



A Primer for Phylogenetic Causal Inference

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Baton Rouge LA

9. Jan. 2026

....but Correlation ≠
Causation!!

Schedule

8:30 - 9:30	Lecture
9:30 - 9:45	Questions/Discussion
9:45 -10:00	Group building/introductions
10:00 - 10:30	Coffee break
10:30 - 11:00	Case Study I
11:00 - 11:30	Case Study II
11:30 - 12:00	Questions/Discussion

A Primer for Phylogenetic Causal Inference

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Why Causal Inference?

Using the term in macroevolution will probably still get you in trouble

It will not save you from “correlation ≠ causation”

We will not fully equip you to solve your own causal problems today

We are not presenting software/method

CI is a logical framework not a single tool

Our goals

Make assumptions & inference strategies explicit

Clarify how CI differs from traditional practice

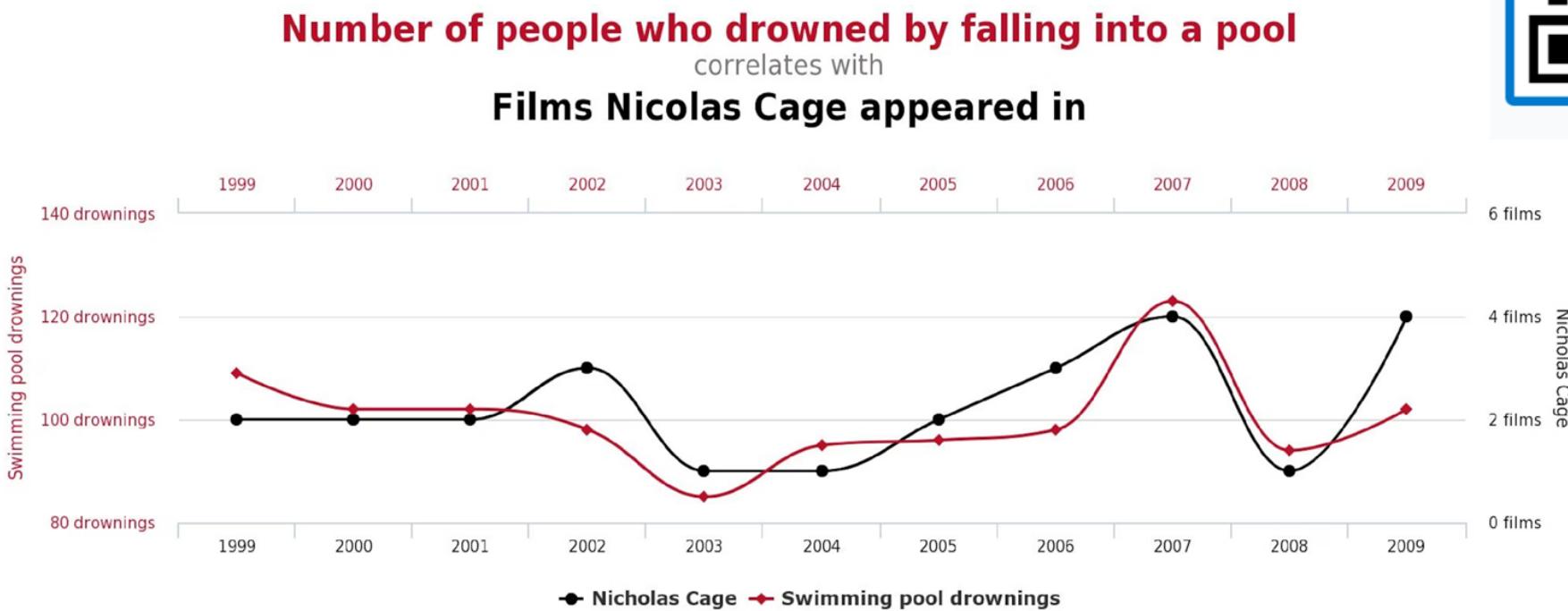
Align familiar comparative methods with causal ideas

Remind that thinking causally helps generate testable predictions

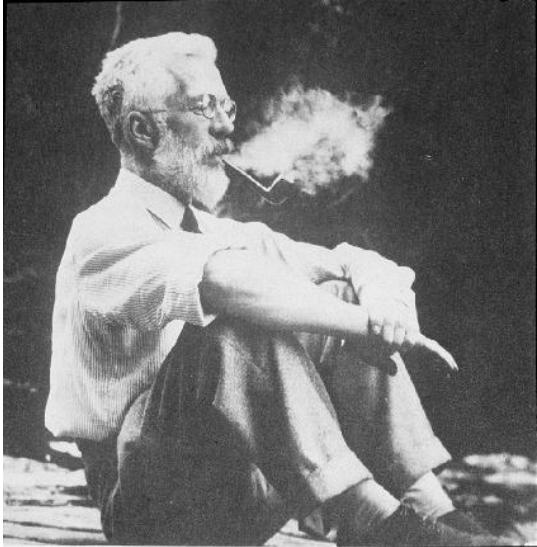
Correlation does not equal causation

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TRUE.



What is the “gold standard” for supporting causal claims in science?



Experiments:
Adding the “do” to science

RA Fisher

Macroevolution = (*mostly*) observational science

Do we care about/talk about causes in macroevolution?

“ecologists [macroevolutionary biologists] often qualify their results verbally to avoid making causal claims—even when their research focus is causal understanding, rather than description...This practice muddies the waters and can create confusion over whether an author is claiming an association or implying causation while allowing themselves plausible deniability.”

-Byrnes & Dee, 2024

Do we care about/talk about causes in macroevolution?

The screenshot shows two separate Google Scholar search queries. The top query is for "causes of diversification" "phylogeny", which yields 365 results. The bottom query is for "drivers of diversification" "phylogeny", which yields 1,570 results. Both searches were completed in 0.08 seconds.

Search Query	Results
"causes of diversification" "phylogeny"	365 results
"drivers of diversification" "phylogeny"	1,570 results

...yes, but we may hide the claims (and assumptions) behind weaker language

What is a cause?

Mathematically, not represented by equations

e.g. Breeder's Equation: $R = h^2 s$

The Evolutionary [R]esponse to selection equals

[h²]eritability x the [s]trength of natural selection

Math rules allow rearranging; e.g. $R/h^2 = s$

Can increasing h^2 decrease s ? NO.

"Do" operator; cause defined as: $P(Y|do(X = x)) \neq P(Y|do(X = x'))$

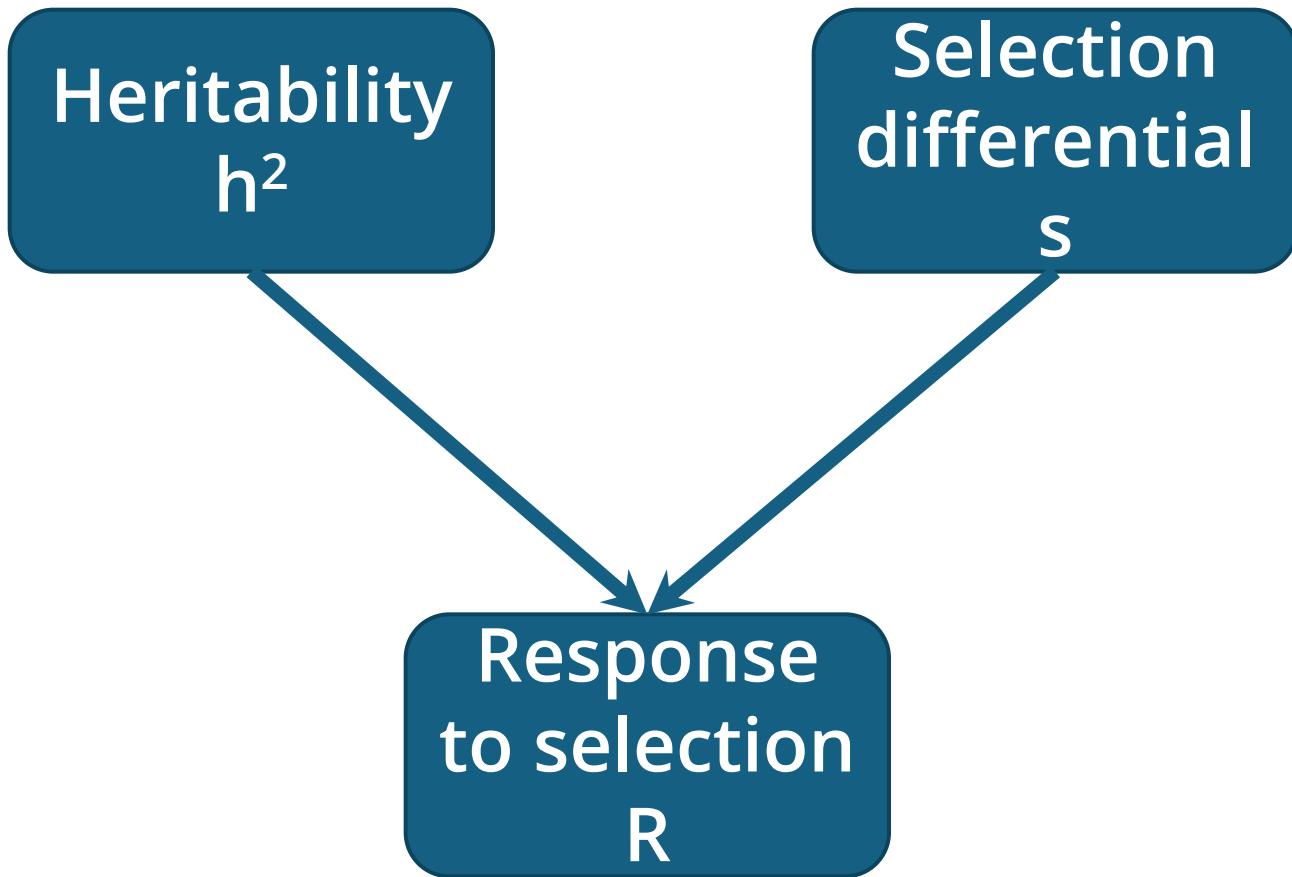
Causal logic of interventions in the Breeder's Equation (for example):

1. h^2 and s are causes of R $P(R|do(s = 0.5)) \neq P(R|do(s = 0))$

2. h^2 is not a cause of s , and vice versa $P(s|do(h^2 = 0.5)) = P(s|do(h^2 = 0))$

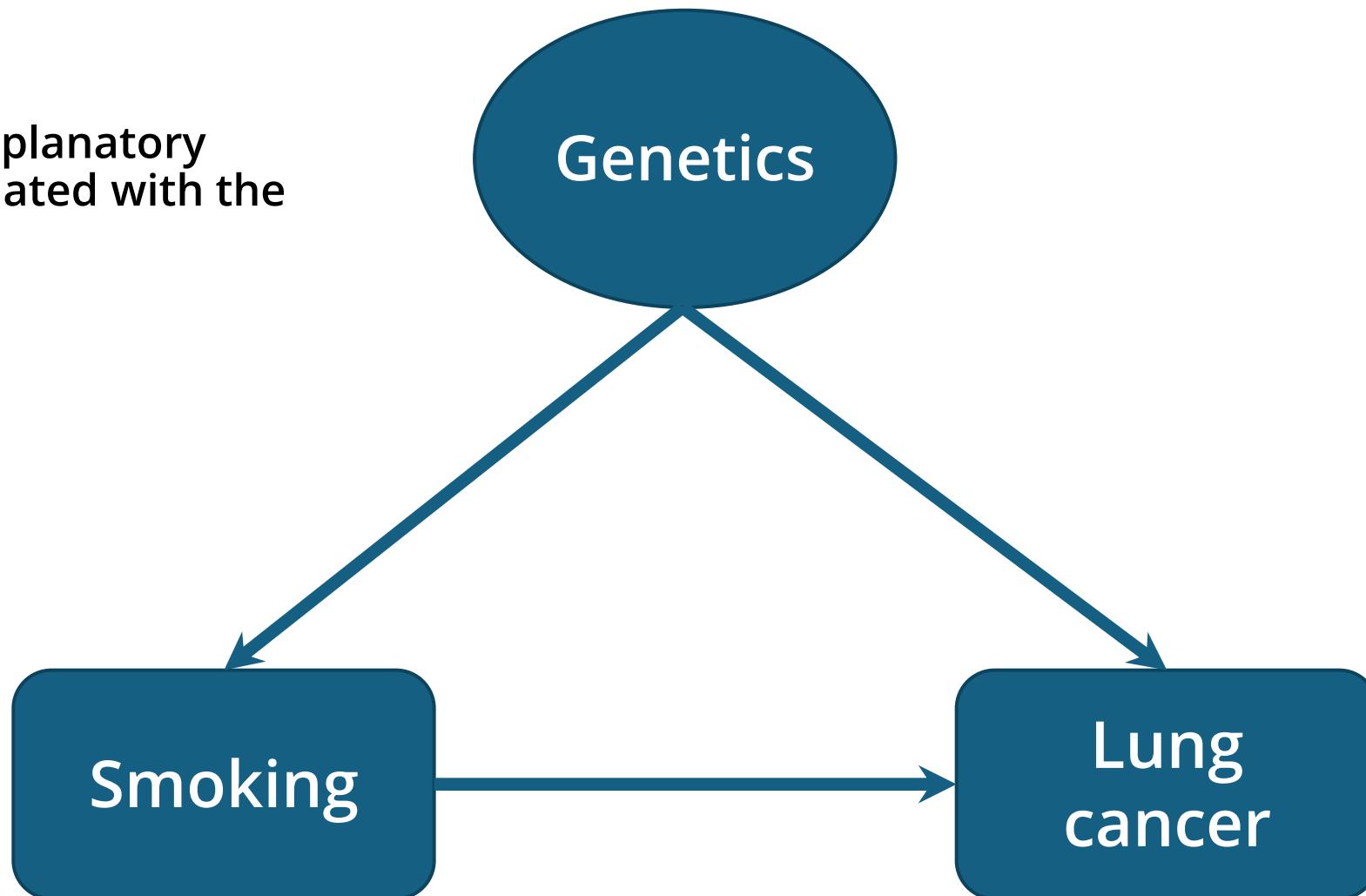
3. Manipulating R doesn't go back in time and affect original h^2 & s
(unclear what this manipulation would even be...)

Directed Acyclic Graph as Causal Models *representing interventions “do()”*

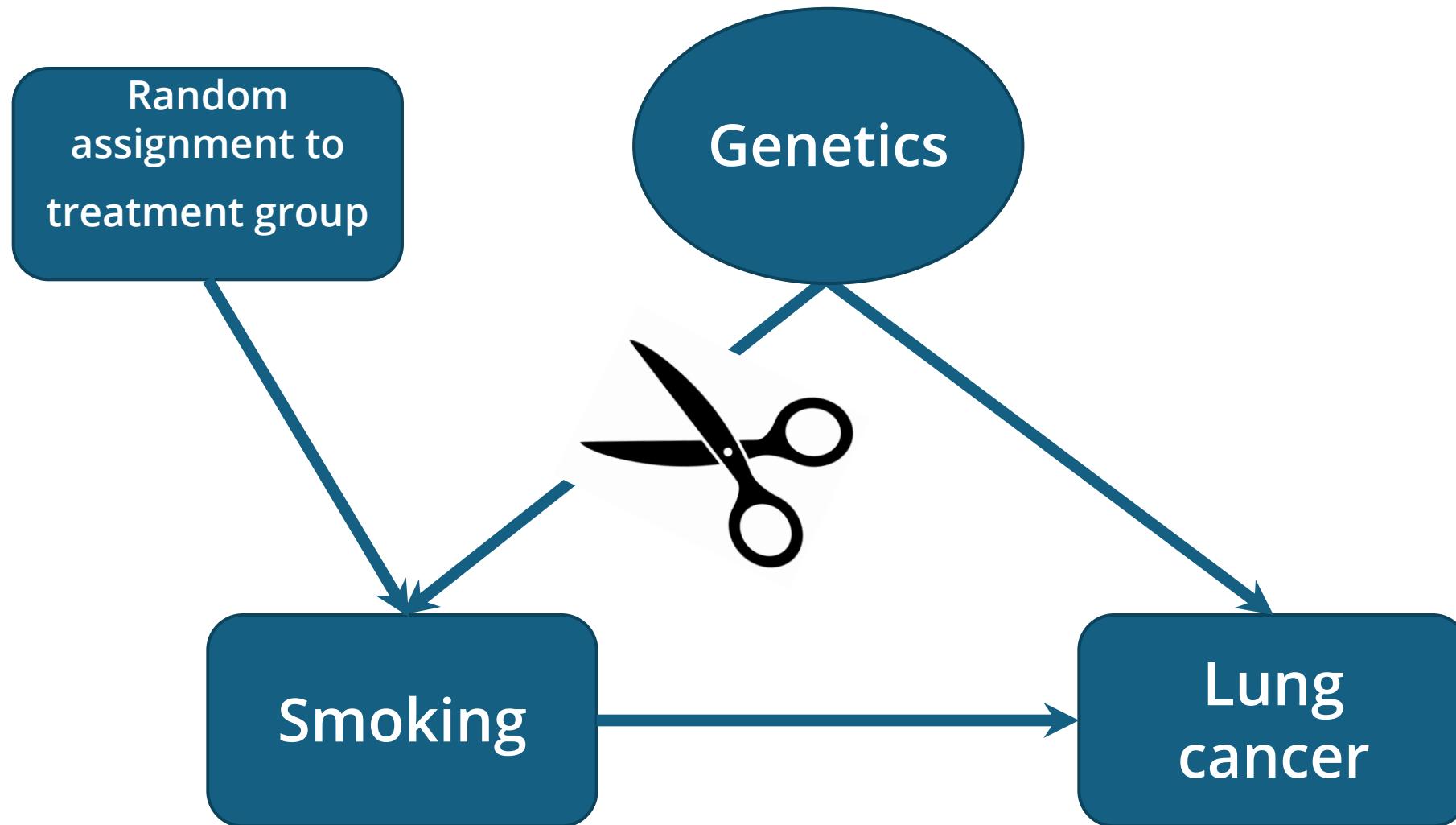


Confounding

Endogeneity - Explanatory variable is correlated with the error term.



