Niche construction

A new dimension for biological and cultural evolution

Marc Feldman

Stanford, July 2006

see book:

Niche Construction: The Neglected Process in Evolution

J. Odling-Smee, K. Laland, M. W. Feldman

Princeton University Press 2003

Definition of Niche Construction:

"*Niche construction* occurs when an organism modifies the feature-factor relationship between itself and its environment by actively changing one or more of the factors in its environment, either by physically perturbing factors at its current location in space and time, or by relocating to a different space-time address, thereby exposing itself to different factors."

(Book, pp.41)

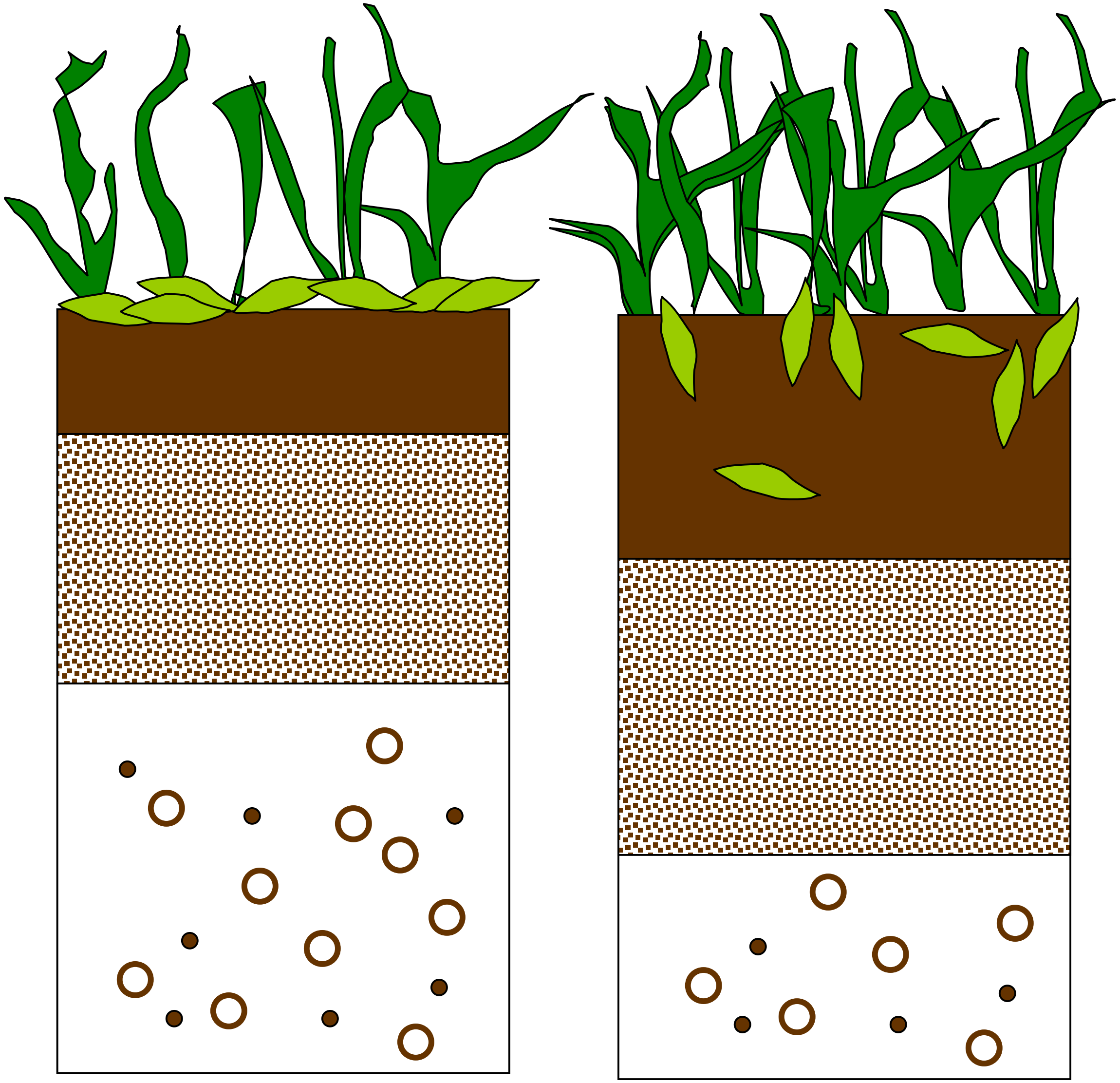
Categories of Niche Construction:

	PERTURBATION	RELOCATION
INCEPTIVE	*Organisms initiate a change in their selective environment by physically modifying their surroundings.* e.g. Emission of detritus	*Organisms expose themselves to a novel selective environment by moving to or growing into a new place.* e.g. Invasion of a new habitat.
COUNTER-ACTIVE	*Organisms counteract a prior change in the environment by physically modifying their surroundings.* e.g. Thermo-regulation of nests.	*Organisms respond to a change in the environment by moving to or growing into a more suitable place.* e.g. Seasonal migration.

Examples of Niche Construction

Beaver's dam.

Examples of Niche Construction



WITHOUT EARTHWORMS

WITH EARTHWORMS

ENHANCED PLANT YIELD

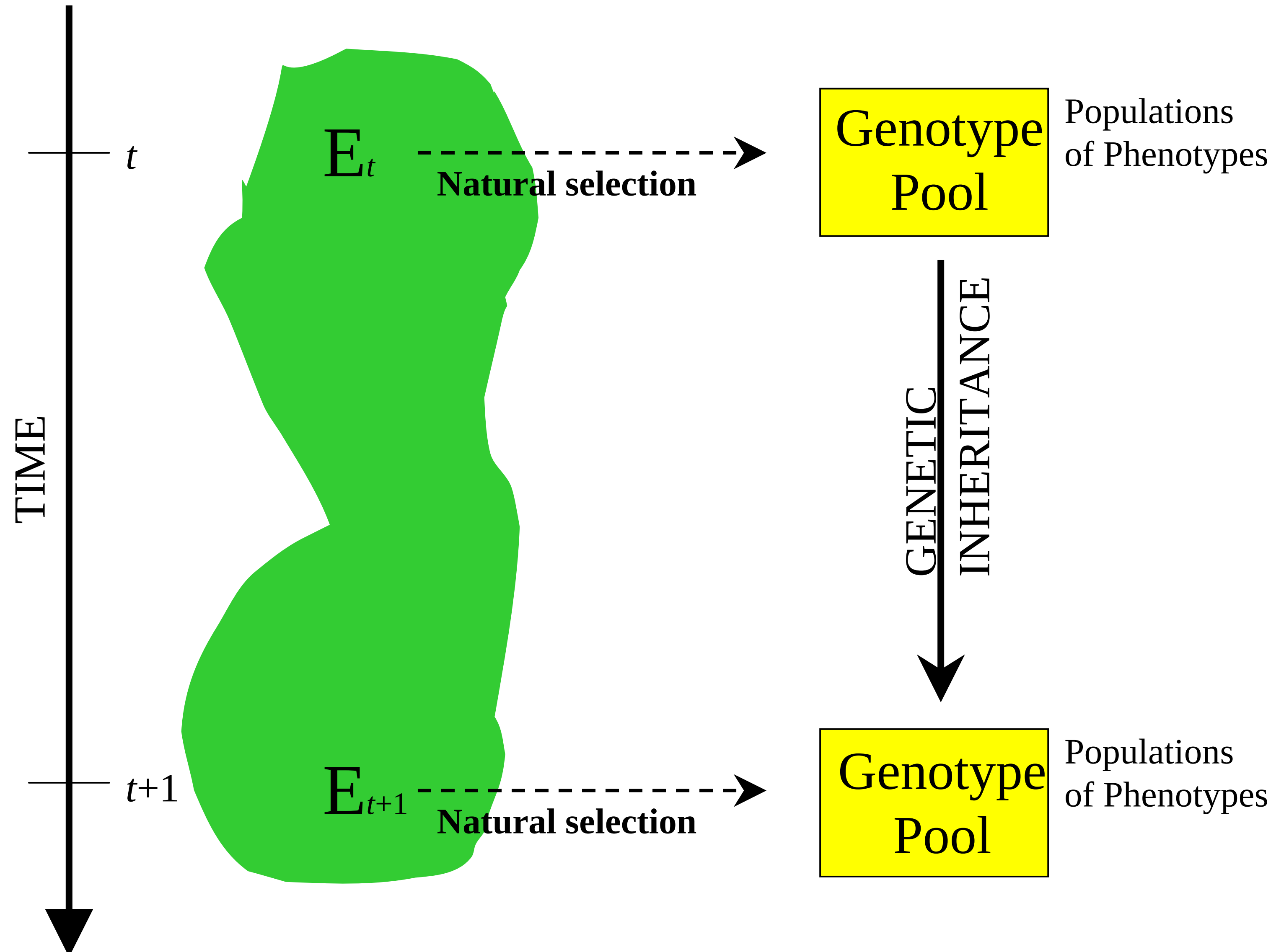
LESS SURFACE LITTER

MORE TOPSOIL

MORE ORGANIC CARBON, NITROGEN AND POLYSACCHARIDES

ENHANCED POROSITY AERATION AND DRAINAGE

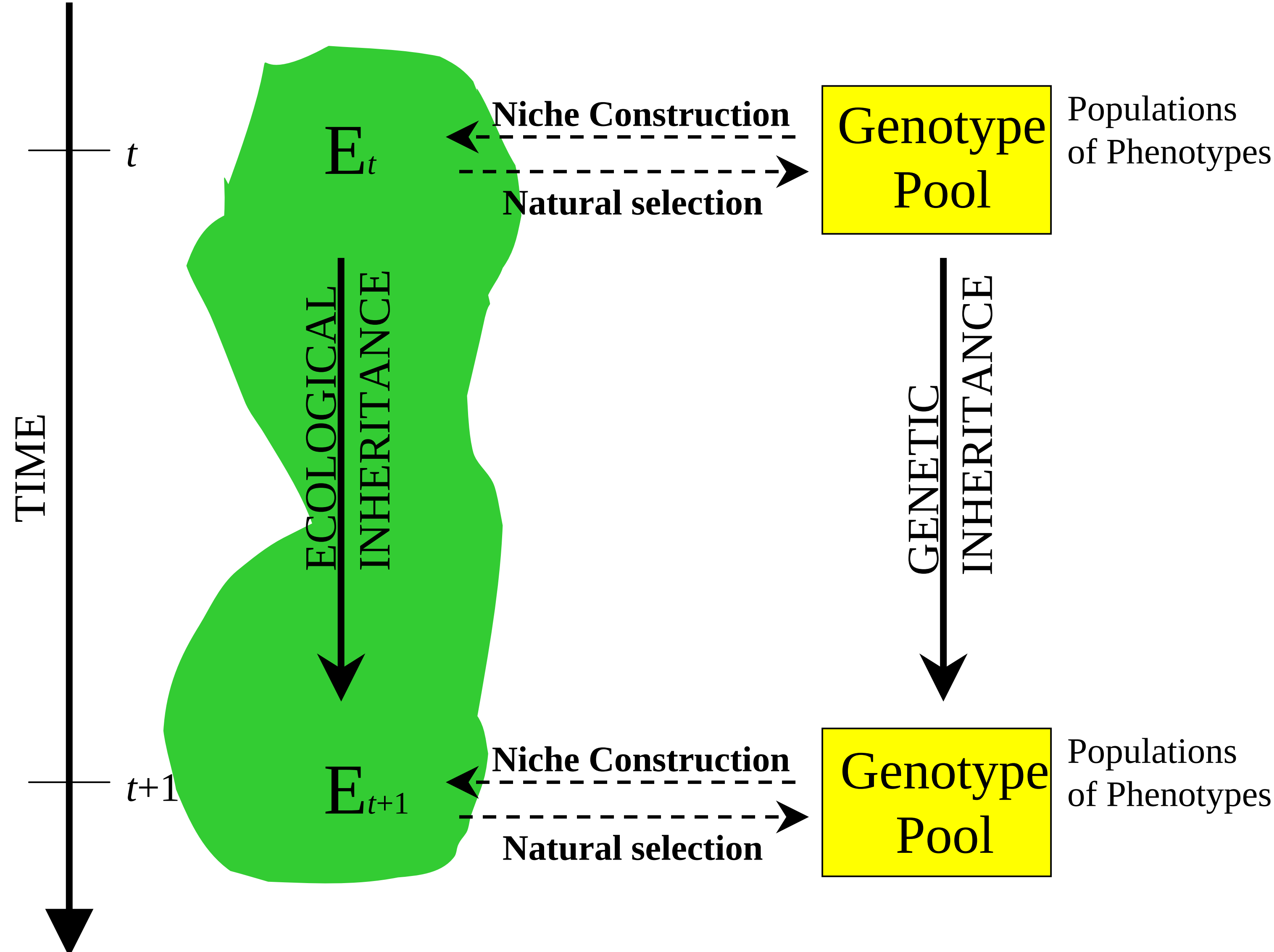
Standard Evolutionary Theory



$dE / dt = g(E)$

$dO / dt = f(O, E)$

Extended Evolutionary Theory



$dE / dt = g(E, O)$ $dO / dt = f(O, E)$ (Lewontin)

Consequences for Adaptation

[Adaptation is] …those transgenerational alterations of the features and capacities of organisms in a lineage that enable them to solve … problems posed by the environment

(Burian, 1992, p.7)

Organisms do not adapt to their environments; they construct them out of the bits and pieces of the external world.

(Lewontin, 1983, p.280)

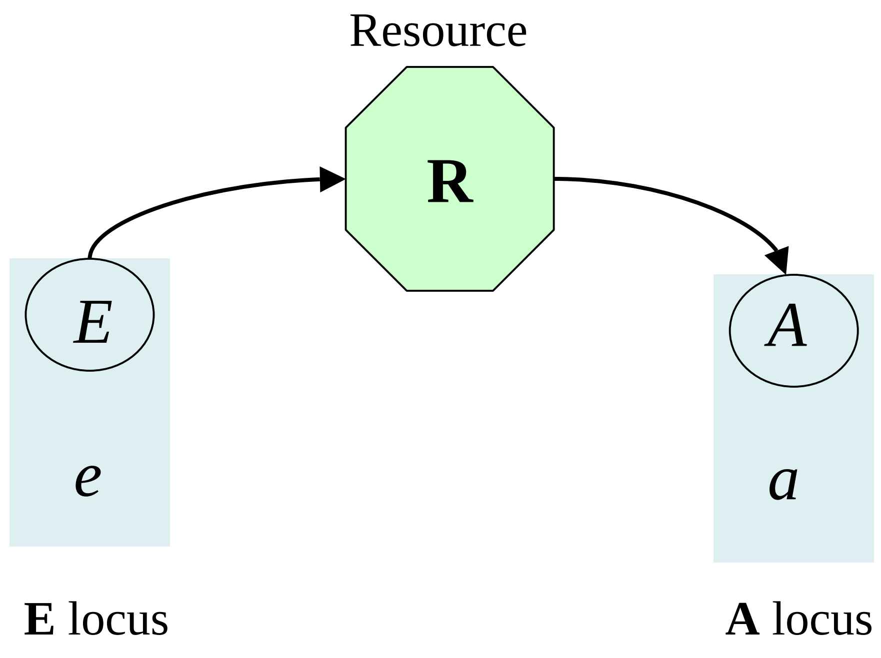
Implications of Niche Construction

1. Evolutionary Theory.
2. Ecology.
3. Human Sciences and Social Dynamics.

Implications of Niche Construction

1. Evolutionary Theory.

   - Model based on 2 locus population genetic theory.



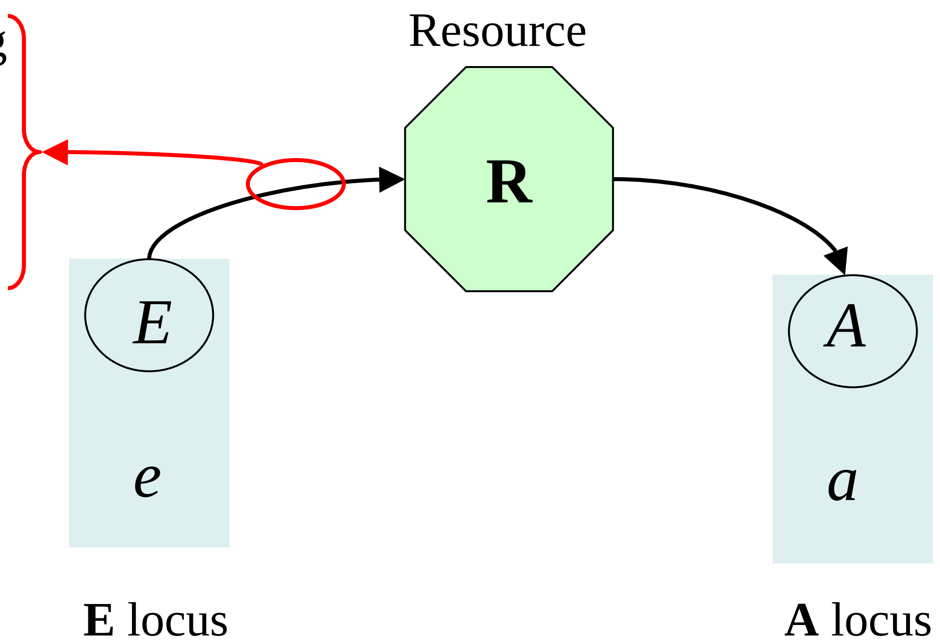
x = frequency(E) p = frequency(A)

Implications of Niche Construction

1. Evolutionary Theory.

- Model based on 2 locus population genetic theory.

