Theories of speciation

Today:

Mathematical theories of speciation:
what do they tell us?

Mathematical theory in evolution, including speciation

- Identify key parameters
- Attempts to simplify – "caricature" processes
- Almost inevitably over-simplifies, because speciation is complex, multilocus

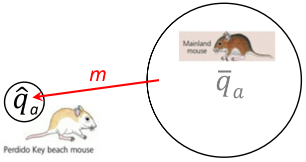
Pairs of coexisting closely related sexual species tend to be:
1) Ecologically different (some sort of divergent, disruptive selection).
2) Tend not to mate together (some sort of assortative mating).

How might that happen, and could it occur with gene flow?

Simplest key model: Haldane's model of migration/selection balance

Additive divergent selection*
against *a* alleles, s

	AA	Aa	aa
* Fitnesses	1	1-s	1-2s

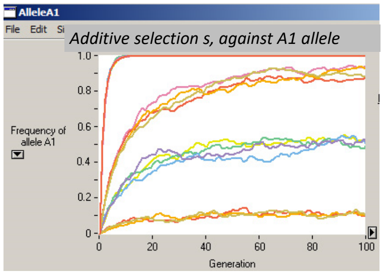


If $m \gg s$ then equilibrium $\hat{q} \approx \bar{q}$ -- "swamping!"

If $m \ll s$ then equilibrium $\hat{q} \approx \frac{m}{s}\bar{q}$ -- migration-selection equilibrium!

Too much gene flow, then no divergence

Selection vs. migration



s = 0.1,
N_e = 1000 :
with ...
m = 0.5
m = 0.1
m = 0.05
m = 0.001

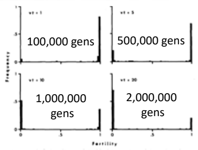
If $m << s$ then *selection "wins," maintains divergence, p* $\approx m/s$
If $m >> s$ then *migration "wins", pops. are "swamped"*

Nei et al. 1983:
Allopatric speciation. Accumulation of incompatibility via genetic drift: stepwise mutation, heterozygote disadvantage

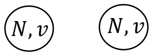
TABLE 1

Fertilities or viabilities for various genotypes in the stepwise mutation model. Here only five alleles are shown

	•	A_{-2}	A_{-1}	A_0	A_1	A_2	•
A_{-2}	•	1	1	0	0	0	•
A_{-1}	•	1	1	1	0	0	•
A_0	•	0	1	1	1	0	•
A_1	•	0	0	1	1	1	•
A_2	•	0	0	0	1	1	•



v is the mutation rate



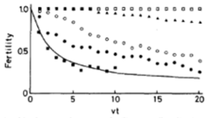
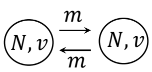


FIGURE 4.—Relationships between the average fertility over all replications ($\bar{f}$) and evolutionary time (vt) for various values of $2Nv$, where $v = 10^{-5}$ is used. The stepwise mutation model is used. The number of replications used is 100. The symbols □, ▲, ○, ● and ■ represent the cases of $N = 5 \times 10^4$, 2.5×10^4, 5×10^3 and 500, respectively. The solid line shows the expected lowest fertility.

Nei et al. 1983

Effect of gene flow



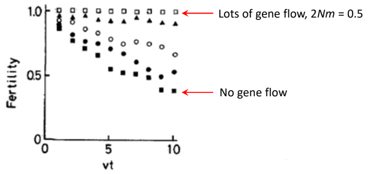


FIGURE 8.—Relationships between the average fertility over all replications ($\bar{f}$) and evolutionary time in the presence of migration. $N = 5000$ and $v = 10^{-5}$ were used. The number of replications is 100. The symbols □, ▲, ○, ● and ■ represent the cases of $2Nm = 0.5, 0.2, 0.1, 0.05$ and 0, respectively.

Conclusions
- Allopatric speciation via drift will be slow!
- Faster in smaller population sizes
- Even moderate gene flow (e.g. parapatry) stops speciation

Nei et al. 1983

Gene flow: maintenance of polymorphism via selection. Dempster's versus Levene's models

example fitnesses

	patch 1	patch 2
AA	$U_1 = 1 + s$	$U_2 = 1$
Aa	$V_1 = 1 + hs$	$V_2 = 1 + ht$
aa	$W_1 = 1$	$W_2 = 1 + t$

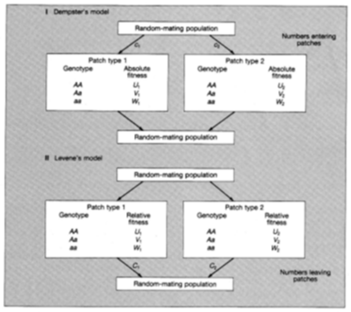


FIG. 4.8. Models used to investigate whether polymorphism can be maintained in a variable environment.

Dempster: No population regulation in patch:

Then heterozygotes must be on average fitter than homozygotes for maintenance of polymorphism, as in a single population

Levene: Population regulation in patch, so that each gives a constant proportion to total population:

Then no matter how strong the selection is in one patch versus another, there will always be some contribution from the least favoured patch that can balance losses, because the population is "topped up".

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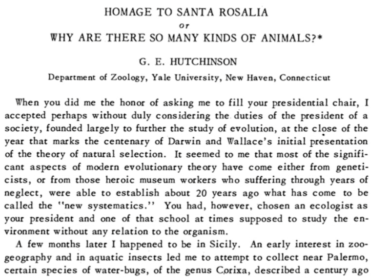
Felsenstein, 1981. Skepticism towards Santa Rosalia, or why are there so *few* kinds of animals*? Evolution 35: 124-138

Reference to a famous paper in ecology by George E. Hutchinson (1959) American Naturalist 93: 145-159.



HOMAGE TO SANTA ROSALIA
or
WHY ARE THERE SO MANY KINDS OF ANIMALS?*

G. E. HUTCHINSON

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When you did me the honor of asking me to fill your presidential chair, I accepted perhaps without duly considering the duties of the president of a society, founded largely to further the study of evolution, at the close of the year that marks the centenary of Darwin and Wallace's initial presentation of the theory of natural selection. It seemed to me that most of the significant aspects of modern evolutionary theory have come either from geneticists, or from those heroic museum workers who suffering through years of neglect, were able to establish about 20 years ago what has come to be called the "new systematics." You had, however, chosen an ecologist as your president and one of that school at times supposed to study the environment without any relation to the organism.

