Some news on the population genetics of partial asexuals

PhD Thesis at INRA Rennes

Katja Reichel

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Organisms

examples for different rates of asexuality


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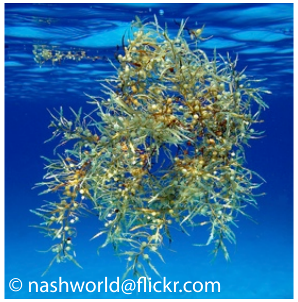
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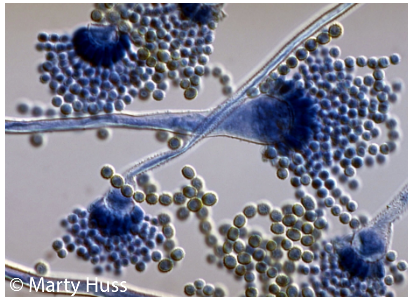
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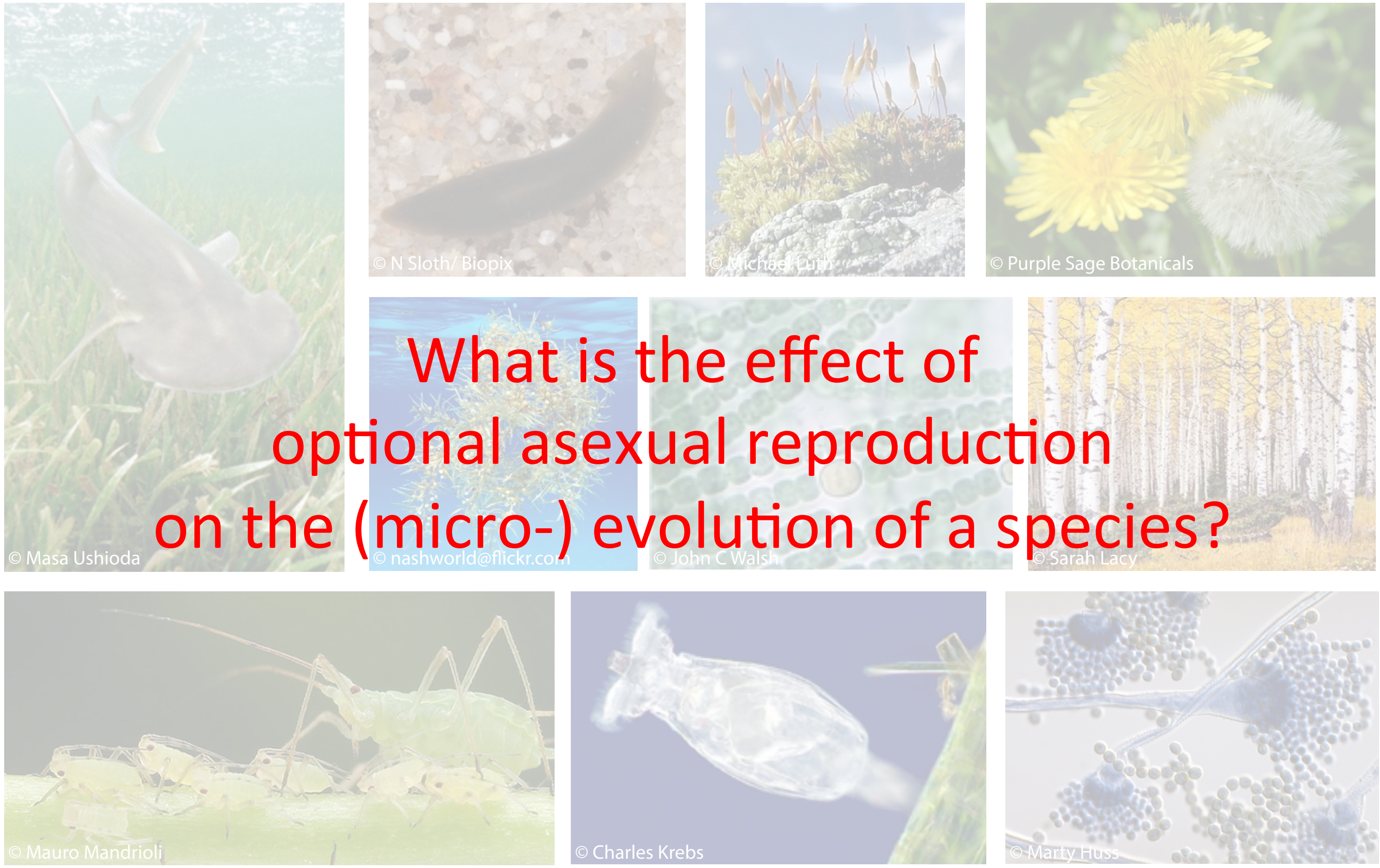
Organisms

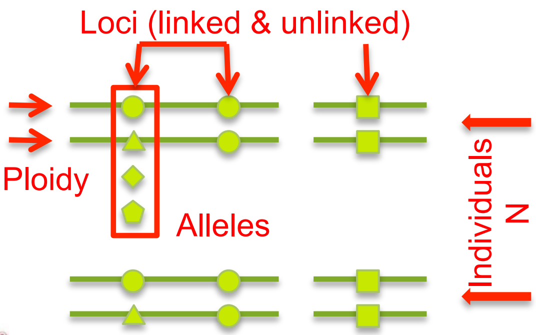
examples for different rates of asexuality