1. Equal weighting
2. Recency effect
3. Primacy effect



x = frequency(E) p = frequency(A)

Implications of Niche Construction

1. Total Resource at time t, $R_t = \sum_{i=t-n+1}^{t} \pi_i x_i$

1. Equal weighting: $\pi_t = \pi_{t-1} = \pi_{t-2} = \ldots = \pi_{t-n+1} = \frac{1}{n}$ → $R_t = \frac{1}{n} \sum_{i=t-n+1}^{t} x_i$

2. Recency effect

3. Primacy effect

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2. Recency effect: $\pi_t > \pi_{t-1} > \pi_{t-2} > \ldots > \pi_{t-n+1}$. → $R_t = \frac{1-\mu}{1-\mu^n} \sum_{i=t-n+1}^{t} \mu^{t-i} x_i$

3. Primacy effect

2. $\pi_t = \frac{1}{\varphi}$, $\pi_{t-1} = \frac{\mu}{\varphi}$, $\pi_{t-2} = \frac{\mu^2}{\varphi}$, . . . , $\pi_{t-n+1} = \frac{\mu^{n-1}}{\varphi}$ where $0 < \mu < 1$, and $\varphi = \frac{1-\mu^n}{1-\mu}$

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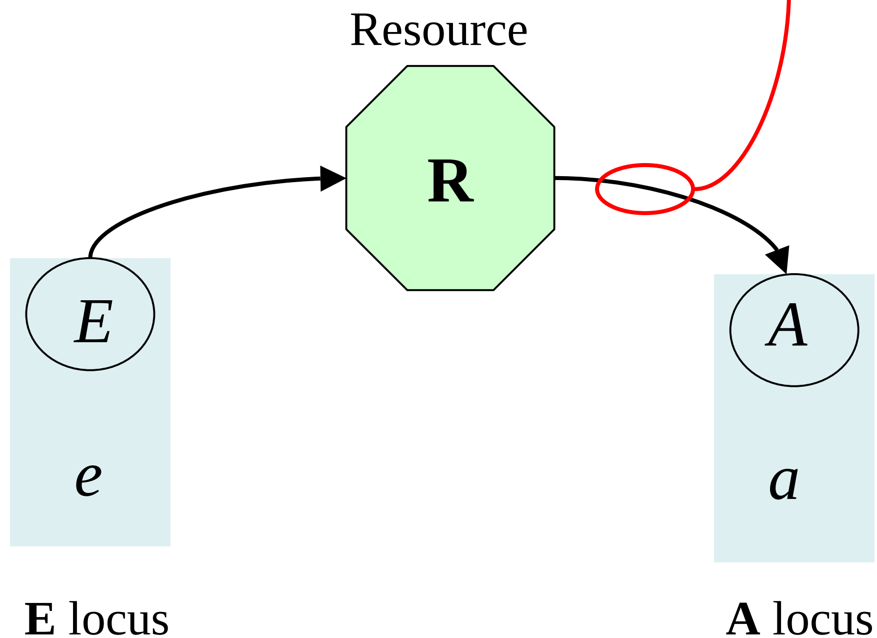
Evolutionary Theory

Fitness Table (for diploid model):

	EE (α_1)	Ee (1)	ee (α_2)
AA (η_1)	$w_{11} = \alpha_1\eta_1 + \varepsilon R^f$	$w_{12} = \eta_1 + \varepsilon R^f$	$w_{13} = \alpha_2\eta_1 + \varepsilon R^f$
Aa (1)	$w_{21} = \alpha_1 + \varepsilon[R(1-R)]^{f/2}$	$w_{22} = 1 + \varepsilon[R(1-R)]^{f/2}$	$w_{23} = \alpha_2 + \varepsilon[R(1-R)]^{f/2}$
aa (η_2)	$w_{31} = \alpha_1\eta_2 + \varepsilon(1-R)^f$	$w_{32} = \eta_2 + \varepsilon(1-R)^f$	$w_{33} = \alpha_2\eta_2 + \varepsilon(1- R)^f$



Where $-1 < \varepsilon < 1$,
$f > 0$.

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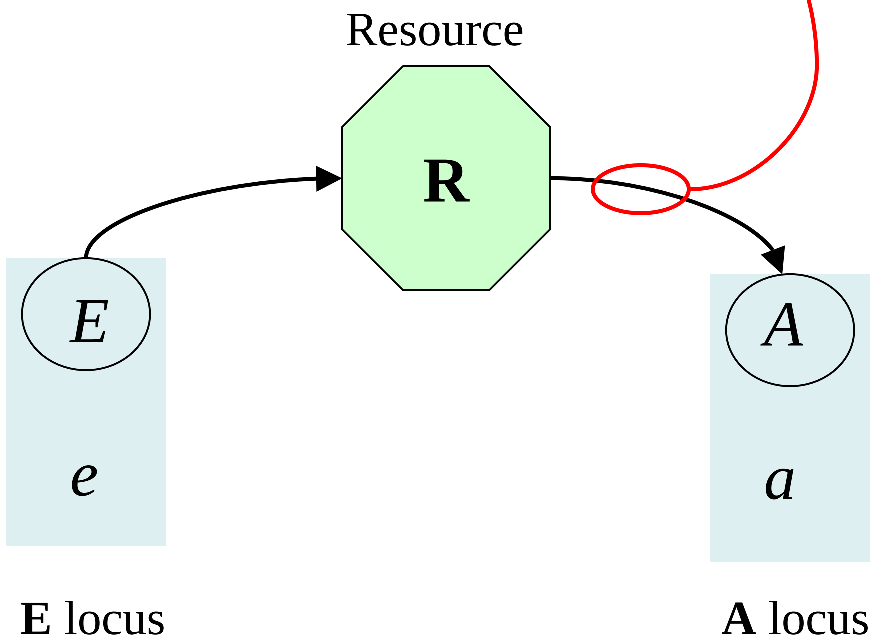

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$f > 0$.

Evolutionary Theory

Frequencies:

	A	a
E	u_1	u_2
e	u_3	u_4

Gametic Recursions:

$$Wu_1' = [u_1(u_1w_{11} + u_2w_{21} + u_3w_{12} + u_4w_{22})] - rw_{22}D \qquad (1)$$

$$Wu_2' = [u_2(u_1w_{21} + u_2w_{31} + u_3w_{22} + u_4w_{32})] + rw_{22}D \qquad (2)$$

$$Wu_3' = [u_3(u_1w_{12} + u_2w_{22} + u_3w_{13} + u_4w_{23})] + rw_{22}D \qquad (3)$$

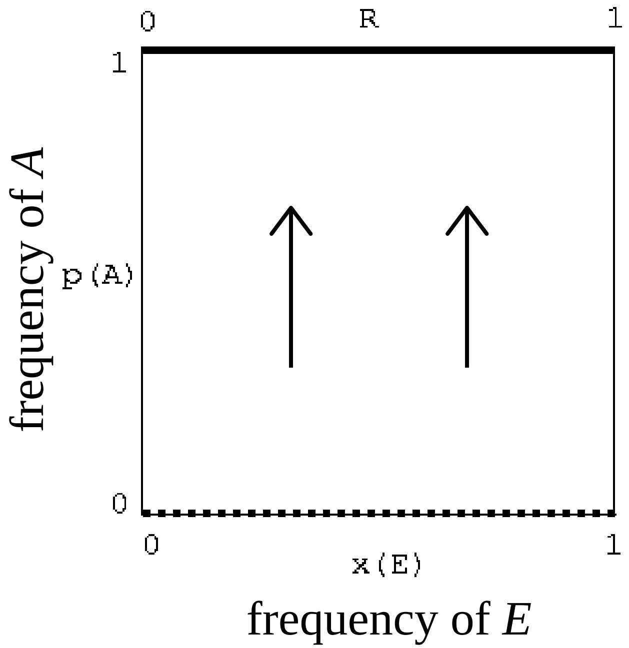
$$Wu_4' = [u_4(u_1w_{22} + u_2w_{32} + u_3w_{23} + u_4w_{33})] - rw_{22}D, \qquad (4)$$

where
r is the recombination rate,
$D = u_1u_4 - u_2u_3$, is the linkage disequilibrium and
W is the sum of the right hand sides.

Evolutionary Theory

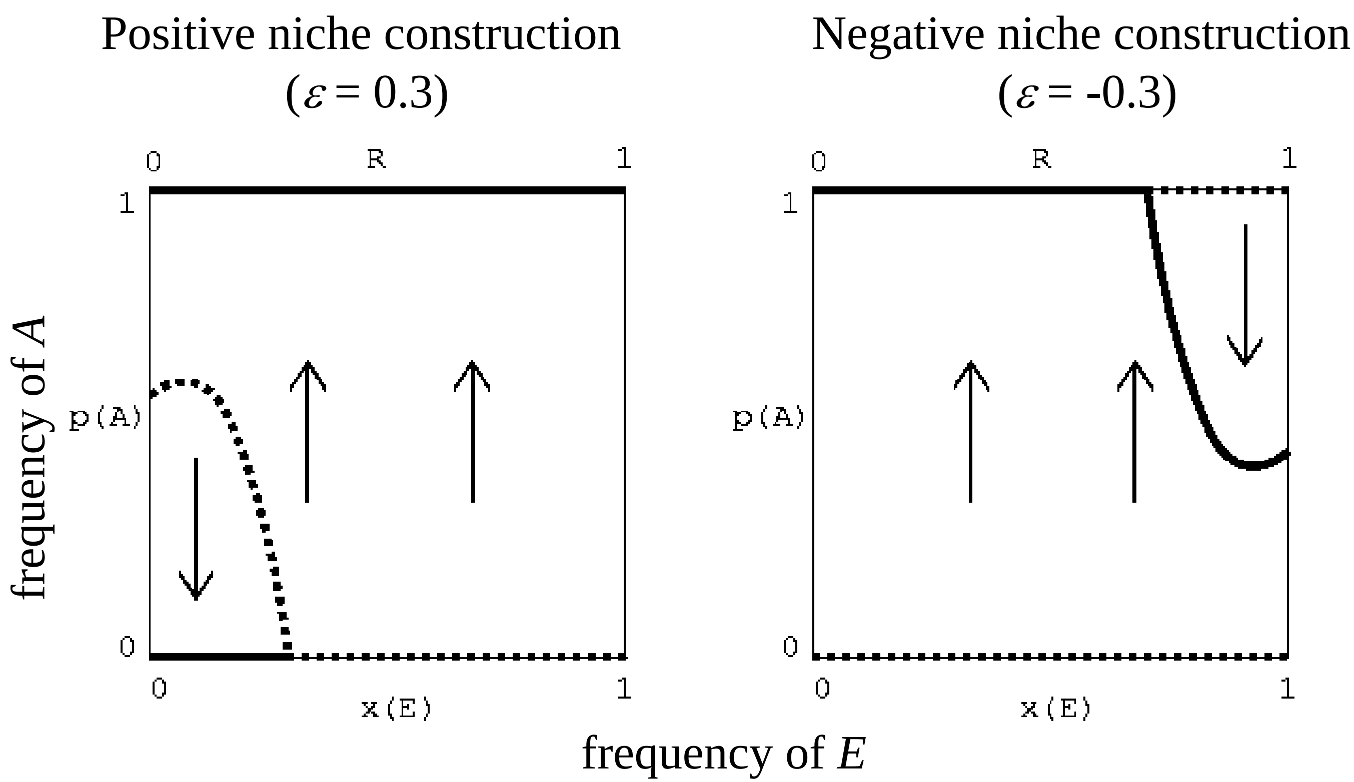
External Selection favoring *A* over *a*:

No niche construction
($\varepsilon = 0$)



Evolutionary Theory

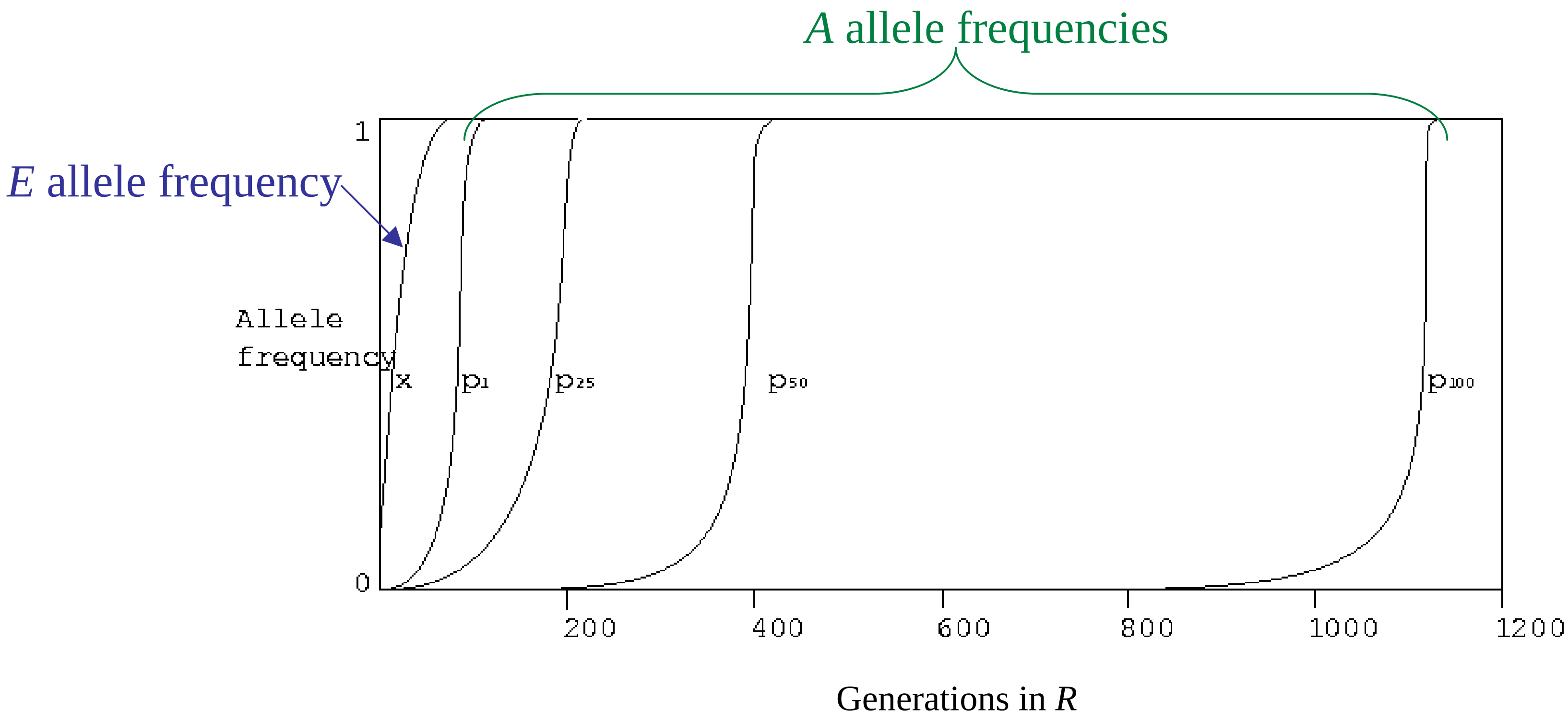
External Selection favoring *A* over *a*:



•Niche construction can generate polymorphic equilibria.

Evolutionary Theory

Illustrating evolutionary inertia:



•Niche construction can generate timelags (especially if primacy effect).

Evolutionary Theory

- Overide external selection pressures
- Create new evolutionary trajectories
- Influence genetic variation (e.g. polymorphic equilibria)
- Generate unusual dynamics (evolutionary inertia, momentum)