Deconfounding via experiments



Deconfounding via conditioning

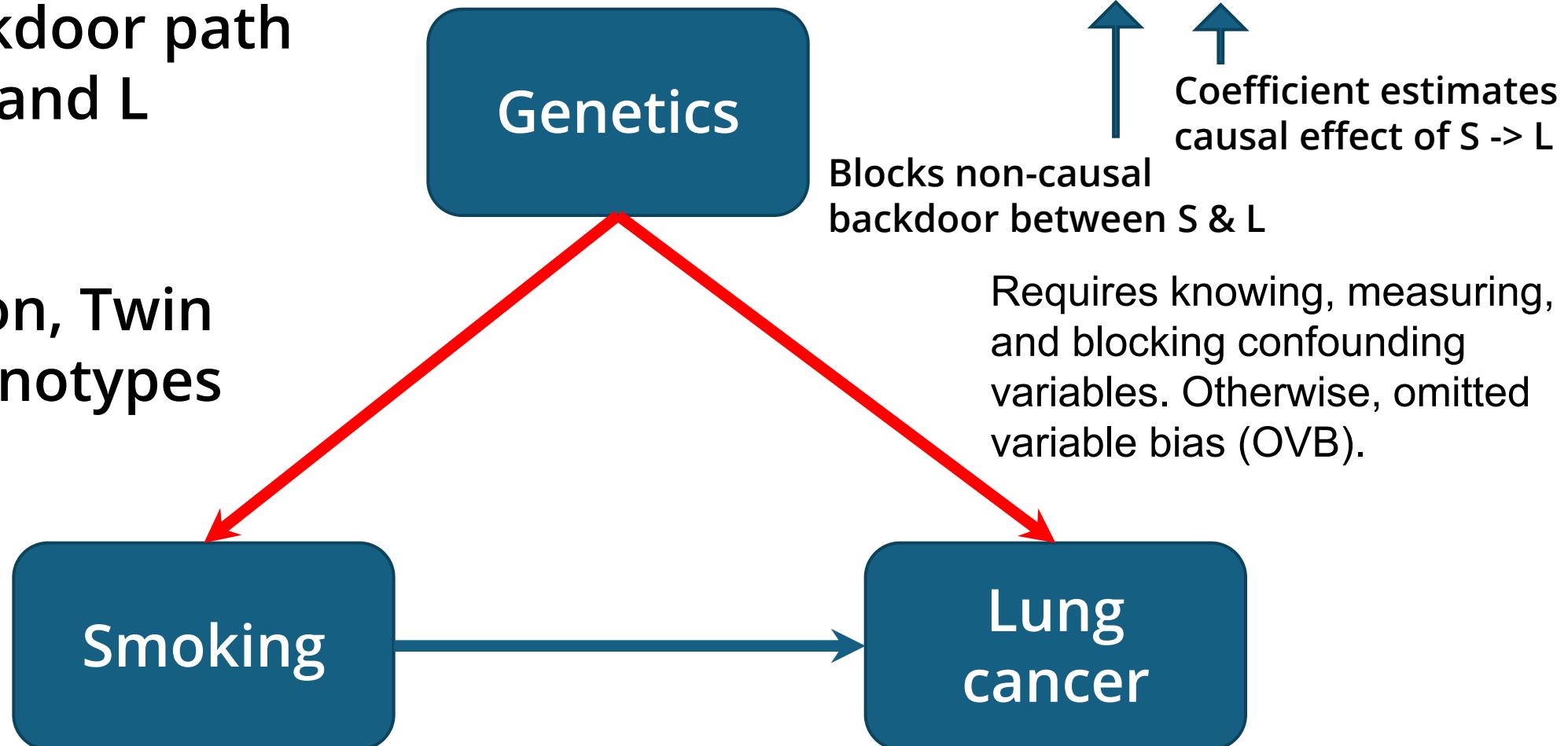
Backdoor adjustment

Conditioning on G

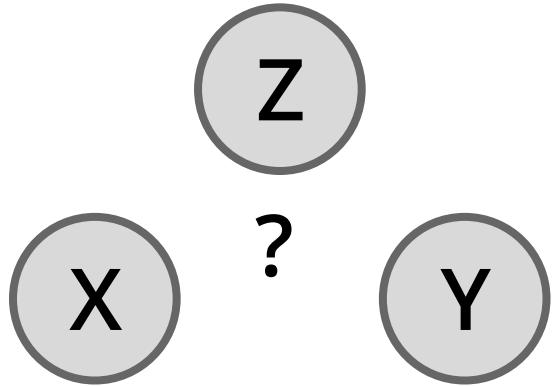
blocks backdoor path

between S and L

Population
stratification, Twin
studies, Genotypes
etc.

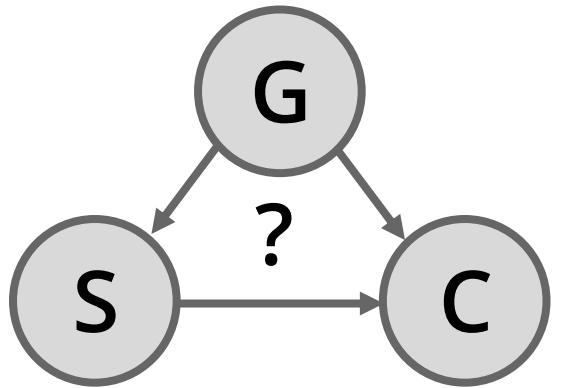


The “Elemental Confounds”



- Basic ways of how three variables can be causally connected
- Rules for how information flows reveal confounding dangers
- Larger DAGs can be deconstructed into these!

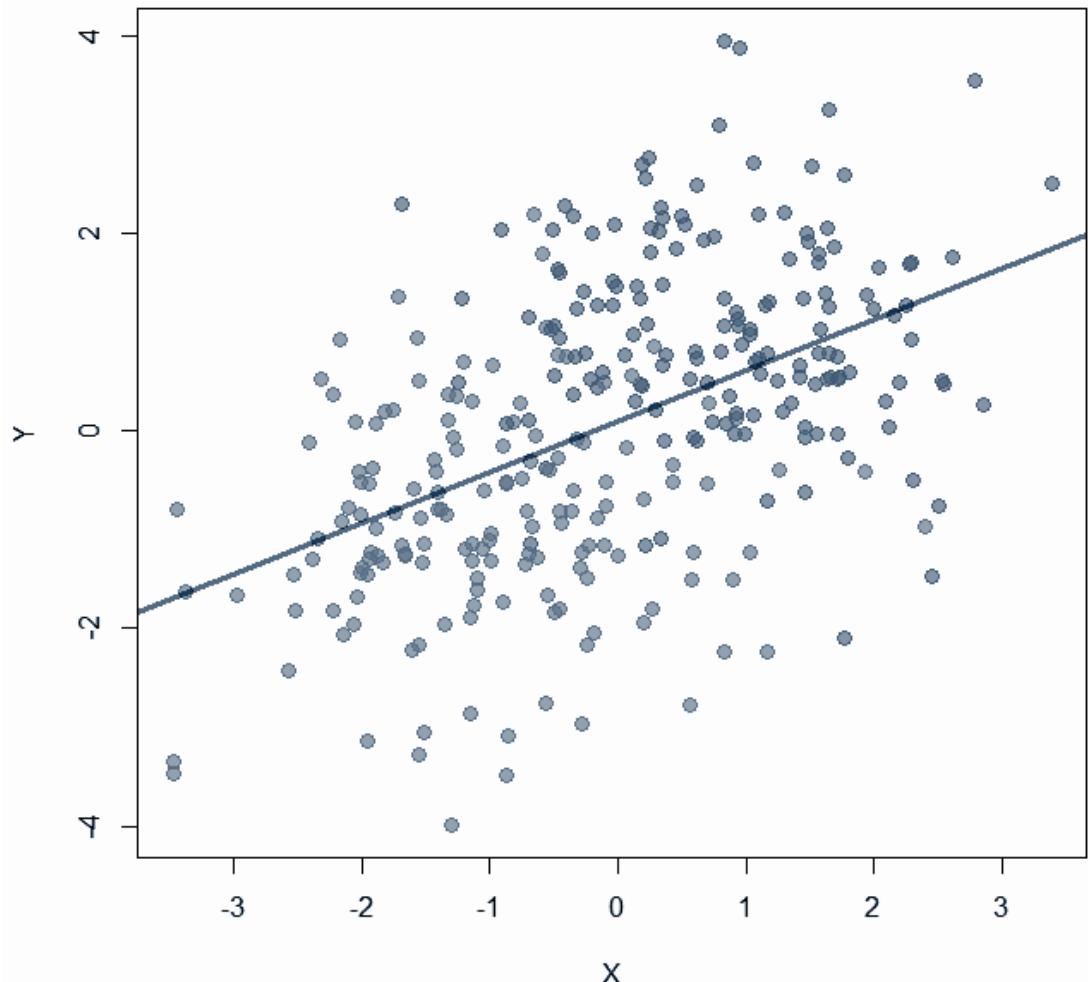
The “Elemental Confounds”



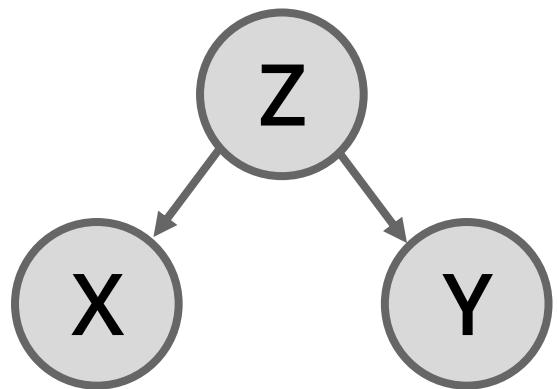
Example:

- Smoking (S) is correlated with cancer (C)
- We wonder whether genetics (G) could affect both

→ Should we include G in our regression?

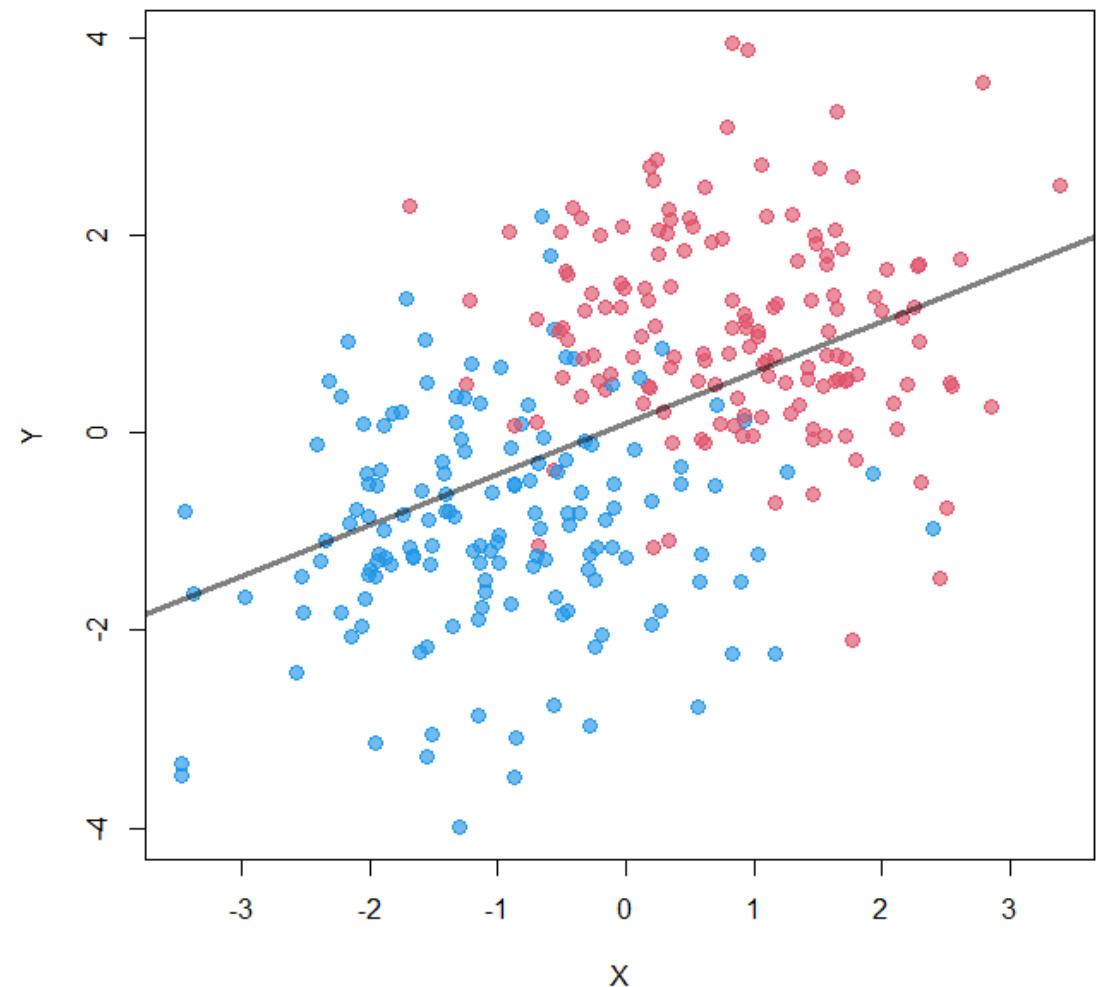


Fork: common cause as confounder

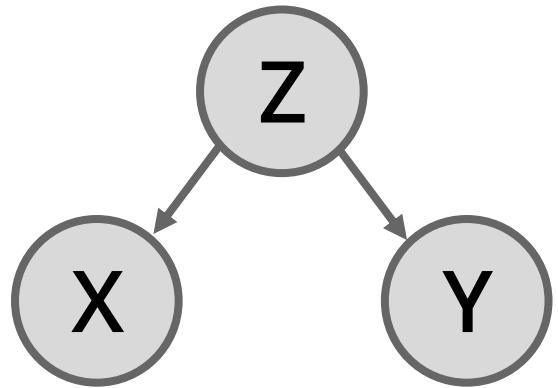


```
lm(formula = Y ~ X)
```

	Estimate	Std. Error	t value	Pr(> t)
(Intercept)	0.09541	0.07345	1.299	0.195
X	0.51315	0.05350	9.592	<2e-16 ***

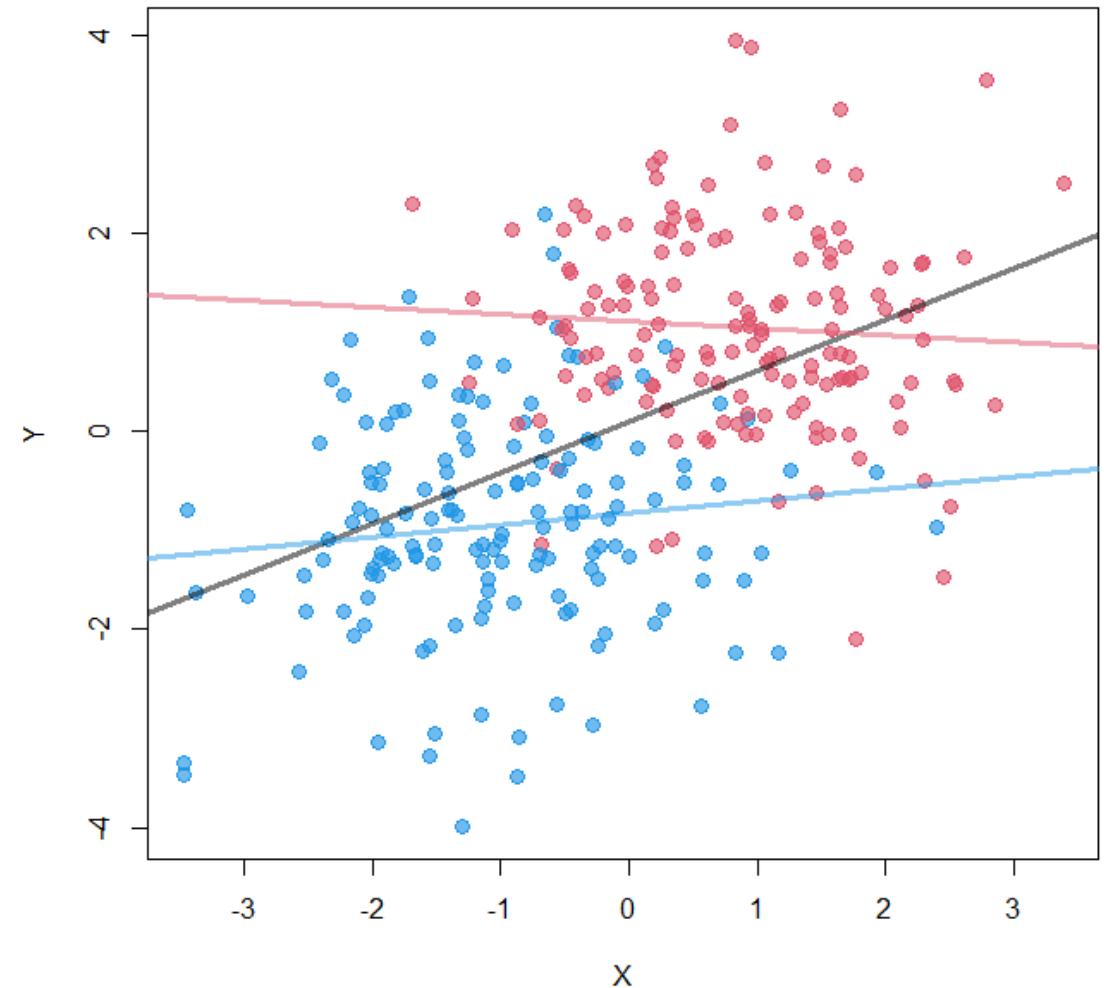


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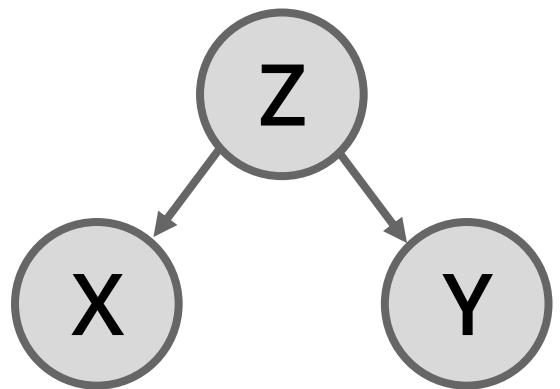


```
lm(formula = Y ~ X + Z)
```