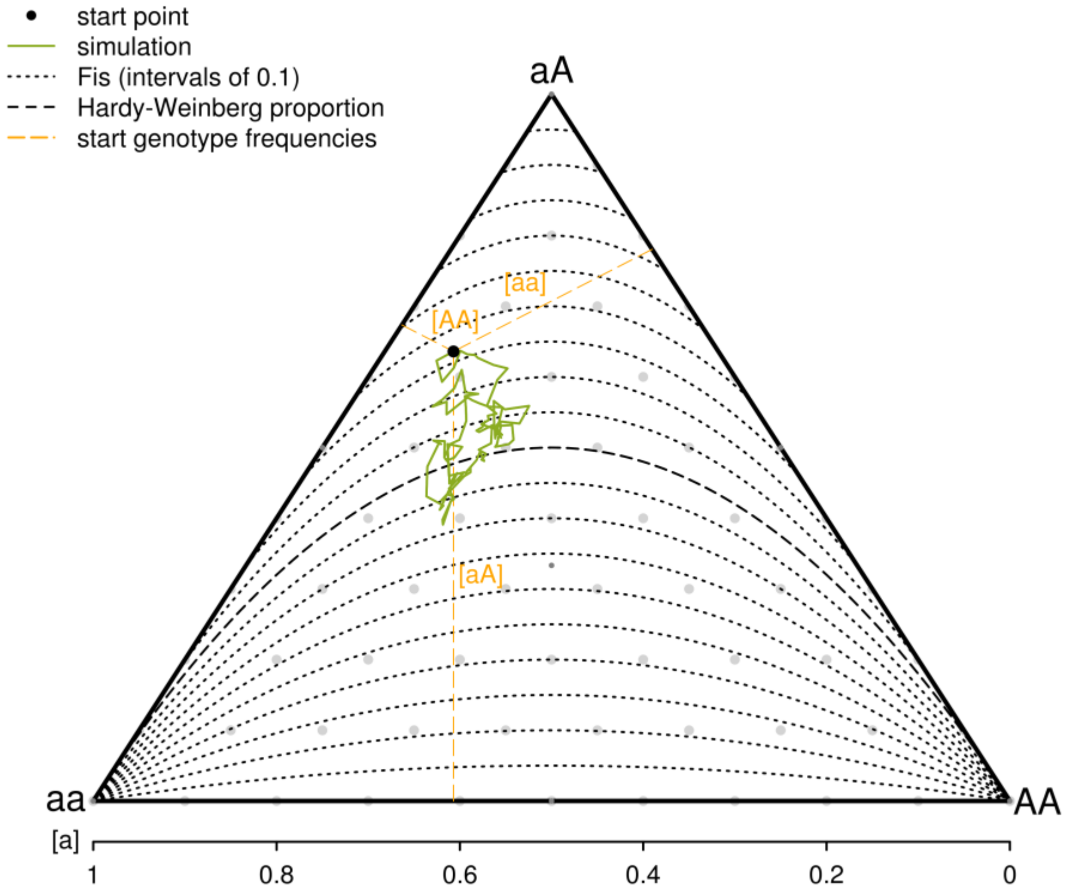


What is the effect of
optional asexual reproduction
on the (micro-) evolution of a species?

Bruno de Finetti

and his diagrams





Markov Chains

the principal idea

generation 1 → reproduction → generation 2 → reproduction → generation 3 …

- mathematically: a (recursive) sequence
  - an example:

    $a_1 = 1$ => generation 1 (first observation)

    $a_{n+1} = a_n + 3$ => reproduction "rule"

    gives {1; 4; 7; 10; 13; 16; 19; 22; 25; 28; 31; 34; 37; …}
  - this series converges to ∞ with a constant positive "speed"
  - none of its members is divisible by 3, but every second is divisible by 2, and (with a skip) every fourth by 4, every fifth by 5, every seventh by 7 …
- to incorporate partial asexuality, we need to change the rules for reproduction!

Some more detail
Model equations

μ … mutation rate
c … asexuality rate

asexual

		do not mutate	mutate	
$p^c(aa)$	=	$(1-\mu)^2 * p(aa)$	$+ \mu(1-\mu) * p(aA)$	$+ \mu^2 * p(AA)$
$p^c(AA)$	=	$(1-\mu)^2 * p(AA)$	$+ \mu(1-\mu) * p(aA)$	$+ \mu^2 * p(aa)$
$p^c(aA)$	=	$((1-\mu)^2 + \mu^2) * p(aA)$	$+ 2\mu(1-\mu) * p(aa)$	$+ 2\mu(1-\mu) * p(AA)$

sexual

$p^s(aa) = ((1-\mu) * p(aa) + \mu * p(AA) + 0.5 * p(aA))^2$ a

$p^s(AA) = ((1-\mu) * p(AA) + \mu * p(aa) + 0.5 * p(aA))^2$ A

$p^s(aA) = 2\,(((1-\mu) * p(aa) + \mu * p(AA) + 0.5 * p(aA)) +$ a

$((1-\mu) * p(AA) + \mu * p(aa) + 0.5 * p(aA)))$ A

which gametes are produced?