A few months later I happened to be in Sicily. An early interest in zoogeography and in aquatic insects led me to attempt to collect near Palermo, certain species of water-bugs, of the genus Corixa, described a century ago

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A concluding sentence from Hutchinson:

"… the reason why there are so many species of animals is at least partly because a complex trophic organization of a community is more stable than a simple one, but that limits are set by the tendency of food chains to shorten or become blurred, by unfavorable physical factors, by space, by the fineness of possible subdivision of niches, and by those characters of the environmental mosaic which permit a greater diversity of small than of large allied species."

Mosaicism of niches: maybe small size of organisms and "fineness of possible subdivision of niches" is related to gene flow among niches?

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Felsenstein (1981), Skepticism towards Santa Rosalia, or why are there so few kinds of animals? :

Introduction p. 124

Speciation in current mathematical models is too easy! Maynard Smith 1966, Dickinson & Antonovics 1973, Caisse & Antonovics 1978, Balkau & Feldman 1973. All show sympatric speciation is possible: speciation is easy!

"…One would expect to find nearly infinite numbers of species. … There would be a balance between speciation and the extinction of small species. …

It is my impression that the number of species in nature is far smaller, and their size [i.e. nos. of individuals] far larger, than such a model would predict."

Maybe there's a genetic constraint on speciation?

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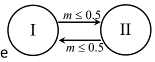
Felsenstein's model p. 125 et seq.



Haploid, deterministic, $N \to \infty$
Levene-type model of population structure
(**soft selection, density-regulated**)

Two loci, *B,C* experience divergent selection, *s,* in two subpopulations (niches), which maintains *B,C* polymorphisms overall, as follows:

	Subpopulation	
Genotype	I	II
BC	$(1 + s)^2$	1
Bc	$1 + s$	$1 + s$
bC	$1 + s$	$1 + s$
bc	1	$(1 + s)^2$

Population regulation: absolute

Within each population, random mating every generation for recombination (*m*=0.5).

Linkage Disequilibrium (LD) forms between B and C in such a Levene model.

Unrelated assortative mating locus, *A*, that causes preferential mating with parameter *d*, as follows:

	A	a	Totals
A	$p^2(1 - d) + pd$	$p(1 - p)(1 - d)$	p
a	$p(1 - p)(1 - d)$	$(1 - p)^2(1 - d) + (1 - p)d$	$1\text{-}p$
Totals	p	$1\text{-}p$	1

"Progress towards speciation" = stable *linkage disequilibrium* between A and B+C.

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What is linkage disequilibrium (LD)

When two genes deviate from the expected two locus equilibrium, the genes are said to be in *linkage **dis**equilibrium* (or *gametic disequilibrium*).

The strength of this deviation is measured by the linkage disequilibrium (LD) coefficient, ***D***. Suppose we have two diallelic loci, A/a and B/b

Observed gametic frequencies		random two locus expectation	+ deviation
p_{AB}	=	$p_A p_B$	+ ***D***
p_{Ab}	=	$p_A(1\text{-}p_B)$	- ***D***
p_{aB}	=	$(1\text{-}p_A)p_B$	- ***D***
p_{ab}	=	$(1\text{-}p_A)(1\text{-}p_B)$	+ ***D***

$\Sigma = 1$

D can vary between a maximum of +0.25 and a minimum of –0.25, but the range is often smaller if the frequency of alleles is not exactly 0.5.
(Because $p_{ij} \geq 0$ for all i, j !).

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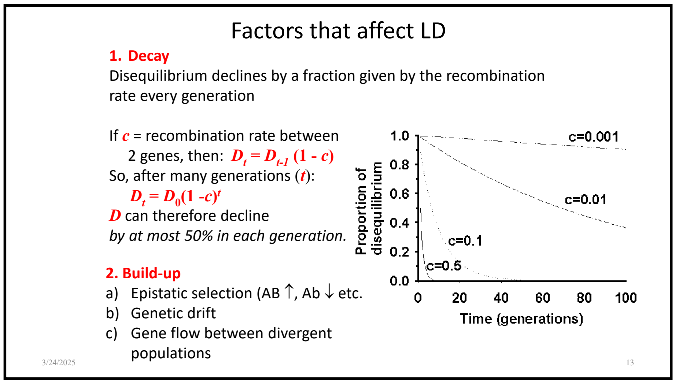


Factors that affect LD

1. Decay

Disequilibrium declines by a fraction given by the recombination rate every generation

If c = recombination rate between 2 genes, then: $D_t = D_{t-1}(1 - c)$

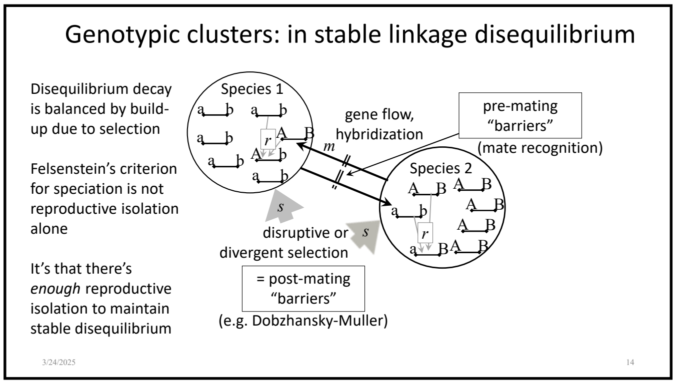
So, after many generations (t):

$D_t = D_0(1 - c)^t$

D can therefore decline *by at most 50% in each generation*.