	Estimate	Std. Error	t value	Pr(> t)
(Intercept)	-0.91314	0.10648	-8.576	5.53e-16 ***
X	0.03208	0.06091	0.527	0.599
Z	1.93154	0.16702	11.565	< 2e-16 ***

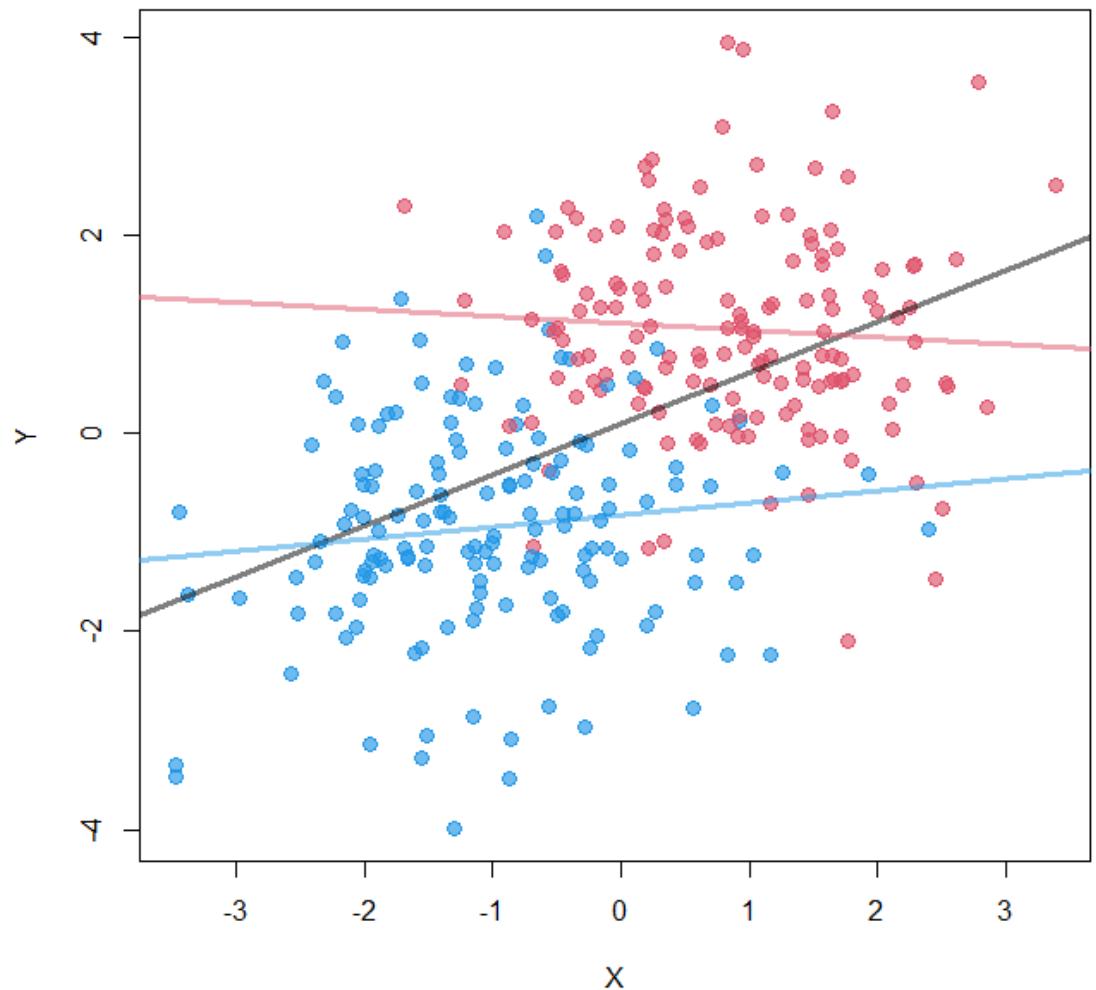


Fork: common cause as confounder

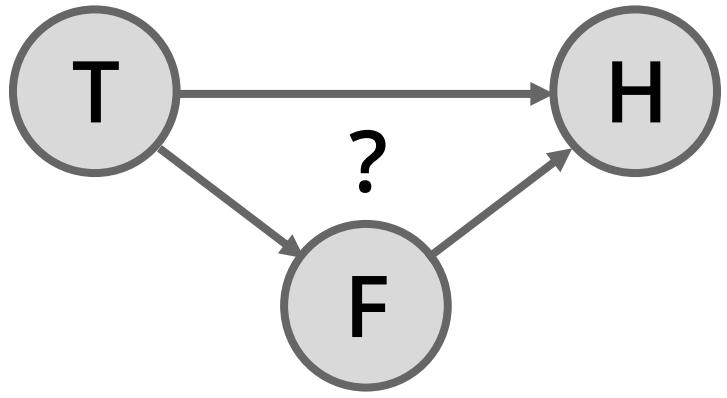


Missing confounder creates
spurious correlation!

	Estimate	Std. Error	t value	Pr(> t)
(Intercept)	-0.91314	0.10648	-8.576	5.53e-16 ***
X	0.03208	0.06091	0.527	0.599
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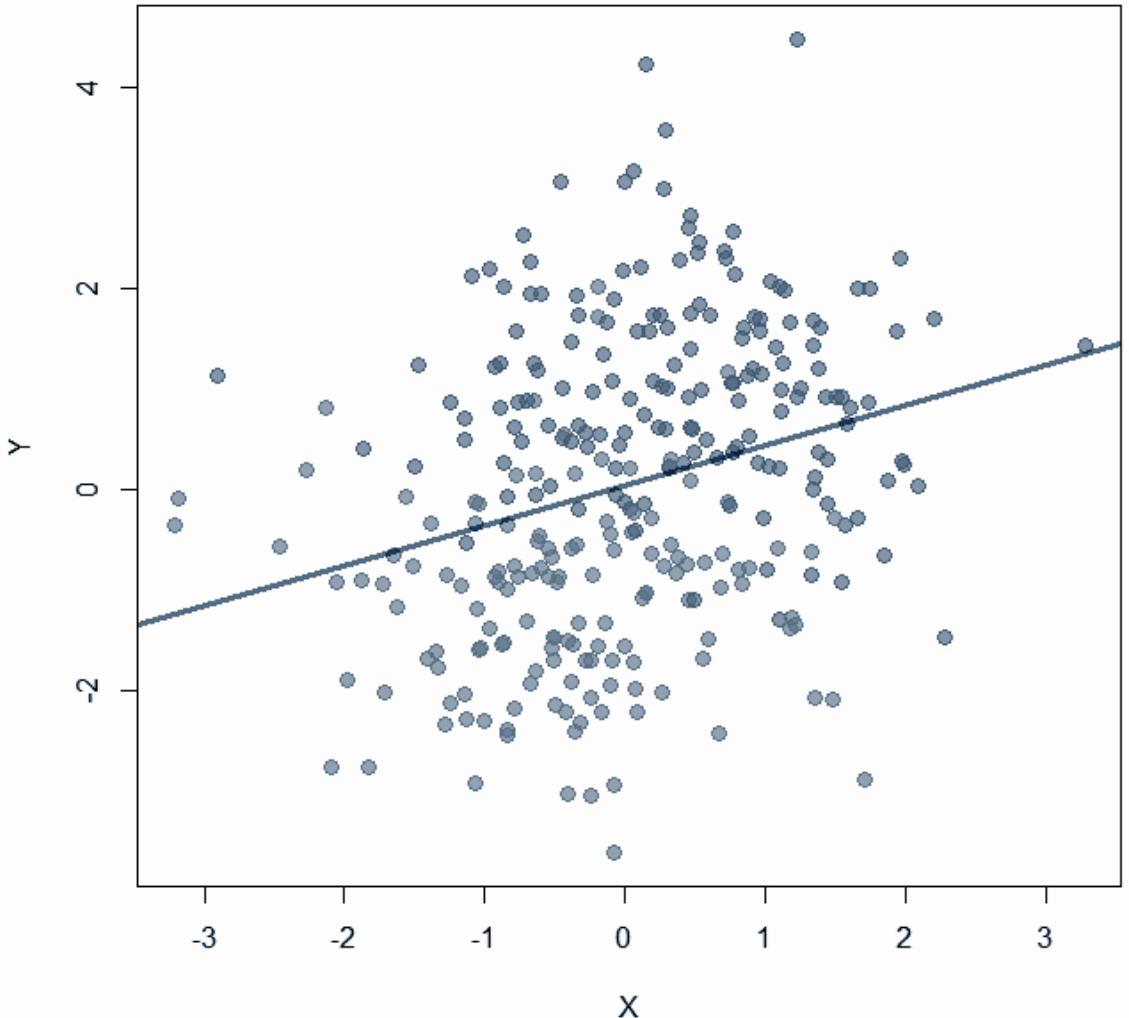
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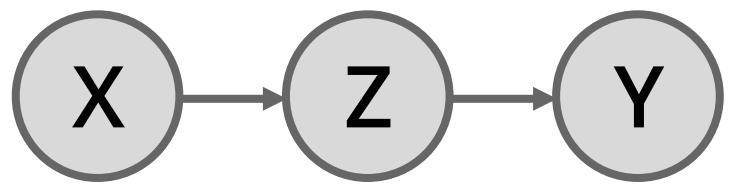
Example:

- Fungus (F) affects plant height (H)
- Experiment to test whether treatment (T) works

→ Should we include F in our regression?



Pipe: mediator

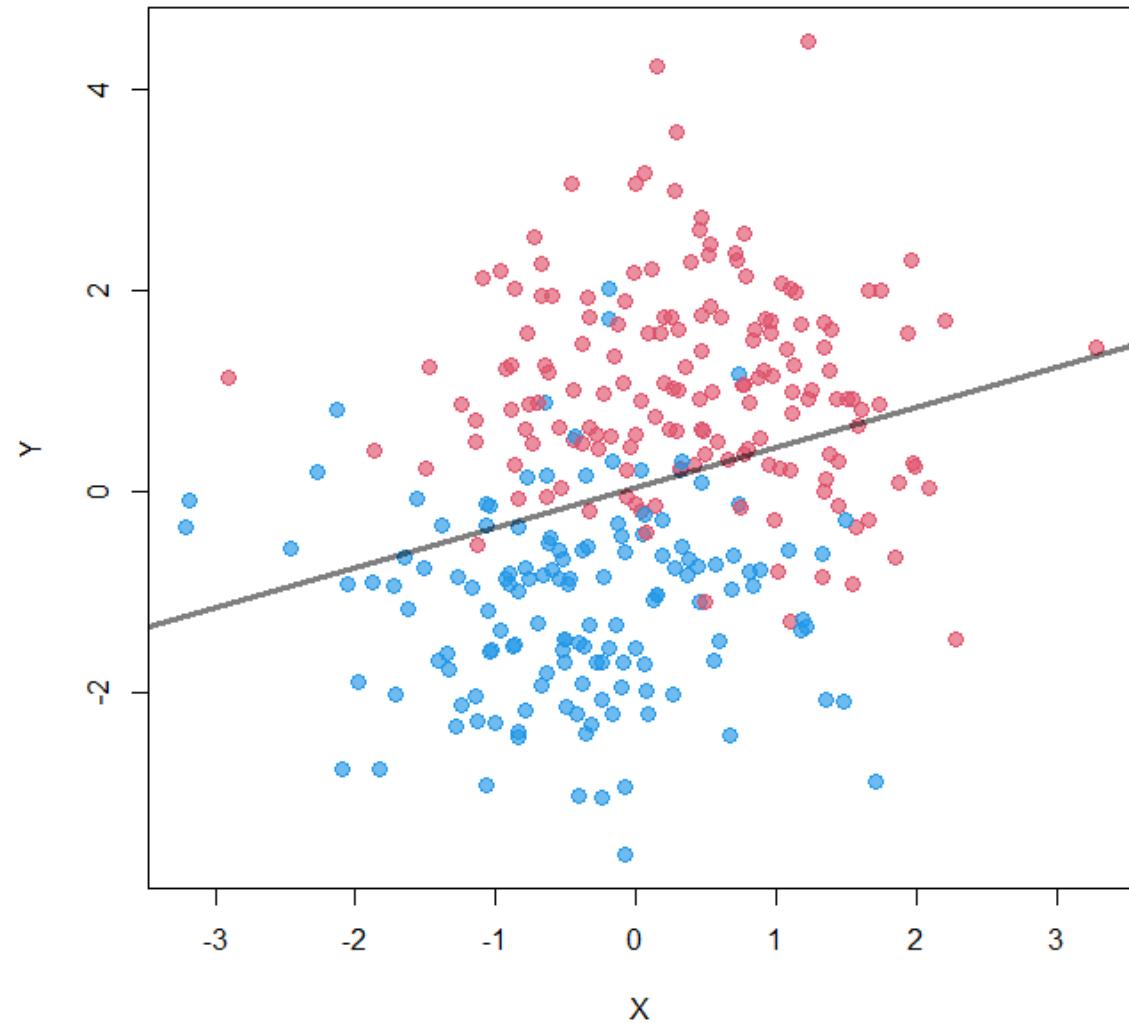


```
lm(formula = Y ~ X)
```

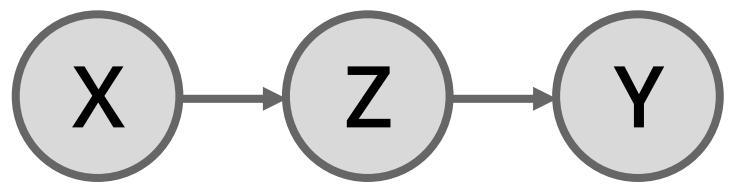
	Estimate	Std. Error	t value	Pr(> t)
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(Intercept)	0.04652	0.08174	0.569	0.57
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X	0.39762	0.08046	4.942	1.29e-06 ***
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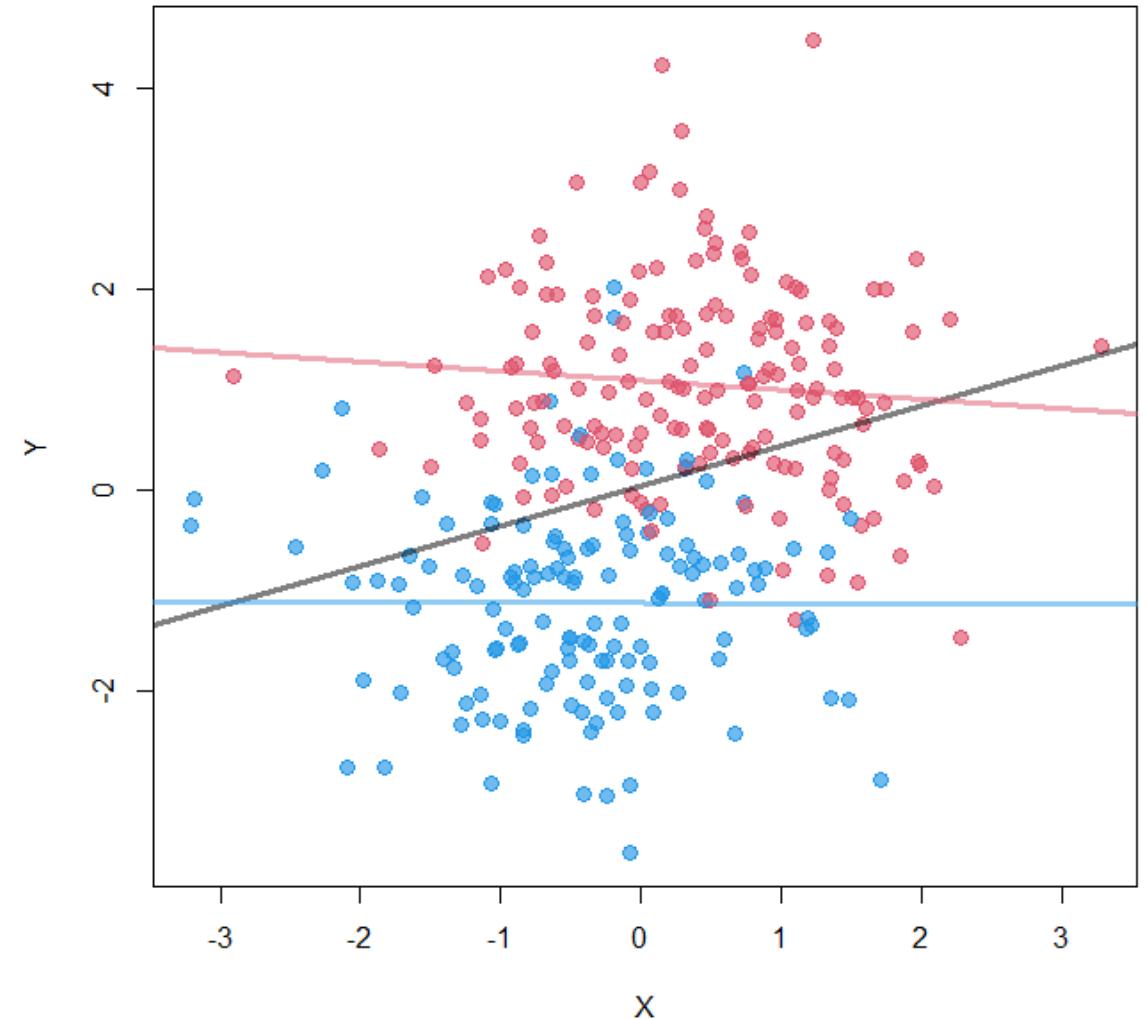