total

$p'(aa) = c * p^c(aa) + (1-c) * (p^s(aa) + 0.5 * p^s(aA))^2$

$p'(AA) = c * p^c(AA) + (1-c) * (p^s(AA) + 0.5 * p^s(aA))^2$

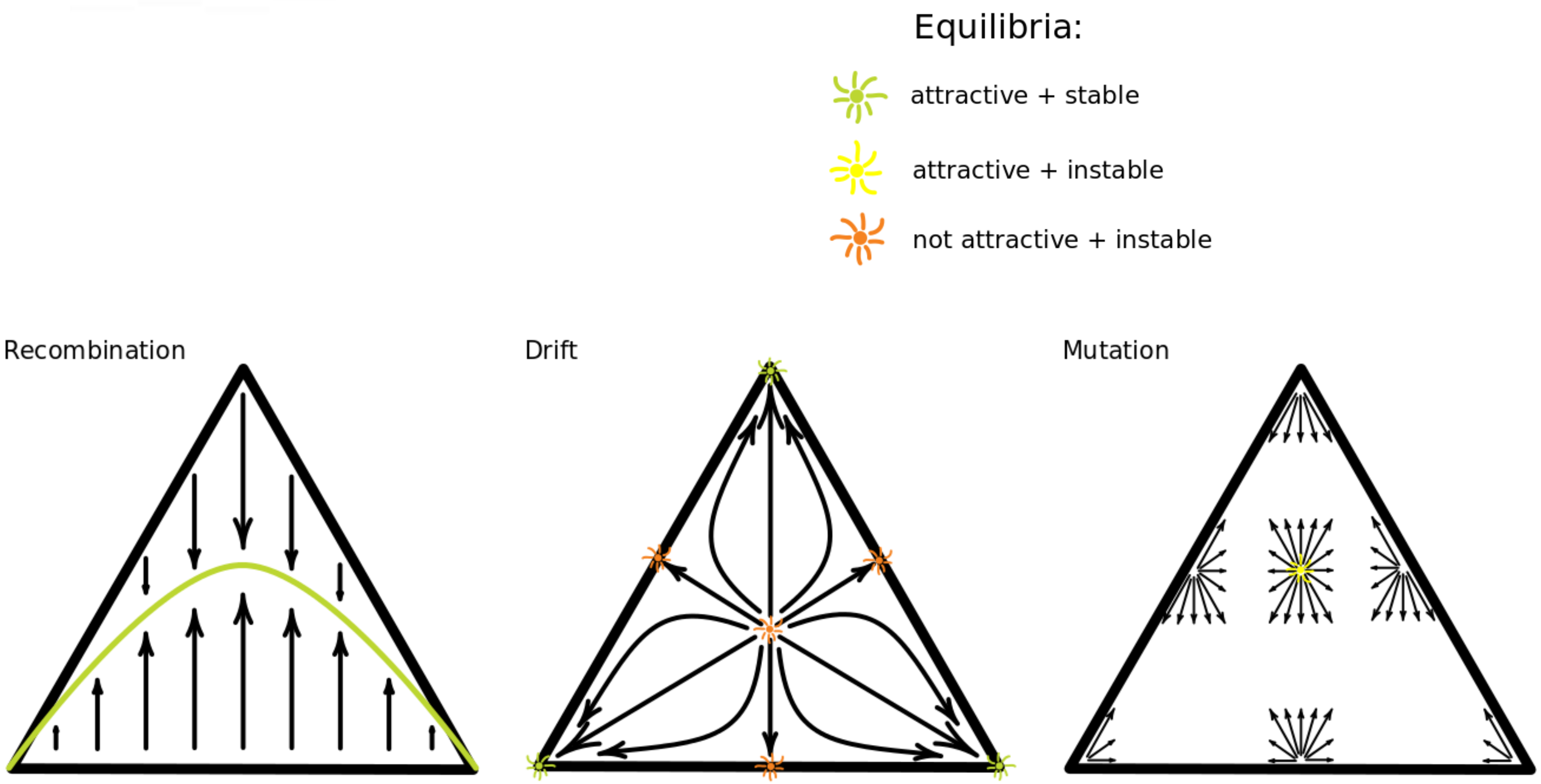
$p'(aA) = c * p^c(aA) + 2 * (1-c) * (p^s(aa) + 0.5 * p^s(aA)) * (p^s(AA) + 0.5 * p^s(aA))$

asexual   *sexual*

which gametes combine?