2. Build-up

a) Epistatic selection (AB ↑, Ab ↓ etc.
b) Genetic drift
c) Gene flow between divergent populations

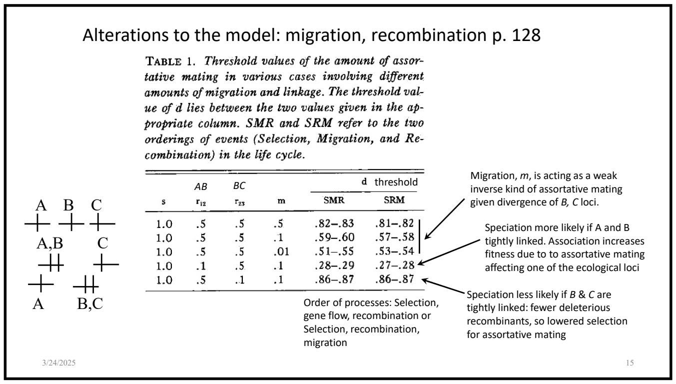


Genotypic clusters: in stable linkage disequilibrium

Disequilibrium decay is balanced by build-up due to selection

Felsenstein's criterion for speciation is not reproductive isolation alone

It's that there's *enough* reproductive isolation to maintain stable disequilibrium



Alterations to the model: migration, recombination p. 128

TABLE 1. *Threshold values of the amount of assortative mating in various cases involving different amounts of migration and linkage. The threshold value of d lies between the two values given in the appropriate column. SMR and SRM refer to the two orderings of events (Selection, Migration, and Recombination) in the life cycle.*

	AB	*BC*		d threshold	
s	r_{12}	r_{23}	m	SMR	SRM
1.0	.5	.5	.5	.82–.83	.81–.82
1.0	.5	.5	.1	.59–.60	.57–.58
1.0	.5	.5	.01	.51–.55	.53–.54
1.0	.1	.5	.1	.28–.29	.27–.28
1.0	.5	.1	.1	.86–.87	.86–.87

Migration, *m*, is acting as a weak inverse kind of assortative mating given divergence of *B, C* loci.

Speciation more likely if A and B tightly linked. Association increases fitness due to to assortative mating affecting one of the ecological loci

Speciation less likely if *B* & *C* are tightly linked: fewer deleterious recombinants, so lowered selection for assortative mating

Order of processes: Selection, gene flow, recombination or Selection, recombination, migration

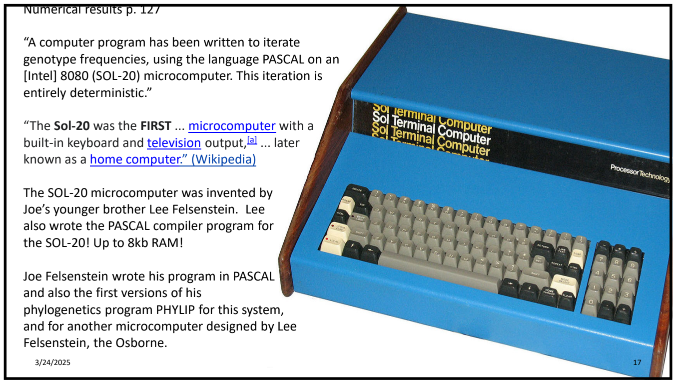
Evolutionary forces, p. 129

How does this work?
Remember, no direct selection on A locus.

Supposing all in perfect linkage disequilibrium, then ABC and abc only.

If rare recombinant Abc were to occur, would mate with ABC, and ABc and AbC offspring would result. Bc and bC are less fit than BC and bc overall, so there is indirect selection against A-(B+C) recombinants.

This is why reduction in recombination rate B-C makes speciation more difficult.
... And why A-B recombination rate reduction makes speciation easier in Table 1.



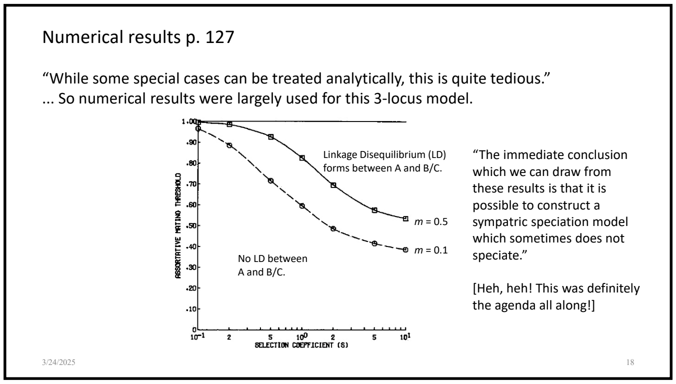
Numerical results p. 127

"A computer program has been written to iterate genotype frequencies, using the language PASCAL on an [Intel] 8080 (SOL-20) microcomputer. This iteration is entirely deterministic."

"The **Sol-20** was the **FIRST** ... microcomputer with a built-in keyboard and television output,[a] ... later known as a home computer." (Wikipedia)

The SOL-20 microcomputer was invented by Joe's younger brother Lee Felsenstein. Lee also wrote the PASCAL compiler program for the SOL-20! Up to 8kb RAM!

Joe Felsenstein wrote his program in PASCAL and also the first versions of his phylogenetics program PHYLIP for this system, and for another microcomputer designed by Lee Felsenstein, the Osborne.



Numerical results p. 127

"While some special cases can be treated analytically, this is quite tedious."
... So numerical results were largely used for this 3-locus model.

"The immediate conclusion which we can draw from these results is that it is possible to construct a sympatric speciation model which sometimes does not speciate."

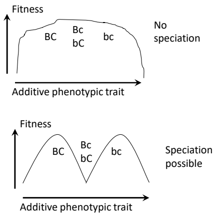
[Heh, heh! This was definitely the agenda all along!]

Mean fitness of BC and bc vs. Bc and bC generalists, p. 136

"When the mean fitnesses of BC and bc, averaged across the two subpopulations, did not exceed the fitnesses of Bc and bC, then sympatric speciation became impossible no matter how strong the assortative mating. In this case the generalists Bc and bC do not have lower average fitness than the specialists.

This suggests that we may be able to relate these fitness patterns to ... niche overlap between the forms adapted to the two subpopulations, and that when this overlap is too great, speciation will not occur."

Niches must be "discrete" relative to quantitative variation so that recombinants/intermediates survive poorly.



