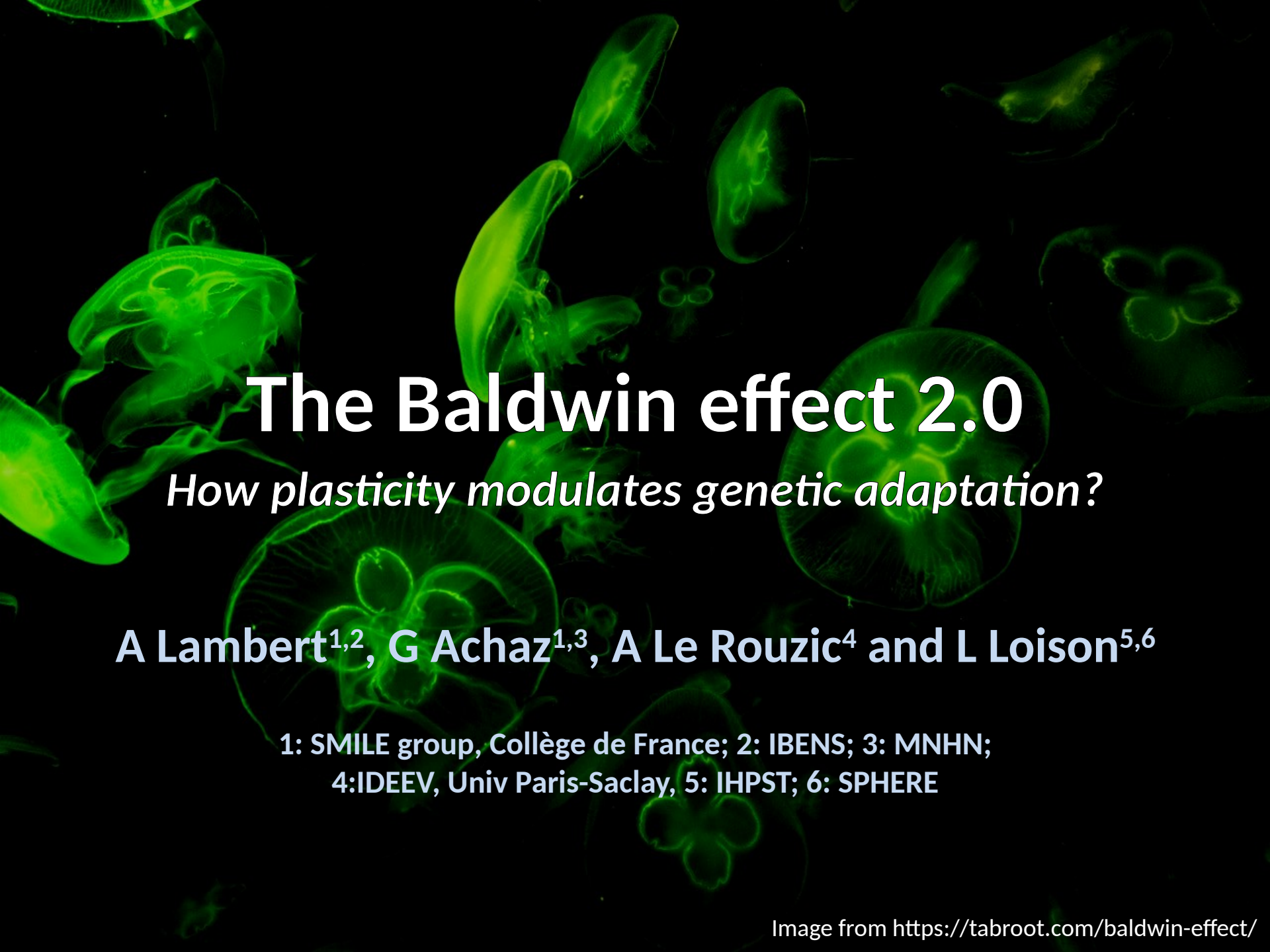


The Baldwin effect 2.0

How plasticity modulates genetic adaptation?

A Lambert[1,2], G Achaz[1,3], A Le Rouzic[4] and L Loison[5,6]

1: SMILE group, Collège de France; 2: IBENS; 3: MNHN;
4:IDEEV, Univ Paris-Saclay, 5: IHPST; 6: SPHERE

Image from https://tabroot.com/baldwin-effect/

Common meaning of…

THE BALDWIN EFFECT

GEORGE GAYLORD SIMPSON
The American Museum of Natural History and Columbia University, New York

HISTORICAL INTRODUCTION

Characters individually acquired by members of a group of organisms may eventually, under the influence of selection, be reenforced or replaced by similar hereditary characters. That is the essence of the evolutionary phenomenon here called "the Baldwin effect."

George G Simpson
1902-1984
Paleontologist

[Simpson, Evolution, 1953]

Year 1896: “A new factor in evolution”

Phenotypic plasticity is about…

James M. Baldwin
1861-1934
Philosopher & psychologist

“keeping single organisms alive – gives the species time to acquire the congenital mechanism for the same functions”

Henry F. Osborn
1852-1936
Paleontologist & geologist

“escaping elimination in the life struggle, [so that] the organism then waits, for a further congenital variation”

Conwy L. Morgan
1852-1936
Zoologist & psychologist

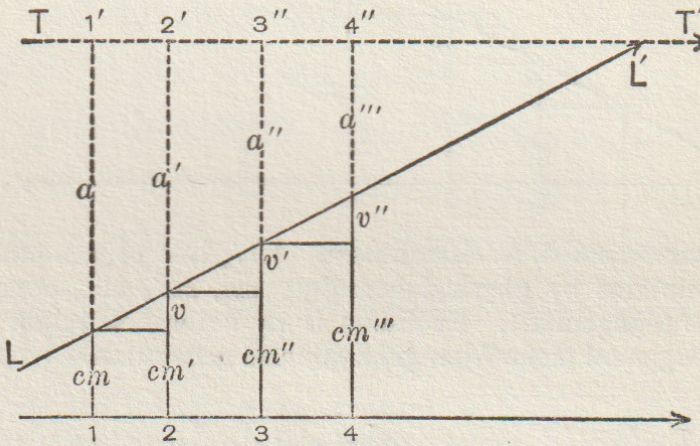
“enabling animals and plants to survive very critical changes in their environments”

1) “Organic selection” [social heredity ; plasticity] prevents extinction
2) The learned trait can be substituted by a “congenital variation”

[Baldwin, *Am Nat*, 1896; Osborn, *Trans. NY Acad Sci.*, 1896; Morgan, 1896]

The Theory of “Orthoplasy”

James M. Baldwin
1861-1934
Philosopher & psychologist



Theory of Orthoplasy. LL′, line of evolution; 1, 2, etc., successive generations by physical heredity; TT′, line of tradition (social transmission); *cm*, *cm′*, etc., congenital mean; *a*, *a′*, etc., accommodations (and modifications) supplementing or screening *cm*, etc.; *v*, *v′*, etc., (congenital) variations added to *cm*, etc., by natural selection. The species is kept alive by *a*, *a′*, etc., and TT′, during the evolution of *cm*. The line TT′, considered as ‘tradition,’ is of varying importance according to the character in question and to the grade of the organism in the scale of life; but if it signify any utility for which the accommodations are necessary, it is always present, and may be called the ‘line of utility.’

[Baldwin, *Development and Evolution* , 1902]

Simpson “clarification”

B.E. can be decomposed in 3 steps

(1) Individual organisms interact with the environment in such a way as systematically to produce in them behavioral, physiological, or structural modifications that are not hereditary as such but that are advantageous for survival, *i.e.*, are adaptive for the individuals having them.

(2) There occur in the population genetic factors producing hereditary characteristics similar to the individual modifications referred to in (1), or having the same sorts of adaptive advantages.

(3) The genetic factors of (2) are favored by natural selection and tend to spread in the population over the course of generations. The net result is that adaptation originally individual and nonhereditary becomes hereditary.

That description of the Baldwin effect is also a more precise definition of the term. At this point it need not be taken for granted that the effect actually occurs or has an essential role in evolution. It may be taken as a hypothesis subject to investigation.

B.E. is not central in Evolution

Seen in a modern context, the Baldwin effect helps to focus attention on a host of problems, especially in developmental (or physiological) genetics, well worthy of further study. It does not, however, seem to require any modification of the opinion that the *directive force* in adaptation, by the Baldwin effect or in any other particular way, is natural selection.

LITERATURE CITED

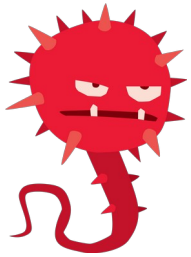
[Simpson, Evolution, 1953]

Evolutionary rescue

Conservation biology

A species becomes *adapted* before *extinction*

- Adapted = genetically fit to the new environment
- Depends on the fitness landscape



Pathogen eradication

A pathogen becomes *resistant* before *complete cleaning*

- Resistant = genetically fit to the new environment
- Depends on the treatment

Evolutionary rescue is a contemporary version of the B.E.