Parameter effects

Explaining what we see



A hypothetical scenario

color synthesis in aphids

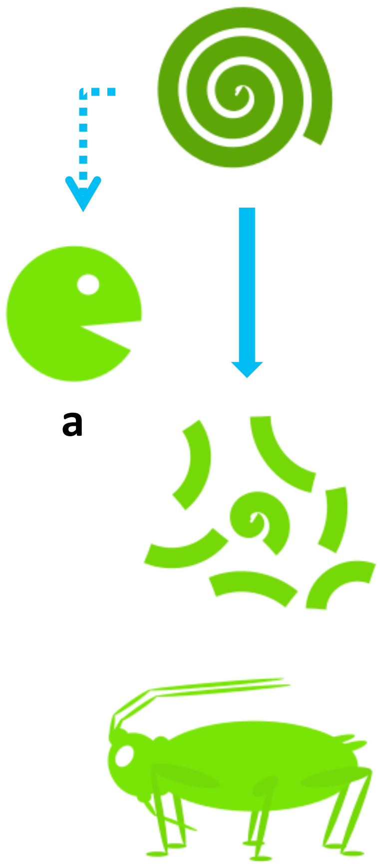
a **toxic substrate**

triggers the synthesis of

an **enzyme**

which transforms it into

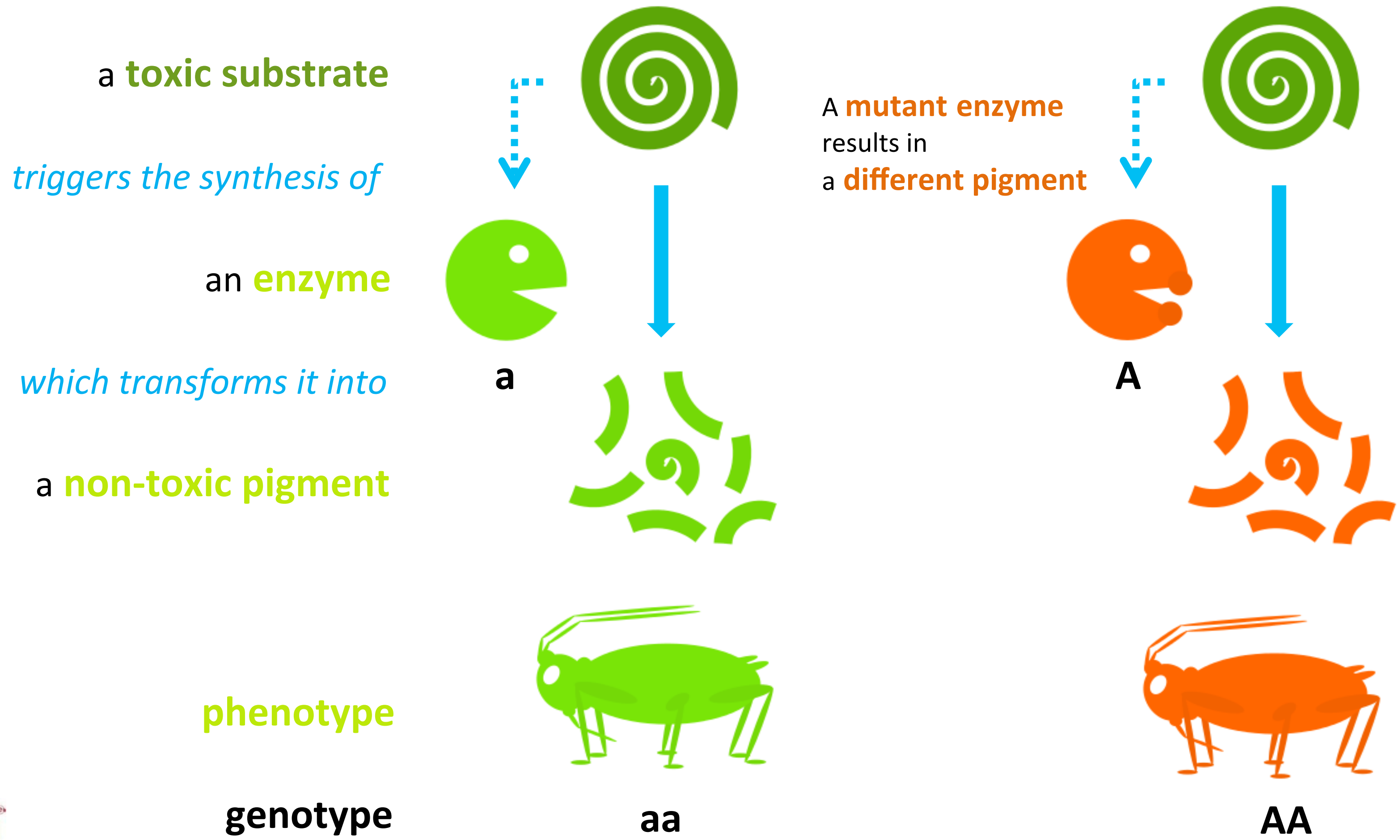