"One-allele" vs. "two-allele" models, pp. 133-135

In Felsenstein's model, sympatric speciation requires very strong selection, and strong assortative mating. Otherwise gene flow with $m = 0.5$ or even $m = 0.1$ swamps recombination and prevents speciation.

Why is sympatric speciation "nearly impossible" in this model, when some previous models show it is likely?

The current model is a two-allele model of assortative mating, in that *A* mates with *A*, and *a* mates with *a*. Progress towards speciation only if one allele, say *A*, becomes associated with *BC*, and the other, say *a*, with *bc*.

One-allele models, in contrast, favor assortative mating by population or by phenotype, with say *A* giving assortative mating within populations, and *a* not. So fixation of *A* is favoured because it enhances fitness in both populations, & does not require linkage disequilibrium between *A* and *BC/bc*, making it easier to attain. Previous models of sympatric speciation have been of this type.

"Allopatry is a situation favorable to either..." one- or two-allele models.

Twenty-one years later:
Mark Kirkpatrick & Virginie Ravigné 2002

Mark Kirkpatrick & Virginie Ravigné

Why do we "still lack a coherent understanding of speciation in terms of population genetic principles"? Three main reasons:

1) Speciation takes a long time.
2) Speciation is complex, many parameters, "spatial structure, non-random mating, epistasis, etc."
3) "Theoreticians have balkanized the subject of speciation." Too many models of many highly specific scenarios.

Surveyed ~ 100 models of speciation via natural selection.
Review is restricted to evolution of prezygotic isolation only.

Kirkpatrick & Ravigné 2002

Mark Kirkpatrick & Virginie Ravigné

Five common elements:

1) A source of disruptive selection (i.e. one that generates linkage disequilibrium
2) A prezygotic isolating mechanism – assortative mating, or mating preference
3) A way to transmit the disruptive selection to the isolating mechanism:
    direct or indirect selection
4) A genetic basis for increased isolation
5) An initial condition. Start near panmixia, or start with strong isolation?

Kirkpatrick & Ravigné 2002

By 2002:

Many mathematical models of speciation!

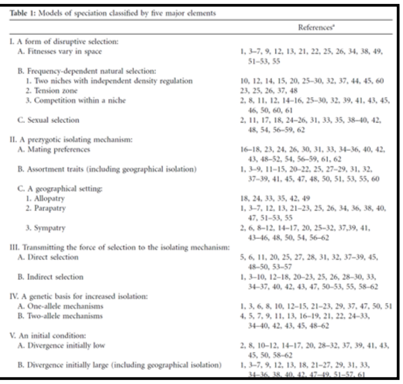


Table 1: Models of speciation classified by five major elements

	References[a]
I. A form of disruptive selection:	
A. Fitnesses vary in space	1, 3–7, 9, 12, 13, 21, 22, 25, 26, 34, 38, 49, 51–53, 55
B. Frequency-dependent natural selection:	
1. Two niches with independent density regulation	10, 12, 14, 15, 20, 25–30, 32, 37, 44, 45, 60
2. Tension zone	23, 25, 26, 37, 48
3. Competition within a niche	2, 8, 11, 12, 14–16, 25–30, 32, 39, 41, 43, 45, 46, 50, 60, 61
C. Sexual selection	2, 11, 17, 18, 24–26, 31, 33, 35, 38–40, 42, 48, 54, 56–59, 62
II. A prezygotic isolating mechanism:	
A. Mating preferences	16–18, 23, 24, 26, 30, 31, 33, 34–36, 40, 42, 43, 48–52, 54, 56–59, 61, 62
B. Assortment traits (including geographical isolation)	1, 3–9, 11–15, 20–22, 25, 27–29, 31, 32, 37–39, 41, 45, 47, 48, 50, 51, 53, 55, 60
C. A geographical setting:	
1. Allopatry	18, 24, 33, 35, 42, 49
2. Parapatry	1, 3–7, 12, 13, 21–23, 25, 26, 34, 36, 38, 40, 47, 51–53, 55
3. Sympatry	2, 6, 8–12, 14–17, 20, 25–32, 37,39, 41, 43–46, 48, 50, 54, 56–62
III. Transmitting the force of selection to the isolating mechanism:	
A. Direct selection	5, 6, 11, 20, 25, 27, 28, 31, 32, 37–39, 45, 48–50, 53–57
B. Indirect selection	1, 3–10, 12–18, 20–23, 25, 26, 28–30, 33, 34–37, 40, 42, 43, 47, 50–53, 55, 58–62
IV. A genetic basis for increased isolation:	
A. One-allele mechanisms	1, 3, 6, 8, 10, 12–15, 21–23, 29, 37, 47, 50, 51
B. Two-allele mechanisms	4, 5, 7, 9, 11, 13, 16–19, 21, 22, 24–33, 34–40, 42, 43, 45, 48–62
V. An initial condition:	
A. Divergence initially low	2, 8, 10–12, 14–17, 20, 28–32, 37, 39, 41, 43, 45, 50, 58–62
B. Divergence initially large (including geographical isolation)	1, 3–7, 9, 12, 13, 18, 21–27, 29, 31, 33, 34–36, 38, 40, 42, 47–49, 51–57, 61

Kirkpatrick & Ravigné

Haploid “toy model” of speciation. Studied the effect of “postzygotic isolation” and “assortative mating”

Two haploid loci, each have + and – alleles that affect a quantitative trait

Allele frequencies are set at 50% (“some form of [disruptive] selection maintains this polymorphism”)

Selection is such that fitnesses of ++, + – , and – – are 1, 1-*S*, and 1

S > 0 is disruptive, *S* < 0 is stabilizing

Fixed assortative mating parameter A: A = 0, is random, A = 1 complete assortment, based on number of + alleles

Kirkpatrick & Ravigné 2002

Kirkpatrick & Ravigné

Using this greatly simplified, haploid two locus “toy model” of speciation, K & R and looked at the effect of disruptive selection and “assortative mating.” Like Felsenstein, K & R are interested in the maintenance of linkage disequilibrium. Even this “toy model” is quite complicated – here’s an analytical result for linkage disequilibrium:

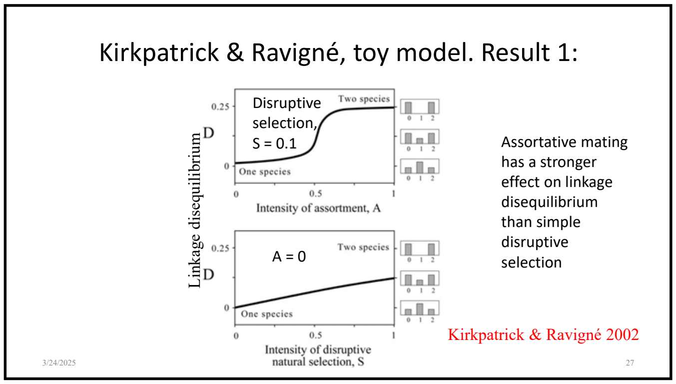
After mating, haploid offspring are produced following free recombination.