Pipe: mediator

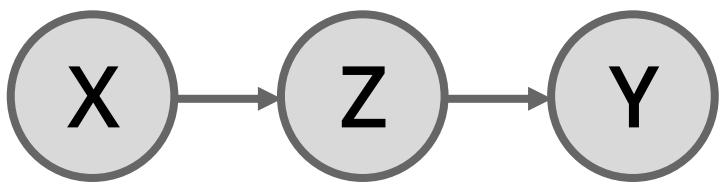


```
lm(formula = Y ~ X + Z)
```

	Estimate	Std. Error	t value	Pr(> t)
(Intercept)	-1.13684	0.08815	-12.897	<2e-16 ***
X	-0.05336	0.06185	-0.863	0.389
Z	2.22261	0.12596	17.645	<2e-16 ***

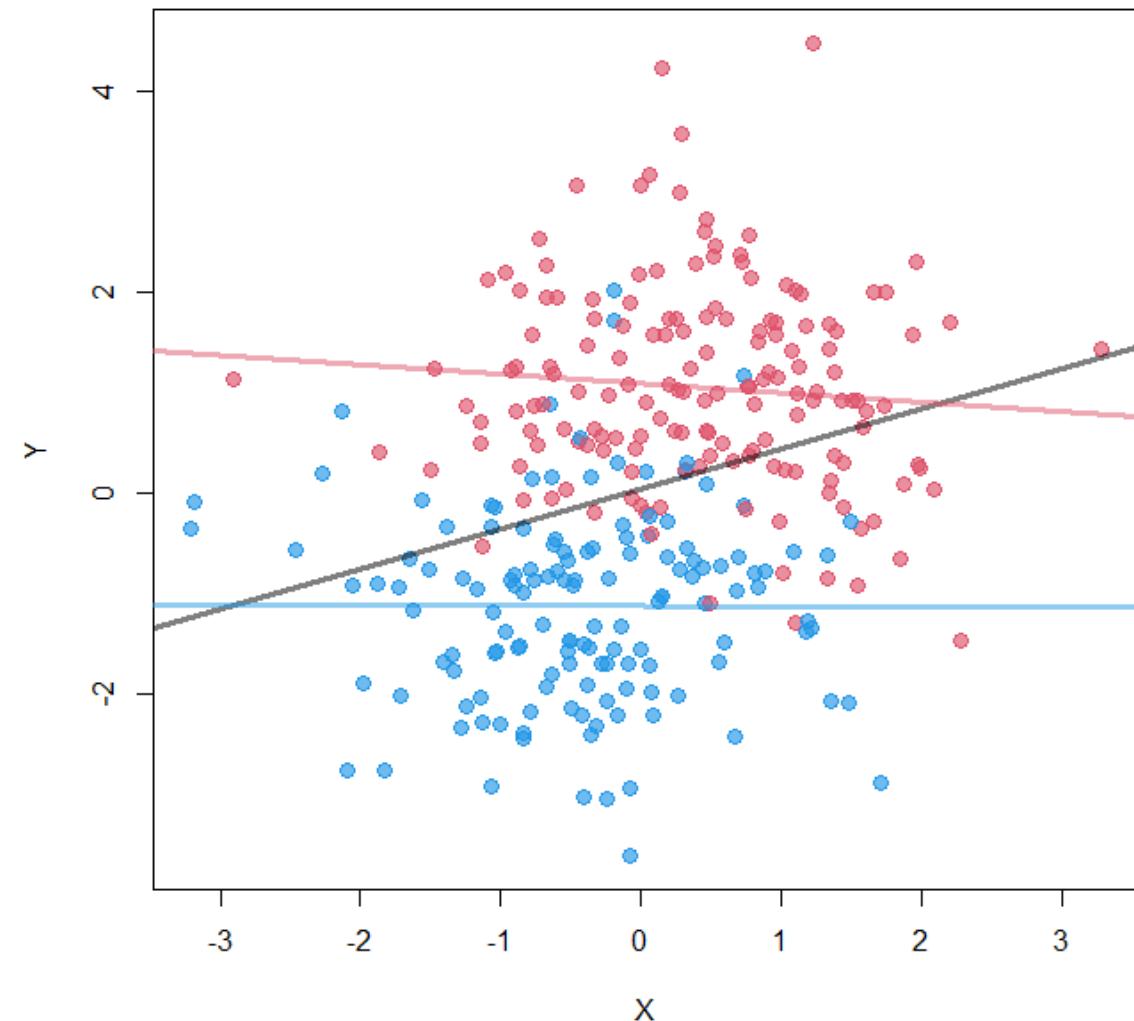


Pipe: mediator

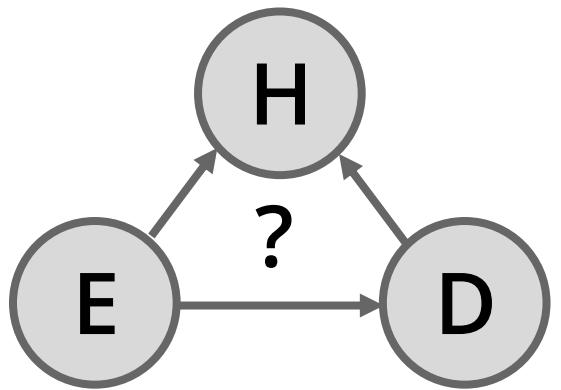


Blocking effects
(Post-treatment bias)

	Estimate	Std. Error	t value	Pr(> t)
(Intercept)	-1.13684	0.08815	-12.897	<2e-16 ***
X	-0.05336	0.06185	-0.863	0.389
Z	2.22261	0.12596	17.645	<2e-16 ***



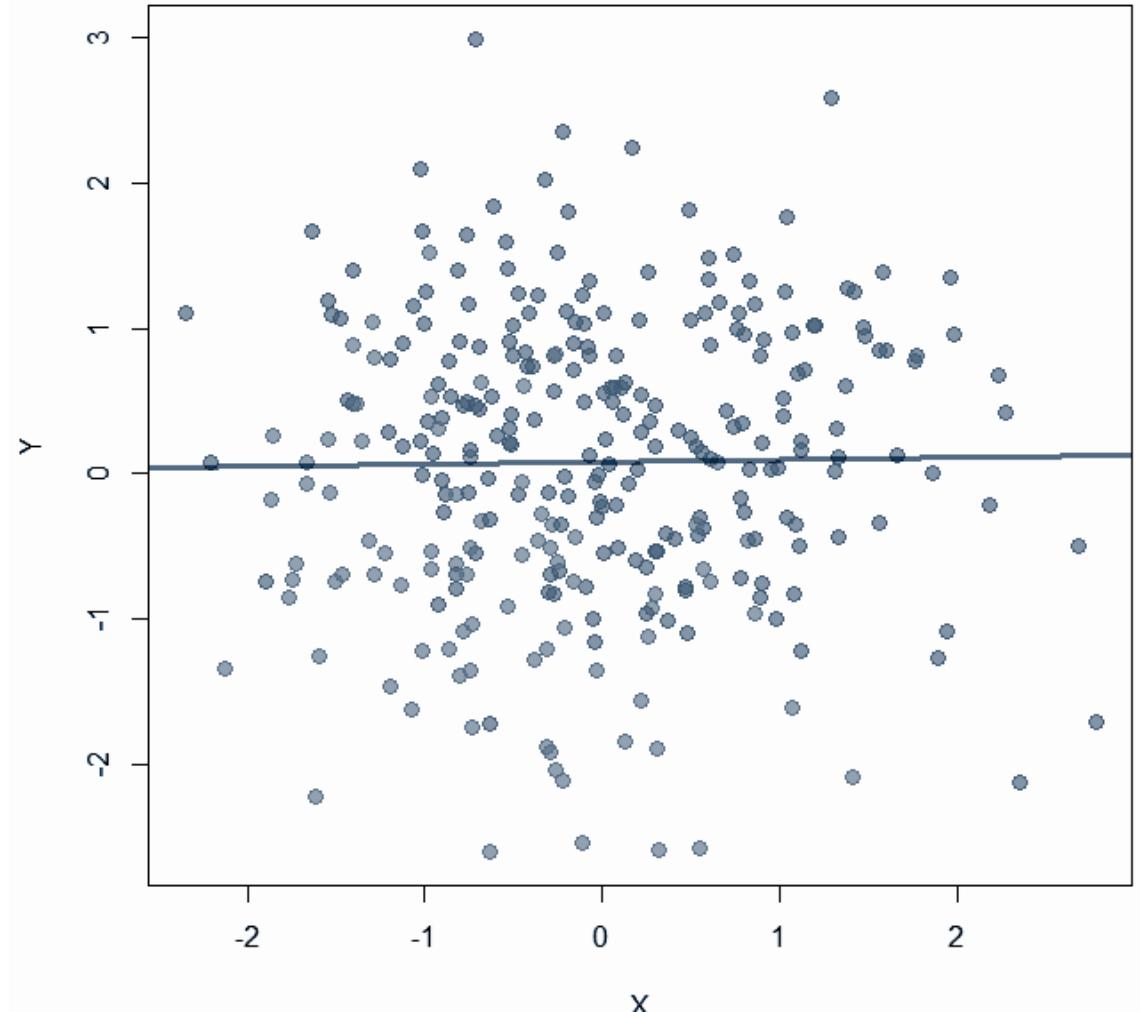
The “Elemental Confounds”



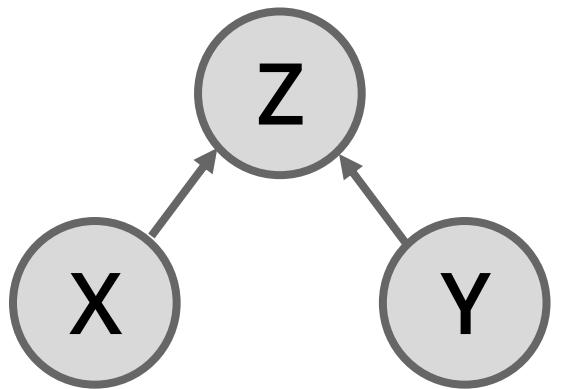
Example:

- Studying health effects of exercise (E) on diet (D)
- Hospitalisation (H) as related proxy for health

→ Should we include H in our regression?

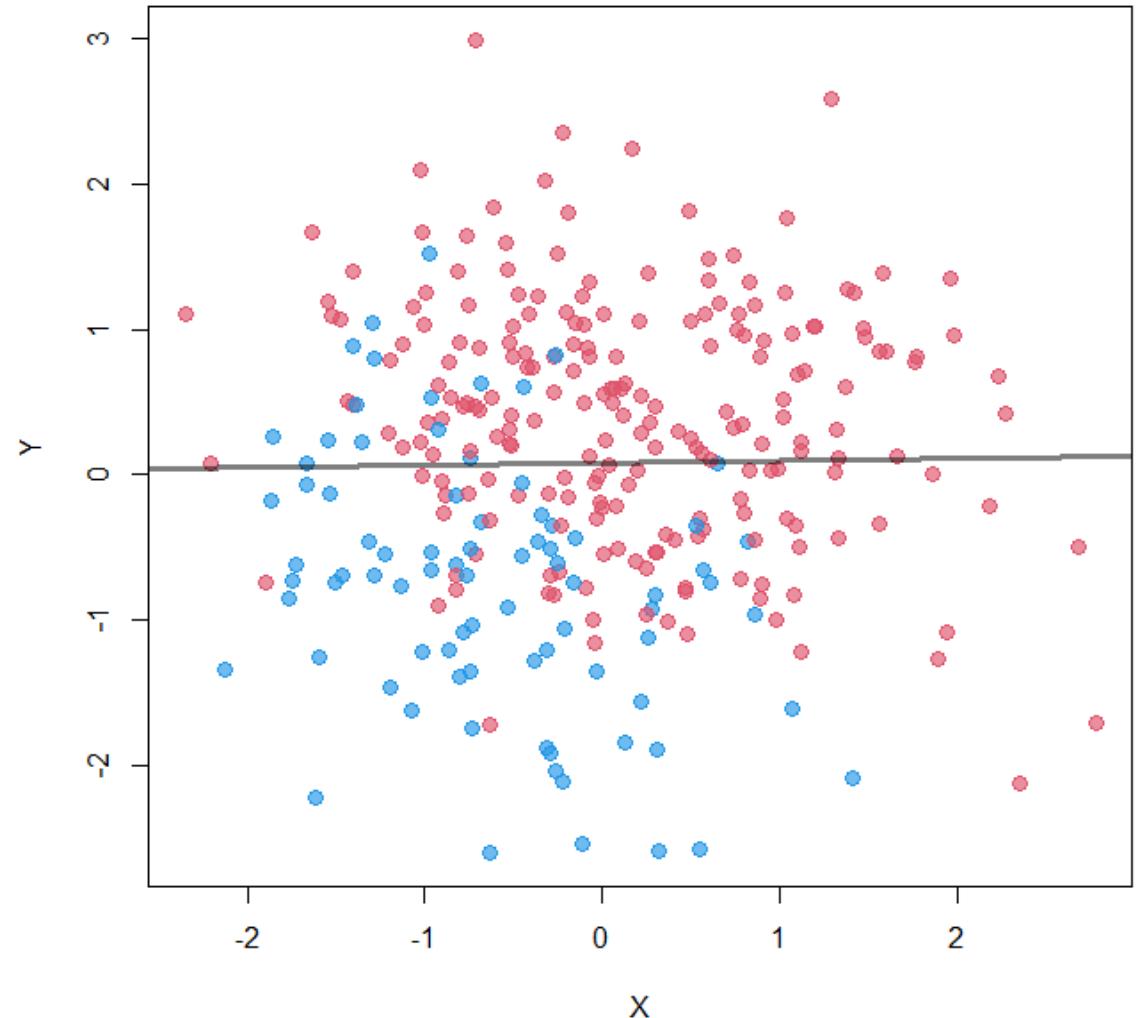


Collider: common effect

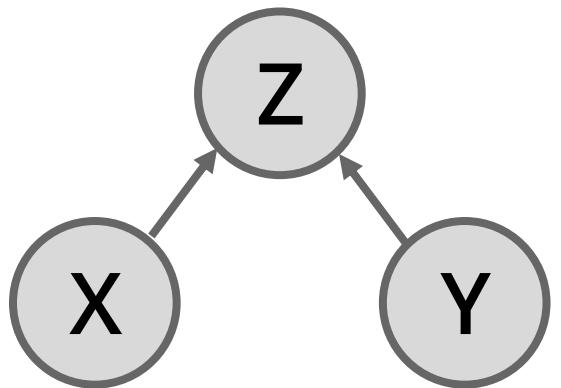


```
lm(formula = Y ~ X)
```

	Estimate	Std. Error	t value	Pr(> t)
(Intercept)	0.08051	0.05753	1.399	0.163
X	0.01505	0.05947	0.253	0.800