a **non-toxic pigment**



phenotype

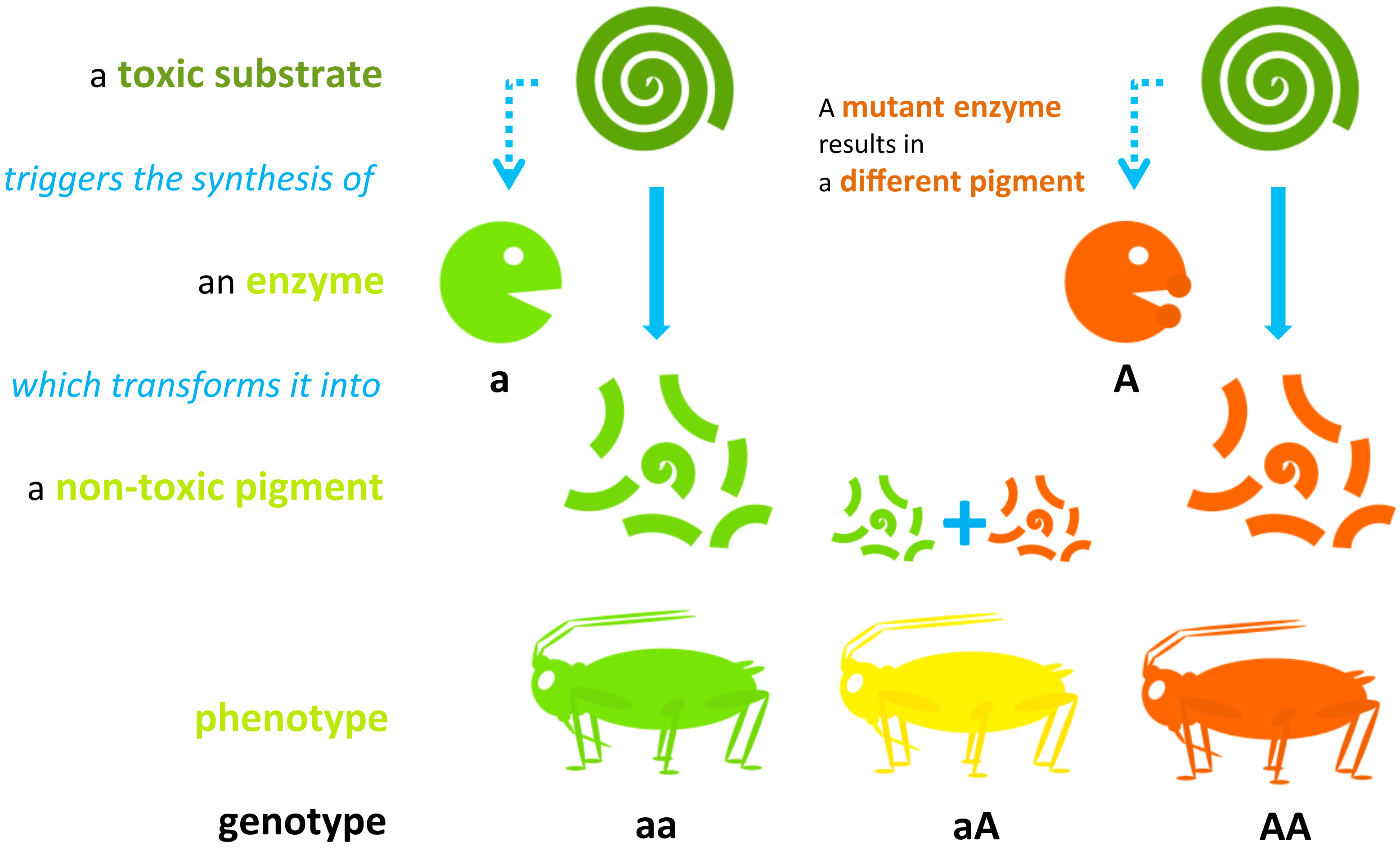
genotype **aa**

A hypothetical scenario
color synthesis in aphids



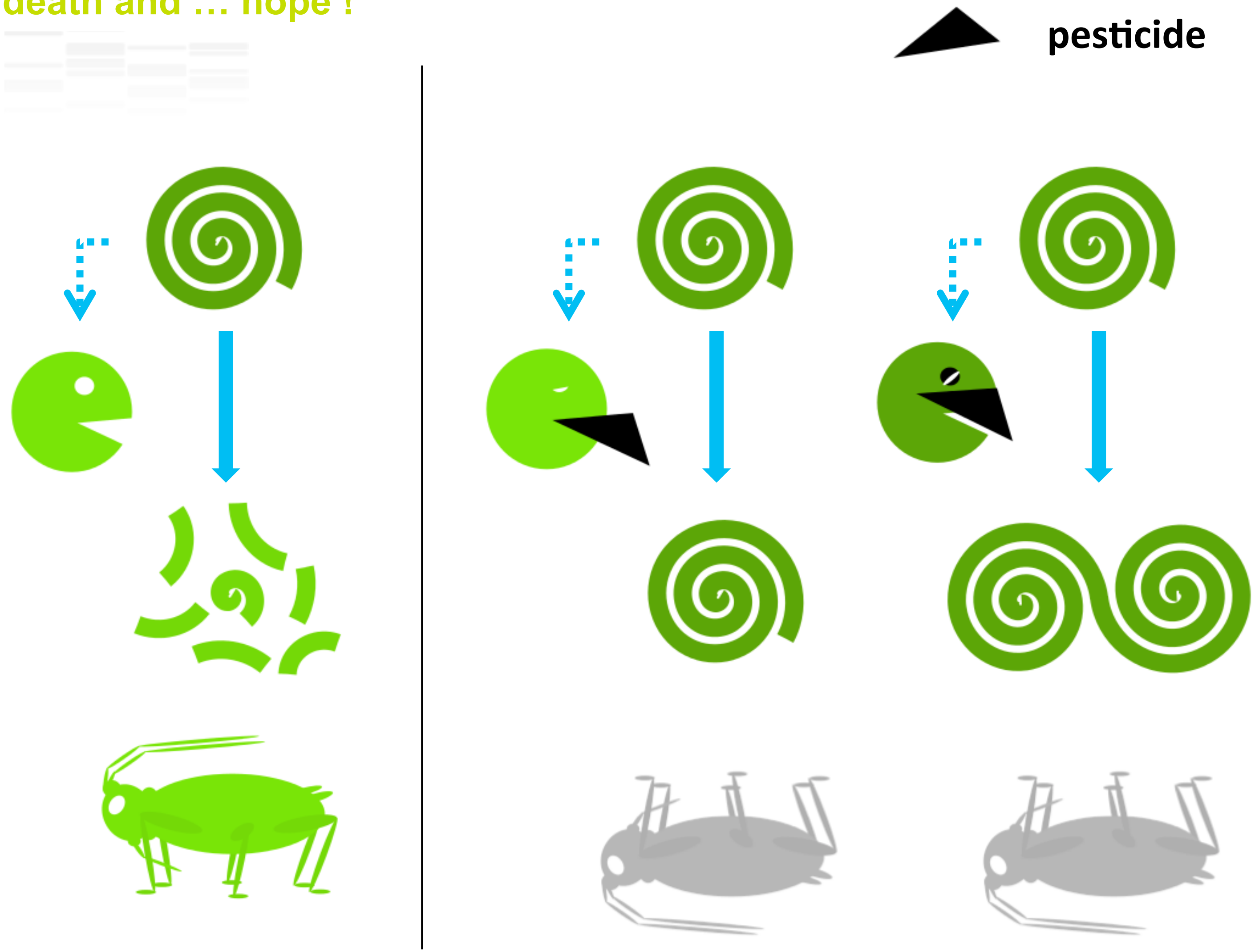
A hypothetical scenario

color synthesis in aphids



Pesticide

death and … hope !