Two conflicting hypotheses

Does adaptive **plasticity facilitate** (by channeling the exploration of the phenotypic space)
or **decelerate** (by masking genetic variation) the **evolution**ary process?

Hypothesis 1. Plasticity facilitates genetic adaptation

- "[Phenotypic variations] may serve as the first step in evolutionary change, not by becoming impressed upon the germ-plasm, but by holding the strain in an environment where mutations tending in the same direction will be selected and incorporated into the constitution" [Huxley, 1942]

Hypothesis 2. Plasticity slows down genetic adaptation

- "If the phenotype is highly plastic, the selection pressure may actually be reduced because there is no selective advantage in changing the genotype when an individual can adjust itself phenotypically to a current condition" [Mayr, 1970 ; see also Delage, 1899]

B.E. is worth re-re-exploring by quantifying these two effects

Minimal stochastic model

1) Population size is finite and can fluctuate

[without extinction, see e.g.: Ancel 1999 ; Ancel 2000 ; Paenke et al., 2007]

2) Plasticity is *optional* but *optimal*

- Plasticity occurs only in a fraction *p* of the population
- Plastic individuals have perfectly fit phenotypes (learned ability = innate ability)
- Plasticity has no cost (learning is for free)

[plasticity=noise, see e.g.: Price et al., 2033 ; Carja & Plotkin, 2019]

3) Genotype leading to adaptive phenotype can be acquired by one mutation event

4) Two environments, two genotypes, two phenotypes

- Phenotype *A* adapted to old envt, maladapted to new envt
- Phenotype *B* adapted to new envt, maladapted to old envt
- Genotype *a* leads to phenotype *A*, genotype *b* to phenotype *B* (*p=0*)

Model definition (formal version)

Two genotypes (*a* and *b*), only one plastic/educable (*a*)

At birth, genotype "*a*" has phenotype "*A*" with probability *(1-p)* in new environment - - "*B*" - - *p* - -

"*b*" - - "*B*" - - *1* in any environment

A birth-death model, with competition

Overall, the "*A*" phenotypes have a birth rate: $n_A\, b_A$

a death rate: $n_A\, d_A + c\, n_A\, (\, n_A + n_B - 1\,) / K$

Overall, the "*B*" ... (replace all "A" by "B")

Mutation: a => b

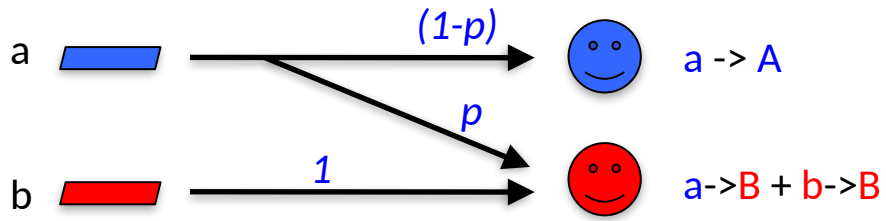
At birth, offspring inherit parent genotype, but genotype "*a*" can mutate to "*b*" with probability μ/K

A simple birth-death model (b_x, d_x, c, K, μ) , with a plasticity twist (p)

Model definition (picture version)

Two genotypes (*a* and *b*), only one plastic/educable (*a*)

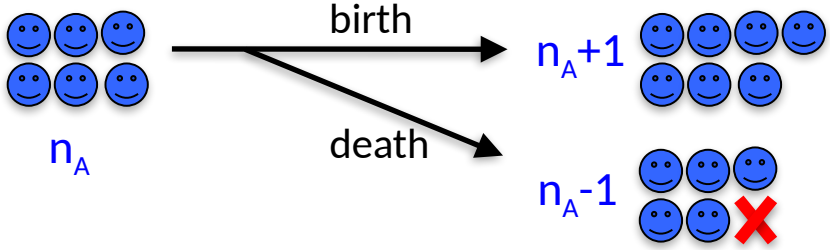
3 kinds of individuals in the red environment