Implications of Niche Construction

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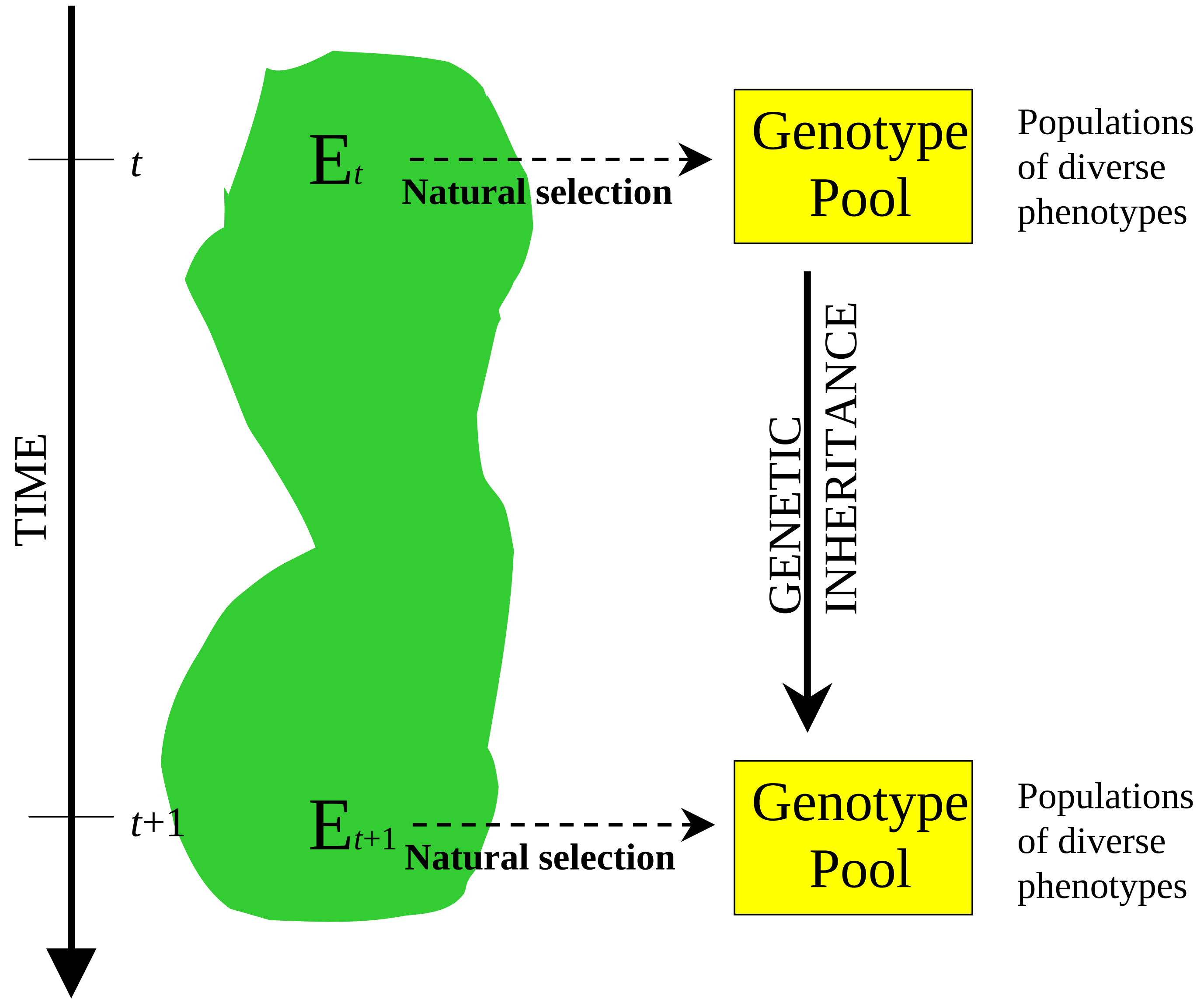
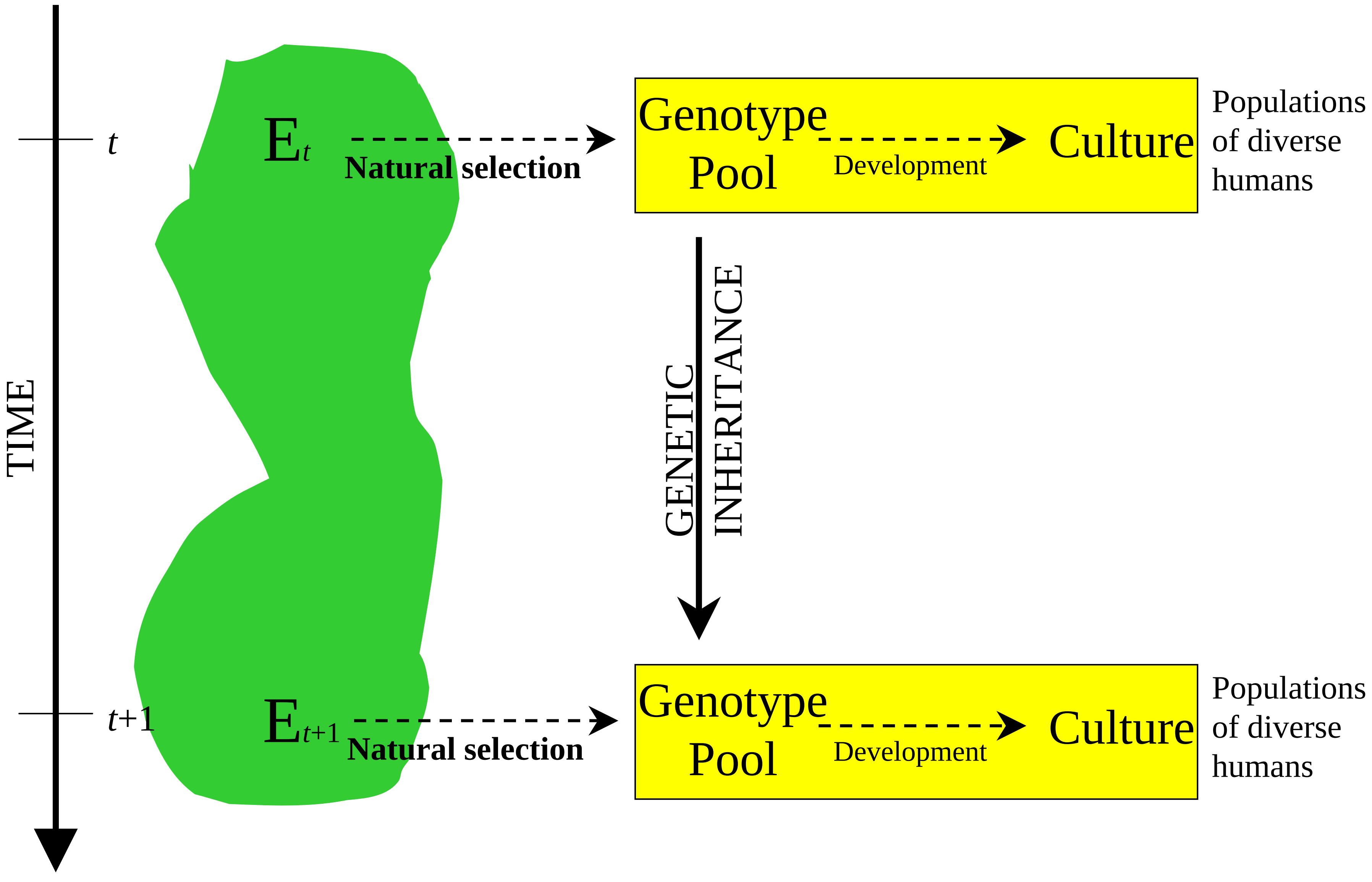
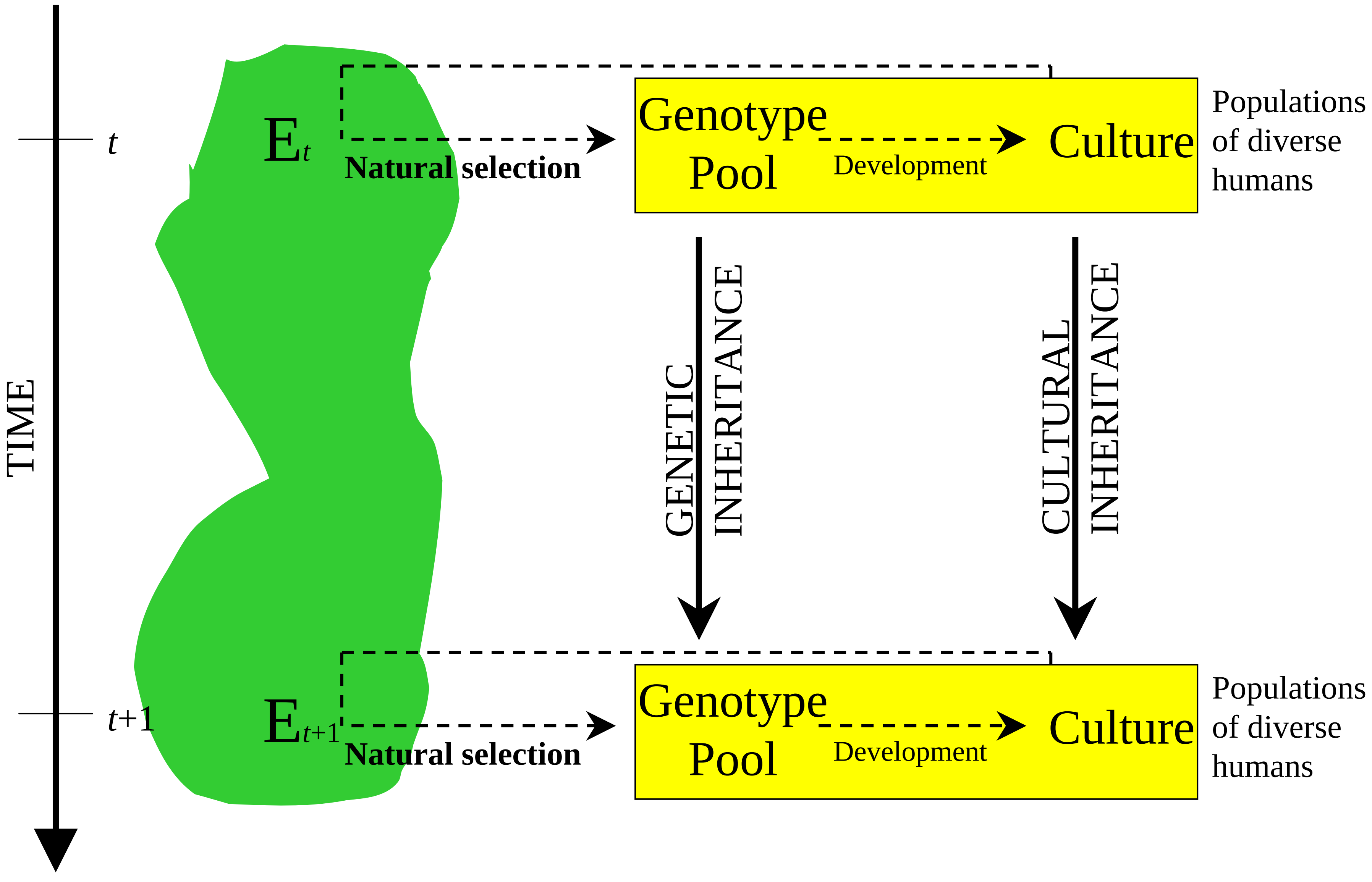
Standard Evolutionary Theory