The dynamics of the linkage disequilibrium are given by

$$\Delta D = \frac{-64(6A^2 - 4A^3 + A^4 - 4AS + 2S^2)D^3 - 16[2A^4 - 8A^3 + 12A^2 - 8A - 3(S-2)S]D^2 - 4[4 + 6A^2 - 4A^3 + A^4 + 4A(S-2) - 4S]D - S(S-2)}{4[6A^2 - 4A^3 + A^4 + 4A(S-2) + 2(S-2)^2 + 32(-4A + 6A^2 - 4A^3 + A^4 + 4S - 2S^2)D + 64(6A^2 - 4A^3 + A^4 - 4AS + 2S^2)D^2]}.$$

Here, *A* is the degree of assortative mating, *S* is the selection pressure, and *D* is linkage disequilibrium.

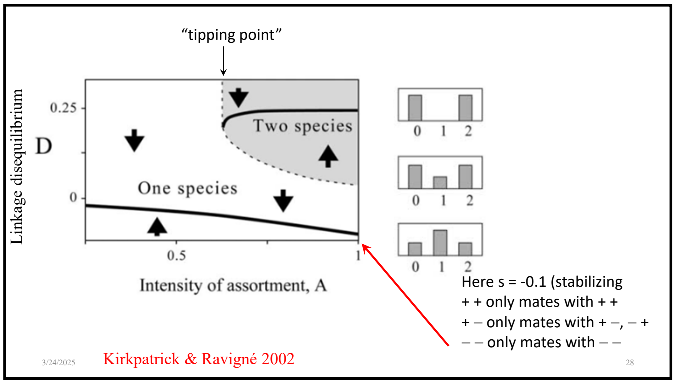
Kirkpatrick & Ravigné 2002



Kirkpatrick & Ravigné, toy model. Result 1:

Assortative mating has a stronger effect on linkage disequilibrium than simple disruptive selection

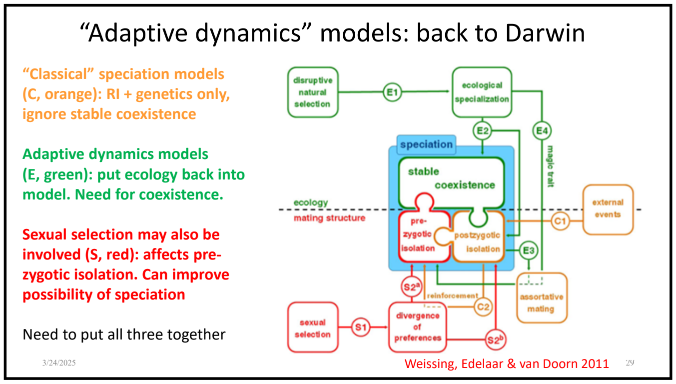
Kirkpatrick & Ravigné 2002



“tipping point”

Here s = -0.1 (stabilizing
+ + only mates with + +
+ – only mates with + –, – +
– – only mates with – –

Kirkpatrick & Ravigné 2002



“Adaptive dynamics” models: back to Darwin

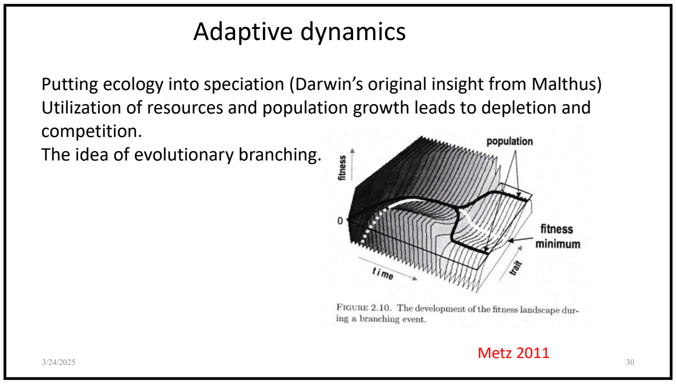
“Classical” speciation models (C, orange): RI + genetics only, ignore stable coexistence

Adaptive dynamics models (E, green): put ecology back into model. Need for coexistence.

Sexual selection may also be involved (S, red): affects pre-zygotic isolation. Can improve possibility of speciation

Need to put all three together

Weissing, Edelaar & van Doorn 2011

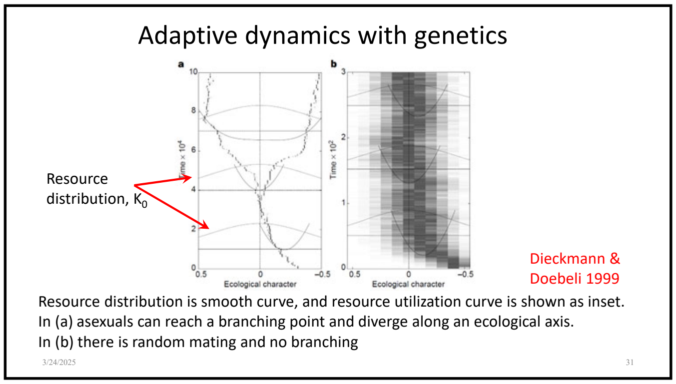


Adaptive dynamics

Putting ecology into speciation (Darwin’s original insight from Malthus)
Utilization of resources and population growth leads to depletion and competition.
The idea of evolutionary branching.