geno>ΦENO

A birth-death model, with competition

Total rates

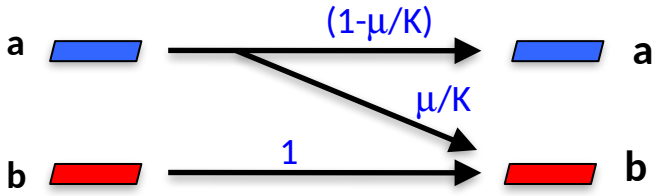


$n_A b_A$

$n_A d_A + c n_A (n_A + n_B - 1)/K$

Mutation: a => b

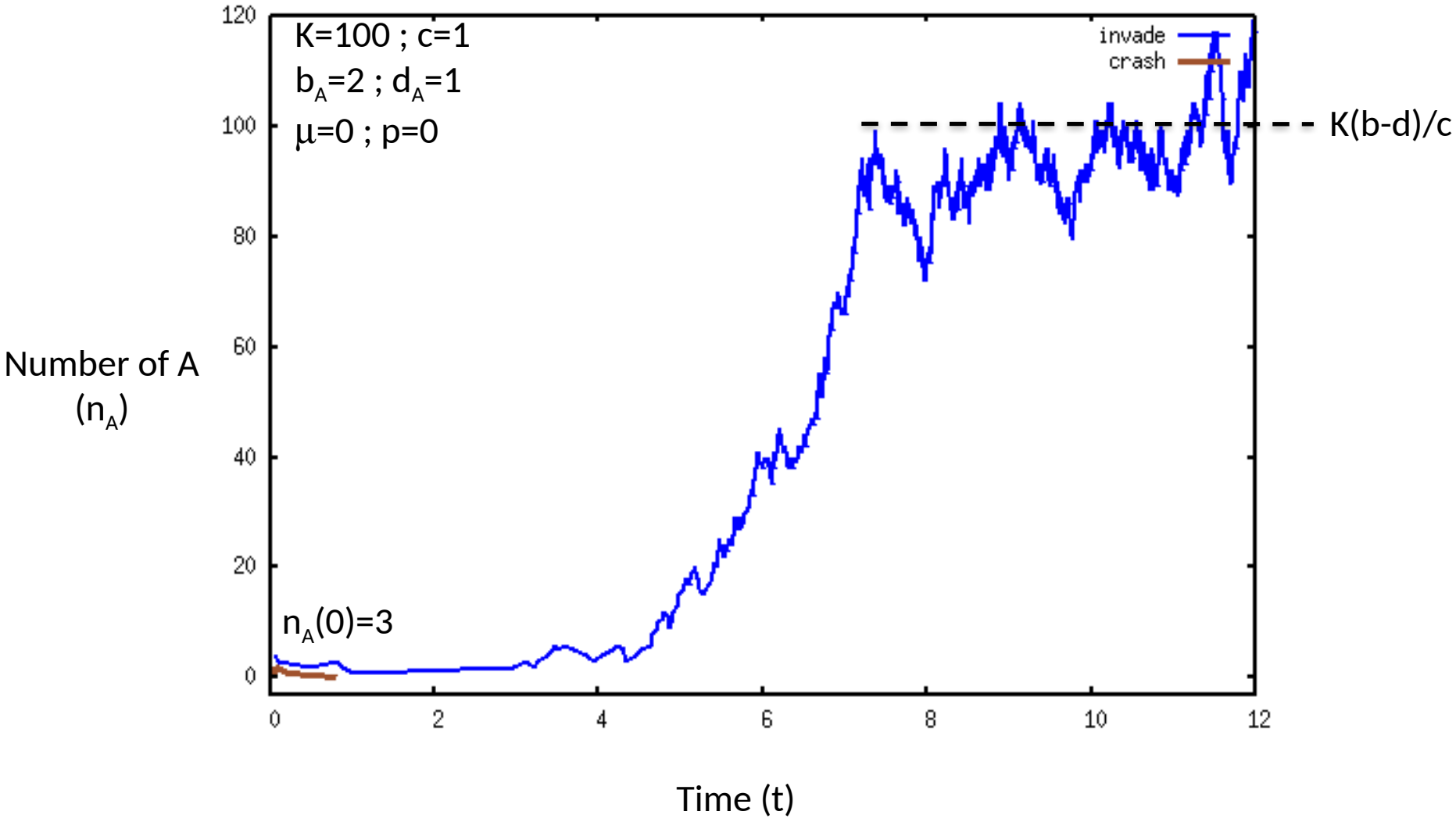
2 genotypes 1 mutable



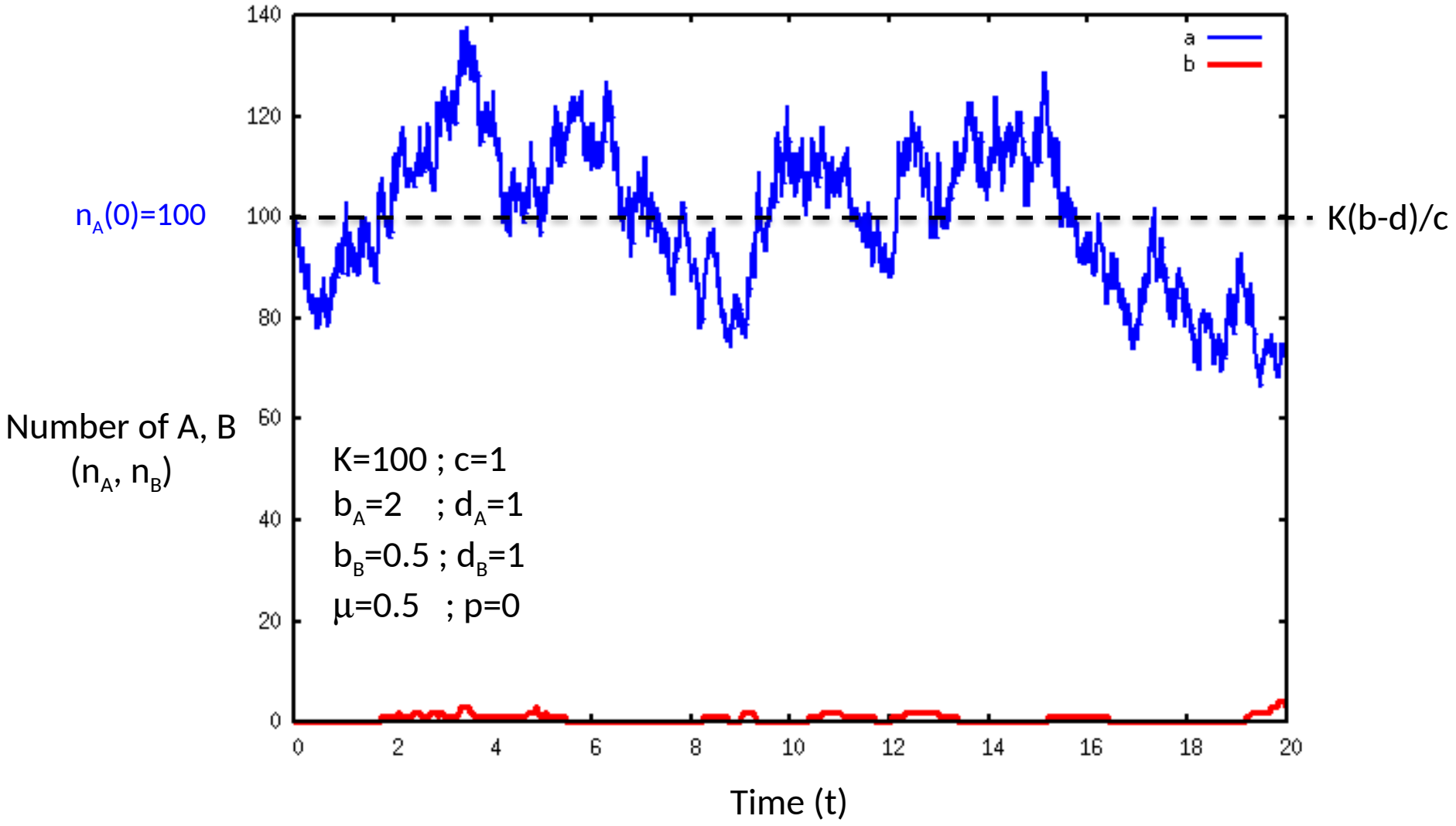
At birth

And now...

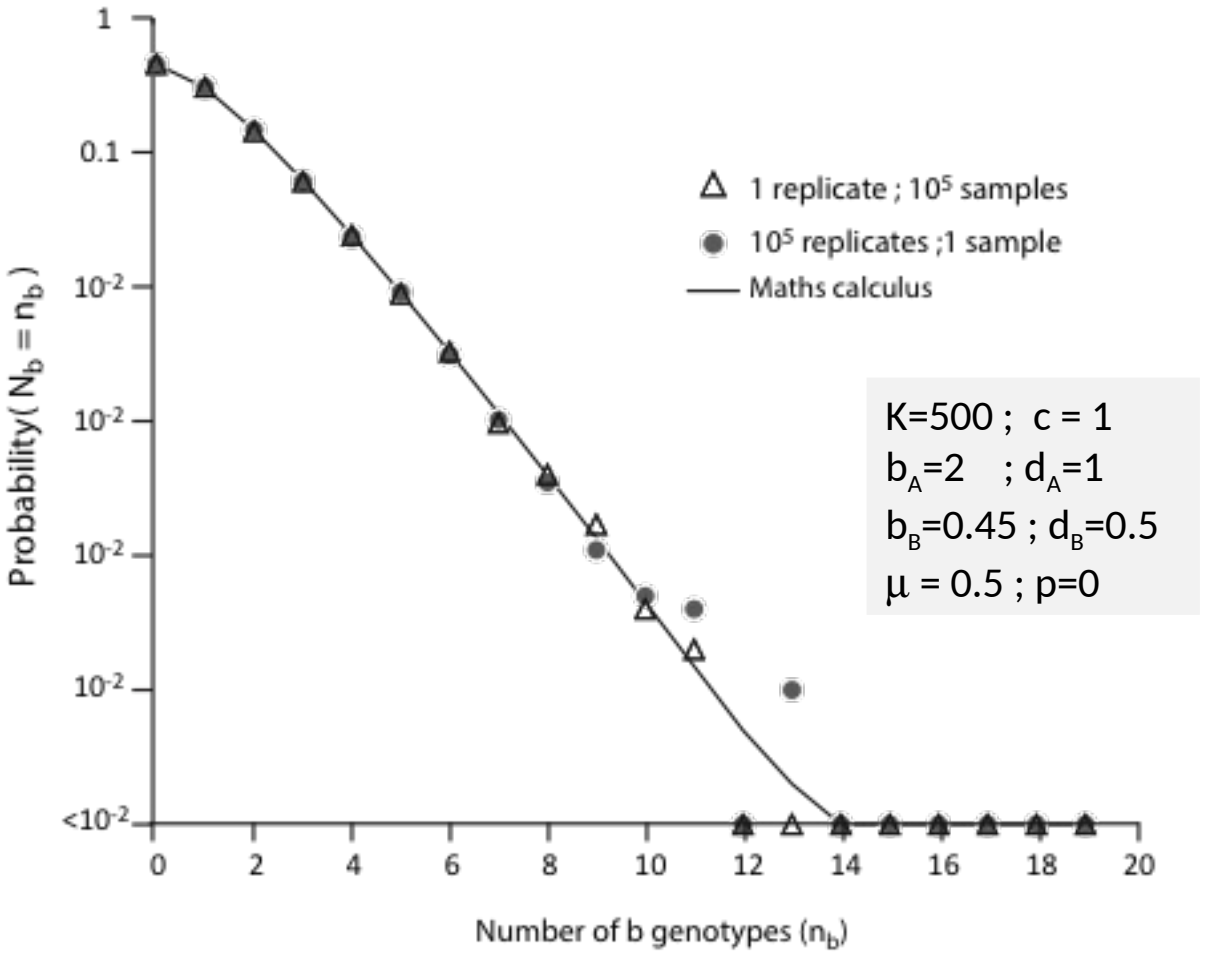

shutterstock
ACTION!