Pesticide

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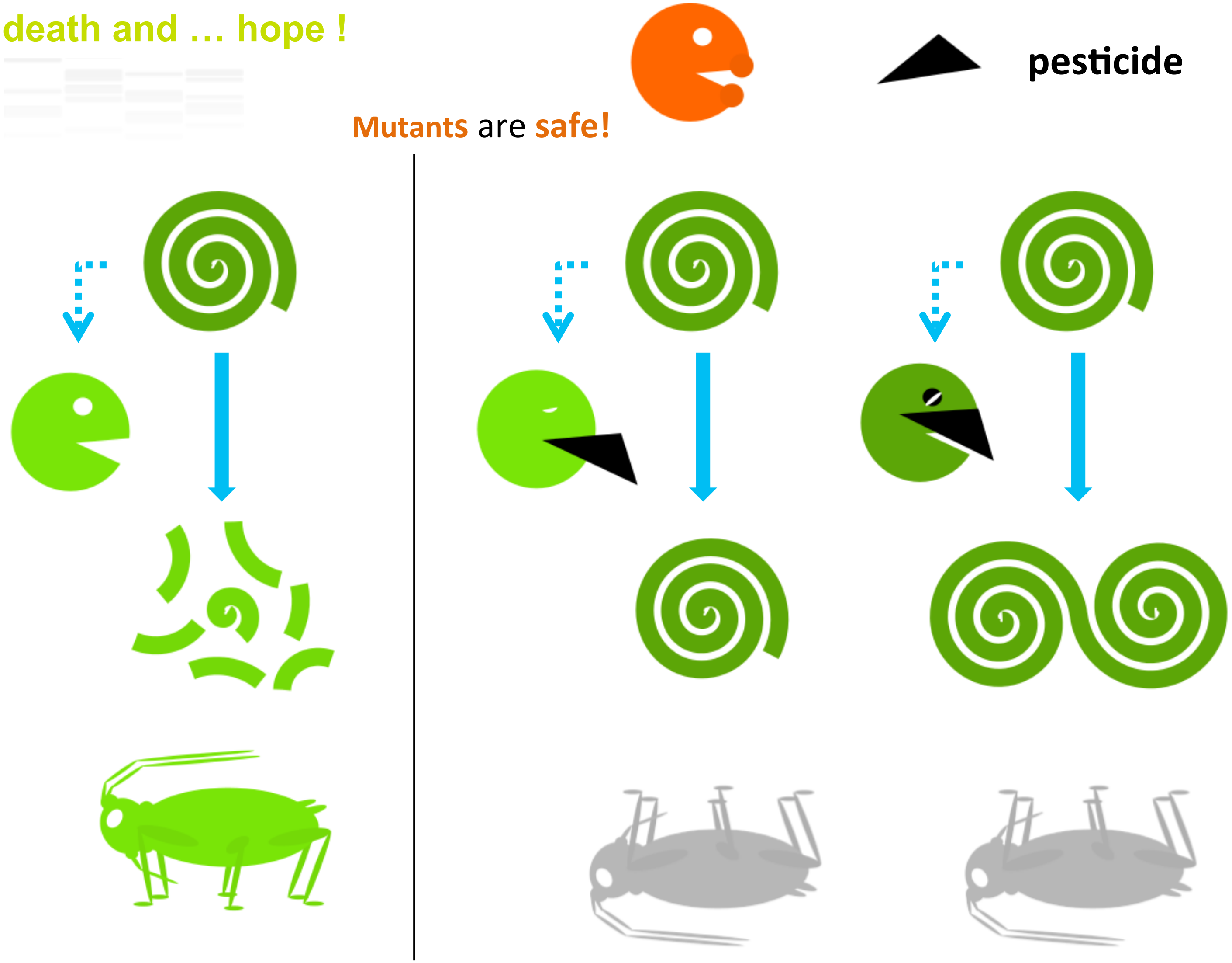
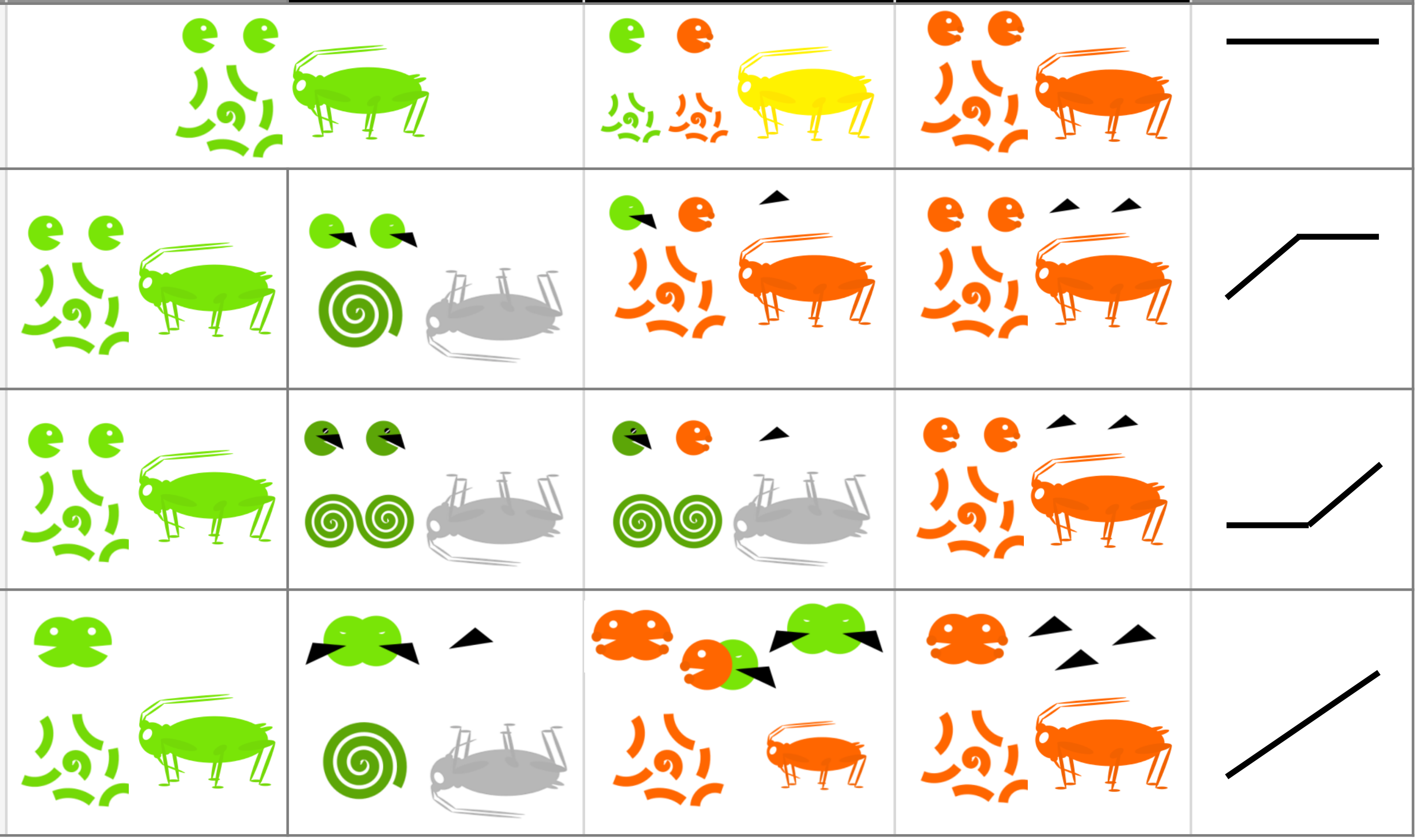