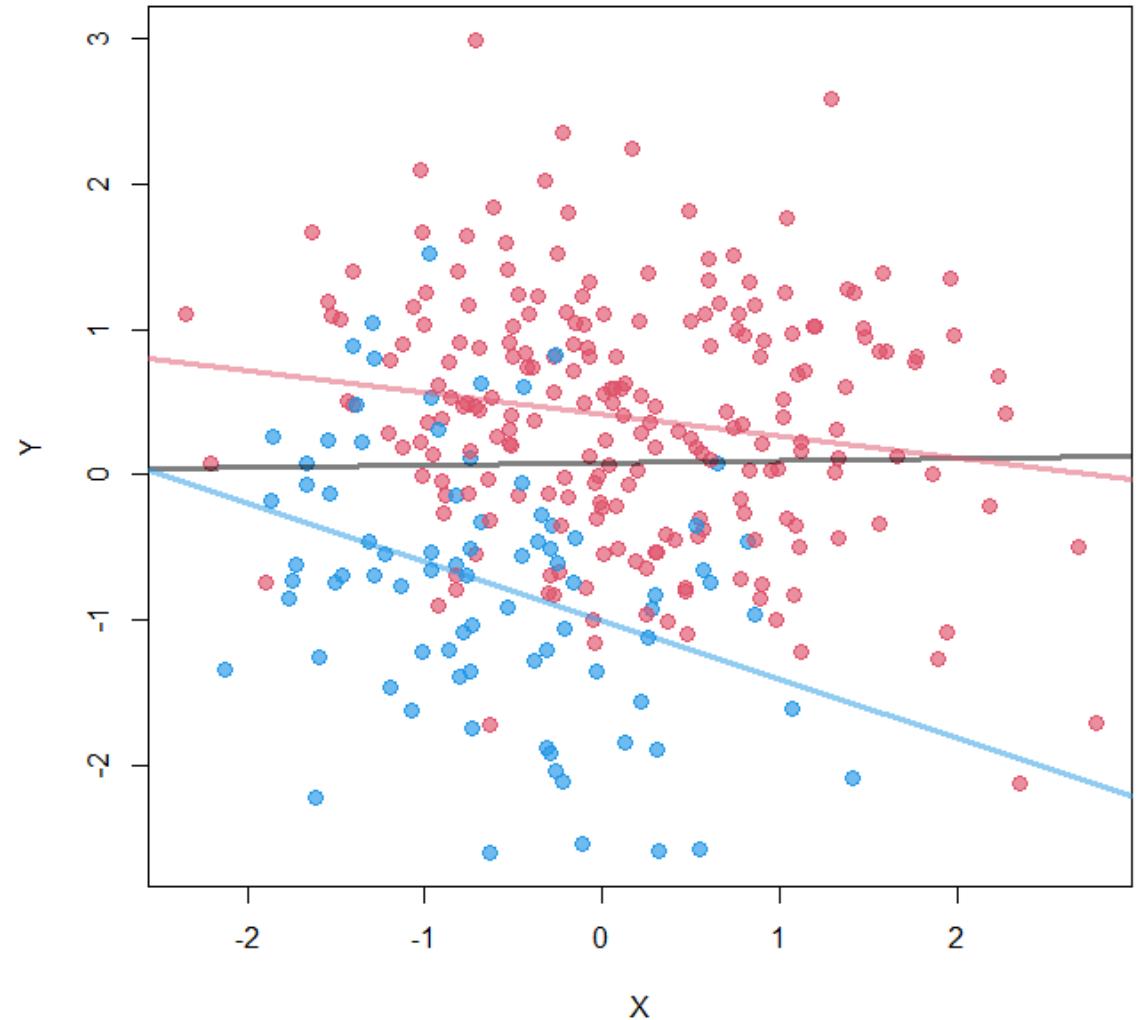
Collider: common effect



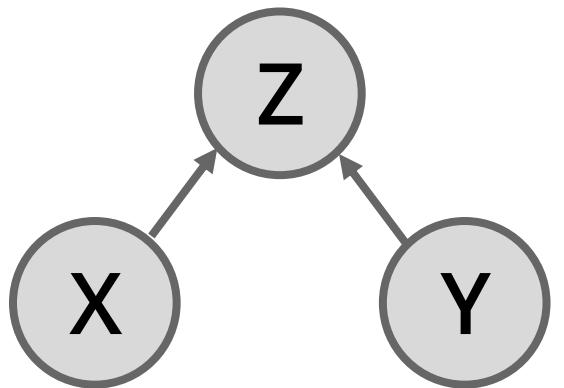
```
lm(formula = Y ~ X + Z)
```

	Estimate	Std. Error	t value	Pr(> t)
--	----------	------------	---------	----------

(Intercept)	-0.87400	0.09826	-8.894	< 2e-16 ***
X	-0.20154	0.05363	-3.758	0.000206 ***
Z	1.29728	0.11625	11.160	< 2e-16 ***

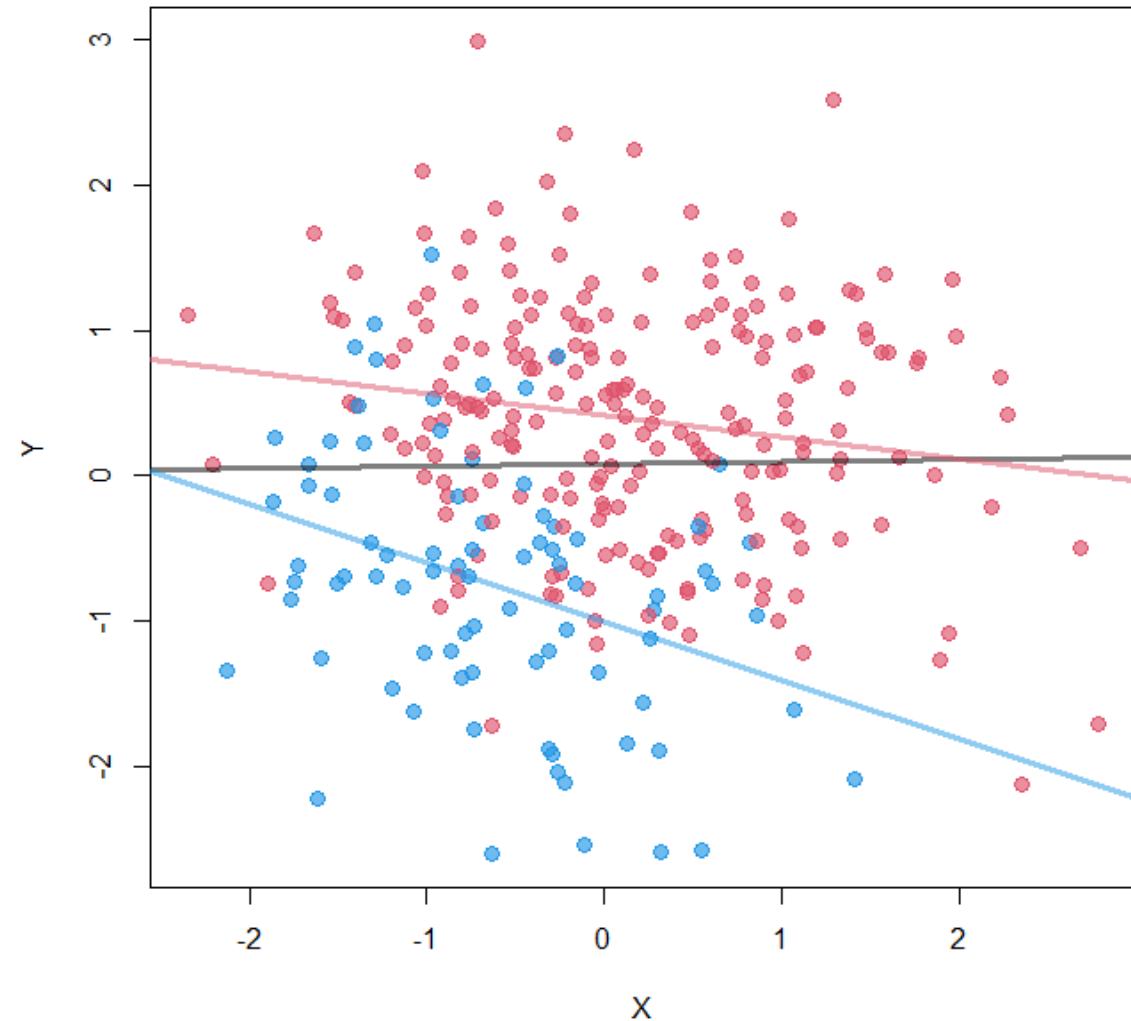


Collider: common effect

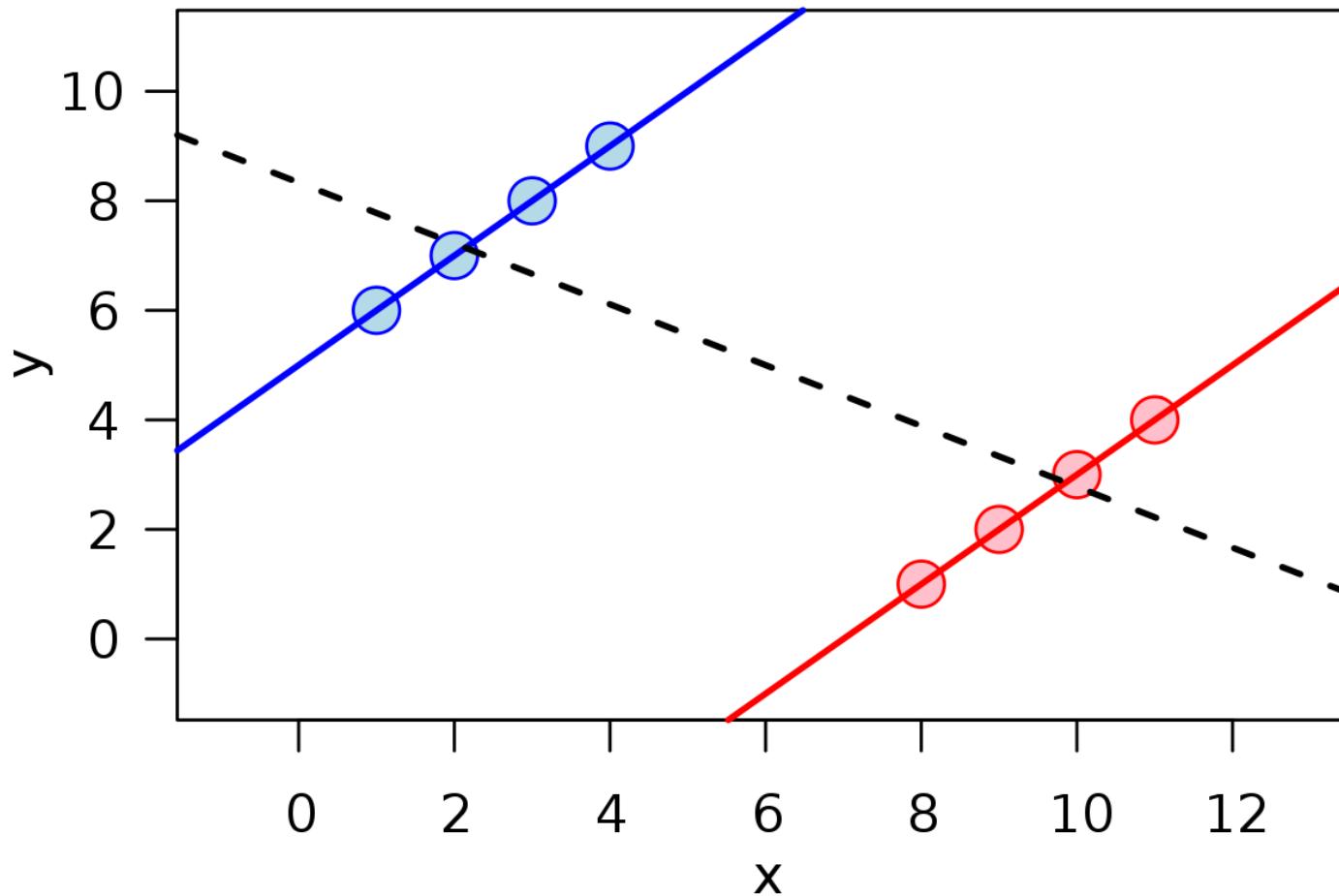


Sampling bias /
Bad (endogenous) controls

	Estimate	Std. Error	t value	Pr(> t)	
(Intercept)	-0.87400	0.09826	-8.894	< 2e-16 ***	
X	-0.20154	0.05363	-3.758	0.000206 ***	
Z	1.29728	0.11625	11.160	< 2e-16 ***	



Simpson's paradox



Simpson's paradox

Male recovery rate

Drug: 60% (18/30)

No drug: 70% (7/10)

Female recovery rate

Drug: 20% (2/10)

No Drug: 30% (9/30)

Combined

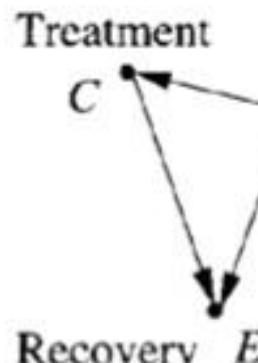
recovery rate:

Drug: 50% (20/40)

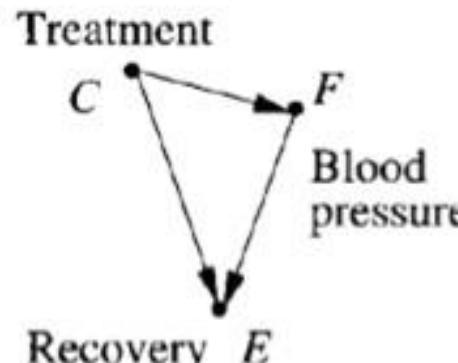
No drug: 40% (16/40)

Causal interpretations

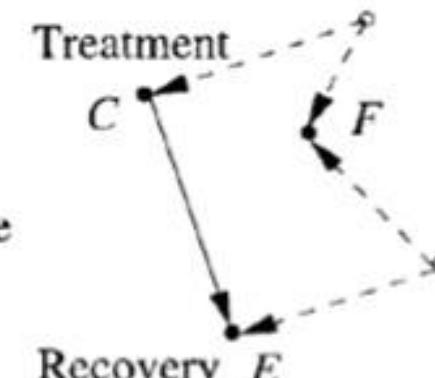
Three causal models capable of generating the data Model (a) dictates use of the **gender-specific tables**, whereas (b) and (c) dictate use of the **combined table**.



(a)



(b)



(c)

Major takeaways of Causal Inference

Prediction vs. Causal Inference

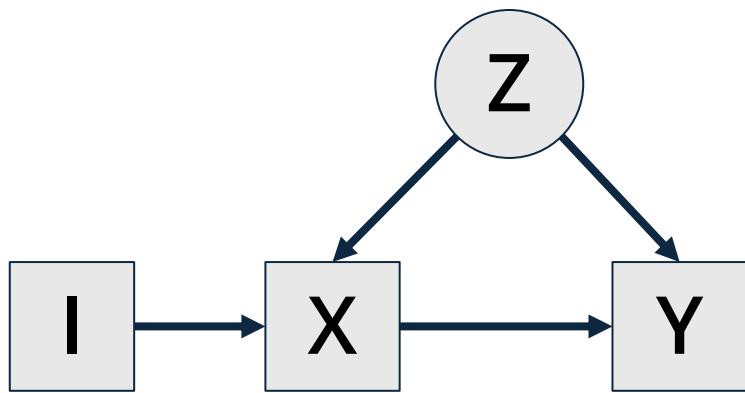
- Including all covariates that improve prediction (e.g. AIC model selection)
- Including covariates justified by the causal hypothesis

What we can do:

- DAGs are hypotheses of generating processes
- Makes causal assumptions explicit & justifies estimation/identification strategy
if the DAG were correct (e.g. Backdoor adjustment)
 - Doesn't solve "cor \neq cause", but CAN REJECT implausible causal hypotheses
 - Leaves *plausible* causal hypotheses & how they could be distinguished
 - More identification strategies available than just Backdoor adjustment
 - (e.g. frontdoor adjustment, instrumental variables)

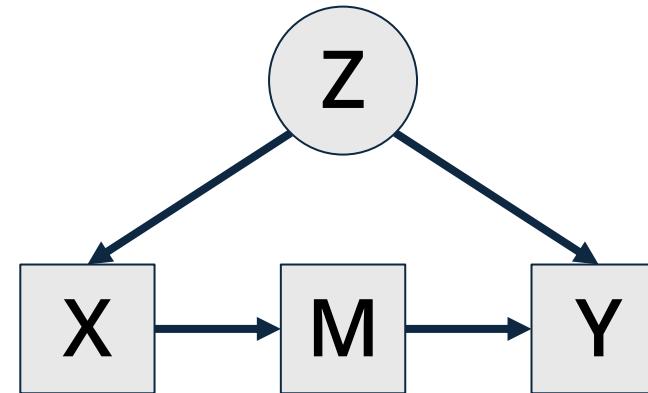
Other options besides backdoor adjustments in Causal inference