K=100 ; c=1
b_A=2 ; d_A=1
μ=0 ; p=0
n_A(0)=3
invade
crash
K(b-d)/c
Number of A (n_A)
Time (t)
0
20
40
60
80
100
120
0
2
4
6
8
10
12

With mutations, a=>b



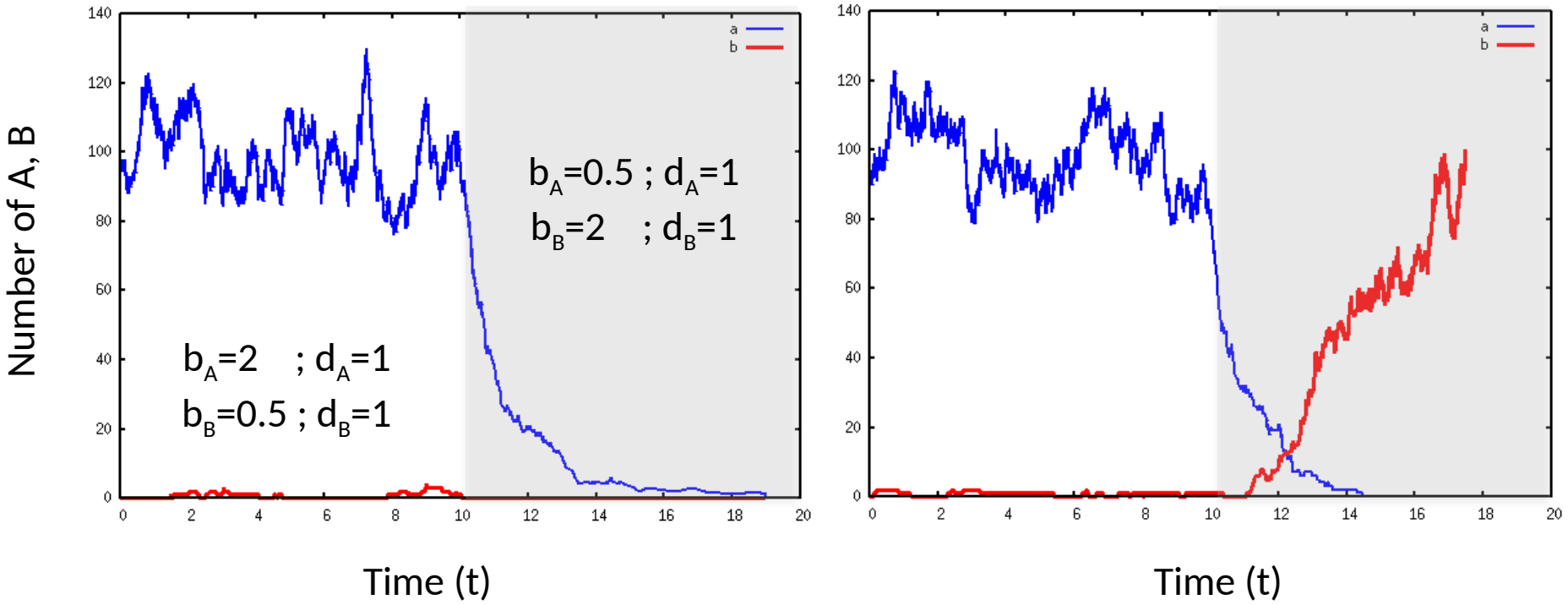
Mutation-selection equilibrium



Stationary distribution of a subcritical birth-death process with immigration

Evolutionary rescue

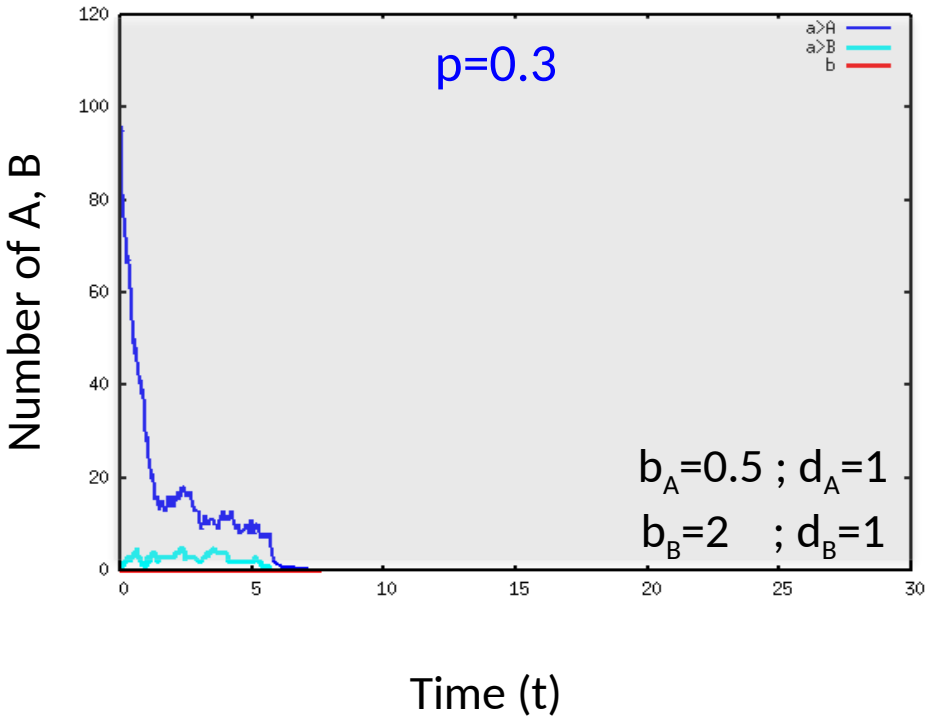
K=100 ; c=1 ; μ=0.5 ; p=0

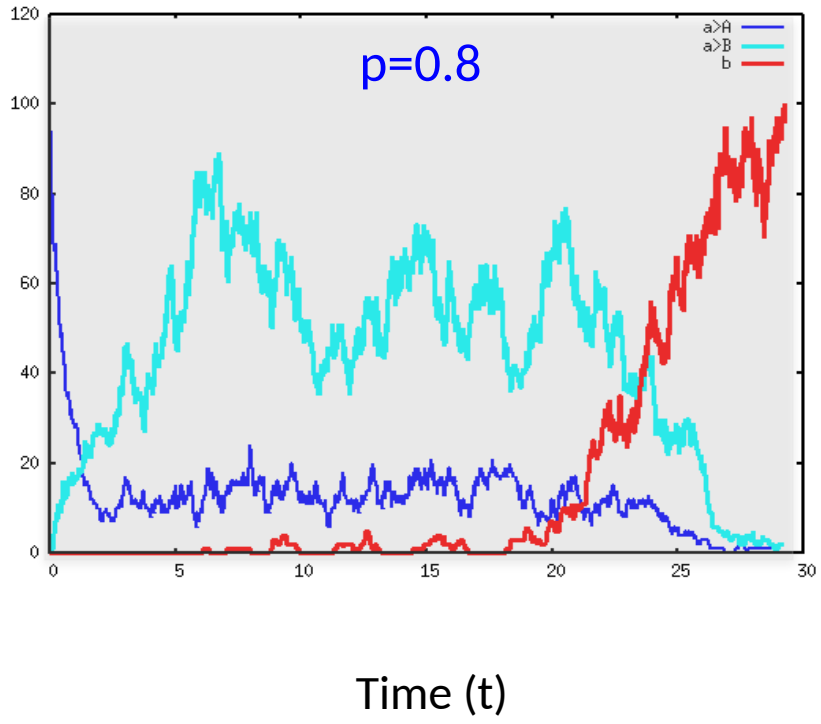


Evolutionary rescue from standing variation or from neo-mutations

Adding plasticity (p>0)

K=100 ; c=1 ; μ=0.5 ;





With strong enough plasticity (ability to learn), “a” genotypes survive longer

Does plasticity help or hinder rescue?

Set-up

Population starts in environment B, with

K = 1000 genotypes "a"

c=1;

b_A=0.5 ; d_A=1 → phenotype A is subcritical (*it goes to extinction*)

b_B=2 ; d_B=1 → phenotype B is supercritical (*it can invade*)

p ranges from 0 (no learning) and 1 (perfect learning)