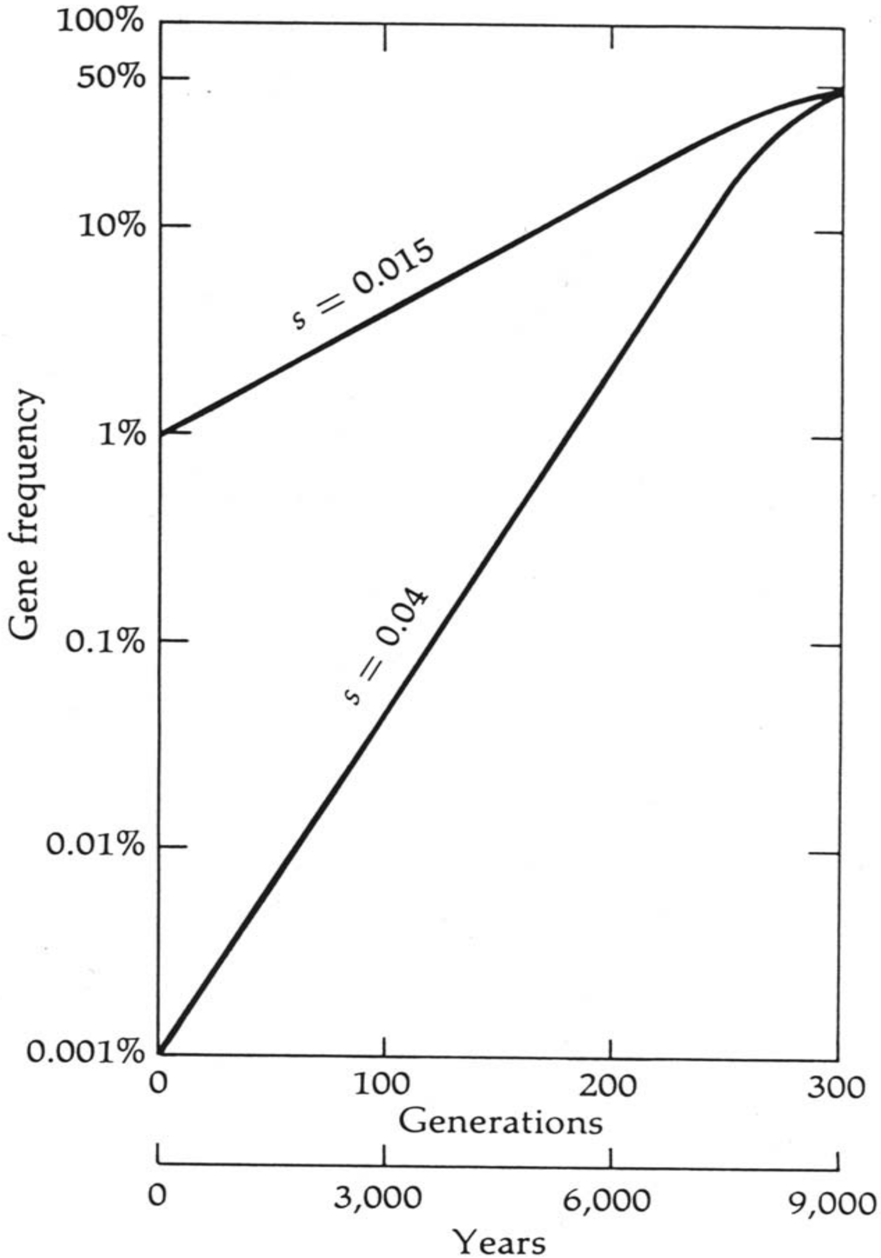
Human Sociobiology

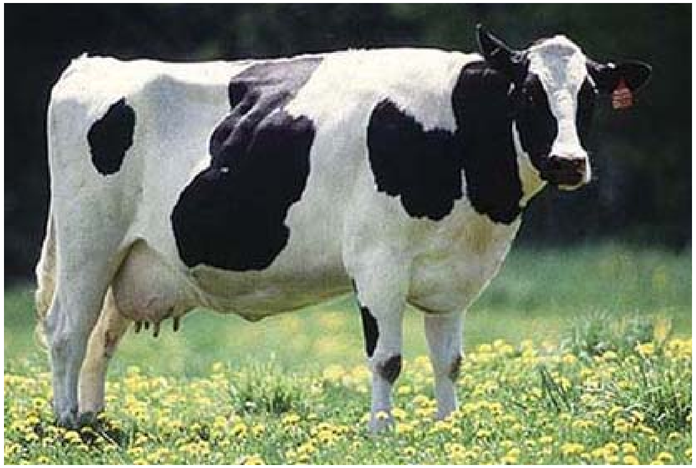


Gene - Culture Coevolution



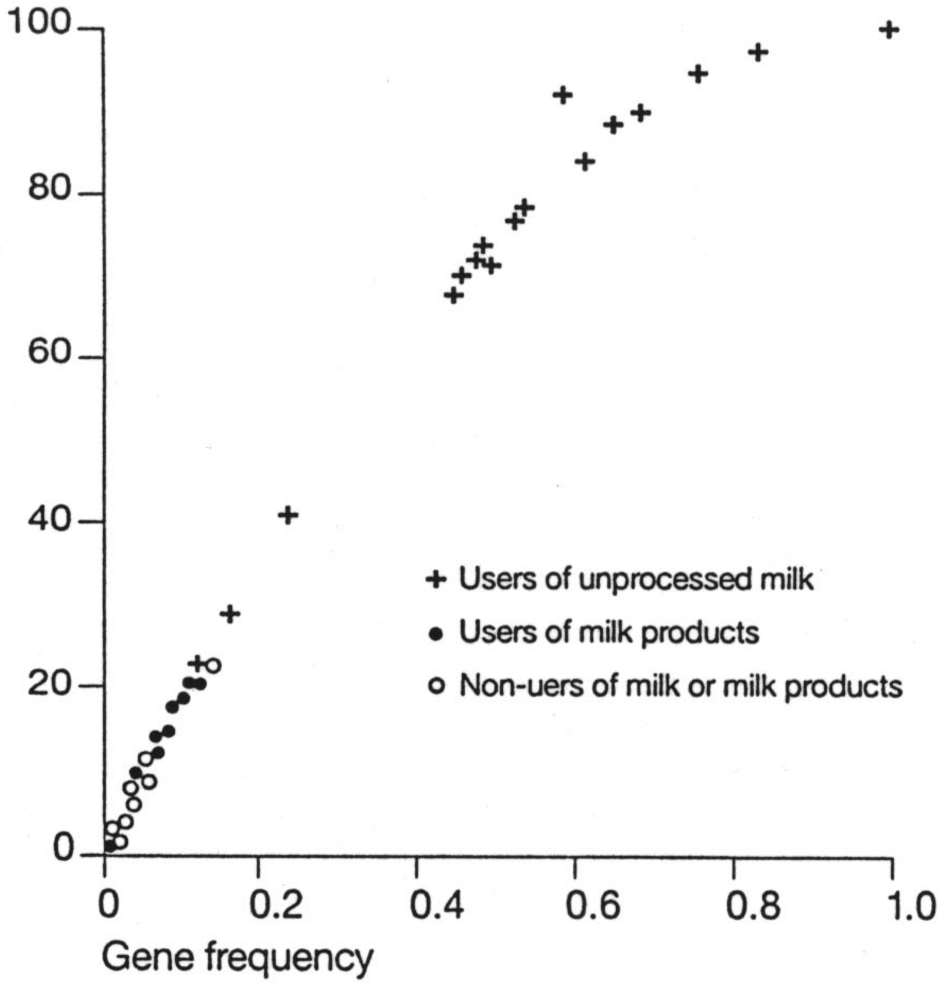
Dairy Farming & Lactose Absorption



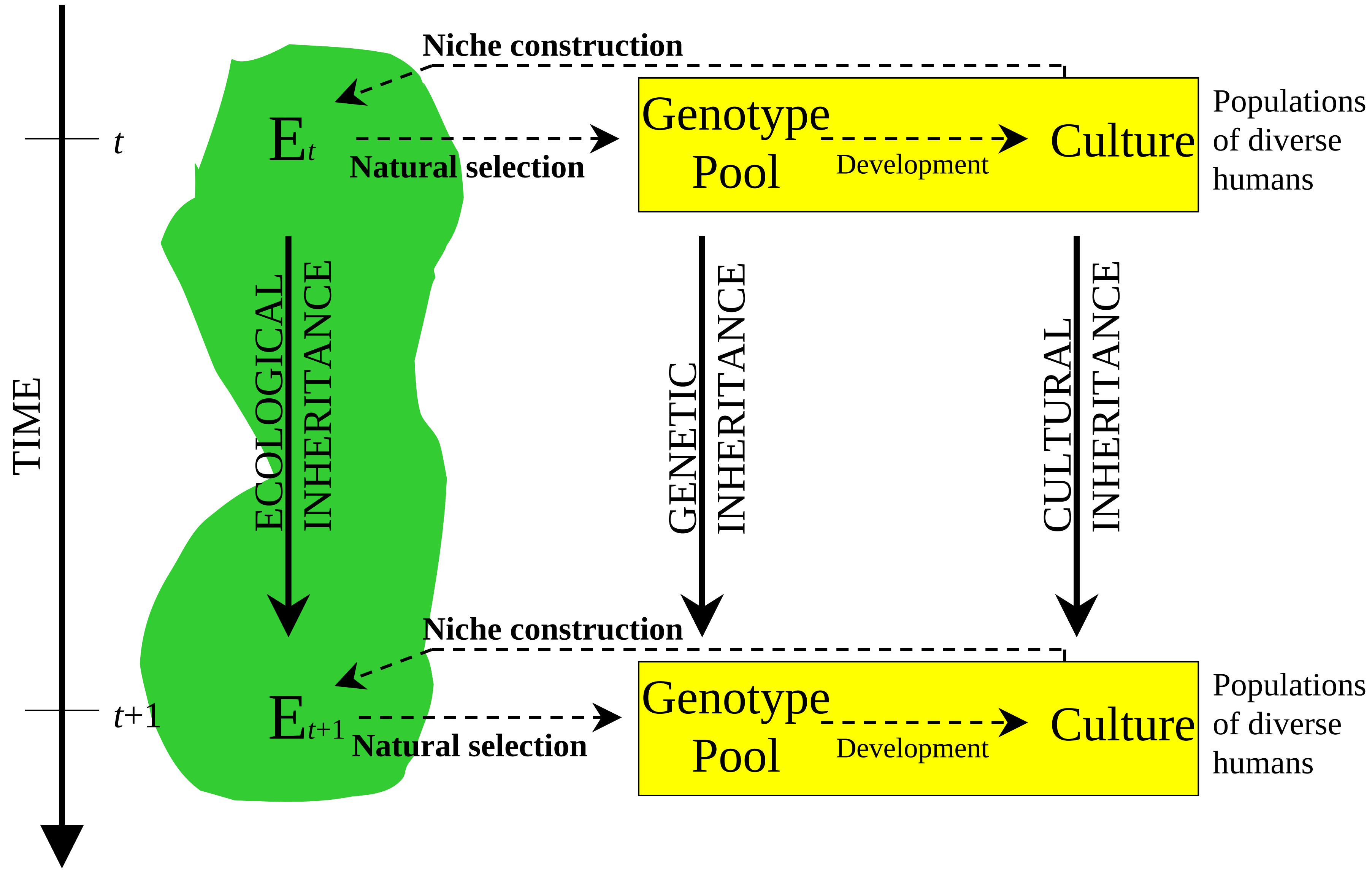


Dairy Farming & Lactose Absorption

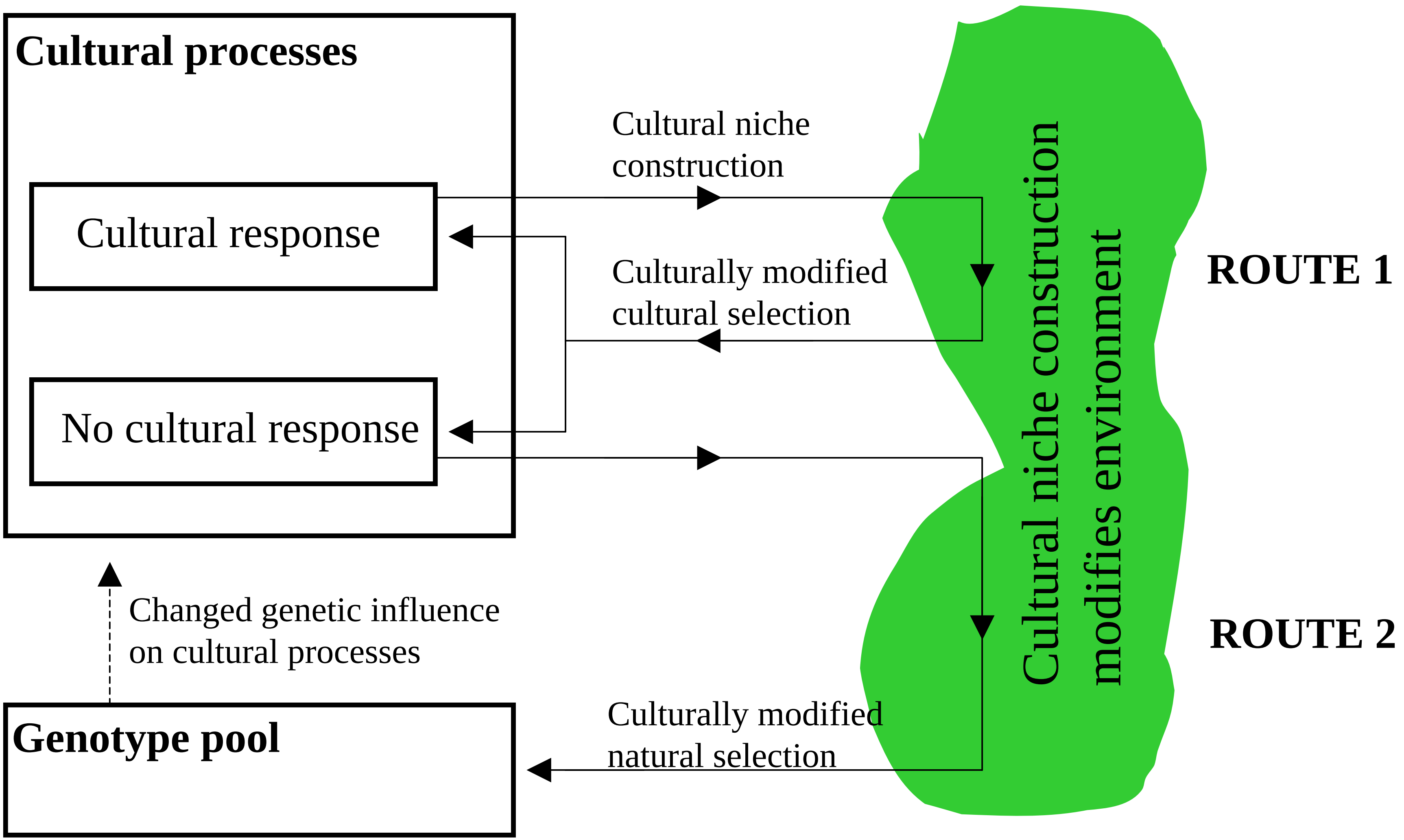
- Genetic signature of cultural niche construction



Triple Inheritance System



Cultural Niche Construction



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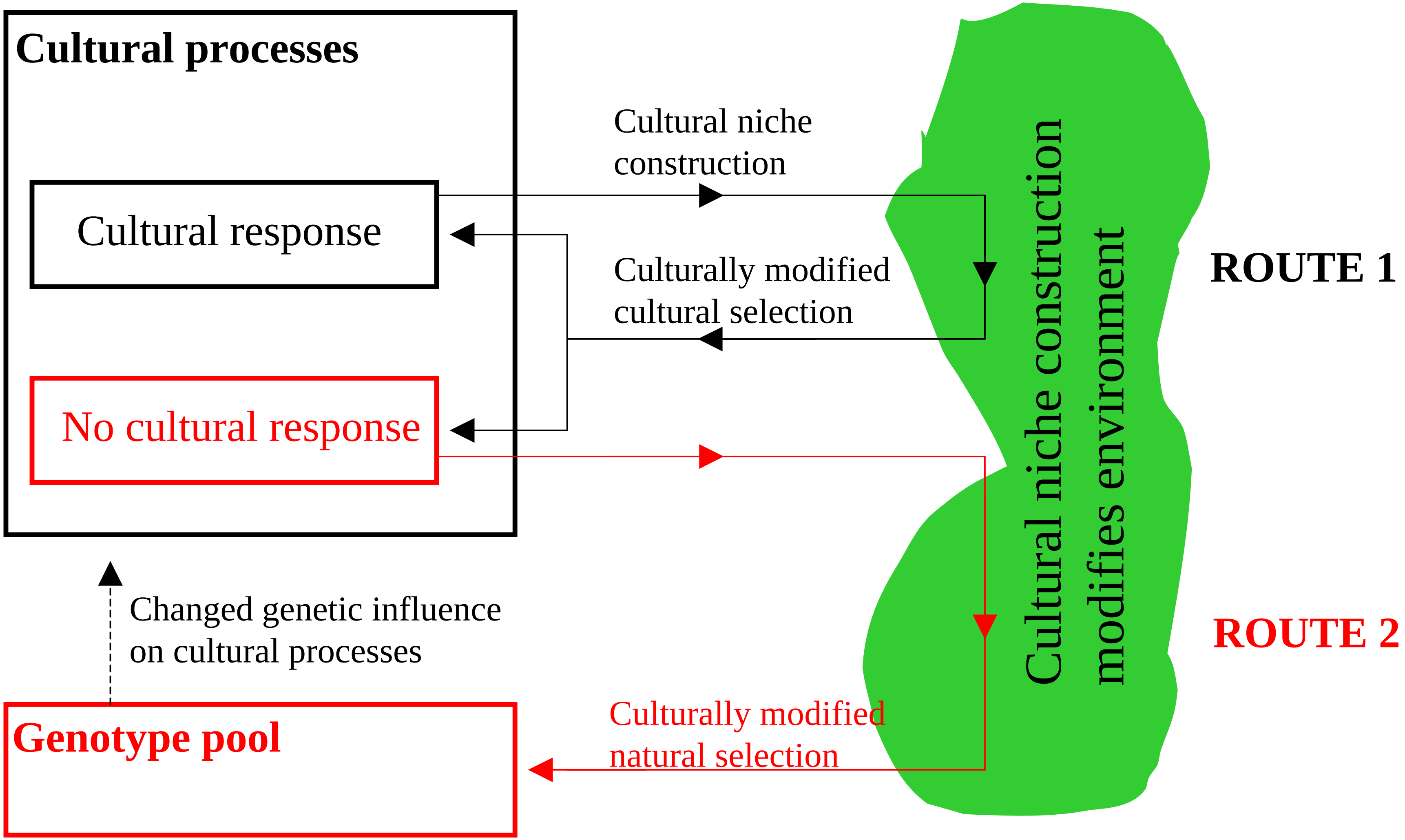
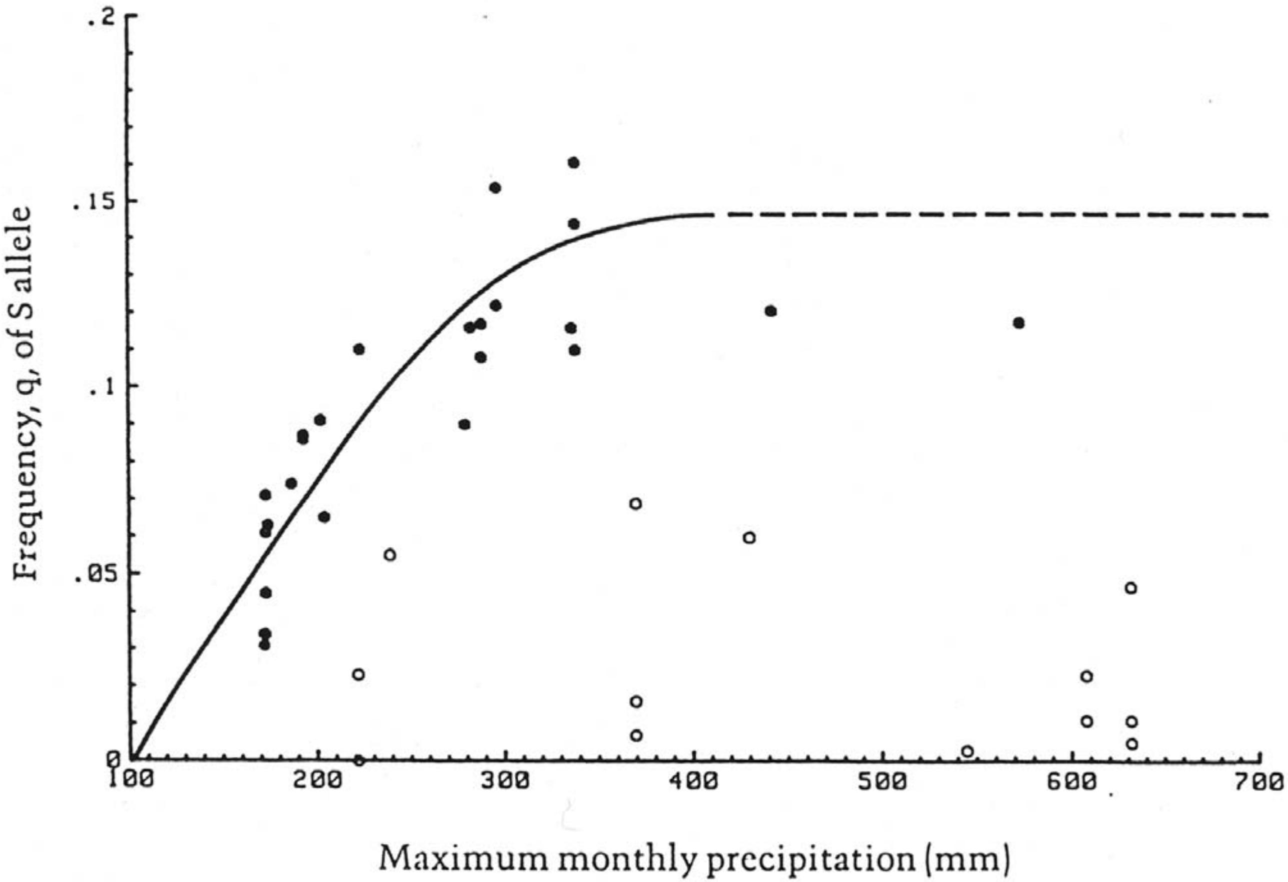
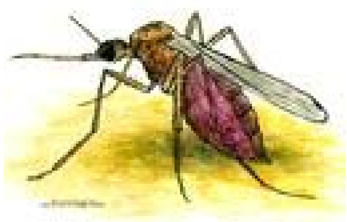


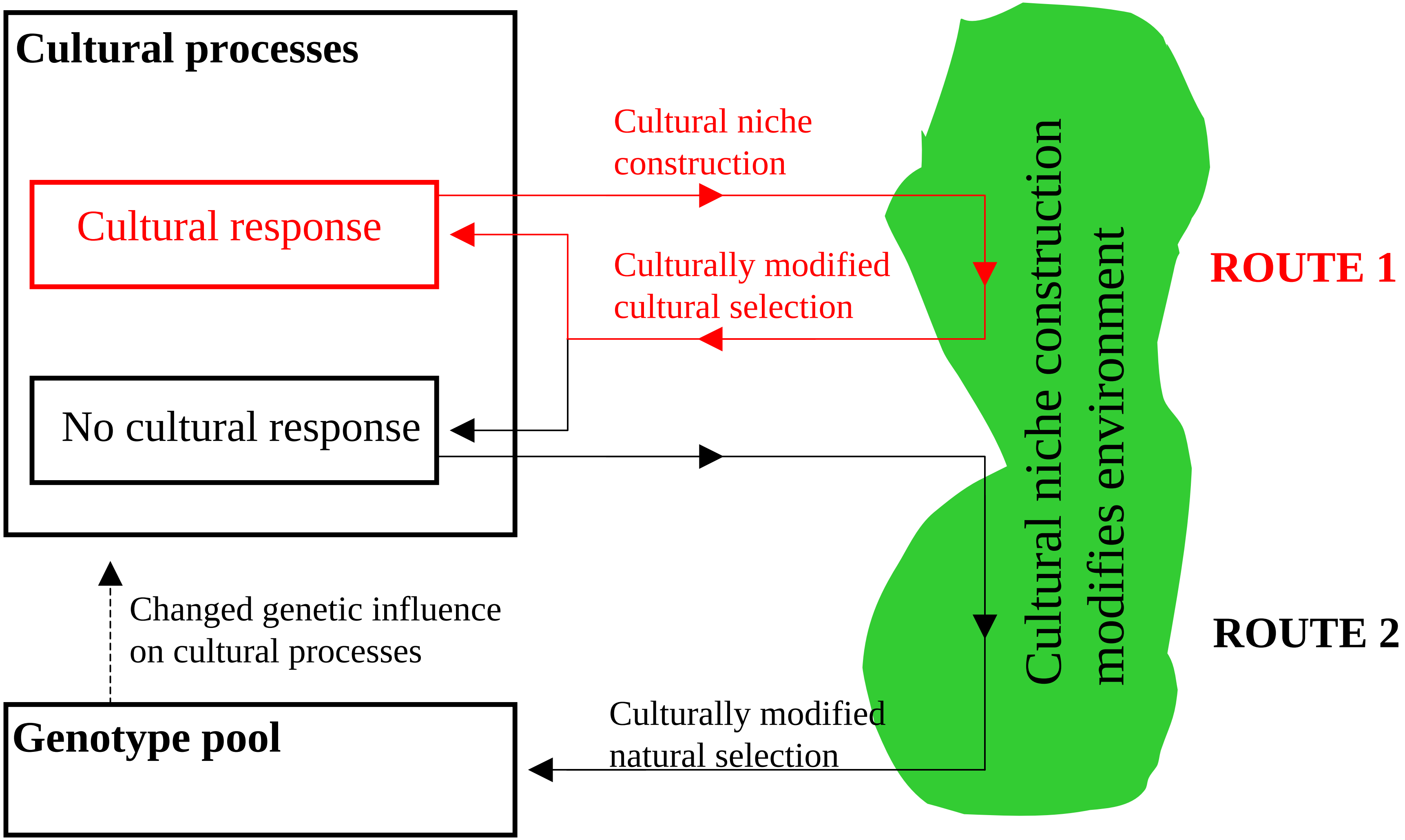
Yam Cultivation & Haemoglobin *S* allele

- Genetic signature of cultural niche construction





Cultural Niche Construction



Cultural Transmission

Vertical:	Parents to children
Oblique:	Older generation to younger generation
Horizontal:	Among members of the same generation

Fitness of phenogenotypes in matrix form

	E	e
AA	$\gamma_{11} + f_{AA}(R)$	$\gamma_{12} + f_{AA}(R)$
Aa	$\gamma_{21} + f_{Aa}(R)$	$\gamma_{22} + f_{Aa}(R)$
aa	$\gamma_{31} + f_{aa}(R)$	$\gamma_{32} + f_{aa}(R)$

Cultural niche construction in vertical transmission rates

Seme 1			Seme 2		
	offspring			offspring	
matings	E	E	matings	H	H
E x E	b_3	$1 - b_3$	H x H	$c_3 + g_3(x)$	$1 - c_3 - g_3(x)$
E x e	b_2	$1 - b_2$	H x h	$c_2 + g_2(x)$	$1 - c_2 - g_2(x)$
e x E	b_1	$1 - b_1$	h x H	$c_1 + g_1(x)$	$1 - c_1 - g_1(x)$
e x e	b_0	$1 - b_0$	h x h	$c_0 + g_0(x)$	$1 - c_0 - g_0(x)$

Frequency of E is x.

Education and fertility reduction

Level of education forms a cultural "niche" in which attitudes towards fertility evolve.

E/*e* levels of education (p = freq. *E*)

A/*a* attitudes towards fertility control (q = freq. *A*)

Transmission of *A* determined by frequency of *E*. Oblique transmission of *A* depends on

$$p(E): \quad \Phi = \mu_0 + \mu_1 p$$

Model of Transmission

I. Education

	offspring	
parents	E	e
E x E	b_3	$1 - b_3$
E x e	b_3	$1 - b_3$
e x e	b_3	$1 - b_3$
e x e	b_3	$1 - b_3$

Model of Transmission

II. Fertility attitude

		offspring	
fertility	parents	A	a
$1 - f$	A x A	$(1 - F)c_3 + F(1 + g)q/z$	$(1 - F)(1 - c_3) + F(1 - q)/z$
$1 - f/2$	A x a	$(1 - F)c_2 + F(1 + g)q/z$	$(1 - F)(1 - c_2) + F(1 - q)/z$
$1 - f/2$	a x A	$(1 - F)c_1 + F(1 + g)q/z$	$(1 - F)(1 - c_1) + F(1 - q)/z$
1	a x a	$(1 - F)c_0 + F(1 + g)q/z$	$(1 - F)(1 - c_0) + F(1 - q)/z$

g measures over-transmission by A.