Instrumental variables



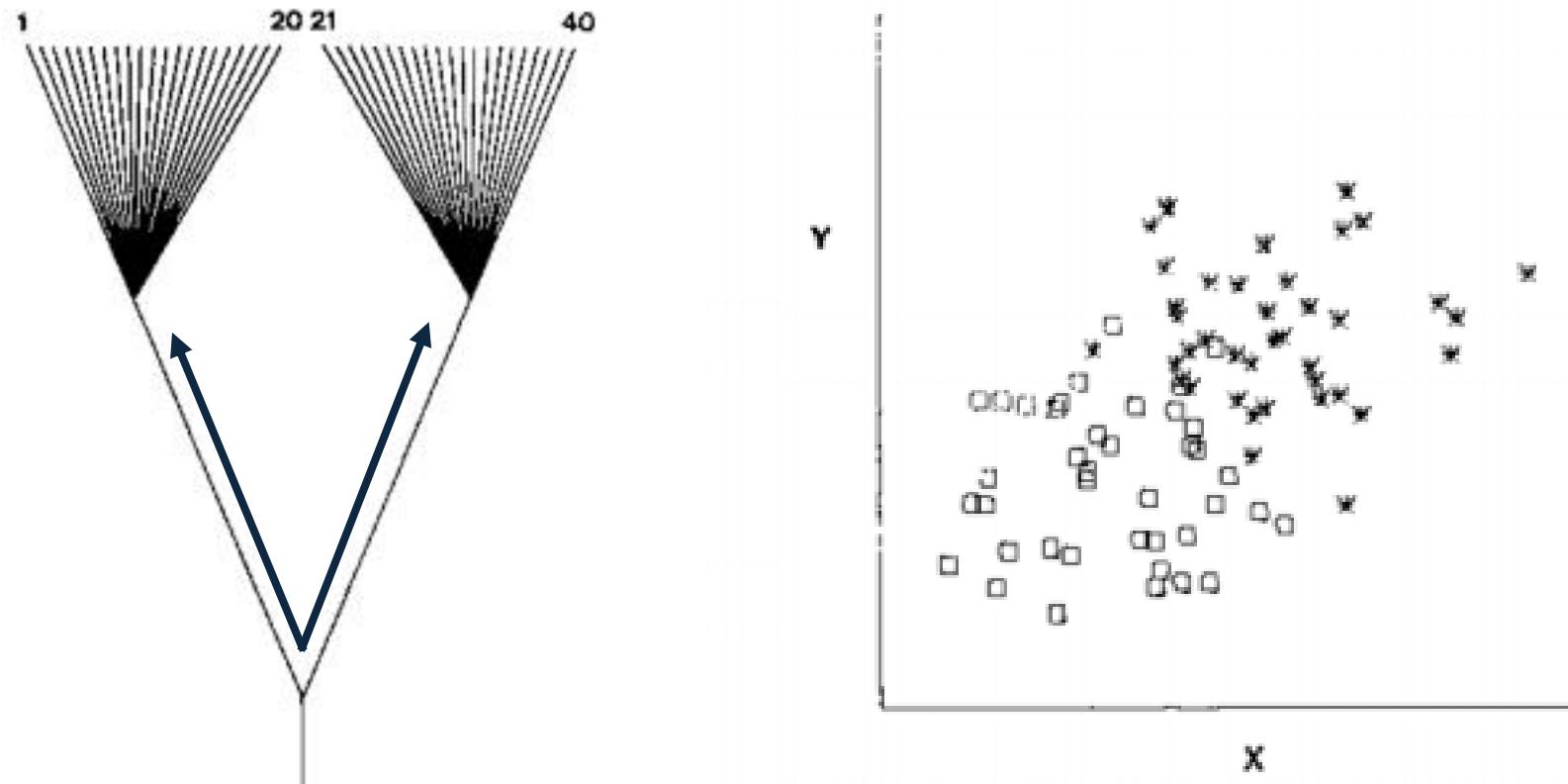
IV: A 3rd variable (I) that only affects Y through its effect on X. (e.g. Tax rate on smoking products)

Front door adjustment

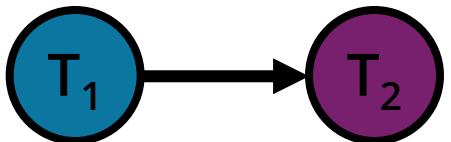
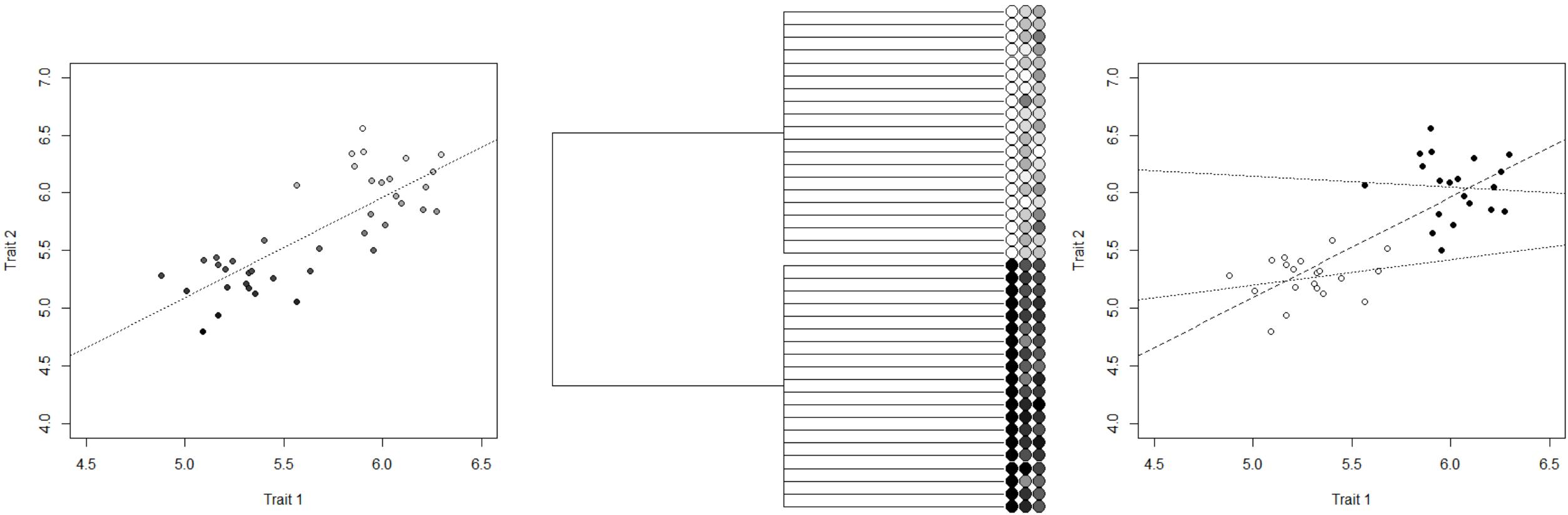


FDA: A 3rd variable (M) that mediates the effect of X on Y. (e.g. Tar levels in lungs mediating the effect of smoking on lung cancer)

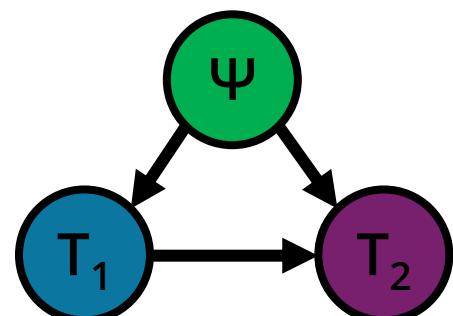
Application to phylogenetic comparative methods



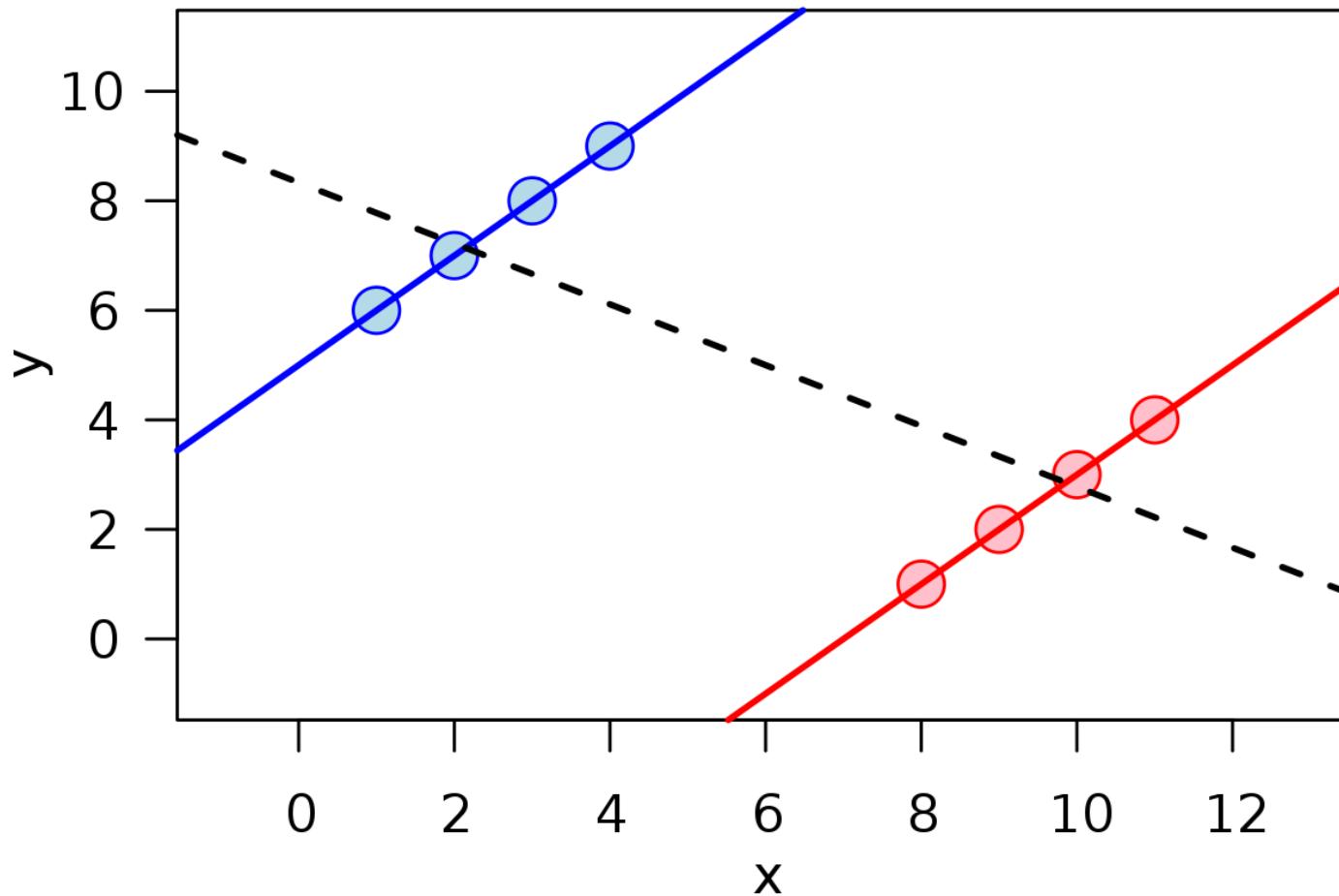
The Felsenstein Scenario

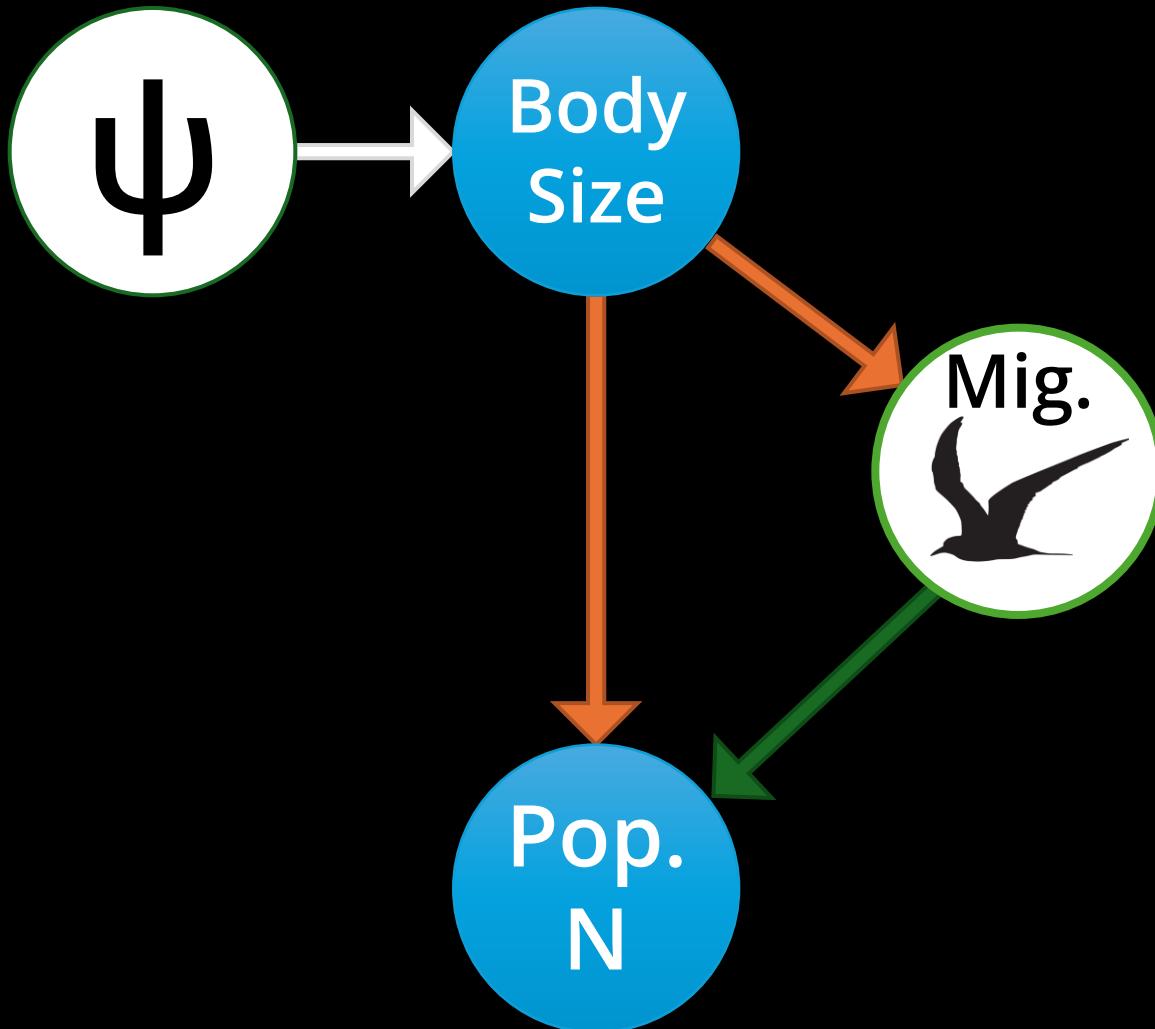


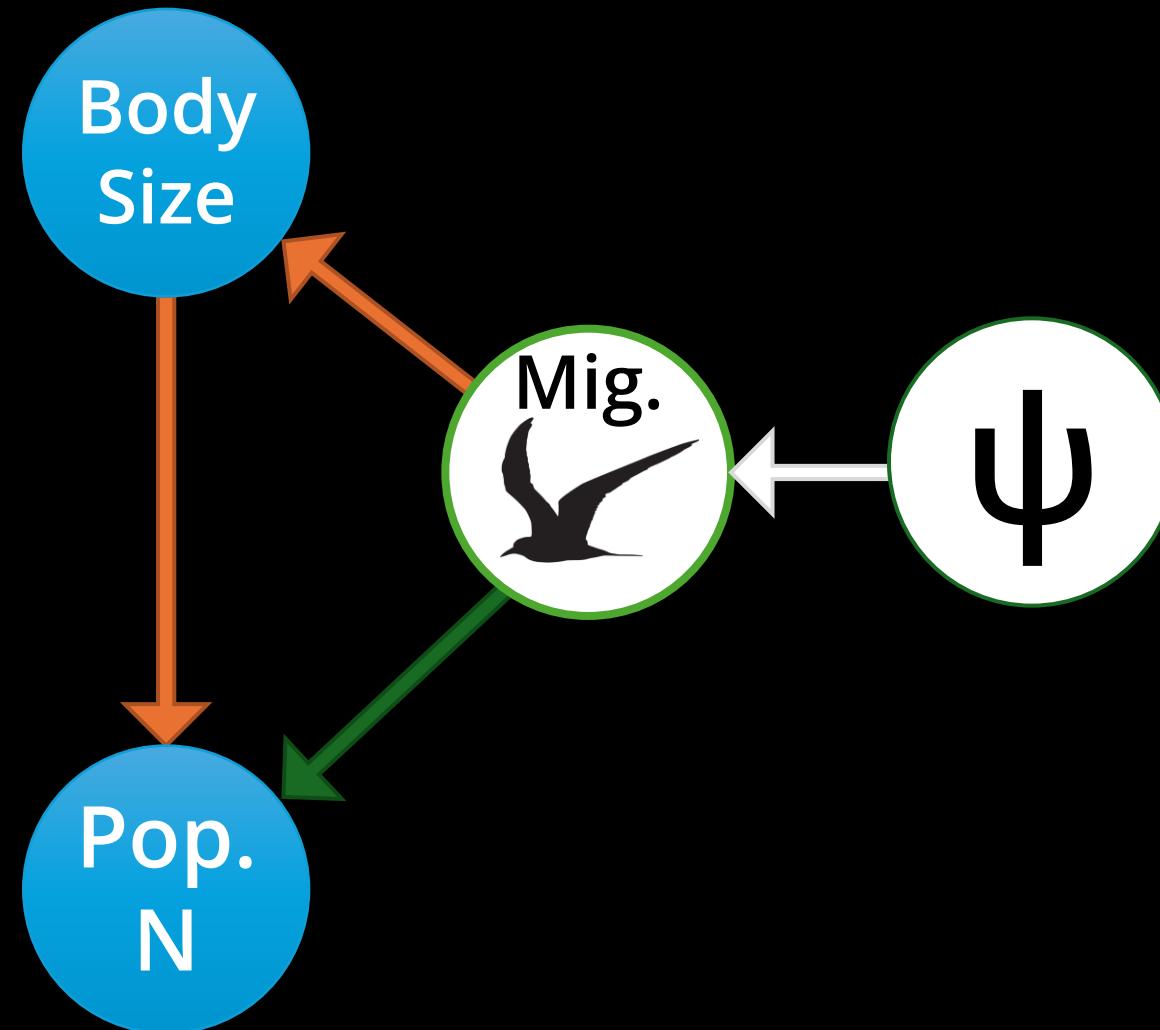
Spurious correlation due to
confounding effect of phylogeny
(endogenous causal variable)