μ ranges from 0 (no "*b*" mutant) to 32/K, in log-scale

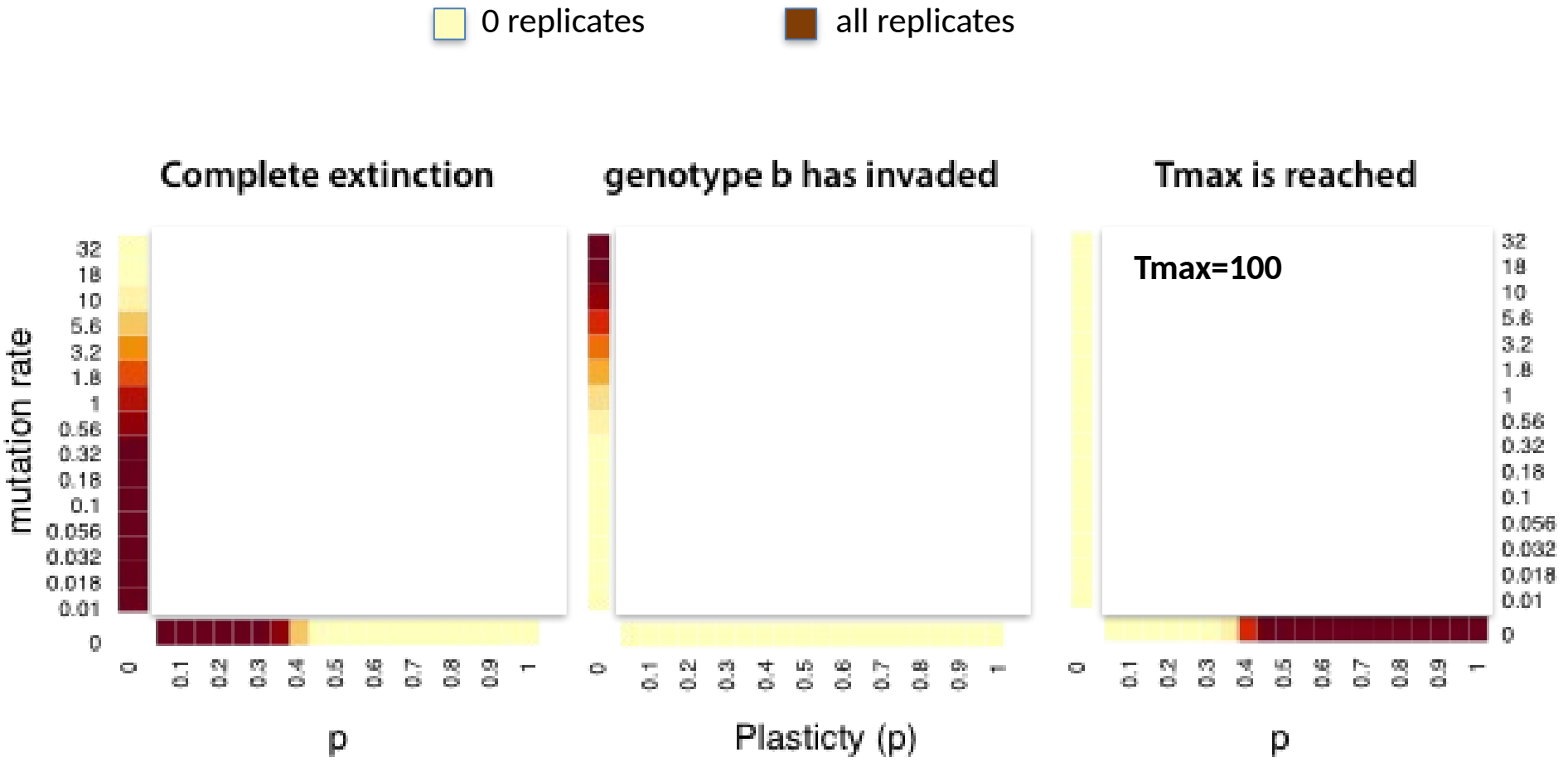
Any of the replicates (1000 per cell) stops when

(1) The whole population has gone extinct

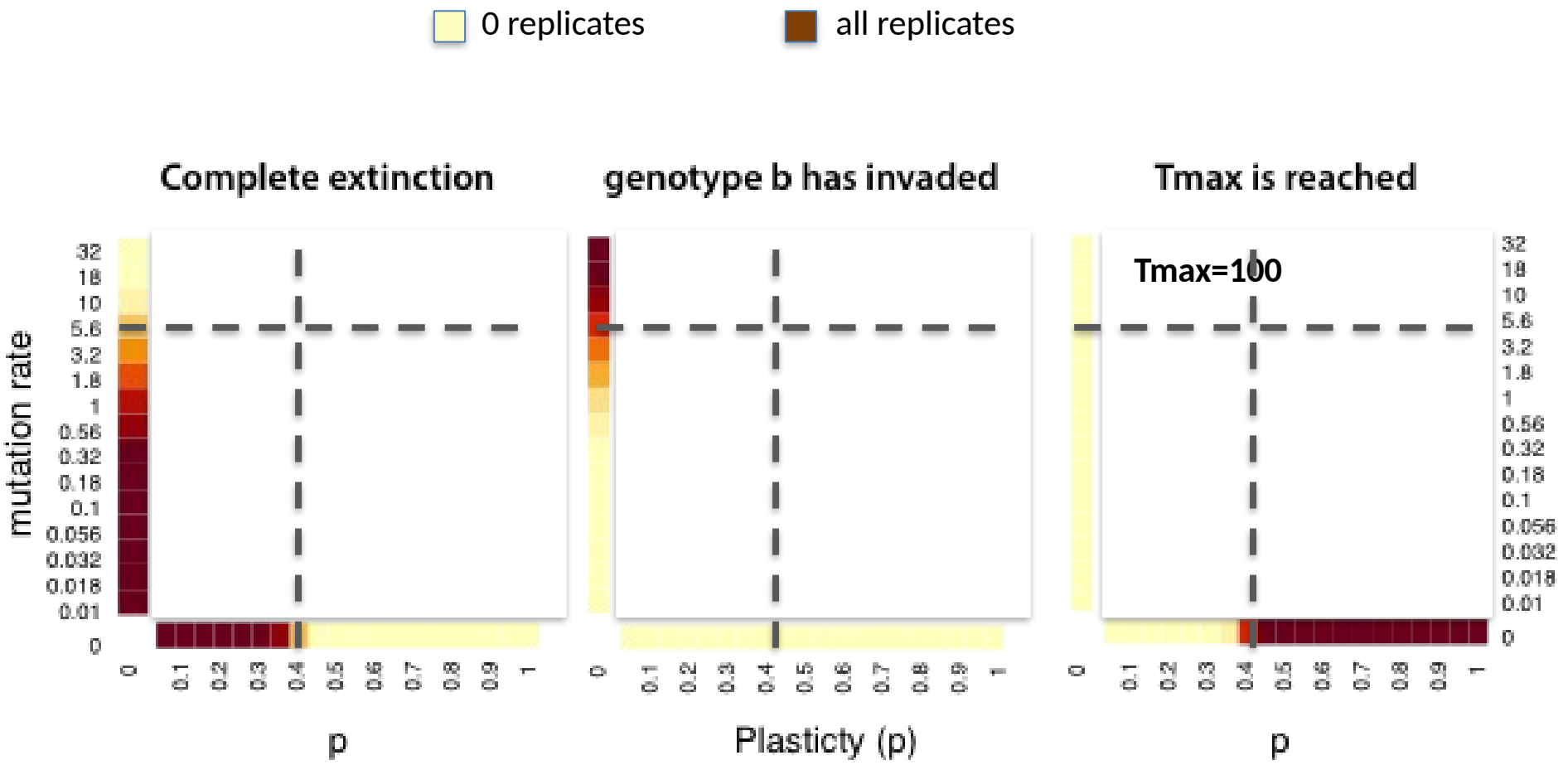
(2) The genotypes "b" have completely invaded ($n_b > K(b-d)/c$)