$z = 1 - q + q(1 + g)$.

III. Mortality

Proportion of juveniles that die before maturity is

$$1 - \nu_0(1 + \nu_1 p)$$

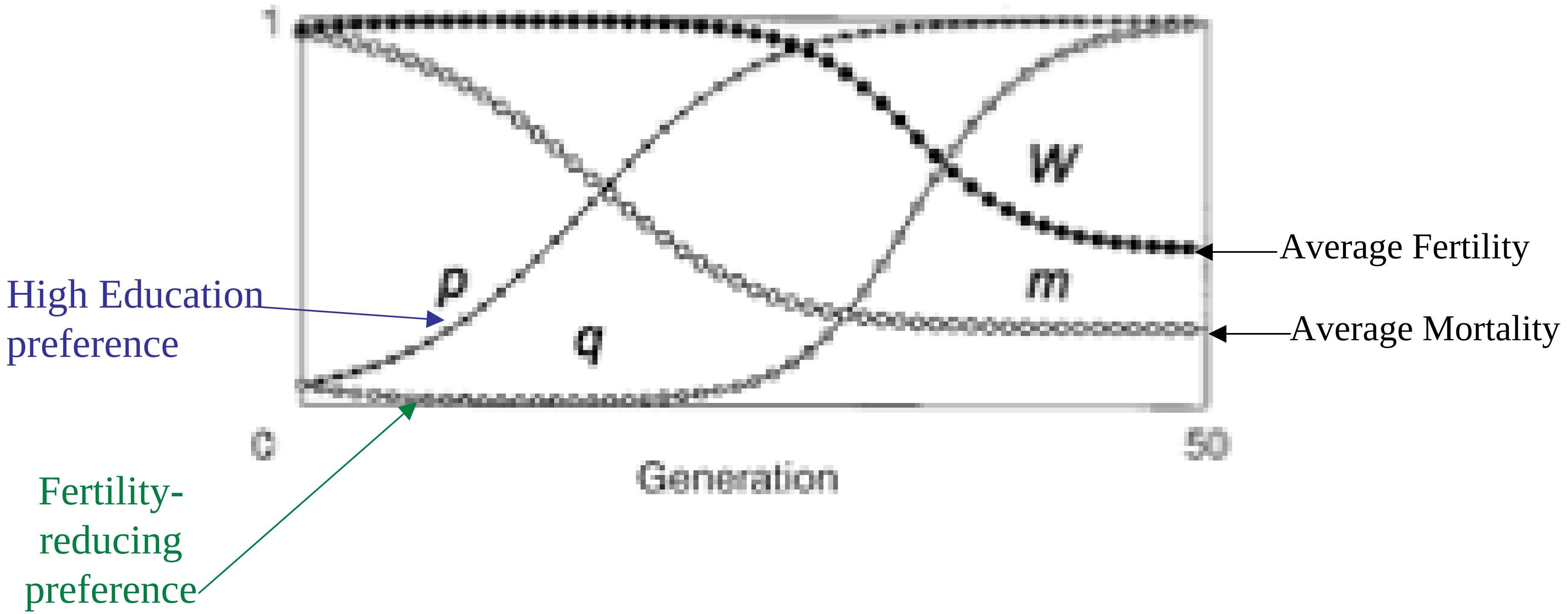
E absent: mortality rate is $1 - \nu_0$

E present: mortality rate is $1 - \nu_0 - \nu_0 \nu_1 p$

Relative mortality: $m = \dfrac{1 - \nu_0(1 + \nu_1 p)}{1 - \nu_0}$

Education and Fertility-Reducing Preference

Education affects the rate of transmission of fertility reduction and mortality reduction.



(Ihara & Feldman 2004)

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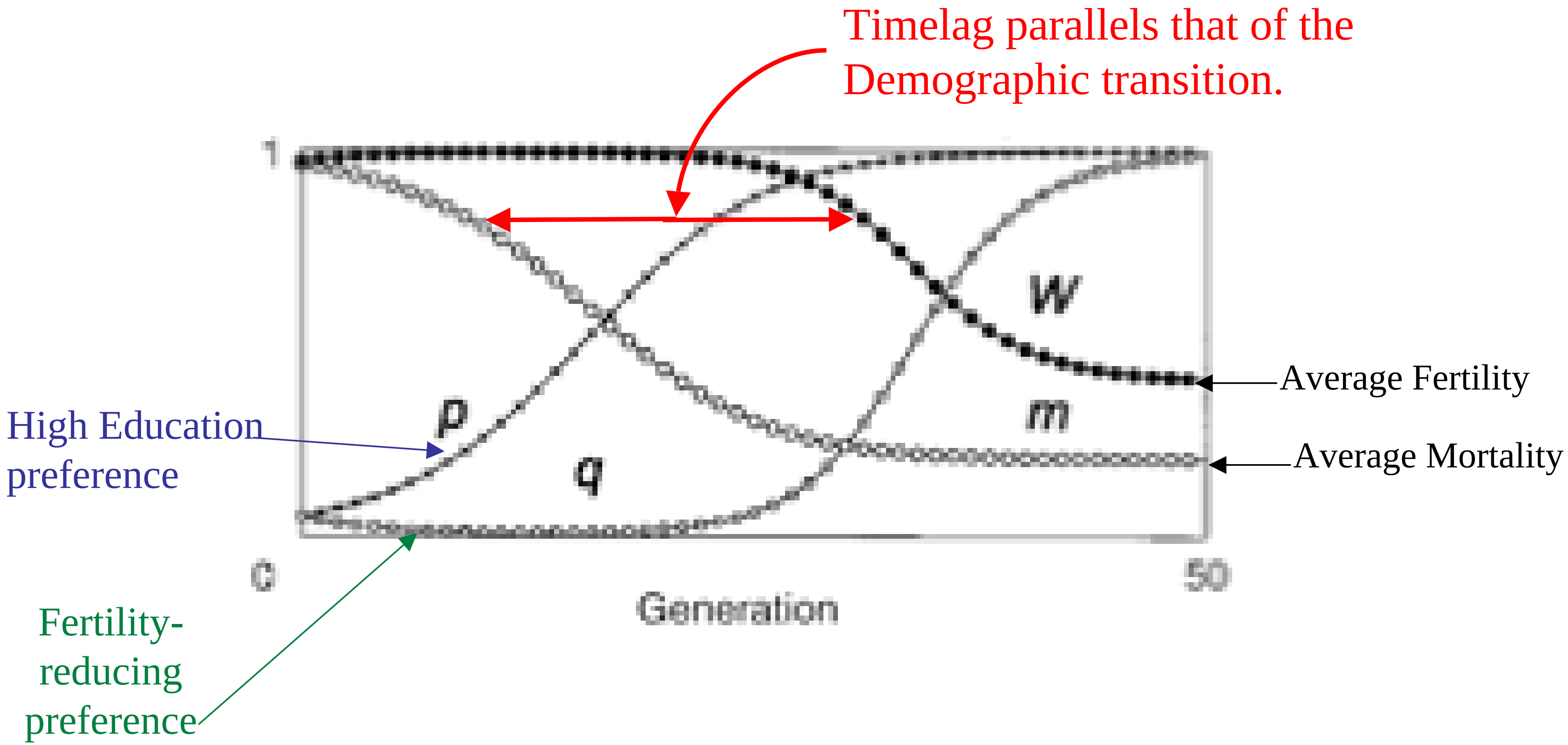
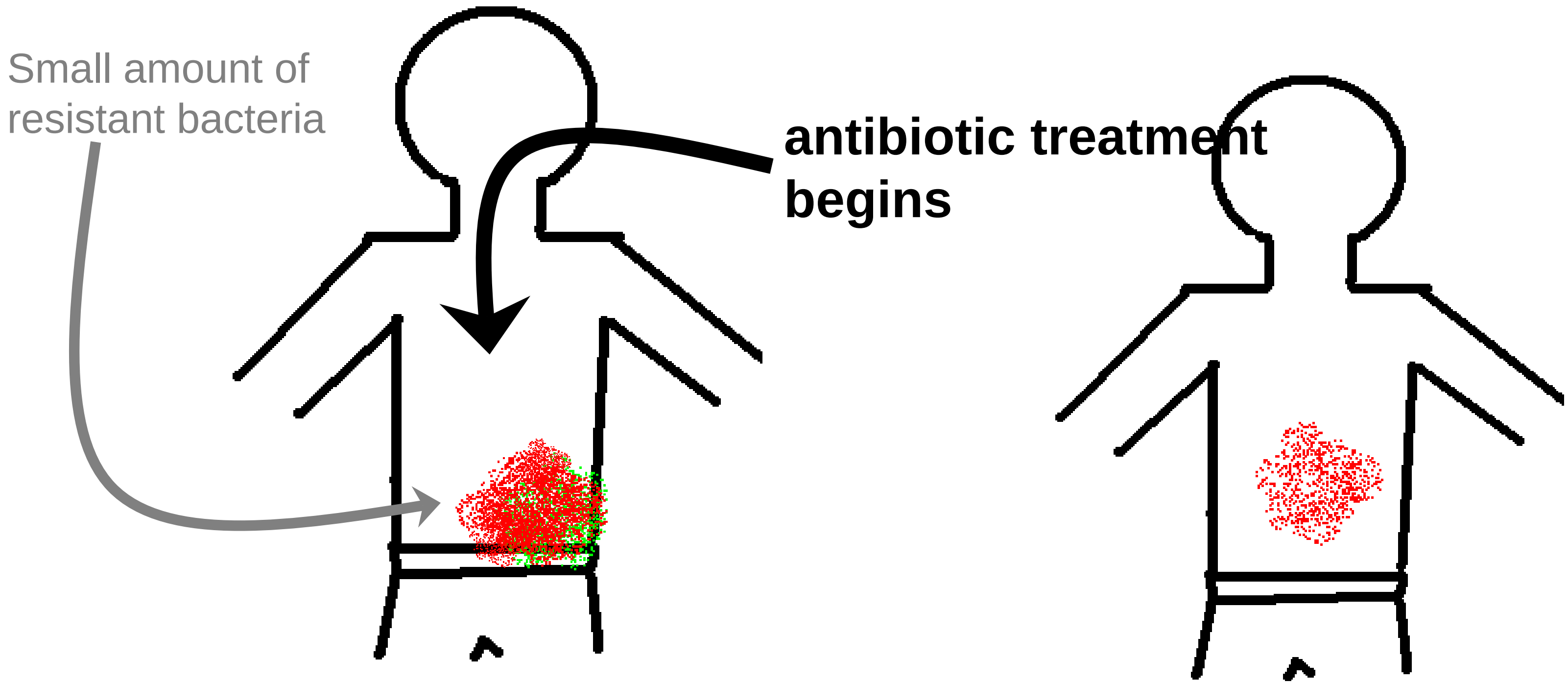


Other examples of cultural/social niche construction and its role in evolution

Trait A (niche)	**Trait B (target)**
Nomadic pastoralism	Consanguineous mating
Social stratification (castes or after military conquest)	(a) Lexicon (b) Meaning of words (c) Marriage rules
Genetic changes → brain changes → language	Group behaviors (hunting, warfare) Knowledge accumulation (agriculture)
Use of antibiotics by humans	Evolution of resistance by pathogens

Niche Construction and the Evolution of Antibiotic Resistance

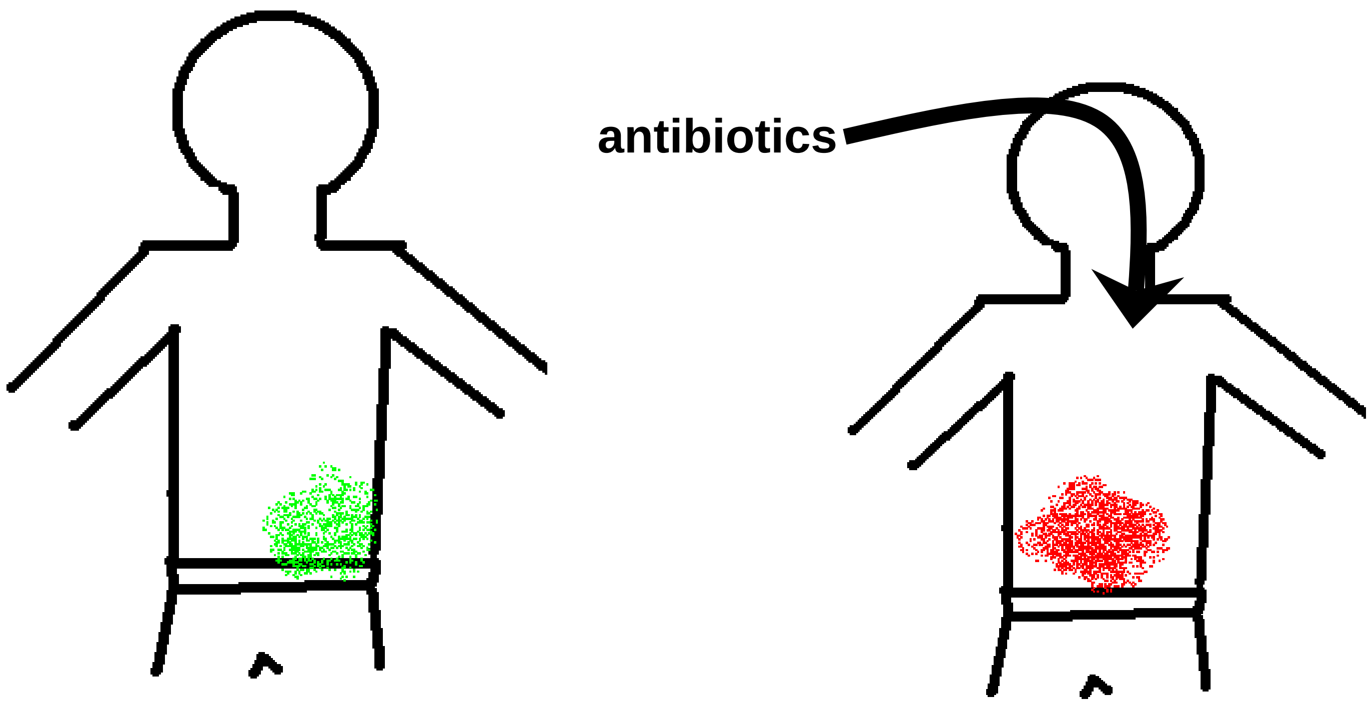
Antibiotic Resistance



antibiotic–sensitive bacteria (green)
antibiotic–resistant bacteria (red)

de novo resistance
primary resistance

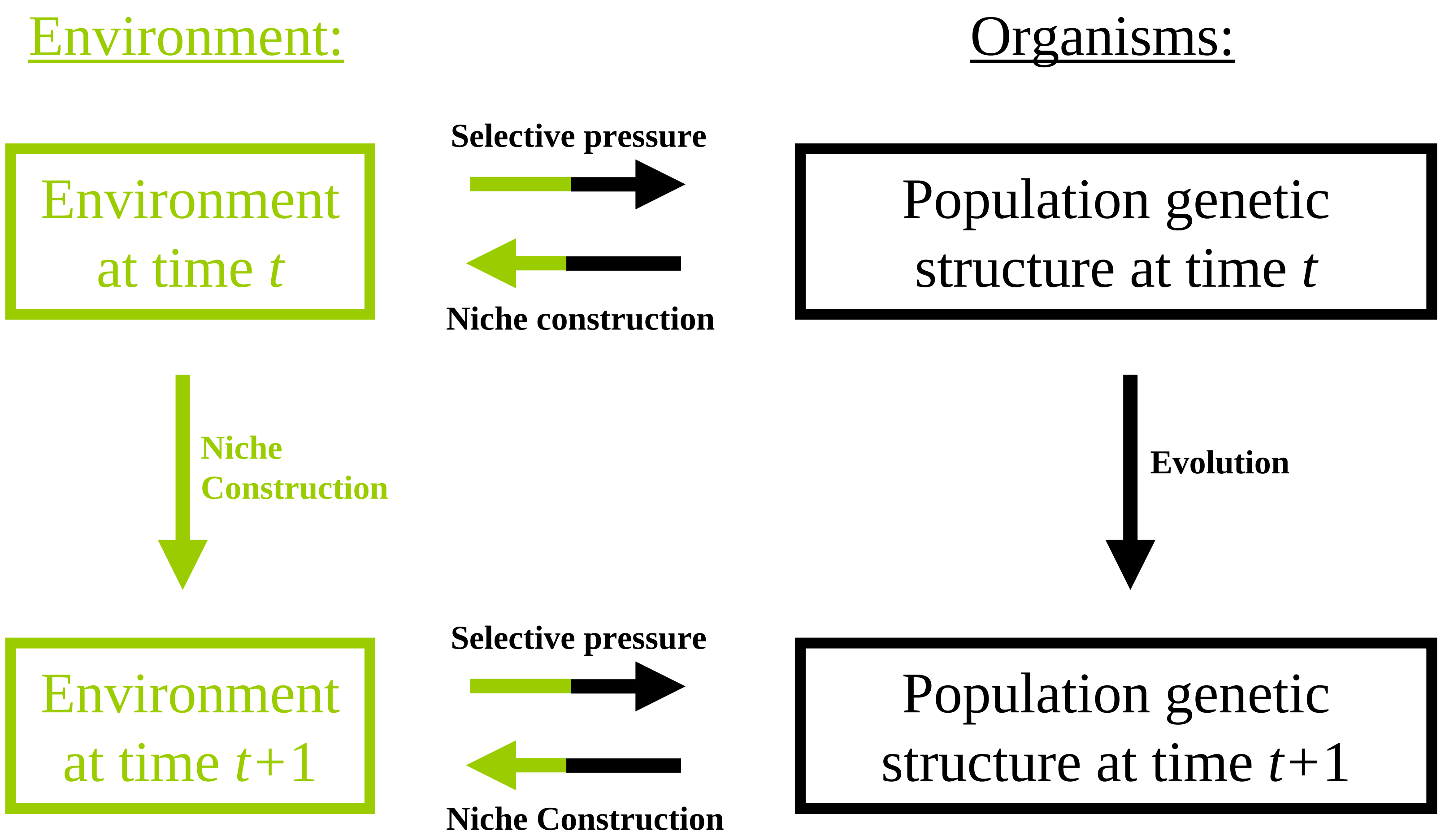
Bacteria experience two different selective environments



wildtype strains are favored in the absence of antibiotics

resistant strains are favored during antibiotic treatment

Niche Construction

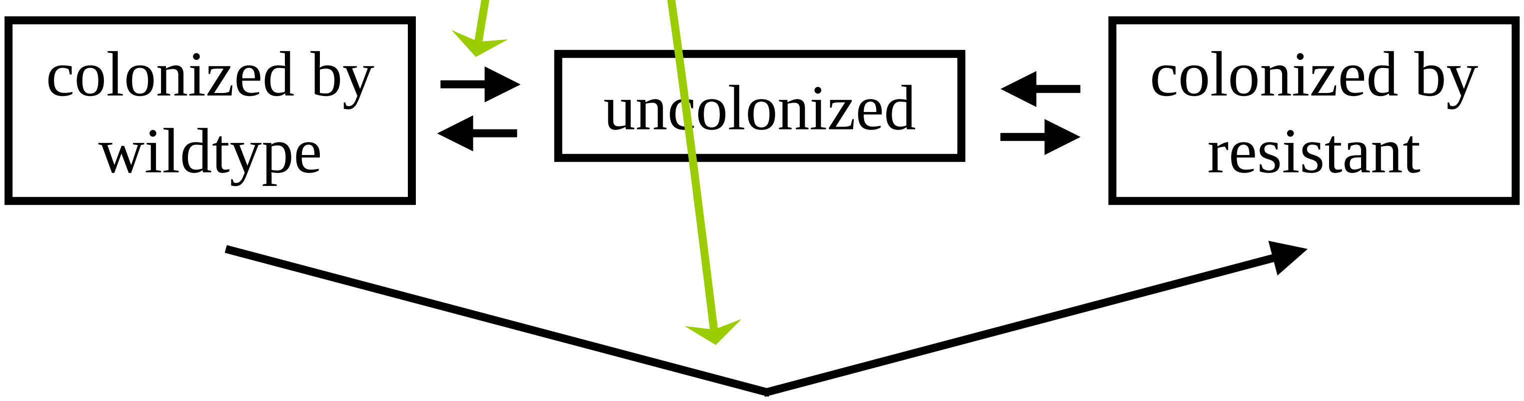


Mathematical Models

Recovery/selection rates are usually affected by antibiotic treatment

Sometimes, there is a constant flow of individuals into “treated” population classes.