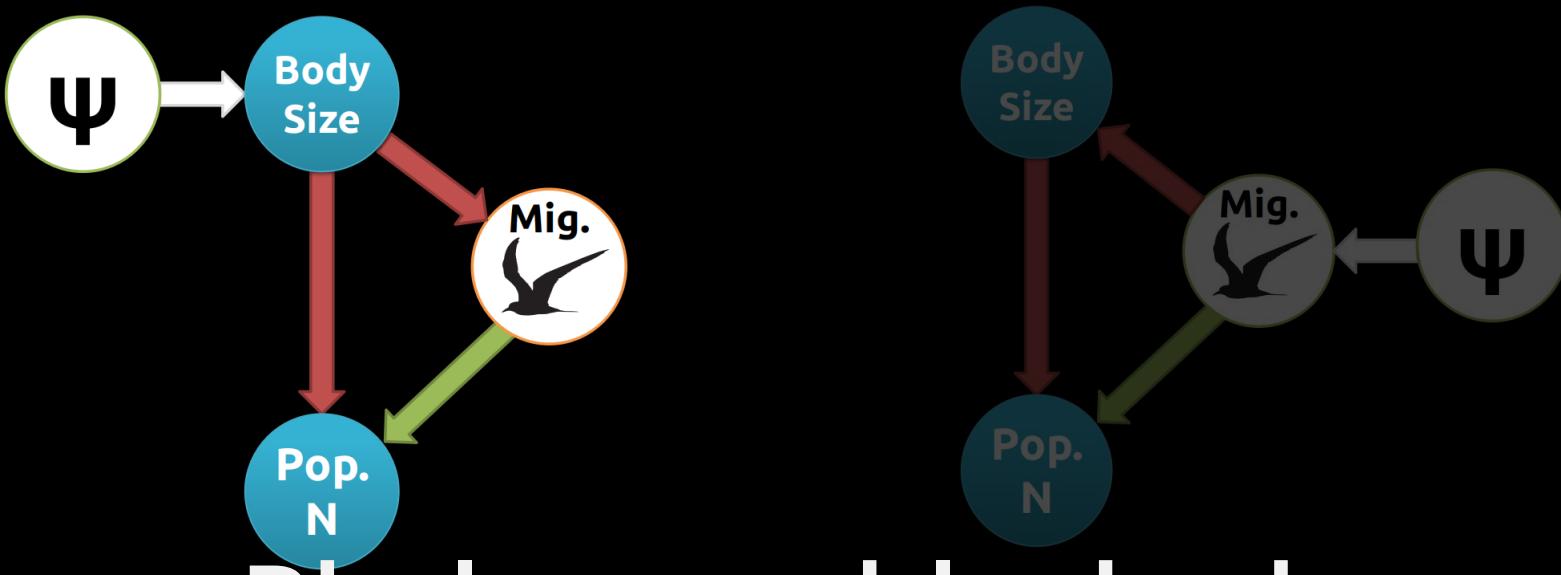


Simpson's paradox

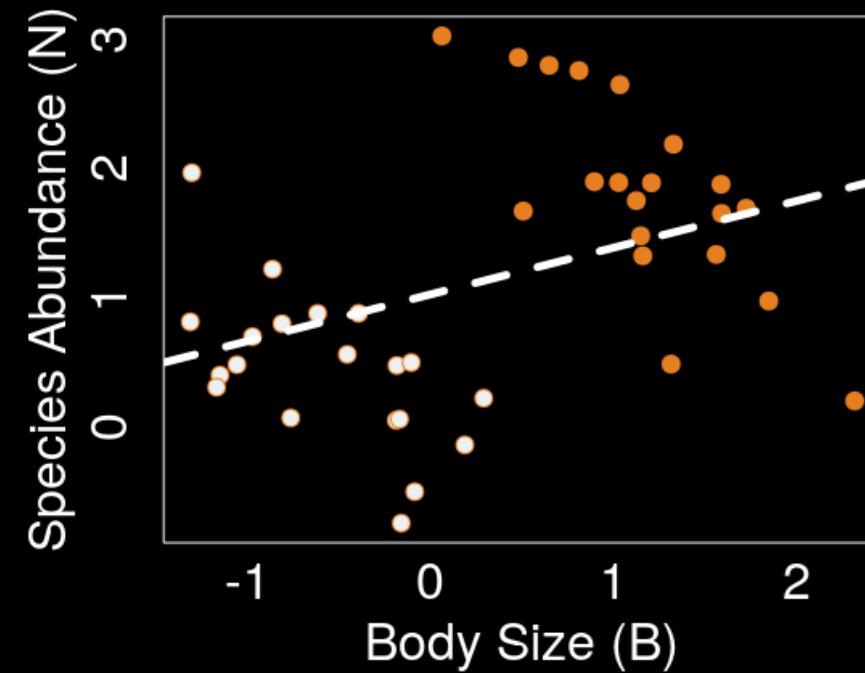
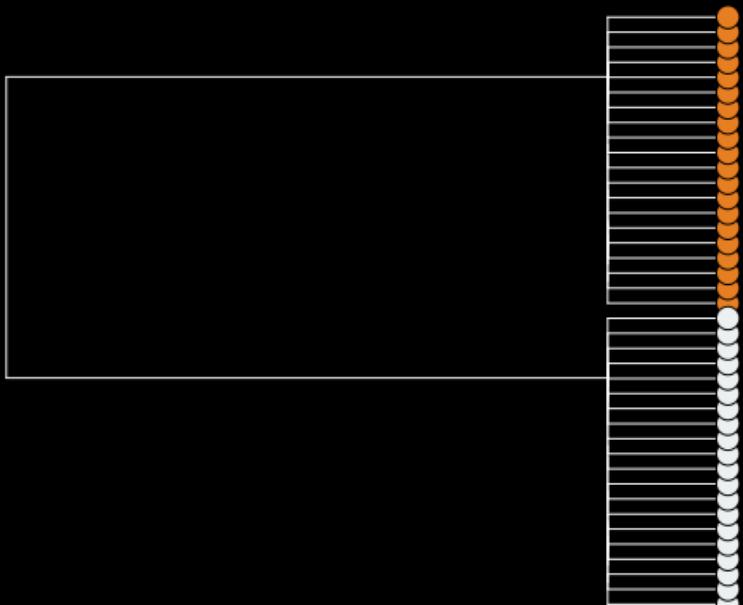


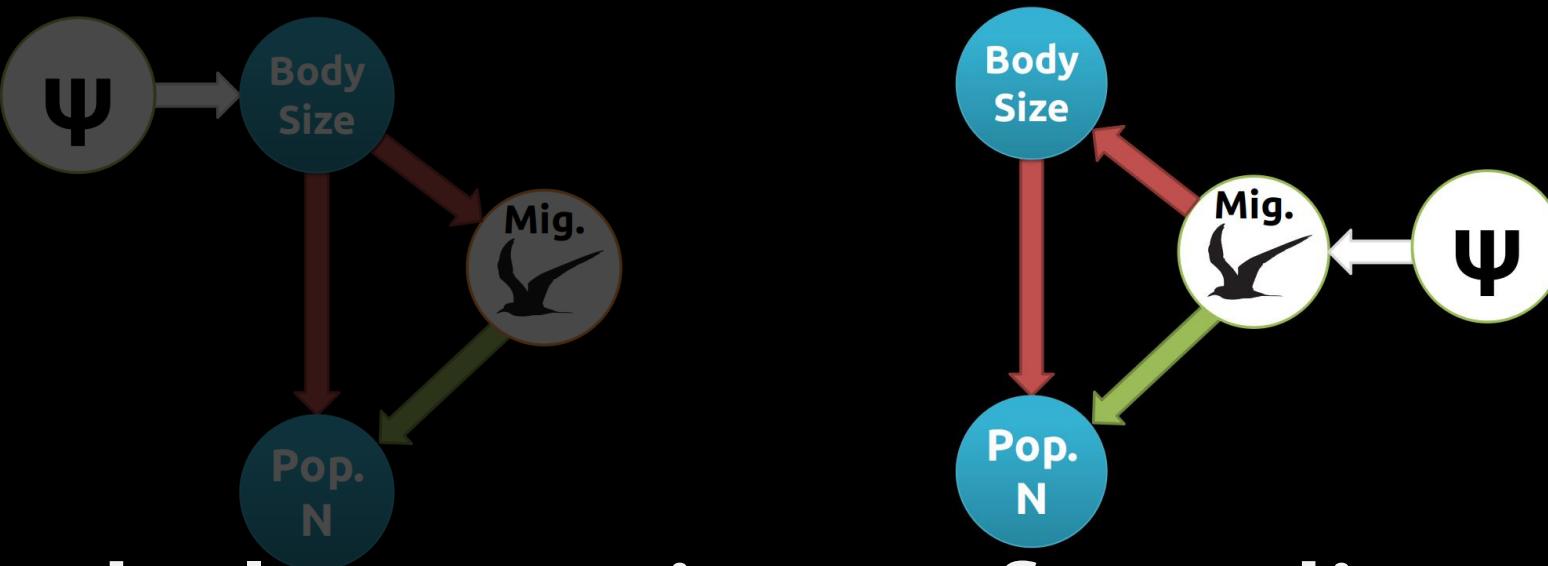




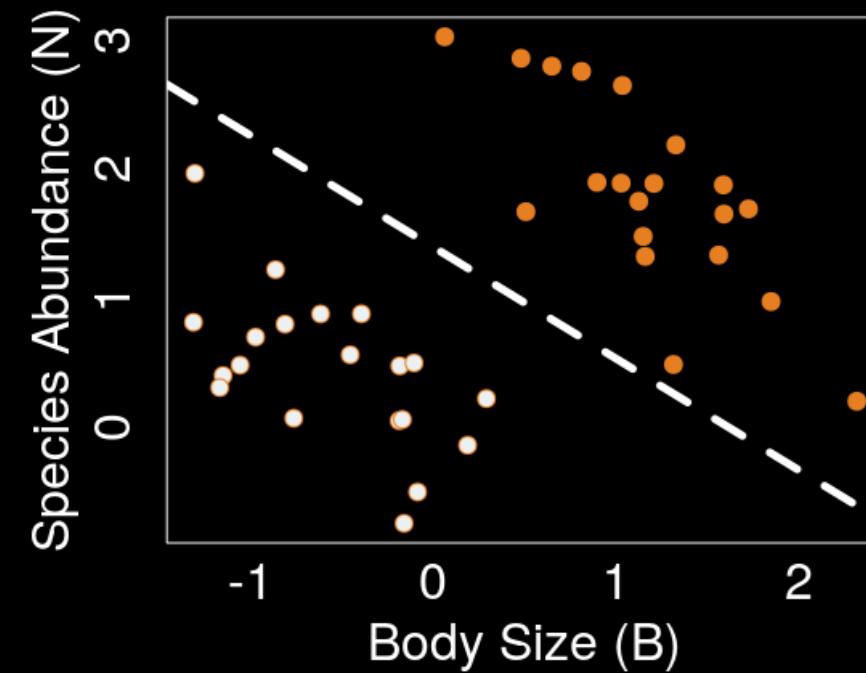
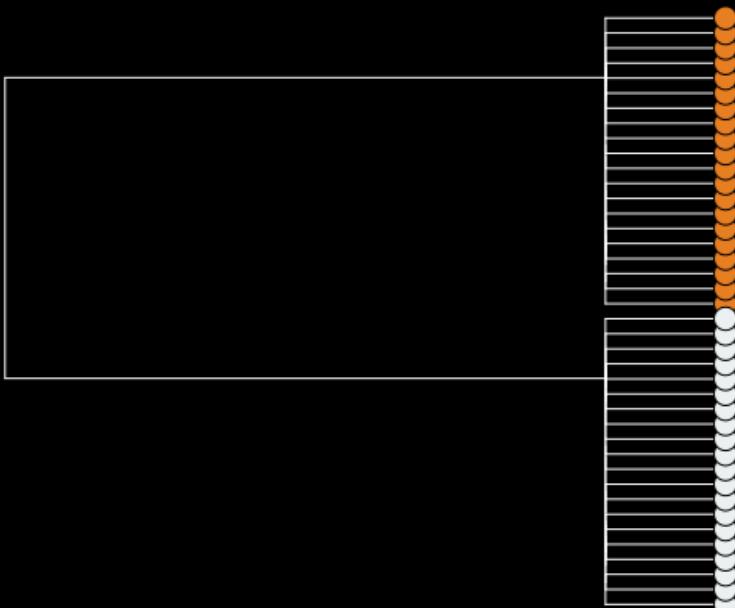


Phylogeny blocked

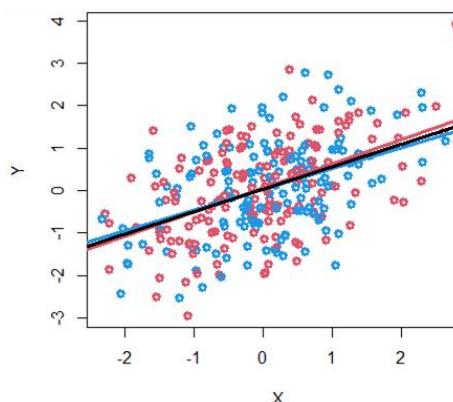
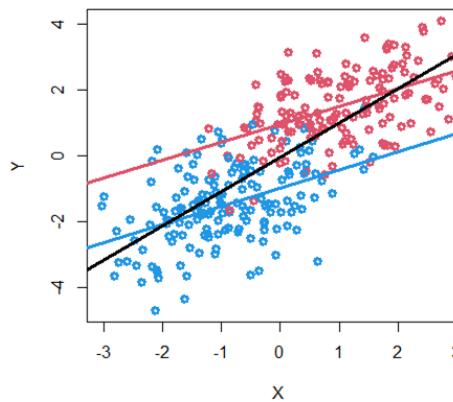
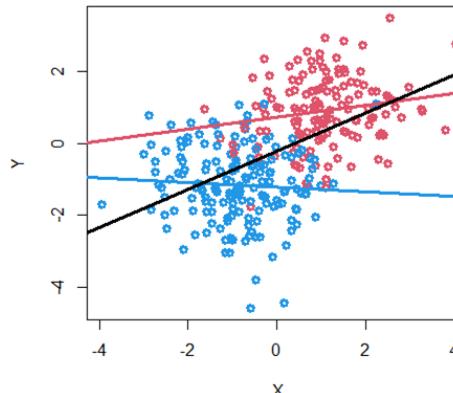
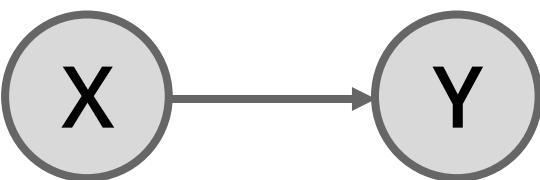
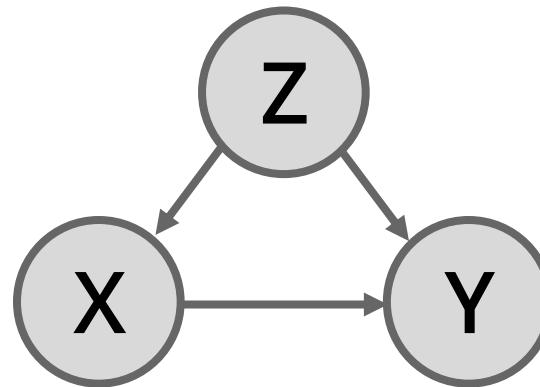
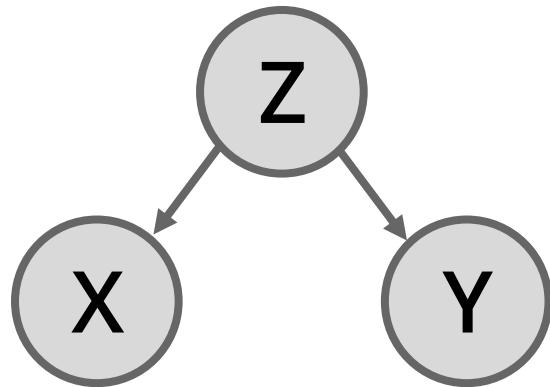




Phylogenetic confounding



d-separation: testing the DAG



DAG implies $X \perp Y$

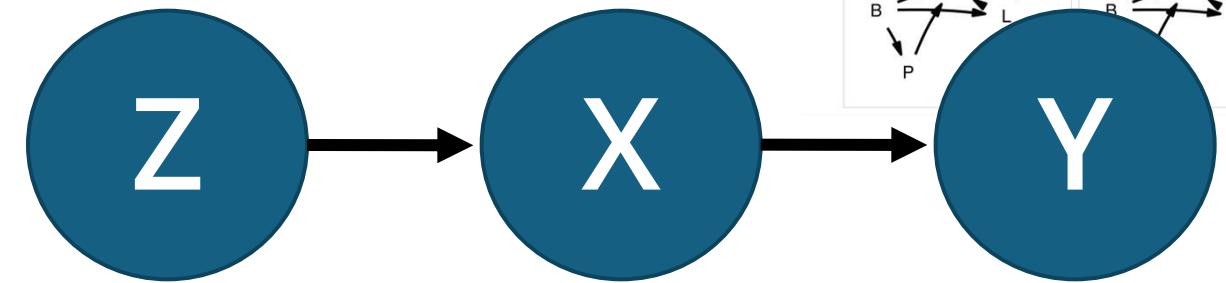
fit: $Y \sim X + Z$

Testing models by a D-sep test.

