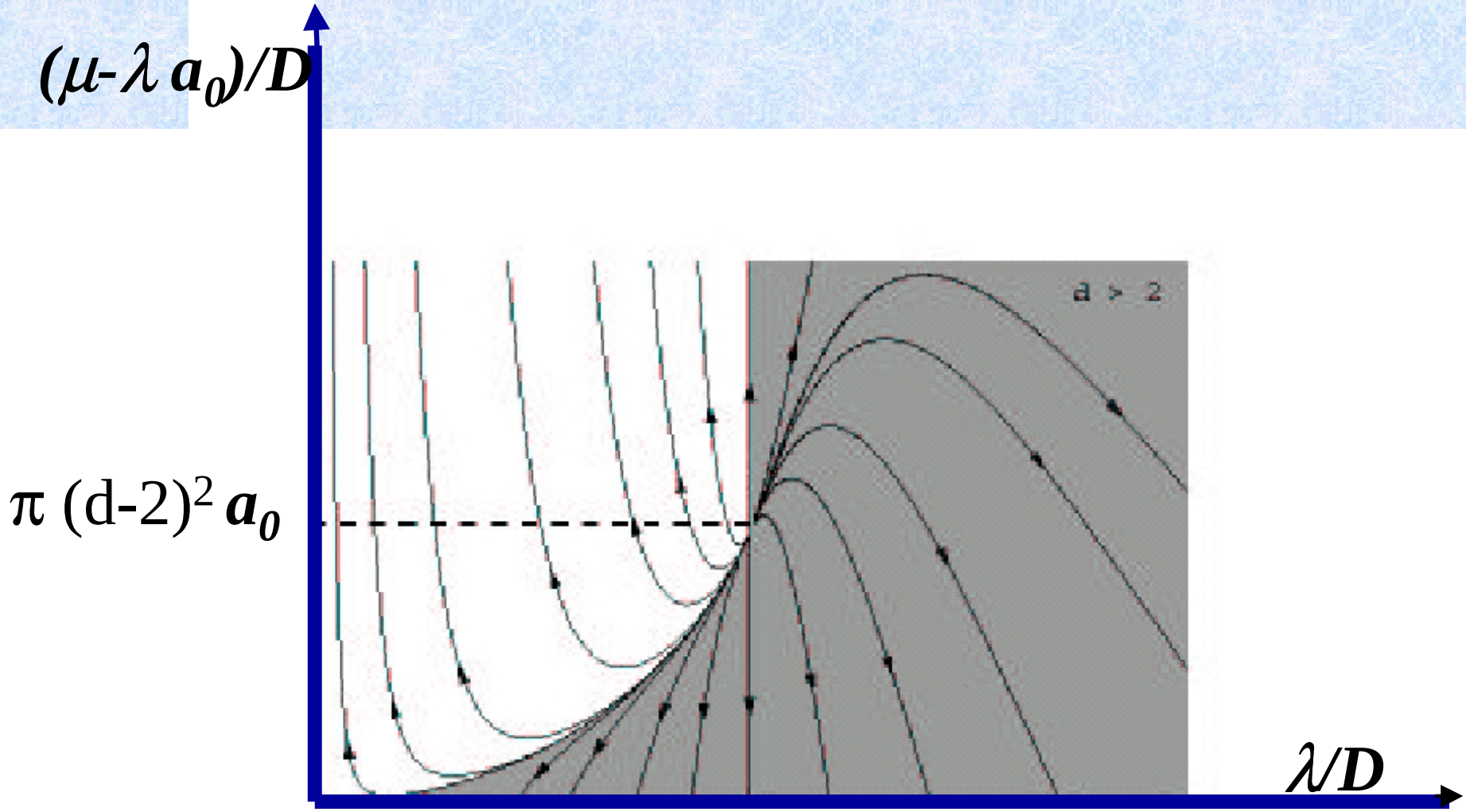


FIG. 4. Lower panel shows flow lines for $d > 2$. Shaded region flows to negative mass ("life"). Upper panel shows flow lines for $d \leq 2$, the whole parameter space flows to negative mass.

Study the effect of one A on $b(x,t)$ on its site of origin x

Ignore for the moment the death and emigration and other A's

Typical duration of each visit: $1/D_a$

Average increase of $b(x,t)$ per visit: e^{λ / D_a}

Probability of return of A by time t (d-dimesional grid) : $P_d(t)$

Expected increase in $b(x,t)$ due to 1 return events: $P_d(t)\, e^{\lambda / D_a}$

If $P_d(\infty)\, e^{\lambda / D_a} > 1$

[Always true in 2 dim !]

Then $\exists\, \tau$ s.t.

$$P_d(\tau)\, e^{\lambda / D_a} = e^{\eta};$$

$$\eta > 0$$