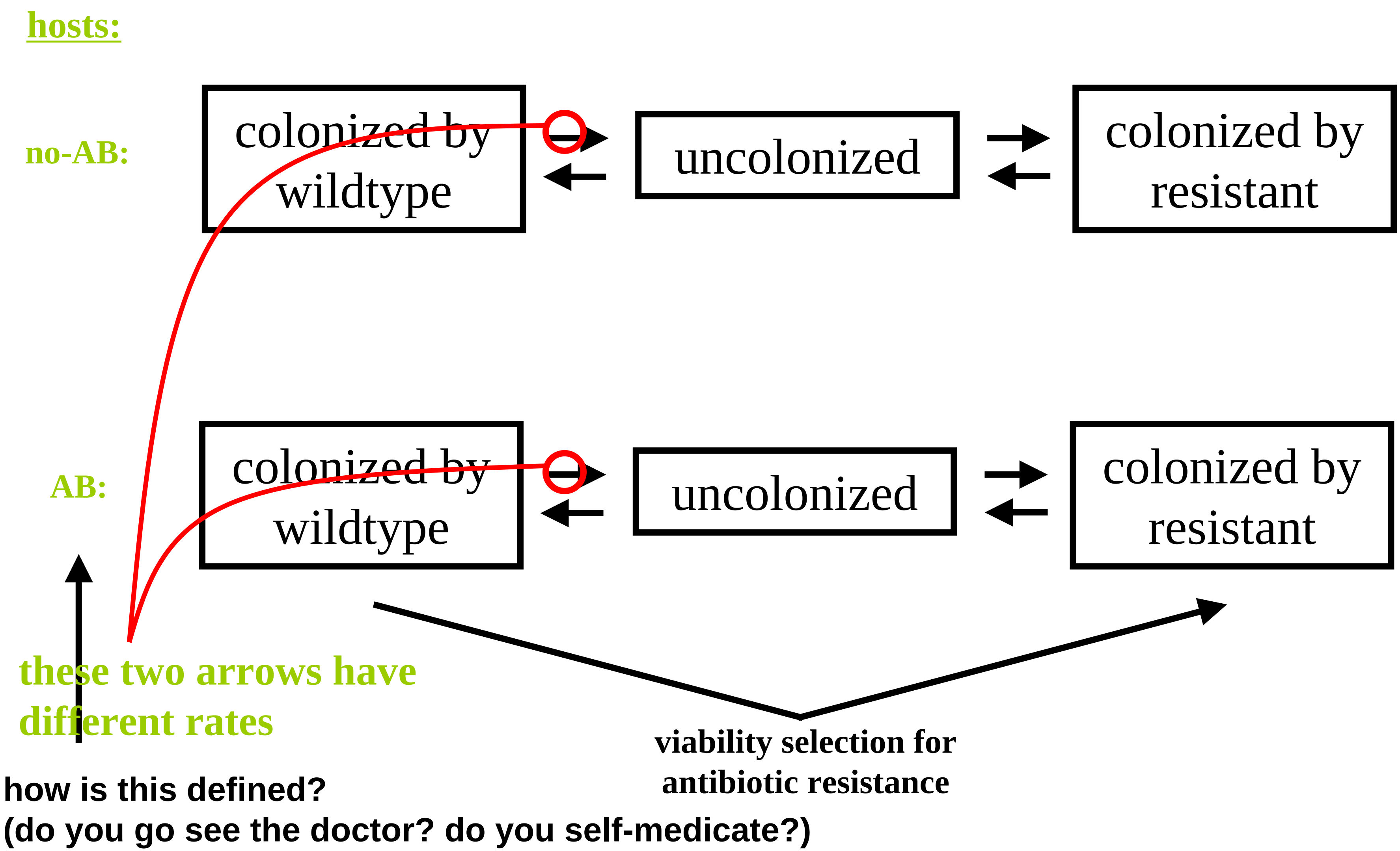
cost of resistance

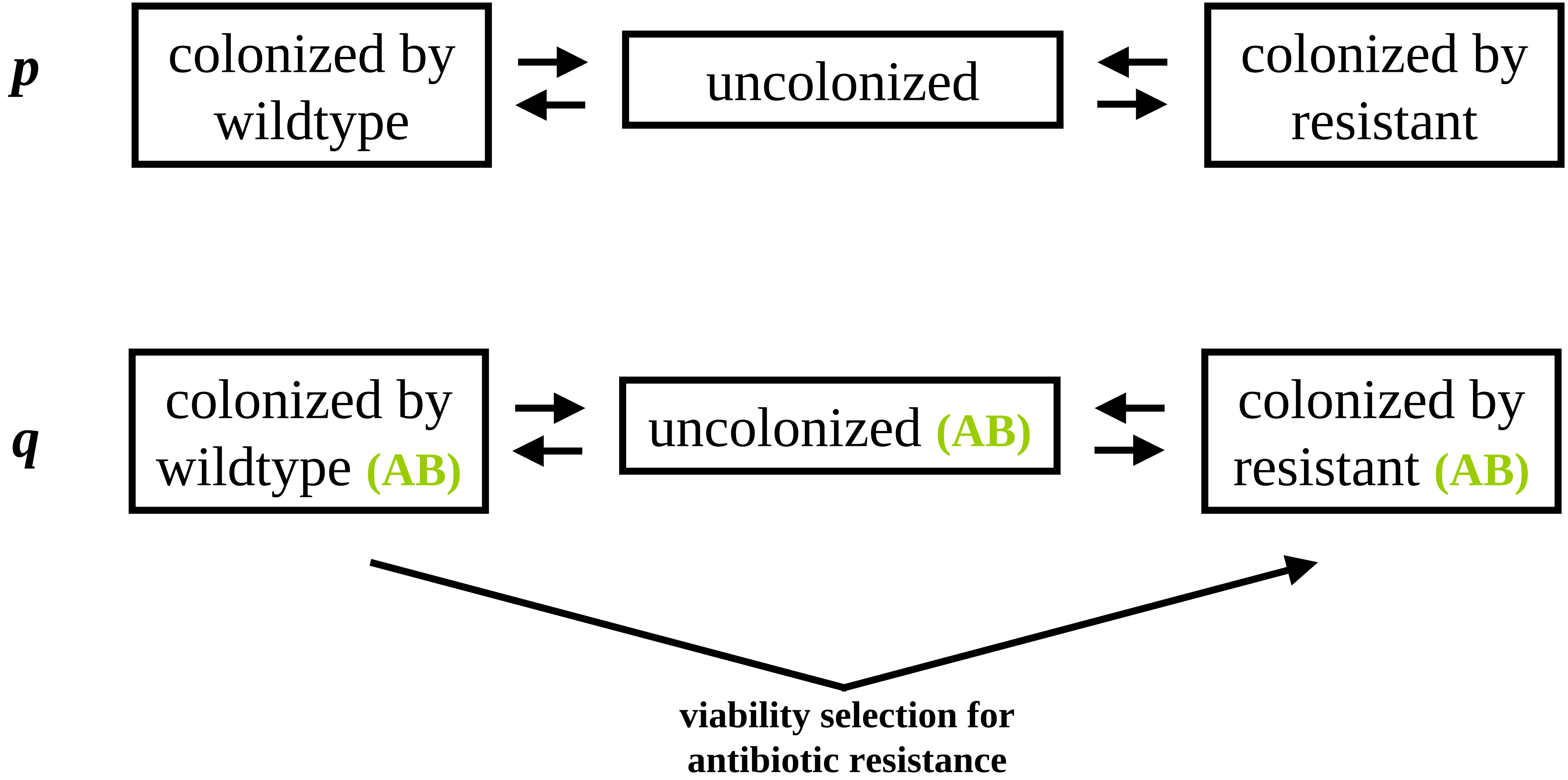


viability selection for antibiotic resistance

(Blower et al 1996, Bonhoeffer et al 1997, Austin et al 1997,1999, Castillo-Chavez and Feng 1997, Levin et al 1997)

Our Model: Two Types of Hosts



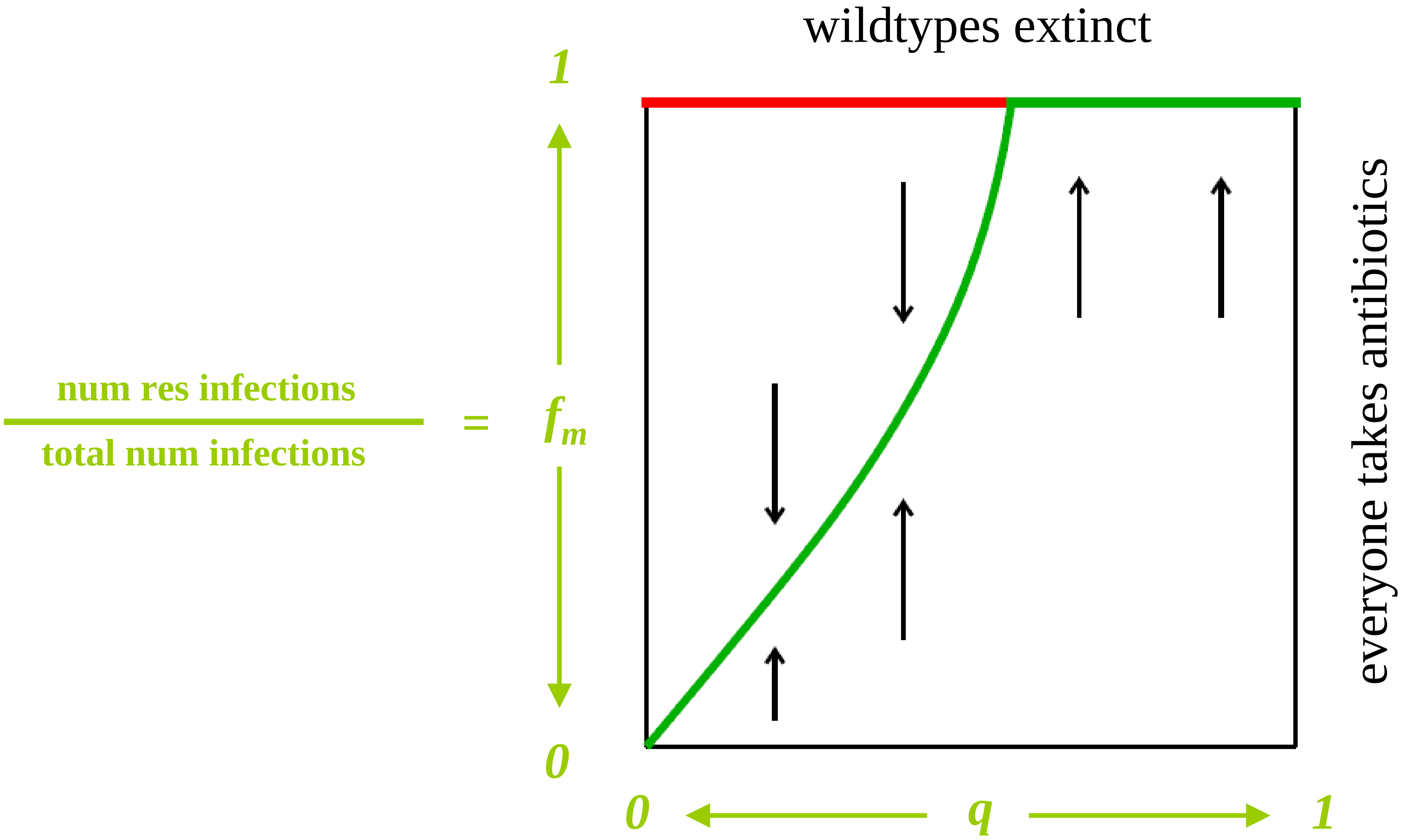


Niche is defined by the ***use*** frequencies *p* and *q*

-- when *q* is low (usage low) wildtypes will be favored

-- when *q* is high (usage high) resistants will be favored

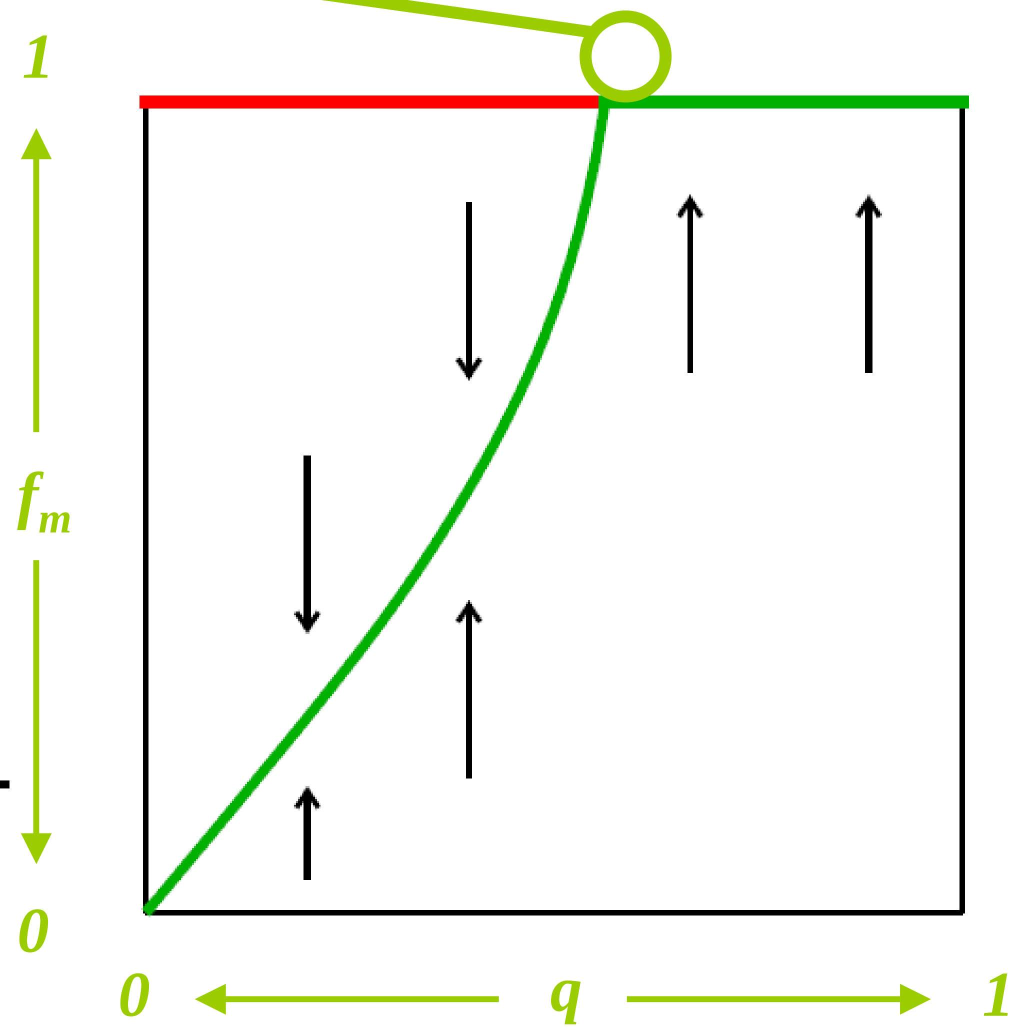
Niche affects dynamics



Niche affects dynamics

There is a critical value q^* beyond which resistance fixes in the population and wildtype antibiotic sensitive bacteria go extinct