Selection

what does it look like

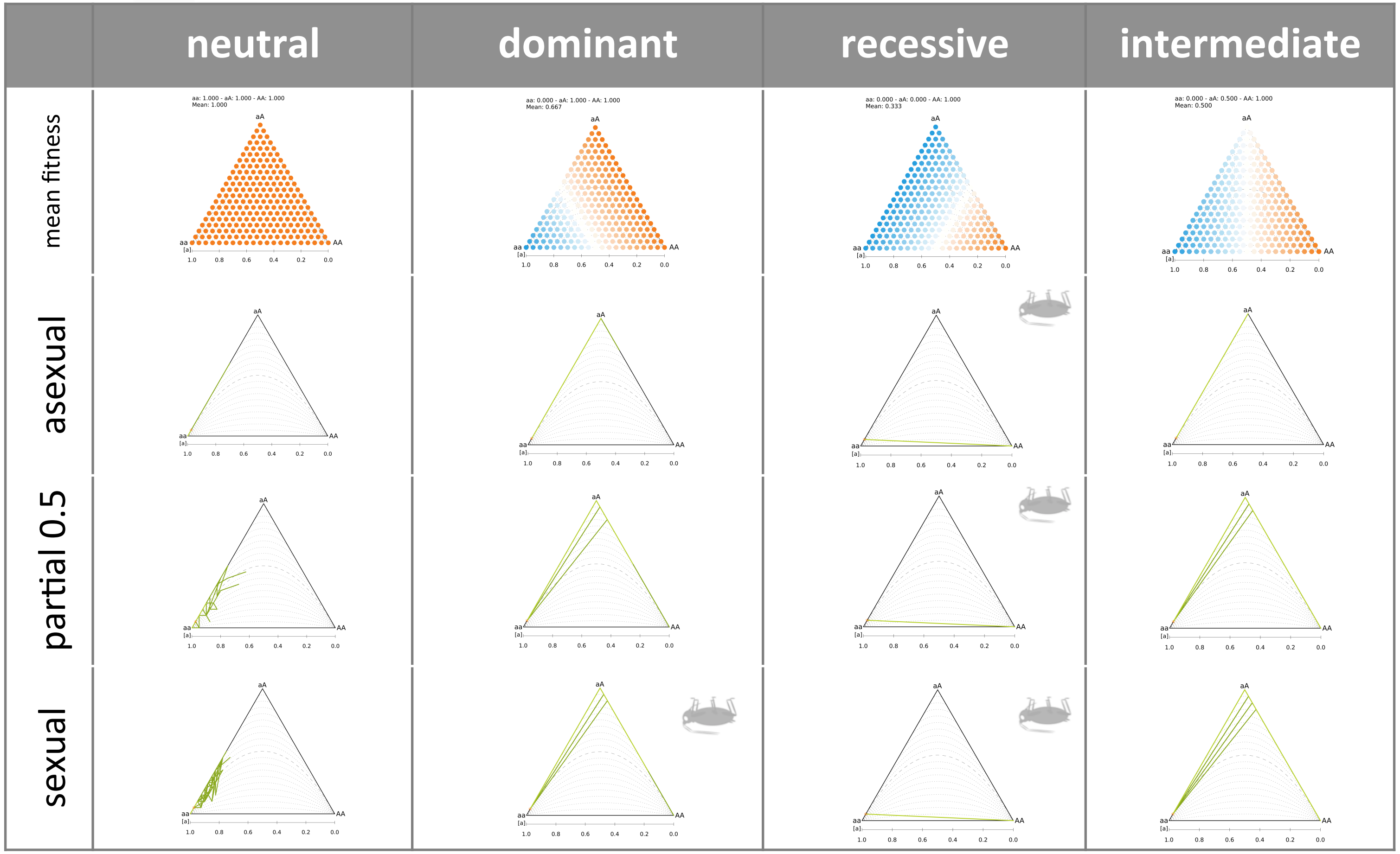
	wildtype aa natural	wildtype aa	heterozygous mutant aA	homozygous mutant AA	symbol
neutral					
dominant					
recessive					
intermediate					