(3) The maximal time (Tmax) is reached

Scanning parameter space

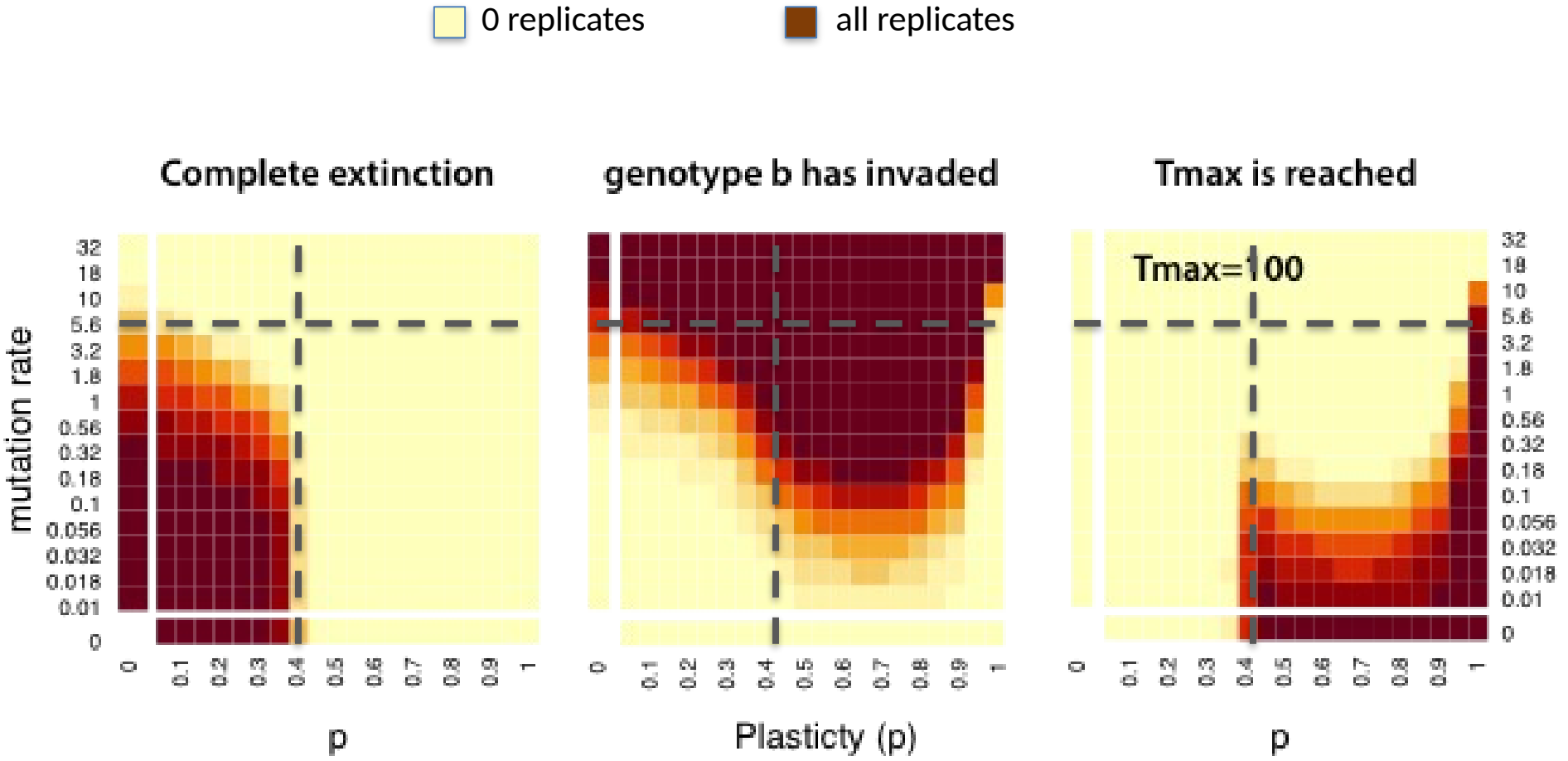


Scanning parameter space

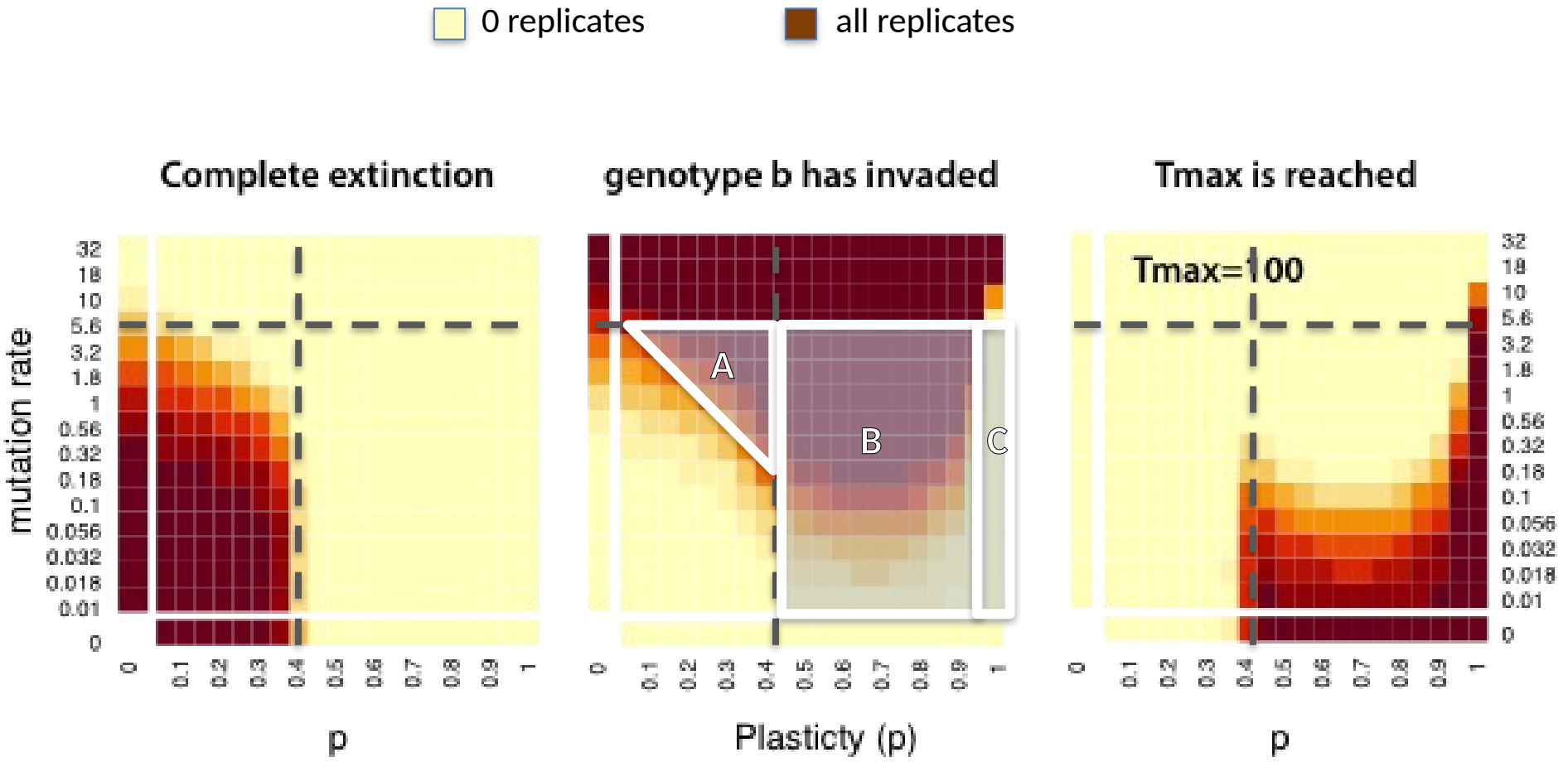


High p = long survival of genotypes ‘a’ ; high μ = frequent evolutionary rescue

Scanning parameter space

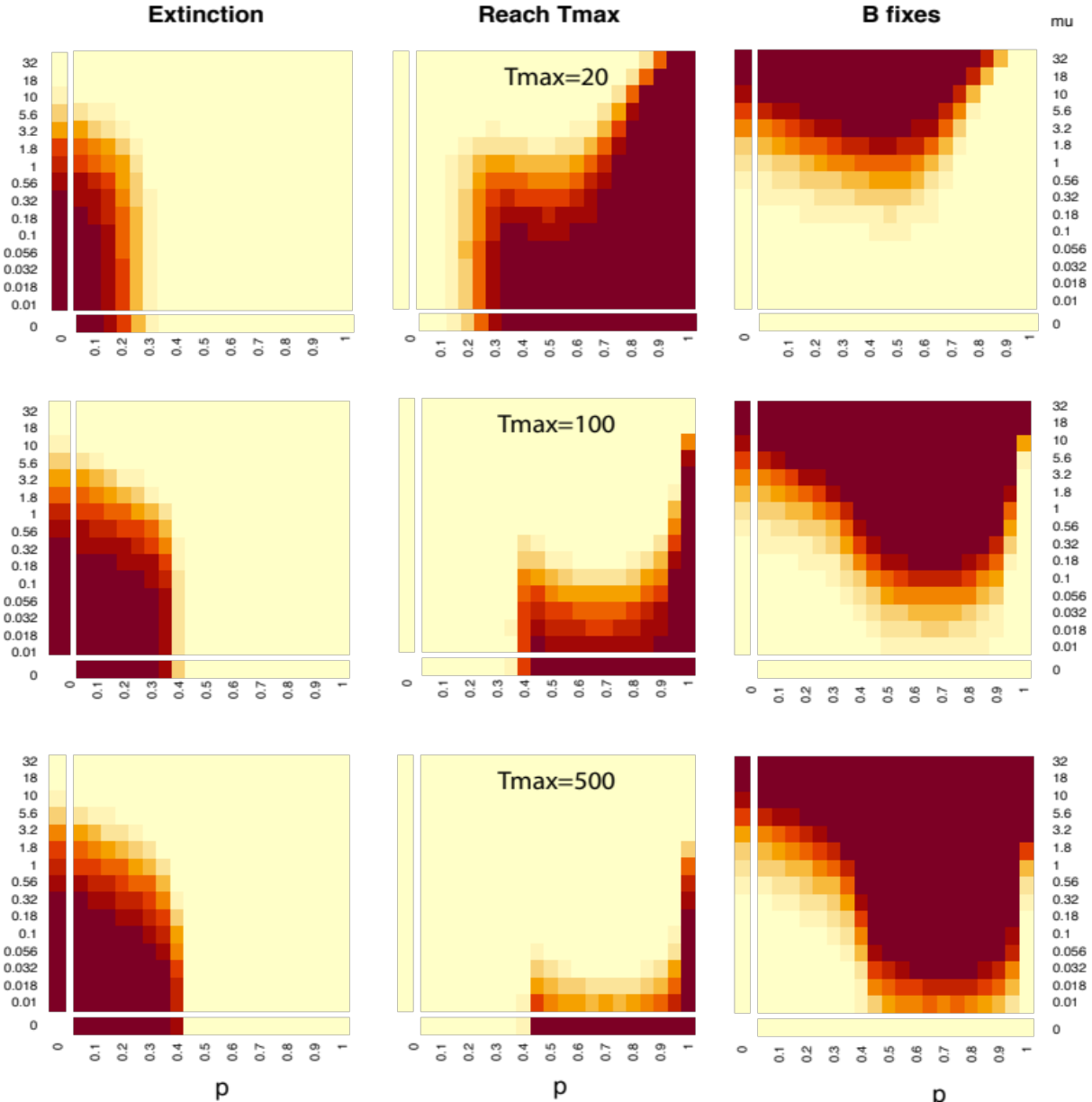


Scanning parameter space