$$q^* = \frac{W_{wt,no\text{-}AB} - W_{res}}{W_{wt,no\text{-}AB} - W_{wt,AB}}$$



Critical value q^*

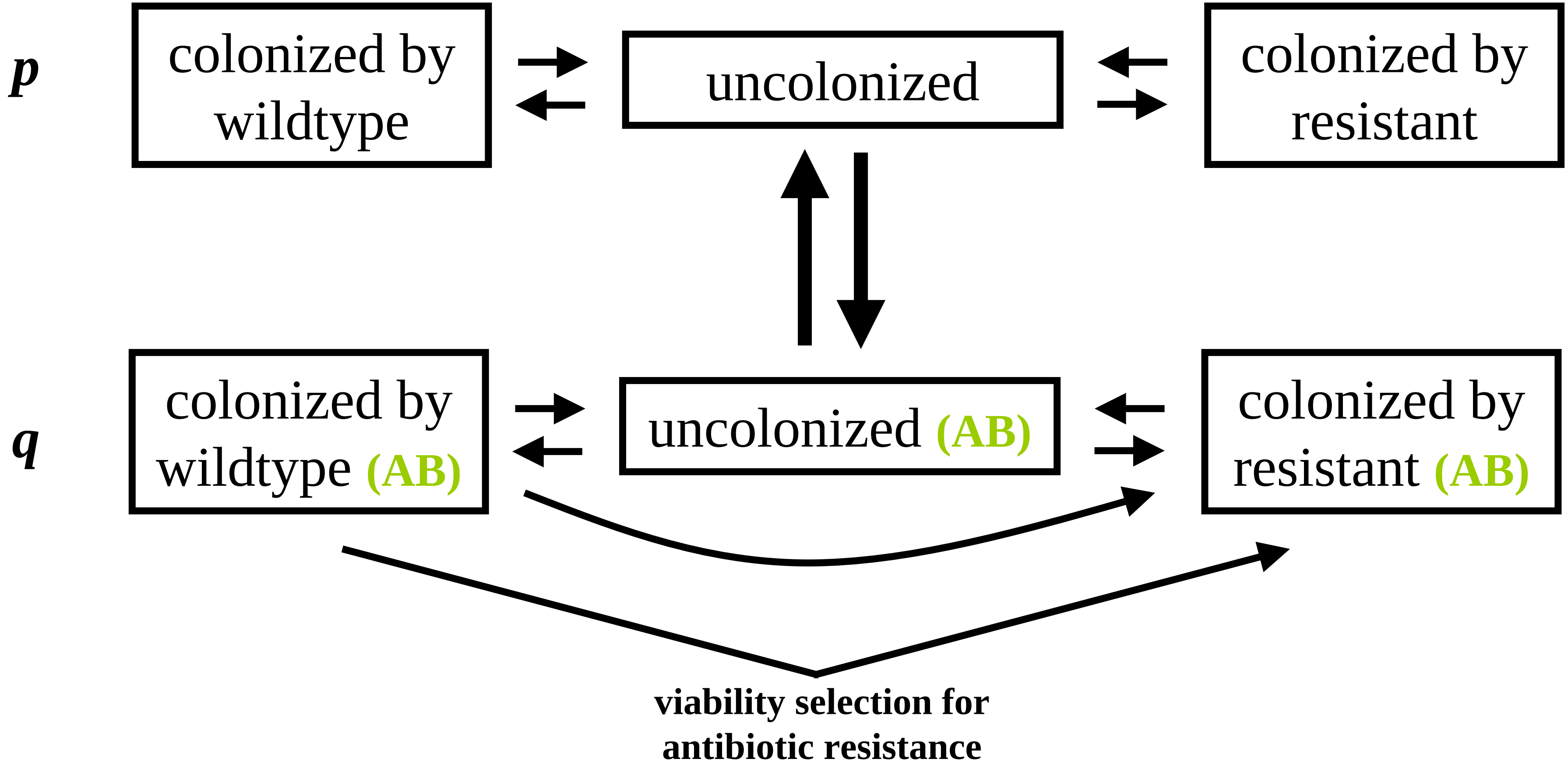
And, our <u>within-host</u> assumptions imply that:

$$q^* < 1$$

i.e. $W_{res} > W_{wt,AB}$

Warning!: what is the definition of fitness?

$$q^* = \frac{W_{wt,no\text{-}AB} - W_{res}}{W_{wt,no\text{-}AB} - W_{wt,AB}} = \frac{\text{cost of resistance}}{\text{cost of undergoing treatment}}$$

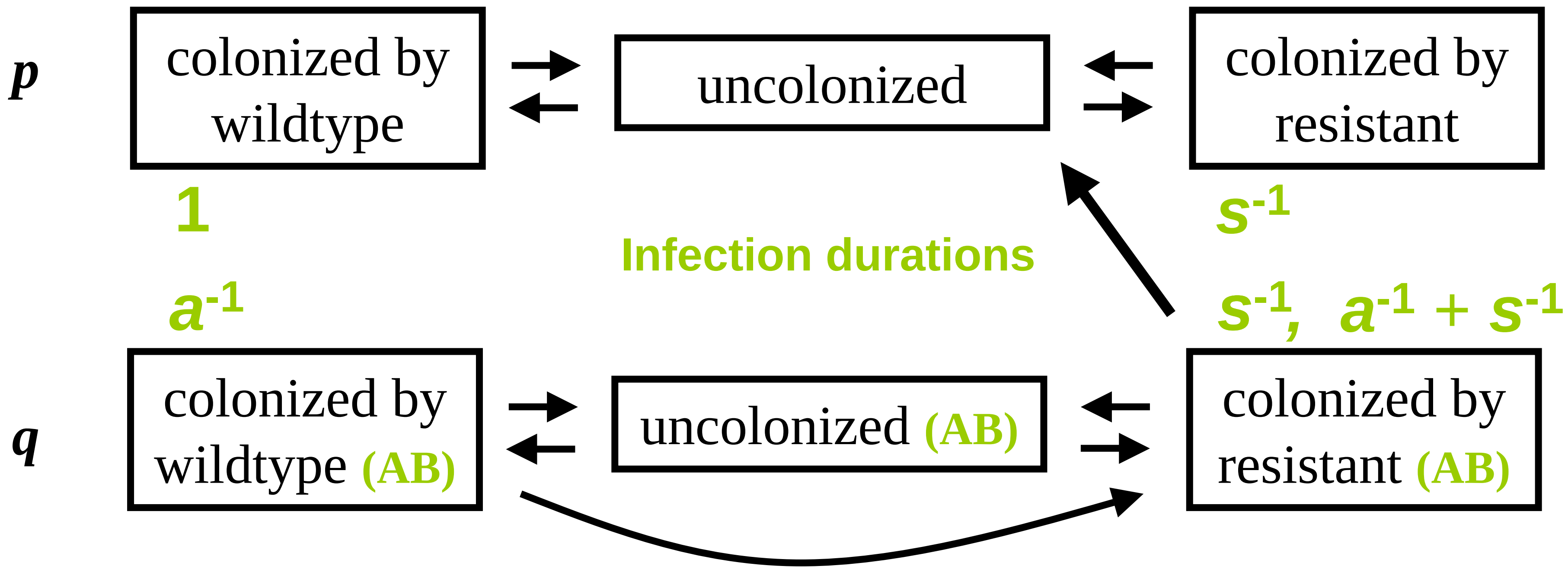


How can the values of *p* and *q* change?

- Humans conform to each others' behavior
- Hosts use/avoid antibiotics based on infection morbidity
- Hosts listen to doctors and public health officials

Experience-Dependent Learning
(EDL)

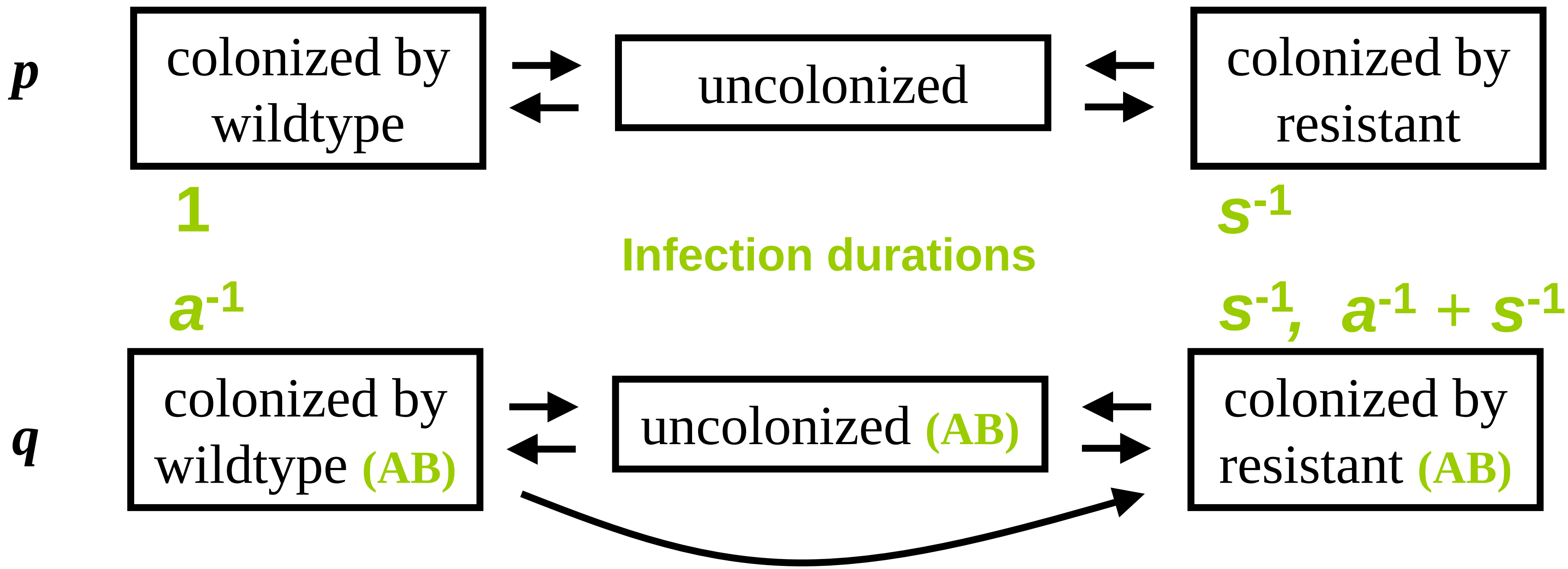
- Hosts will change behaviors upon recovery if they experience a particularly long or severe infection



Experience-Dependent Learning

(EDL)

$$1 > s^{-1} > a^{-1}$$

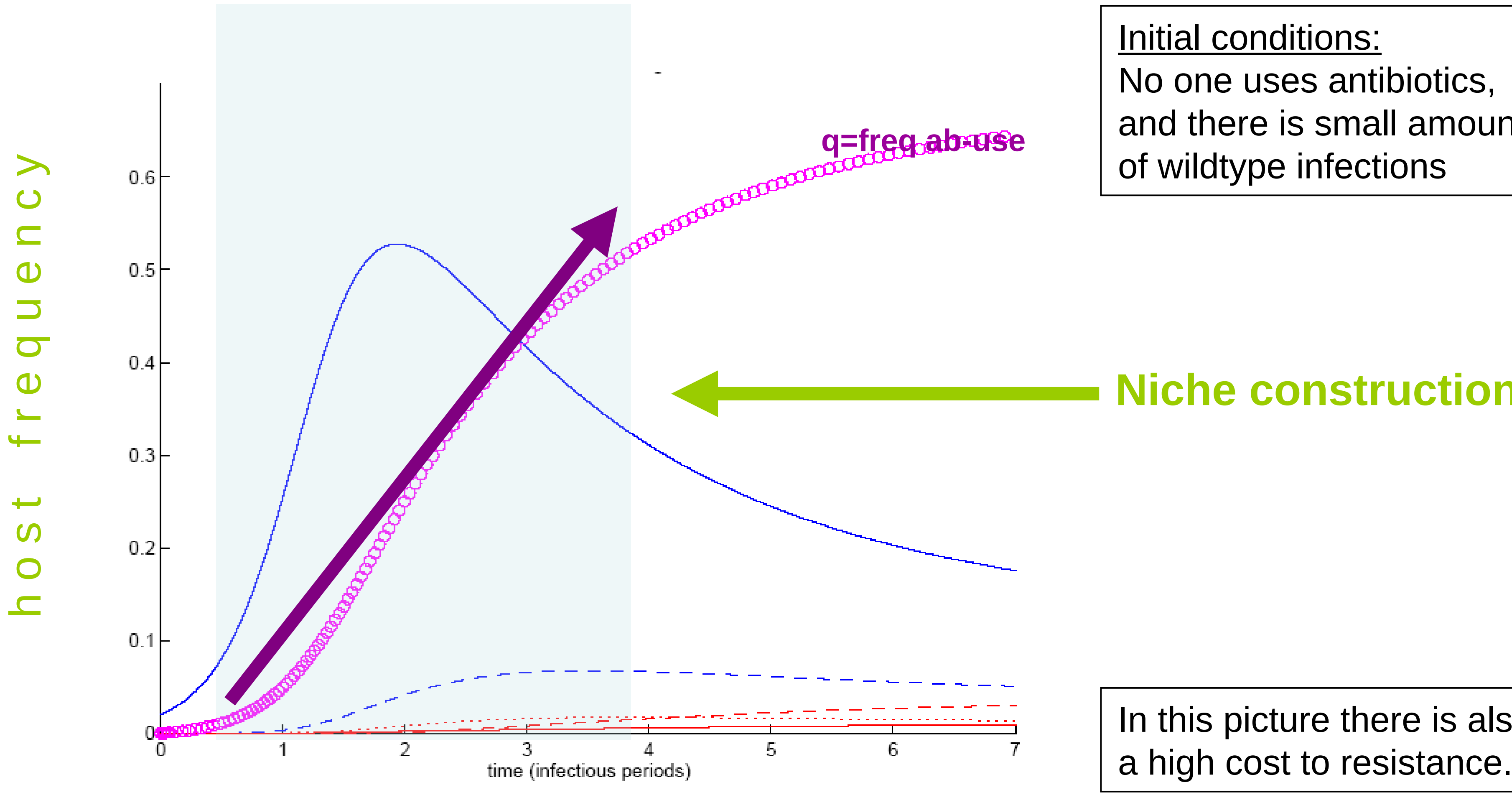


Experience-Dependent Learning
(EDL)

- Notice that long infections by parasite *w* in environment *E*:
  - favor parasite *w*
  - cause the environment to change away from *E*

this means that parasite *w* constructs a niche which favors parasite *m*

Dynamics (strong antibiotics)



Initial conditions:
No one uses antibiotics, and there is small amount of wildtype infections

In this picture there is also a high cost to resistance.

Equilibria

- There always seems to be a unique, stable, globally-attracting equilibrium

- If W_{res} is high enough, resistants fix

- If W_{wt} is high enough, there is polymorphism

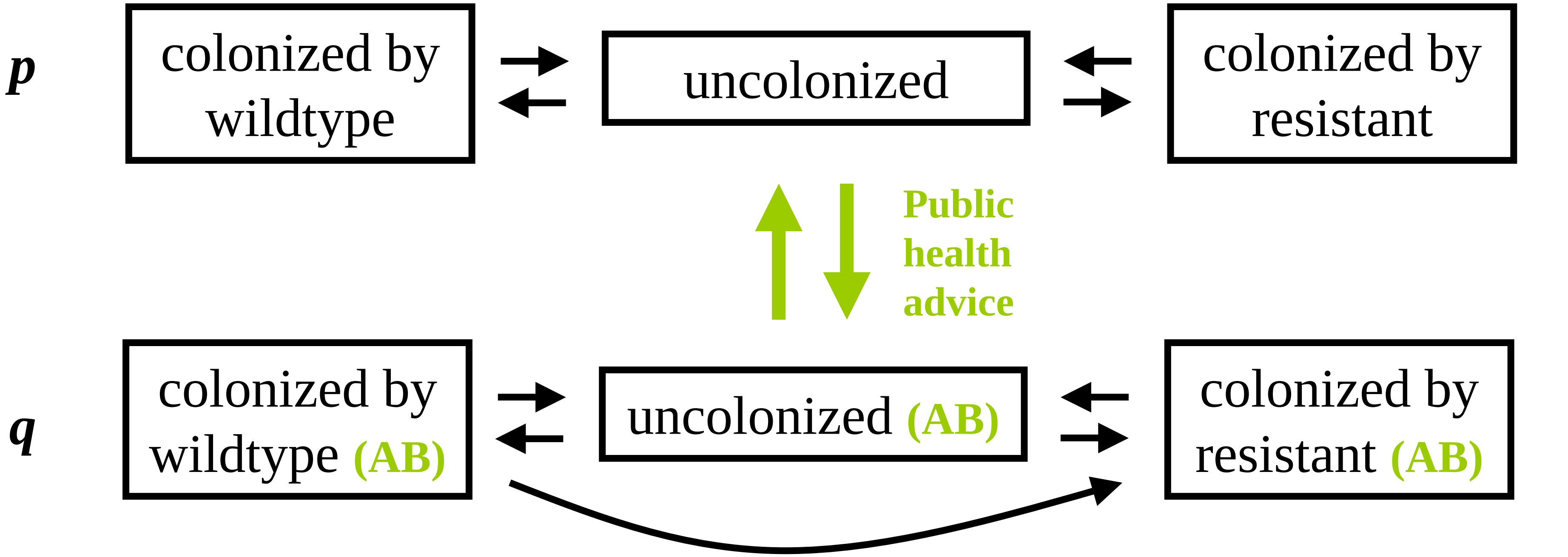
Frequency-Dependent Learning **(FDL)**

- Hosts will change behaviors based on advice from doctors and public health authorities