FIGURE 2.10. The development of the fitness landscape during a branching event.

Metz 2011



Adaptive dynamics with genetics

Resource distribution, K_0

Dieckmann & Doebeli 1999

Resource distribution is smooth curve, and resource utilization curve is shown as inset.
In (a) asexuals can reach a branching point and diverge along an ecological axis.
In (b) there is random mating and no branching

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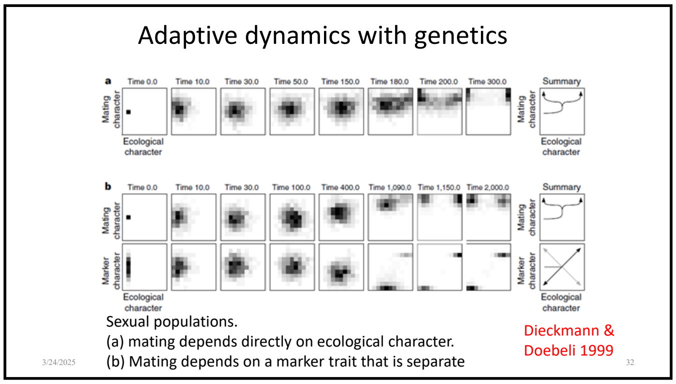

Adaptive dynamics with genetics

Sexual populations.
(a) mating depends directly on ecological character.
(b) Mating depends on a marker trait that is separate

Dieckmann & Doebeli 1999

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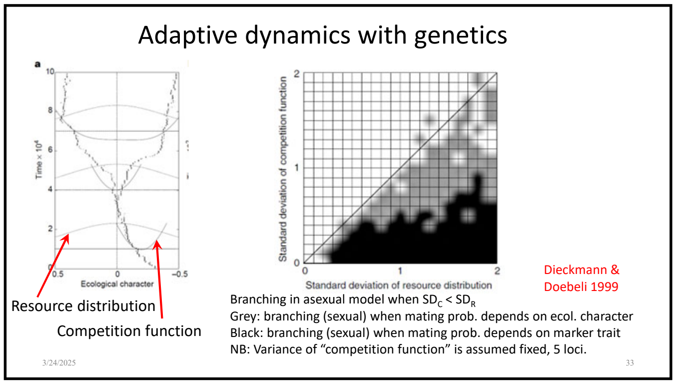

Adaptive dynamics with genetics

Resource distribution

Competition function

Dieckmann & Doebeli 1999

Branching in asexual model when $SD_C < SD_R$
Grey: branching (sexual) when mating prob. depends on ecol. character
Black: branching (sexual) when mating prob. depends on marker trait
NB: Variance of "competition function" is assumed fixed, 5 loci.

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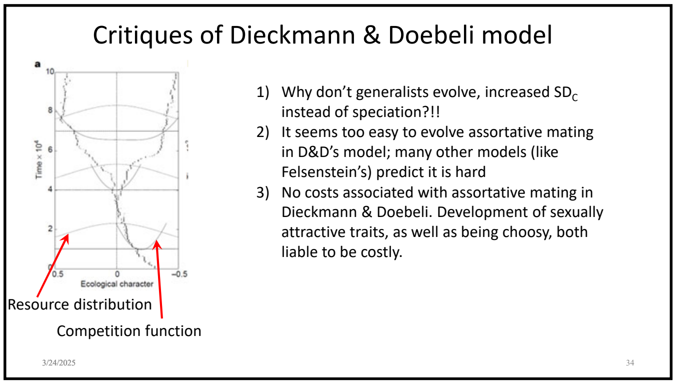

Critiques of Dieckmann & Doebeli model

Resource distribution

Competition function

1) Why don't generalists evolve, increased SD_C instead of speciation?!!
2) It seems too easy to evolve assortative mating in D&D's model; many other models (like Felsenstein's) predict it is hard
3) No costs associated with assortative mating in Dieckmann & Doebeli. Development of sexually attractive traits, as well as being choosy, both liable to be costly.

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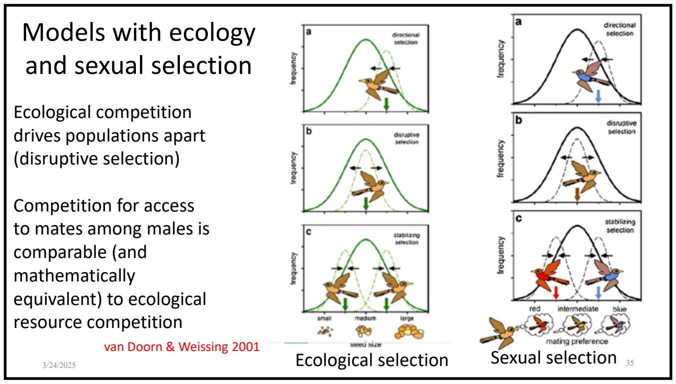

Models with ecology and sexual selection

Ecological competition drives populations apart (disruptive selection)

Competition for access to mates among males is comparable (and mathematically equivalent) to ecological resource competition

van Doorn & Weissing 2001

Ecological selection

Sexual selection

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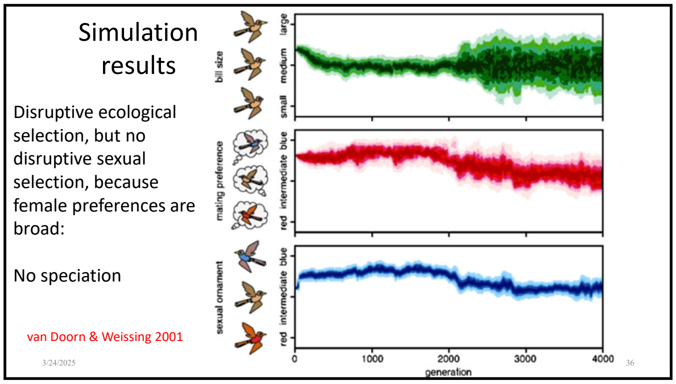

Simulation results

Disruptive ecological selection, but no disruptive sexual selection, because female preferences are broad:

No speciation

van Doorn & Weissing 2001

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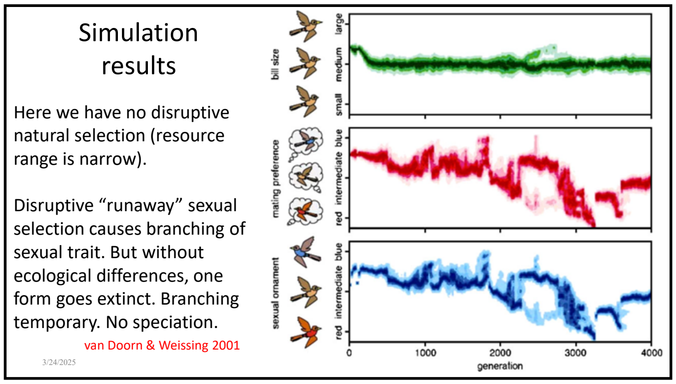
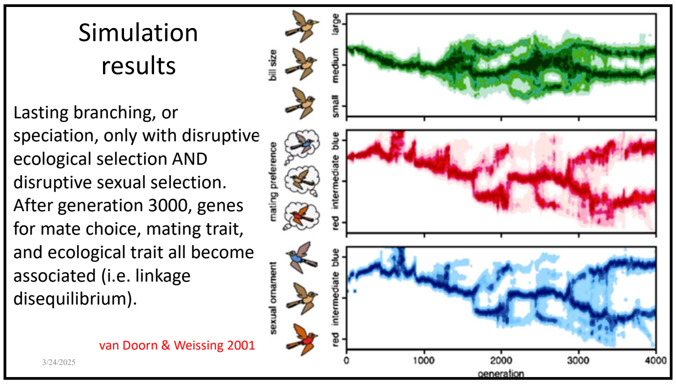


Simulation results

Here we have no disruptive natural selection (resource range is narrow).

Disruptive "runaway" sexual selection causes branching of sexual trait. But without ecological differences, one form goes extinct. Branching temporary. No speciation.

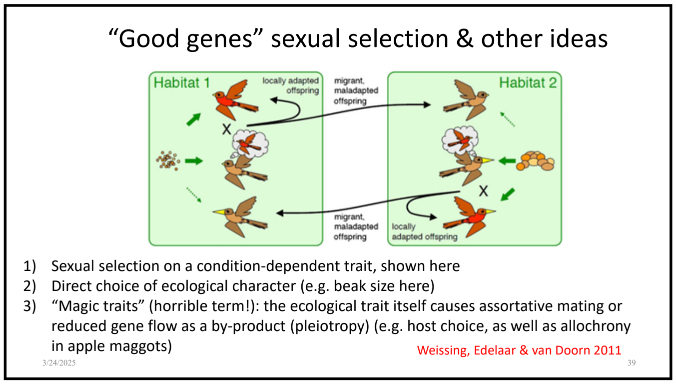
van Doorn & Weissing 2001



Simulation results

Lasting branching, or speciation, only with disruptive ecological selection AND disruptive sexual selection. After generation 3000, genes for mate choice, mating trait, and ecological trait all become associated (i.e. linkage disequilibrium).

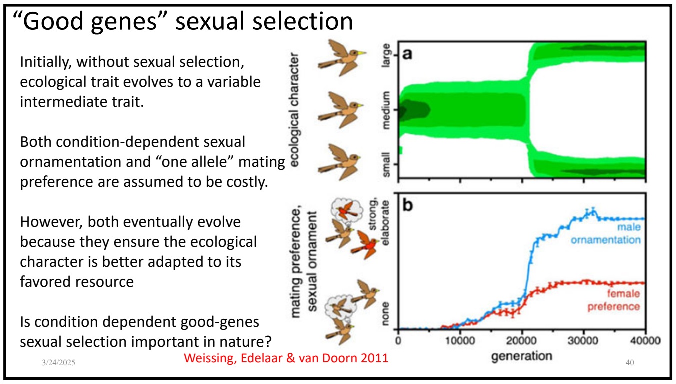
van Doorn & Weissing 2001



"Good genes" sexual selection & other ideas

1) Sexual selection on a condition-dependent trait, shown here
2) Direct choice of ecological character (e.g. beak size here)
3) "Magic traits" (horrible term!): the ecological trait itself causes assortative mating or reduced gene flow as a by-product (pleiotropy) (e.g. host choice, as well as allochrony in apple maggots)

Weissing, Edelaar & van Doorn 2011



"Good genes" sexual selection

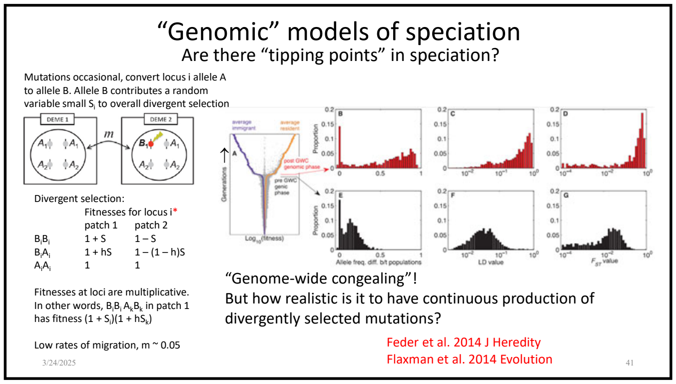
Initially, without sexual selection, ecological trait evolves to a variable intermediate trait.

Both condition-dependent sexual ornamentation and "one allele" mating preference are assumed to be costly.

However, both eventually evolve because they ensure the ecological character is better adapted to its favored resource

Is condition dependent good-genes sexual selection important in nature?

Weissing, Edelaar & van Doorn 2011



"Genomic" models of speciation

Are there "tipping points" in speciation?