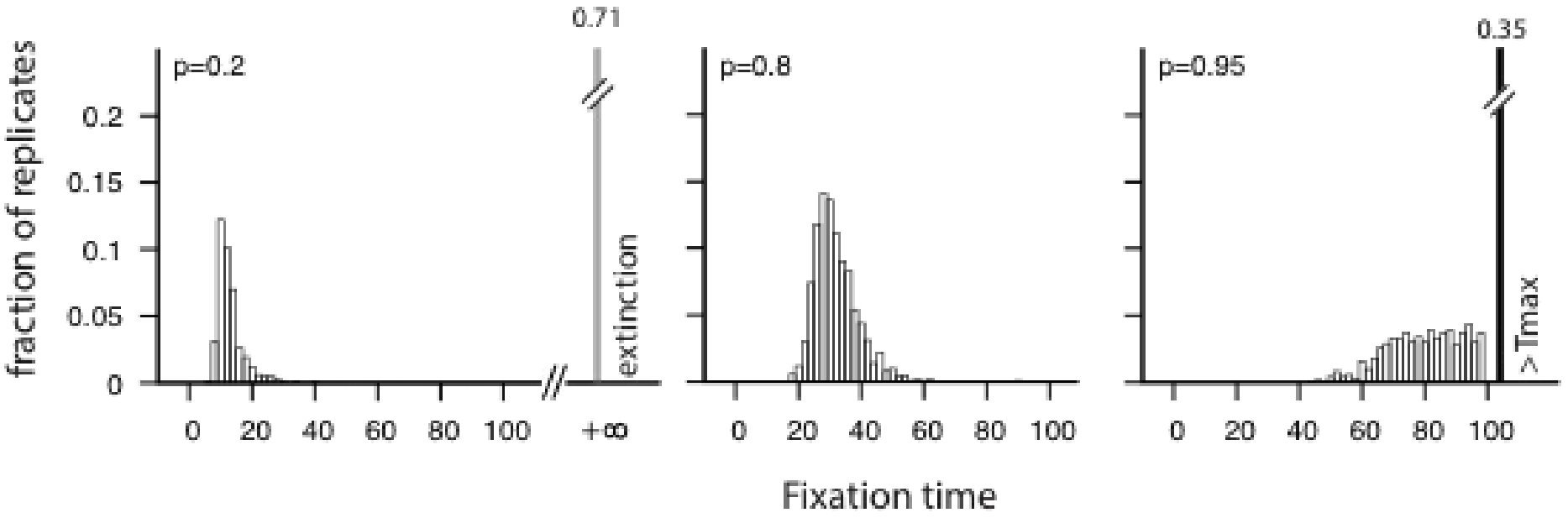


Fixation of genotype “b” is either accelerated (A), made possible (B) or slown down (C)

Increasing Tmax

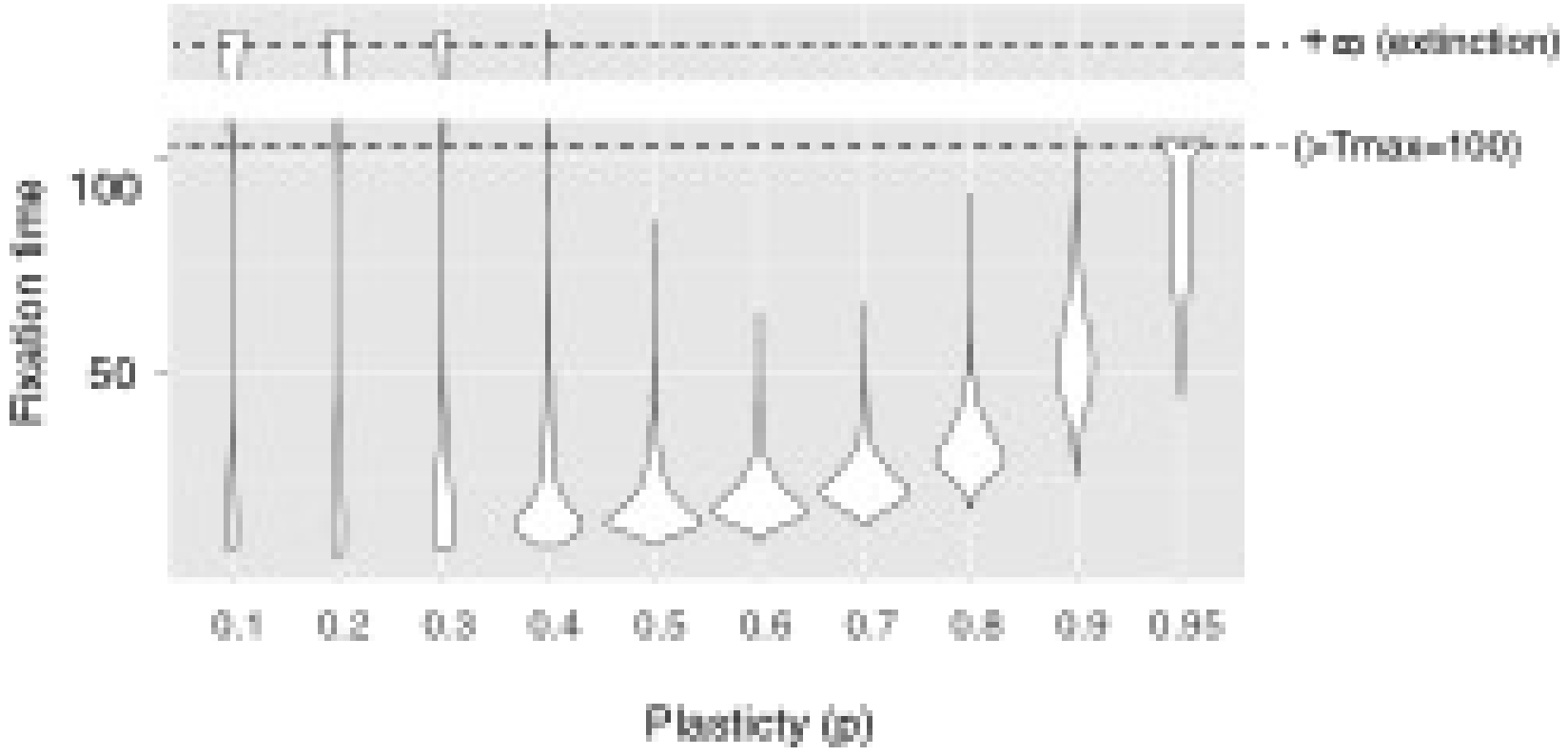


Fixation time of adaptive allele “b”



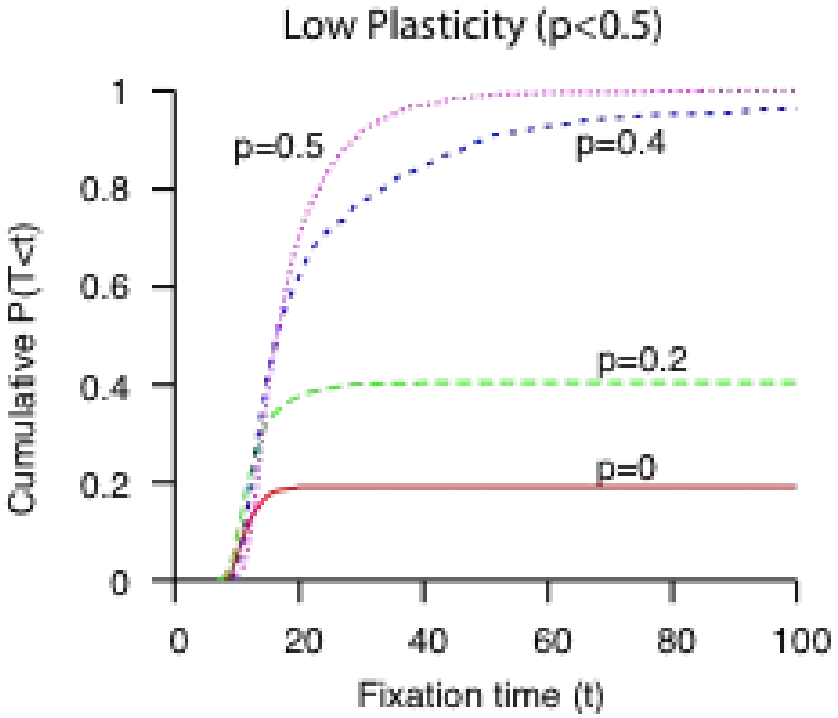
Is there is an “optimal” value for plasticity/learning ability?