High levels of resistance → reduced prescribing rates

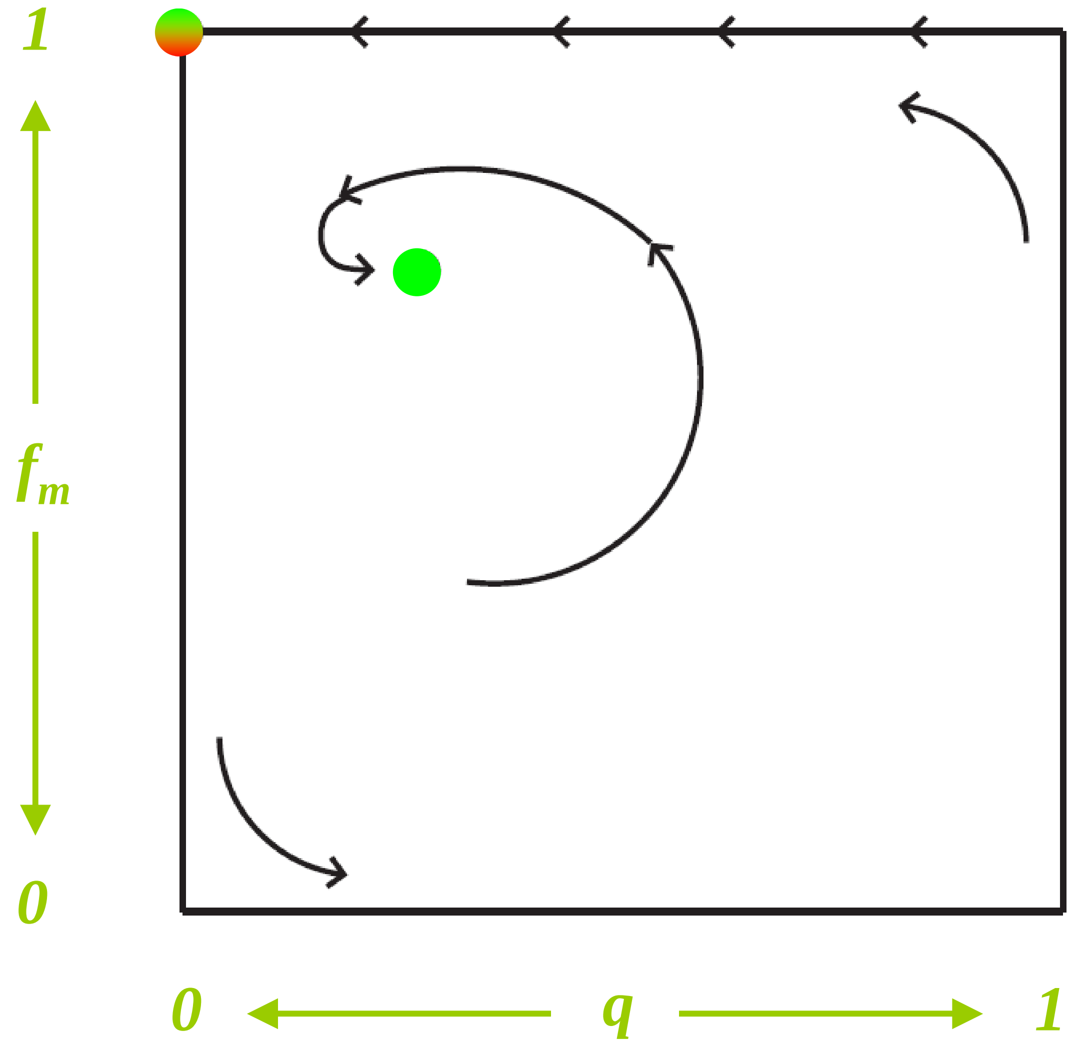
Low levels of resistance → increased prescribing rates

Frequency-Dependent Learning **(FDL)**



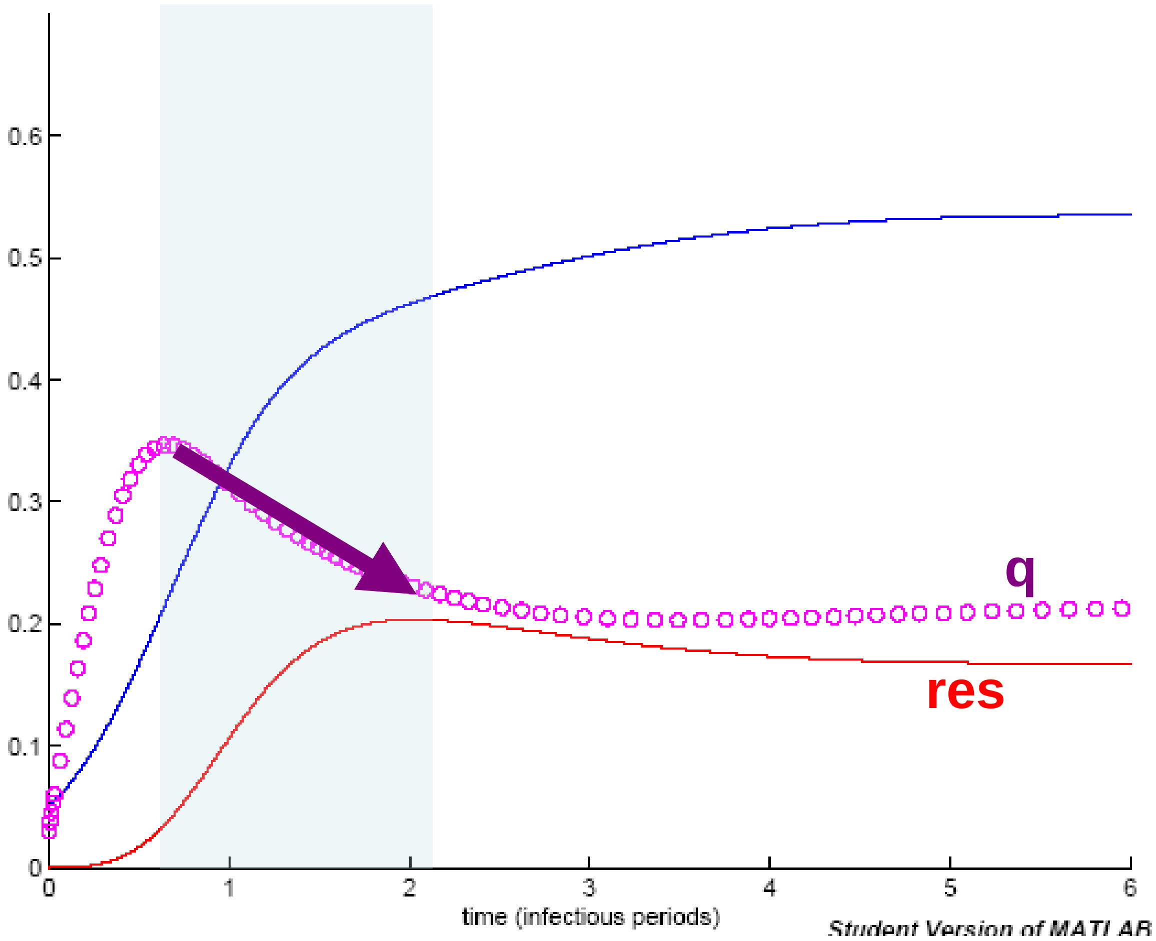
Frequency-Dependent Learning **(FDL)**

There appears always to be a globally-attracting unique stable equilibrium



Niche Construction

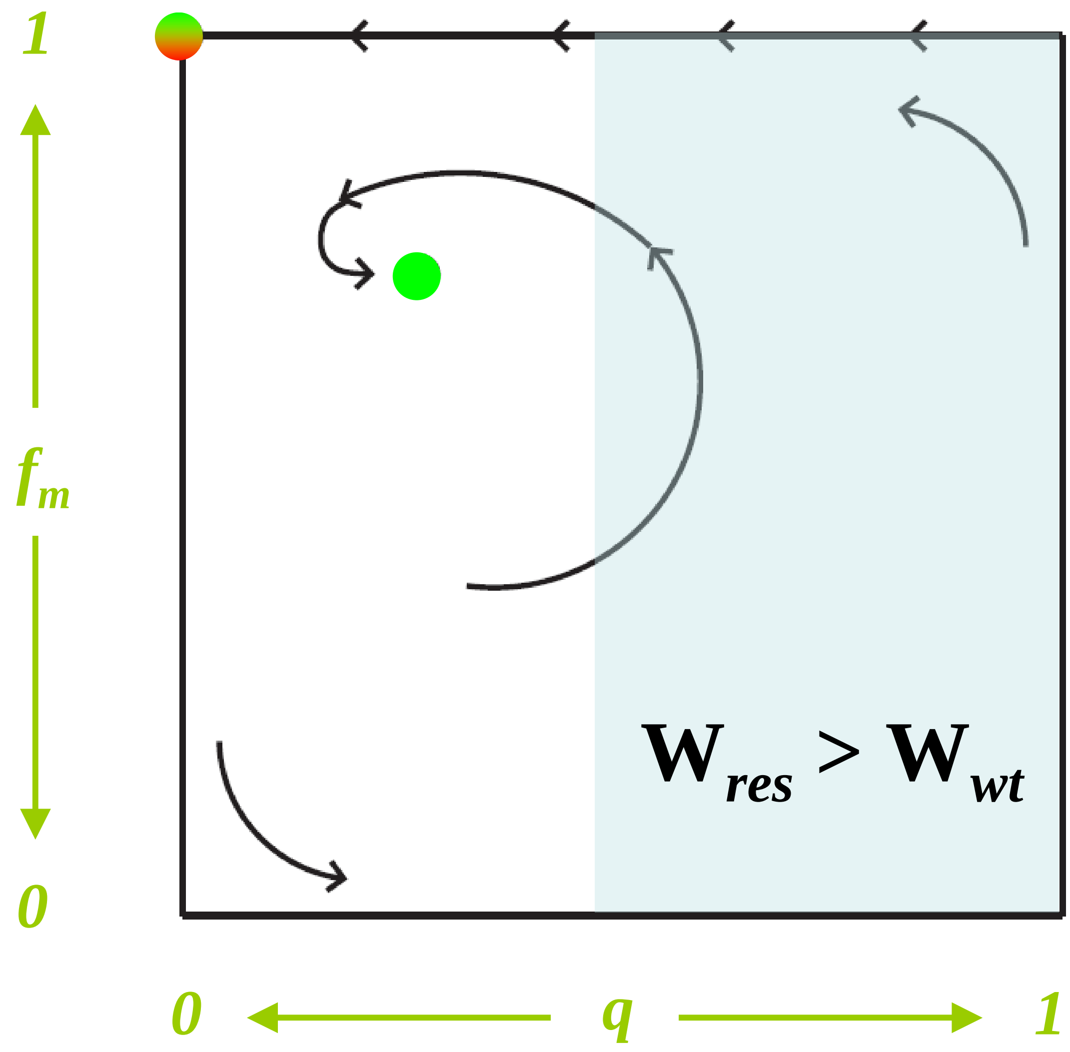
(Strong public health recommendations against AB-use)



<u>Initial conditions:</u>
No one uses antibiotics, and there is small amount of wildtype infections

Niche Construction

Here, we have "polymorphism where none is expected." (Laland et al, 1996, 1999)



Public Health Implications

Table 1 | **Resistance in key pathogens in England and Wales (1997–2002)**

Pathogen and antibiotic	1997	1998	1999	2000	2001	2002
Mostly community-acquired						
Streptococcus pneumoniae/penicillin*	6.9	3.6	5.4	4	2.6	2.8
Streptococcus pneumoniae/erythromycin*	11.6	11.8	12.3	12.8	12	12.9
Escherichia coli/ciprofloxacin‡	3.4	3.7	3.7	4.5	6	8.6
Neisseria gonorrhoeae/ciprofloxacin*	–	–	–	2	3	10
Mostly hospital-acquired						
Staphylococcus aureus/methicillin*	31.7	33.9	36.7	42	43.6	42.9
Klebsiella pneumoniae/ciprofloxacin*	8.4	7.8	7.1	10.9	8.7	9.4
Klebsiella pneumoniae/ceftazidime*	7.4	5.9	5.4	9.9	8.1	10.5
Enterococcus faecium/vancomycin*	19.9	23.9	23.1	29.9	19.3	17.1

*Based on results for bacteraemia isolates in England and Wales collected under a surveillance system described in REF. 53. ‡Based on GRASP surveillance[11]

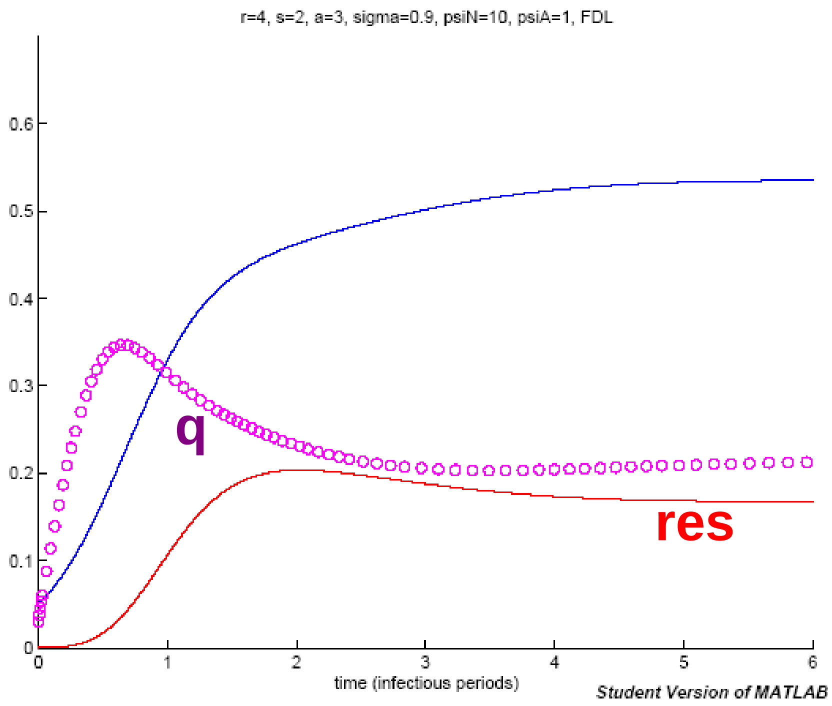
(Livermore 2004)

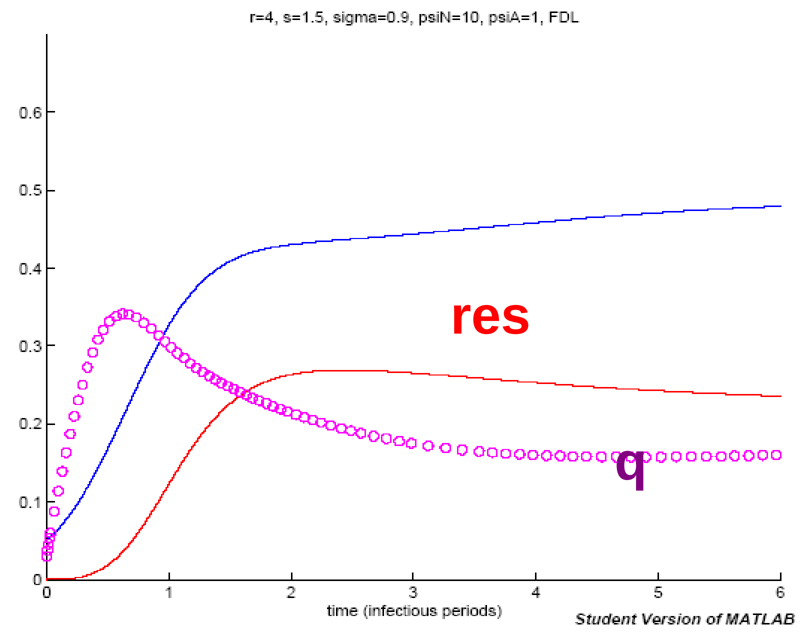
After >20% reduction in AB-prescribing.

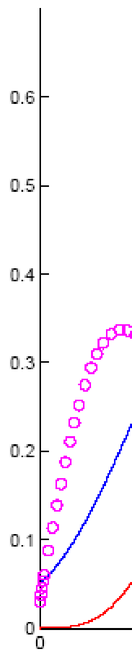
Compensatory Mutations

here, resistants have
acquired beneficial mutations

her
eve



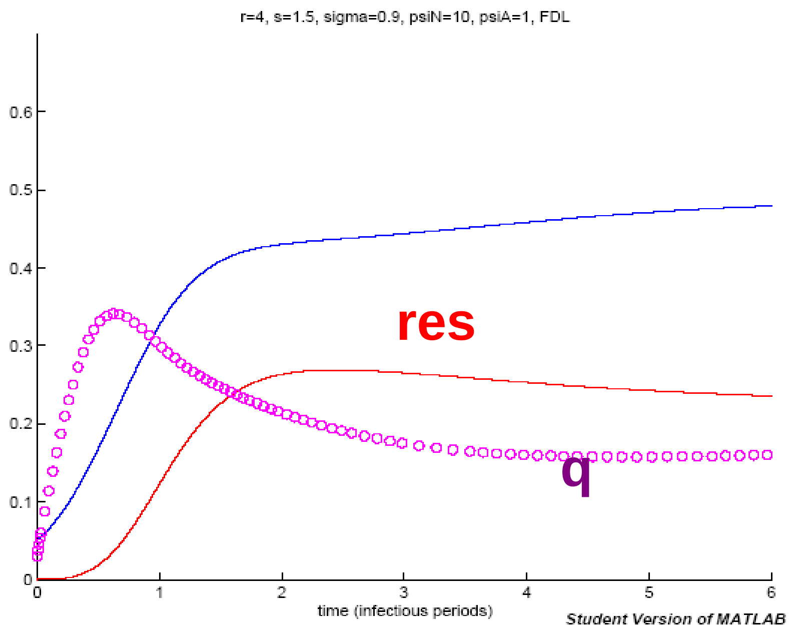




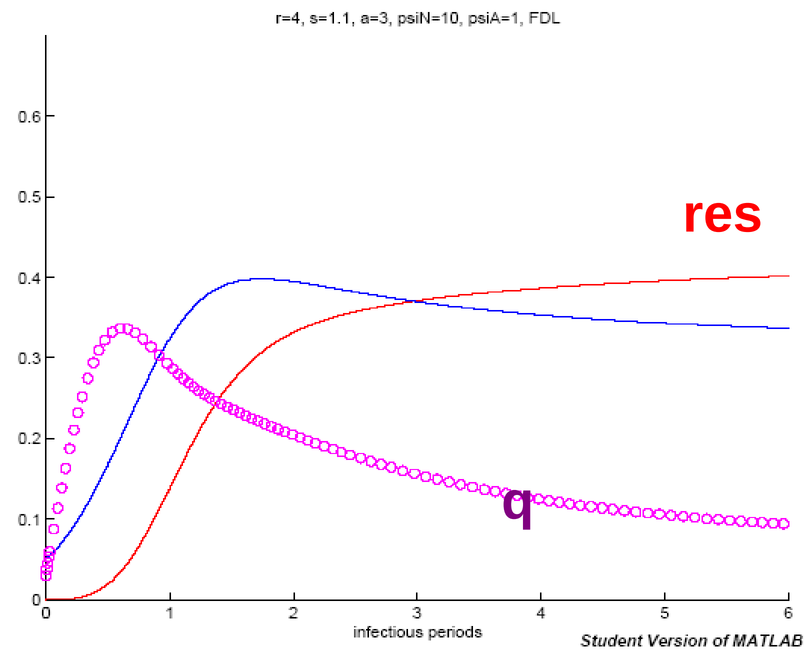
*q** = 0.14

Compensatory Mutations

here, resistants have acquired beneficial mutations



here, resistants have acquired even more beneficial mutations



$q^* = 0.14$