Mutations occasional, convert locus i allele A to allele B. Allele B contributes a random variable small S_i to overall divergent selection

Divergent selection:

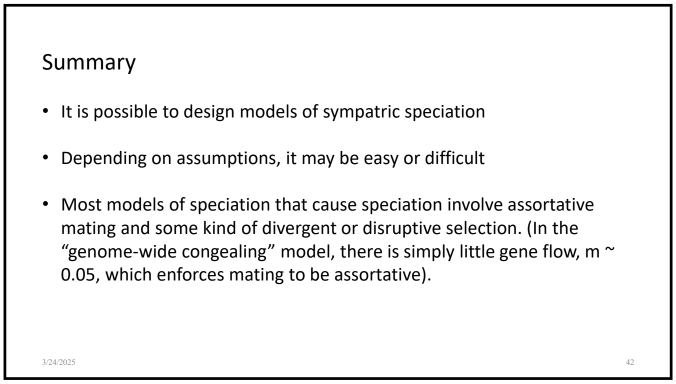
	Fitnesses for locus i*	
	patch 1	patch 2
B_iB_i	1 + S	1 – S
B_iA_i	1 + hS	1 – (1 – h)S
A_iA_i	1	1

Fitnesses at loci are multiplicative. In other words, $B_iB_i\,A_kB_k$ in patch 1 has fitness $(1 + S_i)(1 + hS_k)$

Low rates of migration, m ~ 0.05

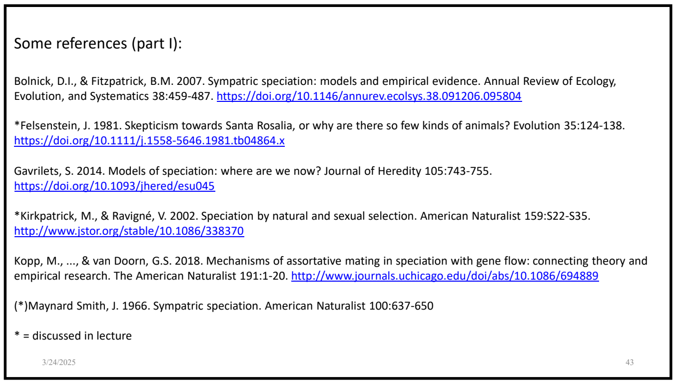
"Genome-wide congealing"!
But how realistic is it to have continuous production of divergently selected mutations?

Feder et al. 2014 J Heredity
Flaxman et al. 2014 Evolution



Summary

- It is possible to design models of sympatric speciation
- Depending on assumptions, it may be easy or difficult
- Most models of speciation that cause speciation involve assortative mating and some kind of divergent or disruptive selection. (In the "genome-wide congealing" model, there is simply little gene flow, m ~ 0.05, which enforces mating to be assortative).



Some references (part I):

Bolnick, D.I., & Fitzpatrick, B.M. 2007. Sympatric speciation: models and empirical evidence. Annual Review of Ecology, Evolution, and Systematics 38:459-487. https://doi.org/10.1146/annurev.ecolsys.38.091206.095804

*Felsenstein, J. 1981. Skepticism towards Santa Rosalia, or why are there so few kinds of animals? Evolution 35:124-138. https://doi.org/10.1111/j.1558-5646.1981.tb04864.x

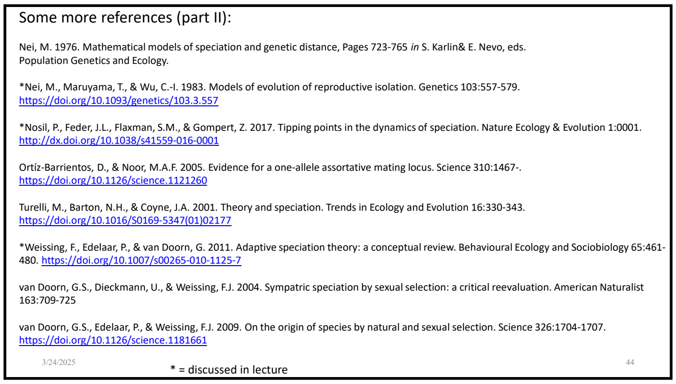
Gavrilets, S. 2014. Models of speciation: where are we now? Journal of Heredity 105:743-755. https://doi.org/10.1093/jhered/esu045

*Kirkpatrick, M., & Ravigné, V. 2002. Speciation by natural and sexual selection. American Naturalist 159:S22-S35. http://www.jstor.org/stable/10.1086/338370

Kopp, M., ..., & van Doorn, G.S. 2018. Mechanisms of assortative mating in speciation with gene flow: connecting theory and empirical research. The American Naturalist 191:1-20. http://www.journals.uchicago.edu/doi/abs/10.1086/694889

(*)Maynard Smith, J. 1966. Sympatric speciation. American Naturalist 100:637-650

* = discussed in lecture



Some more references (part II):

Nei, M. 1976. Mathematical models of speciation and genetic distance, Pages 723-765 *in* S. Karlin& E. Nevo, eds. Population Genetics and Ecology.

*Nei, M., Maruyama, T., & Wu, C.-I. 1983. Models of evolution of reproductive isolation. Genetics 103:557-579. https://doi.org/10.1093/genetics/103.3.557

*Nosil, P., Feder, J.L., Flaxman, S.M., & Gompert, Z. 2017. Tipping points in the dynamics of speciation. Nature Ecology & Evolution 1:0001. http://dx.doi.org/10.1038/s41559-016-0001

Ortíz-Barrientos, D., & Noor, M.A.F. 2005. Evidence for a one-allele assortative mating locus. Science 310:1467-. https://doi.org/10.1126/science.1121260

Turelli, M., Barton, N.H., & Coyne, J.A. 2001. Theory and speciation. Trends in Ecology and Evolution 16:330-343. https://doi.org/10.1016/S0169-5347(01)02177

*Weissing, F., Edelaar, P., & van Doorn, G. 2011. Adaptive speciation theory: a conceptual review. Behavioural Ecology and Sociobiology 65:461-480. https://doi.org/10.1007/s00265-010-1125-7

van Doorn, G.S., Dieckmann, U., & Weissing, F.J. 2004. Sympatric speciation by sexual selection: a critical reevaluation. American Naturalist 163:709-725

van Doorn, G.S., Edelaar, P., & Weissing, F.J. 2009. On the origin of species by natural and sexual selection. Science 326:1704-1707. https://doi.org/10.1126/science.1181661

* = discussed in lecture