Simulation I

all non-resistant individuals die

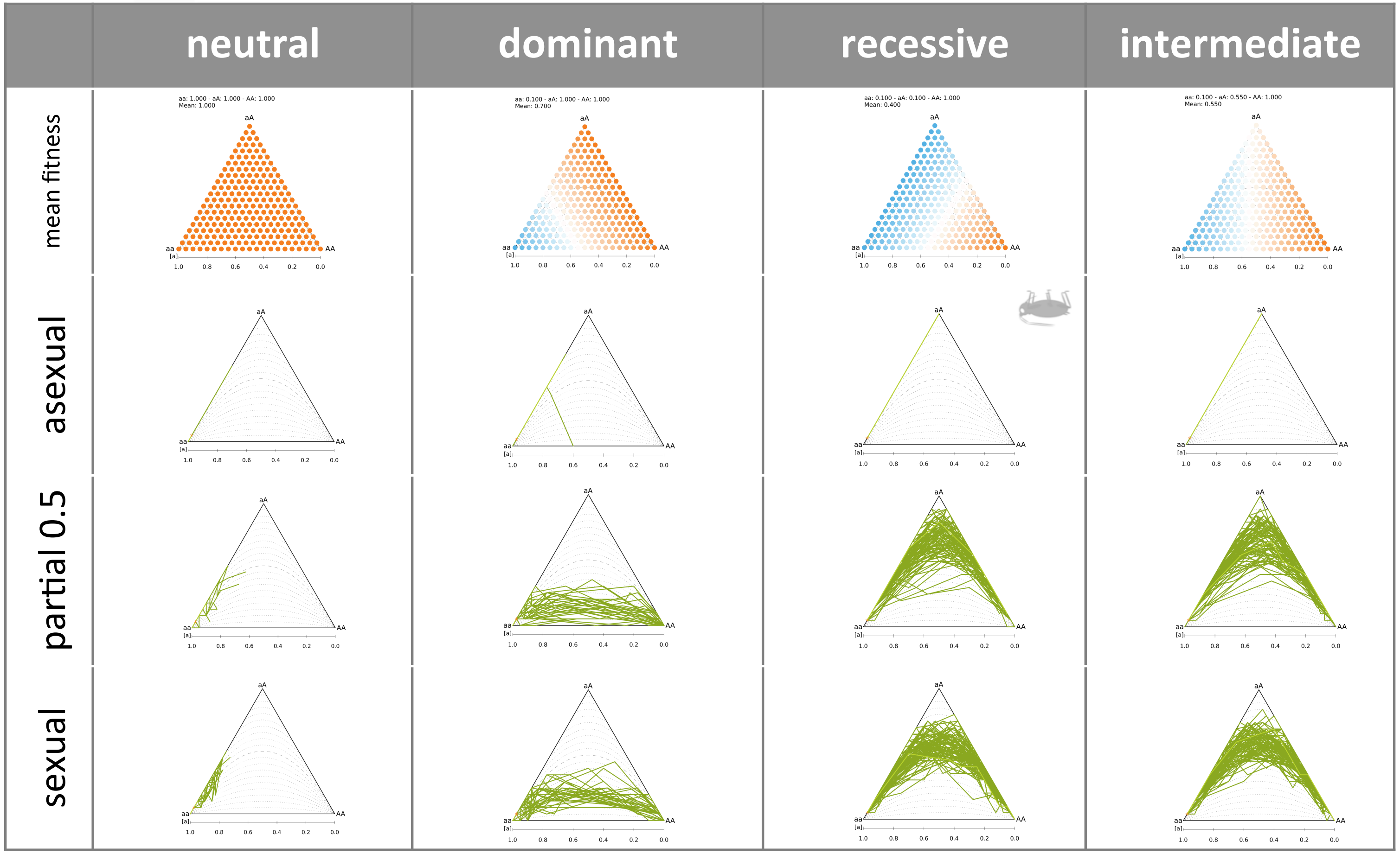
μ … 10^{-6}
N … 20



Simulation II

10% of the non-resistant individuals survive

μ … 10^{-6}
N … 20



Conclusions

pesticides and evolution

Pesticides

- responses to pesticide treatment depend on the mode of action of the pesticide and the reproductive mode of the pest species
- one copy of a "resistance allele" may be enough, in particular if the treatment is not even 100% effective against the non-resistant genotypes – pesticides are not a long-term option
- if a resistant genotype exists … it will spread!

Evolution

- adaptive evolution depends on the underlying "fitness landscape" and the reproductive mode of the adapting species
- one adapted individual may be sufficient to very quickly invade a new environment, especially if there are also some non-adapted mating partners around
- "killing" a species takes quite some force

Outlook

still to come

extending the model

- more alleles – multidimensional fitness landscape
- more loci – crossing over & linkage disequilibrium
- modeling genome evolution

extending the analysis

- times to fixation
- redefining population genetic parameters
- Can we infer the rate of asexual reproduction from genetic data?

Thank you

everyone

… in particular:

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