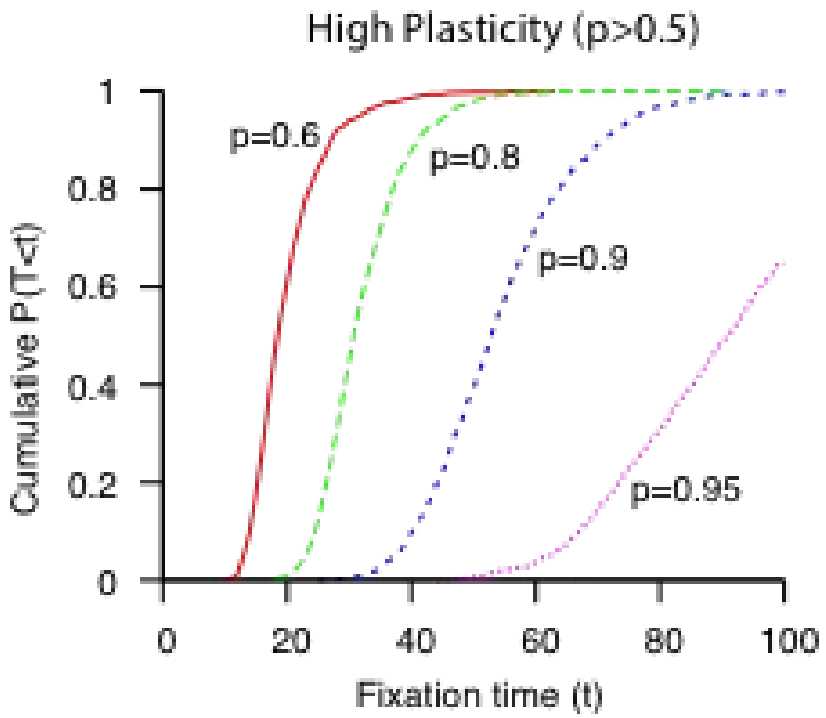
Fixation time of adaptive allele “b”



There is an “optimal” value for plasticity/learning ability!

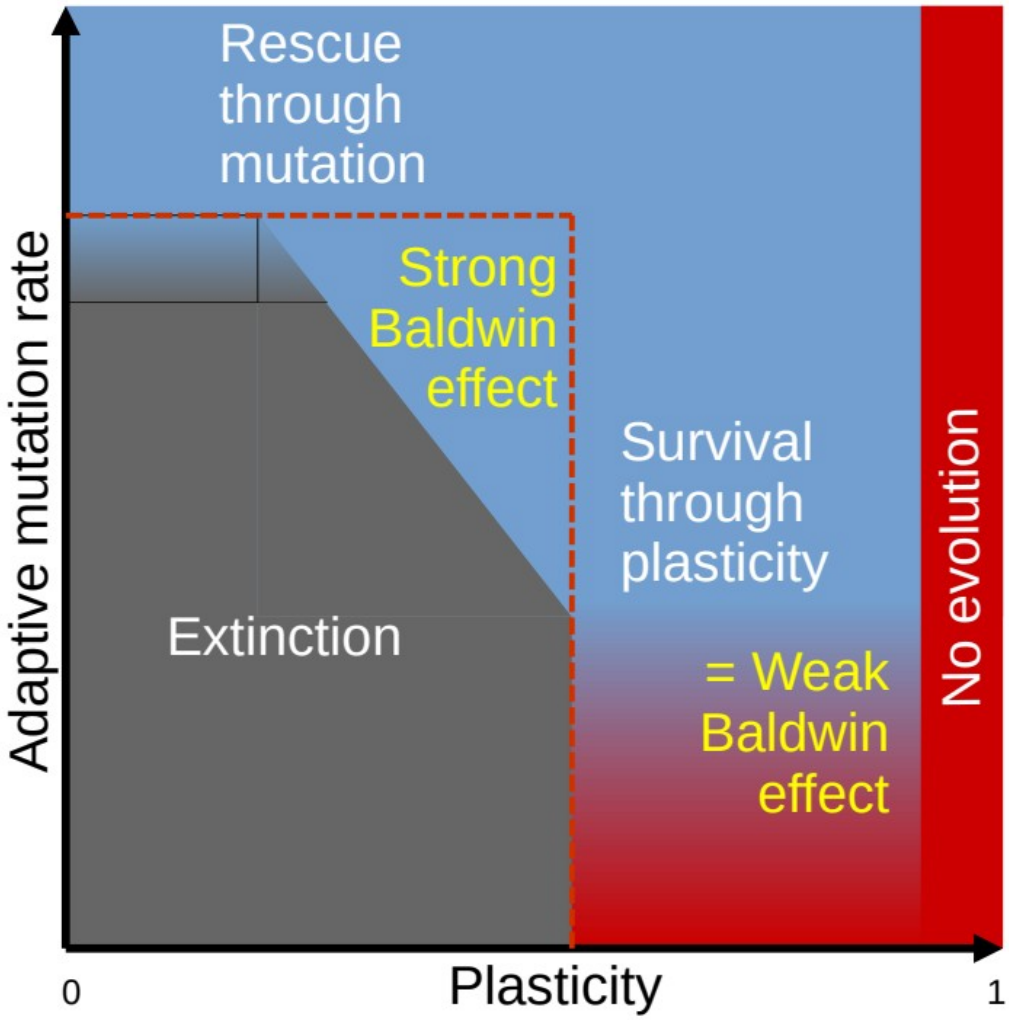
Fixation time of adaptive allele “b”





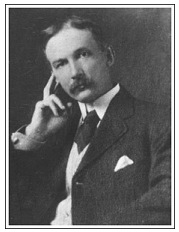
Low plasticity = many extinctions ; High plasticity = slows/prevents fixation of b

Weak vs Strong Baldwin Effect



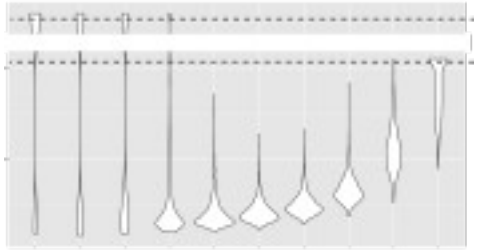
Conclusion

Clarification




Plasticity is sometimes “noise”, sometimes “learning ability”
Baldwin (1896)’s “organic selection” is learning
Simpson (1953) shifted the focus of BE and turned down the importance of population survival
Plasticity prevents extinction
Plasticity masks selection pressure

Baldwin Effect reborn (2.0)



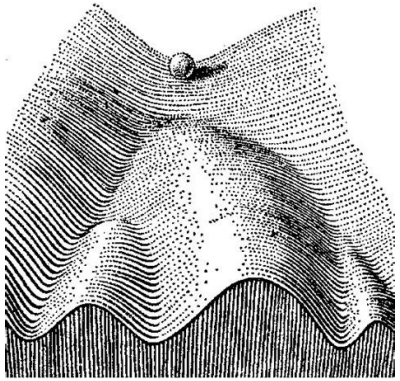
(a) Plasticity prevents extinction

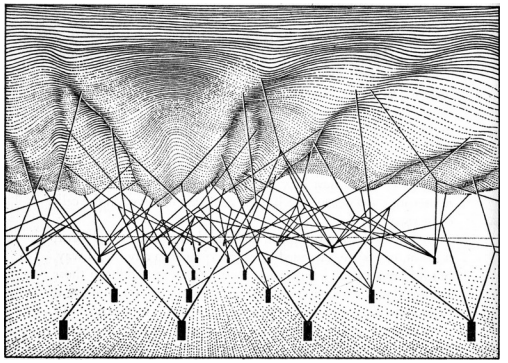
(b) For non-extinct populations, learning slows down fixation of “congenital variants”

(a+b) Intermediate *organic selection* maximizes *genic selection*

Genetic assimilation

Conrad H Waddington
1905-1975
developmental biologist,
paleontologist,
geneticist,
embryologist
and philosopher





Waddington
40's-60's

Genetic assimilation for *Canalization*

after Waddington
60's - present

Genetic assimilation (no specific process)

[e.g. Waddington, *Nature*, 1942; Waddington, *Evolution*, 1953; Waddington, *Adv in Genetics*, 1961
For a discussion on the difference(s) between BE and GA, see Loison, Paradigmi, 2020]