Which model best predicts conditional independencies in the data?

Phylopath

(Hardenberg & Gonzalez-Voyer 2013; Gonzalez-Voyer & Hardenberg 2014; van der Bijl 2018)



Bottom line:

Phylopath allows evaluation of d-separation among a specific set of causal models, but has strong assumptions about model and causes of phylogenetic residual covariance.

Assumes residual covariance is only noise, not causal signal

Assumes only present-day values matter for causation (causal lags don't exist)

Works only if both the DAG and residual model are correct (e.g. Large shifts will violate BM, but may not indicate the DAG itself is wrong, but lead to rejection of the DAG)

D-sep

Test for Conditional independence of:

$$Y|X \perp Z$$

$$Y|X \sim Z \text{ using PGLS (e.g. BM, BM + } \lambda, \text{ OU)}$$



If DAG is plausible,
coefficient not significant

What is this doing?

Accounts for expected phylogenetic covariance of residuals under model

Because PGLS, all historical effects mediated through present-day trait values. Ancestral confounders unrelated to Z affecting Y|X.

Large events/shifts likely to induce significance due to BM model violations (could use robust regression etc, but assumes specific causes of outliers=>i.e. replaces one causal assumption with another)



Can we use causal “do” logic across a broader set of causal scenarios?

Omitted Variable Bias

So far, we've considered if confounder is measured.

What if it is not known and/or measured?

Modeling correlation in error structure based on phylogeny
= phylogenetic random effects

Random effects assumption: Random effects must not be correlated with any covariates in the regression; or else estimates are biased.

Sister pair analyses

Identify independent sister clades that differ in X

Test for a difference in Y

Sample N = Number of pairs

Benefits from a CI perspective:

Conditions on shared due to
confounders due to common
ancestry (see “econometric fixed effects
design” of Byrnes & Dee 2024)

Downside:

Doesn’t use all the information in the
phylogeny, smaller sample size and
fewer degrees of freedom

Assumes confounders are constant
within sister comparisons

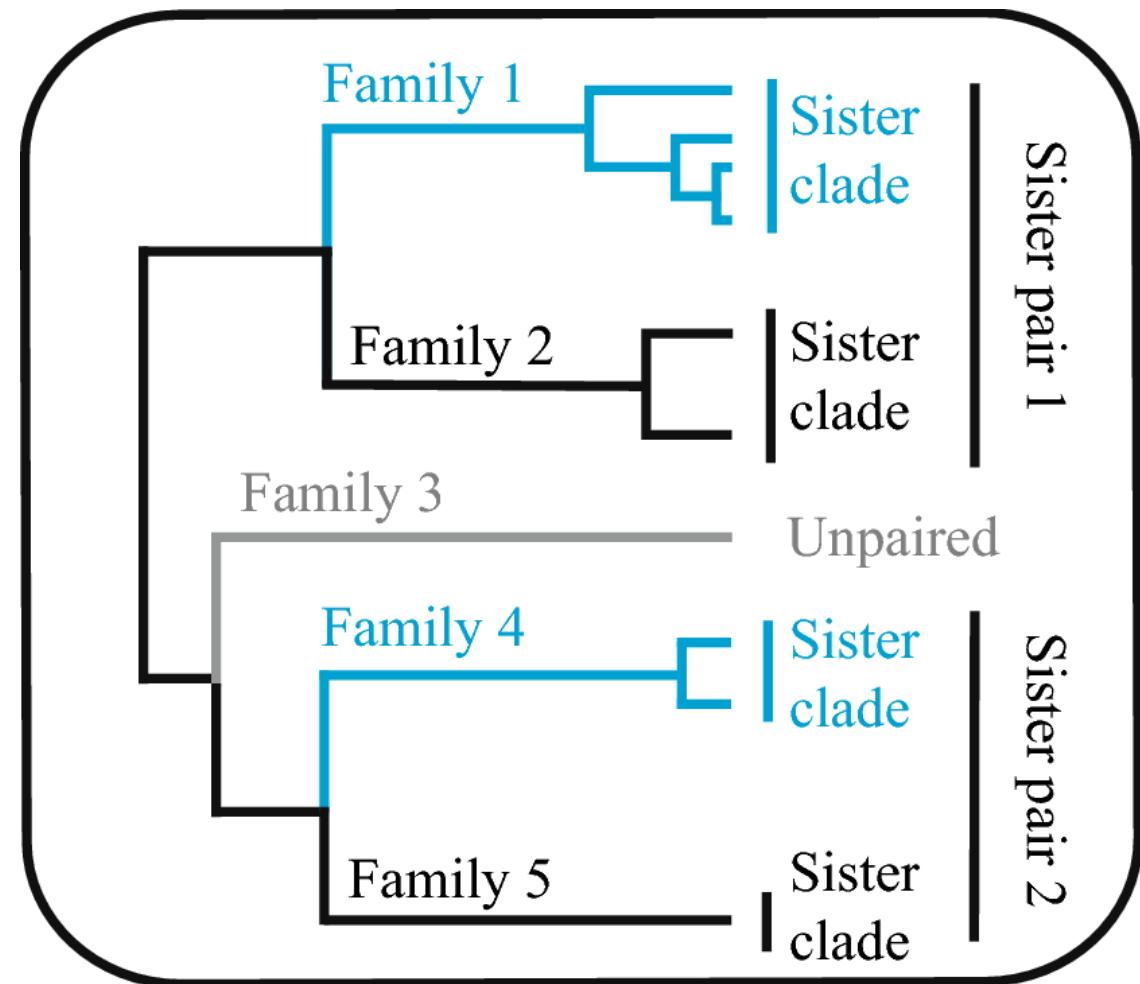
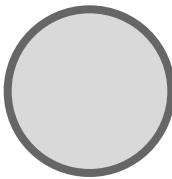


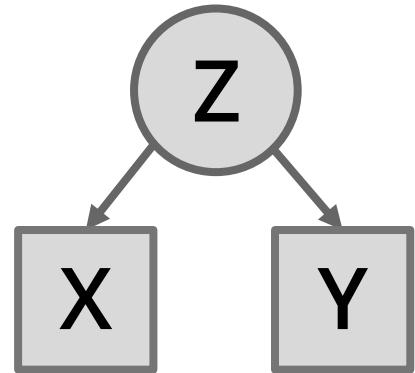
Figure from: Ritchie, A. M., Hua, X., & Bromham, L. (2022). Diversification rate is associated with rate of molecular evolution in ray-finned fish (actinopterygii). *Journal of Molecular Evolution*, 90(2), 200-214.



Observed

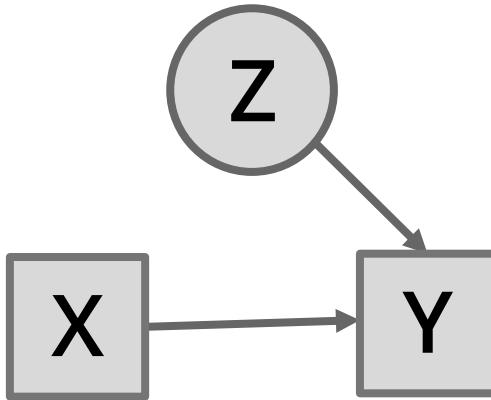


Omitted



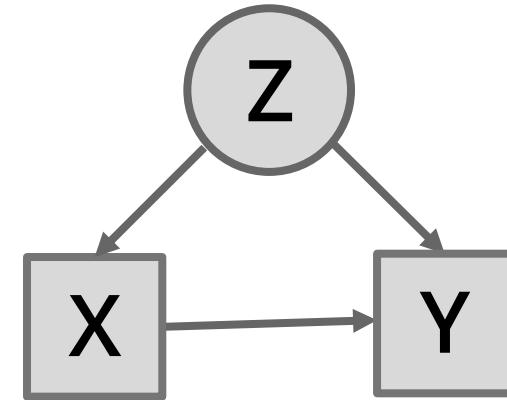
mvBM

(generating process
that inspired PICs.
Covariance estimator)



PGLS

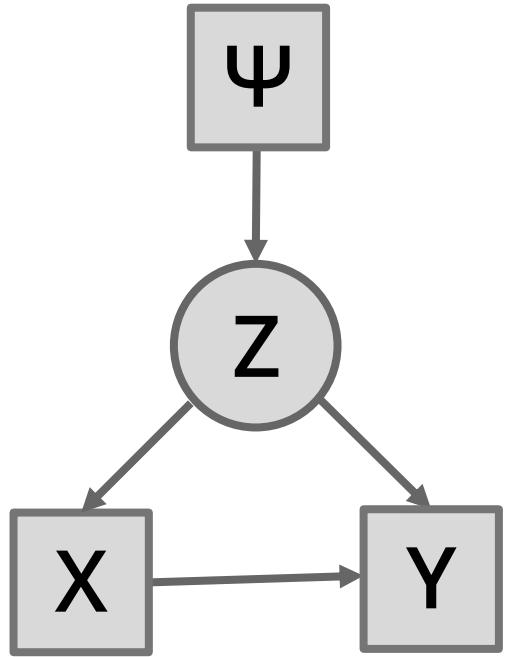
(Assumption of a PGLS
that would identify causal
effect of X on Y)



PGLS

w/endogeneity

(Estimate of causal effect of X on
Y biased! Covariance estimator)



$$\lambda_X = 0$$

$$\lambda_Y > 0$$

$$X \perp \Psi$$

$\lambda_X = 0$, means that X is consistent with ancestry independence

$$Y \mid X \perp \Psi$$

$\lambda_{Y|X} > 0$, means that the residual variation IS correlated with ancestry

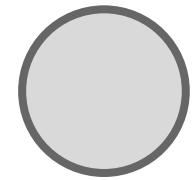
$$\lambda_{Y|X} > 0$$

Subject to statistical estimation caveats, suggests that the backdoor path between $Z \rightarrow X$ may not be open!

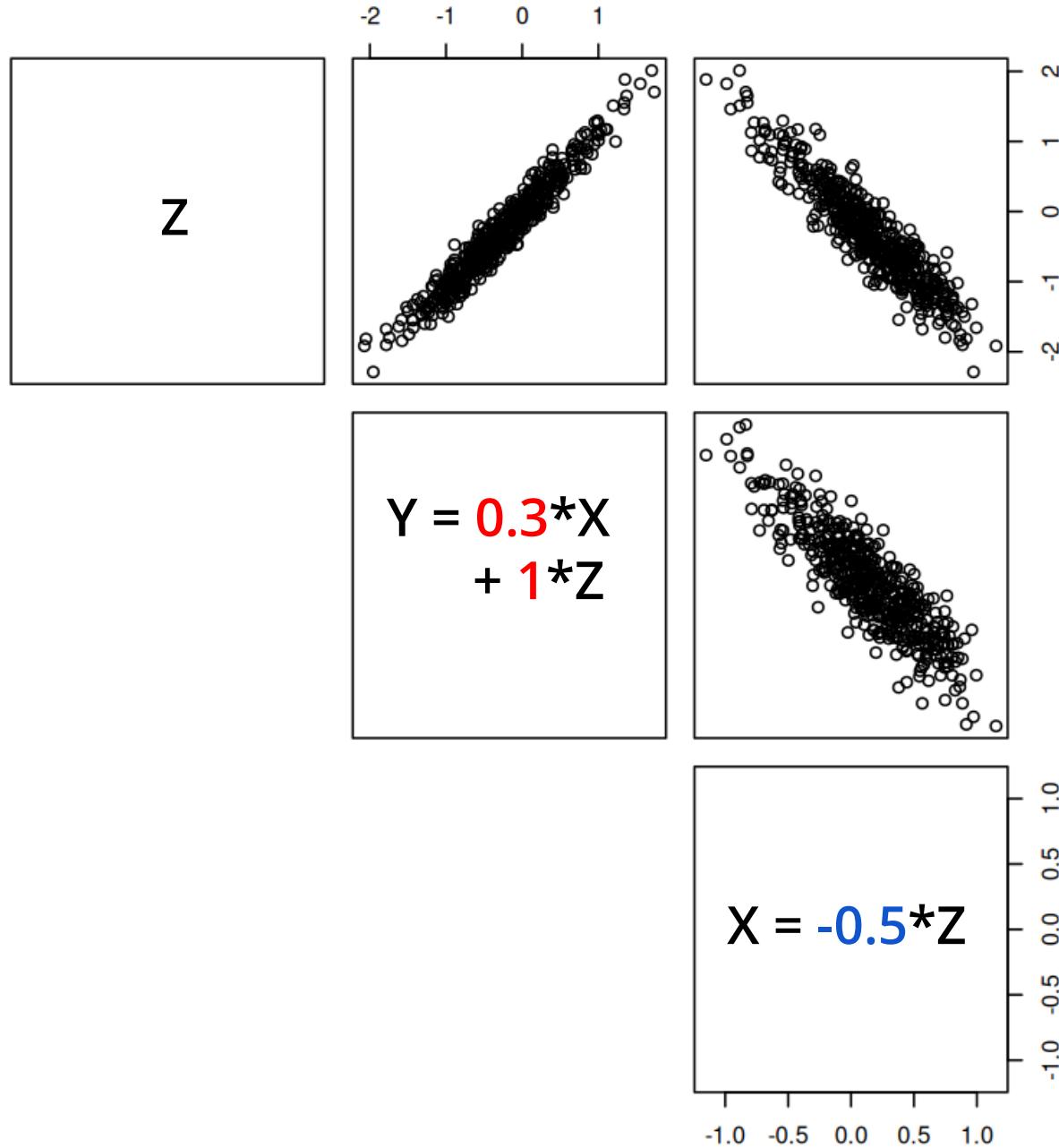
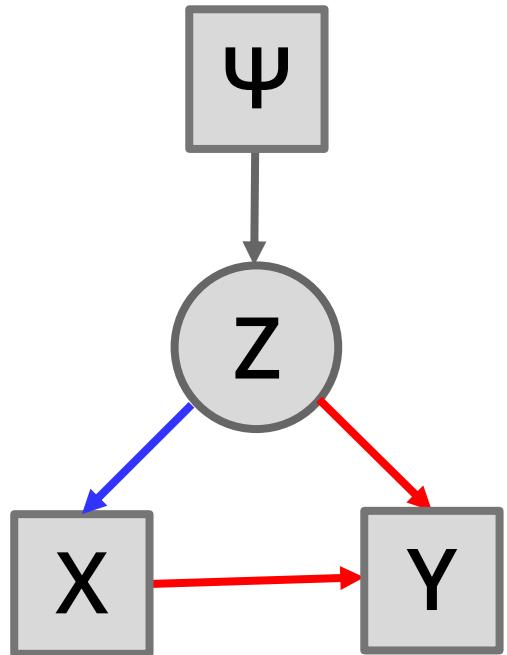
$X \rightarrow Y$ being a causal effect is *plausible*.



Observed



Omitted



```
> phylolm(Y ~ X+Z, phy=tree, model="lambda")
```

Mean tip height: 1

Parameter estimate(s) using ML:

lambda : 1e-07

sigma2: 0.02427564

Coefficients:

	Estimate	StdErr	t.value	p.value
(Intercept)	0.0062104	0.0077854	0.7977	0.4254
X	0.2484793	0.0435549	5.7050	2.001e-08 ***
Z	0.9954576	0.0228658	43.5349	< 2.2e-16 ***

Signif. codes: 0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

R-squared: 0.9681

Adjusted R-squared: 0.9679

Note: p-values and R-squared are conditional on lambda=1e-07.

```
> phylolm(Y ~ X, phy=tree, model="lambda")
```

Mean tip height: 1

Parameter estimate(s) using ML:

lambda : 0.2231165

sigma2: 0.104564

Coefficients:

	Estimate	StdErr	t.value	p.value	
(Intercept)	-0.022783	0.053491	-0.4259	0.6703	
X	-1.409503	0.037800	-37.2880	<2e-16	***

Signif. codes: 0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

R-squared: 0.7363

Adjusted R-squared: 0.7358

Note: p-values and R-squared are conditional on lambda=0.2231165.

REVIEW

Grime Review: Phil Grime's Impact on the Present and Future of Plant Ecology

Phylogenetically conservative trait correlation: Quantification and interpretation

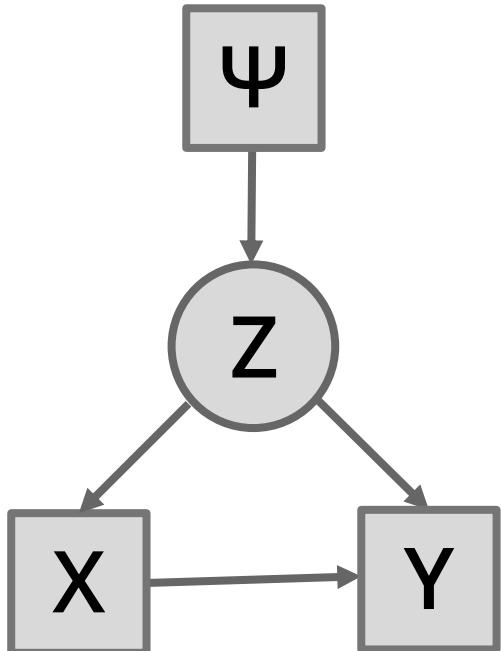
Mark Westoby¹  | Luke Yates²  | Barbara Holland³ | Ben Halliwell²

TABLE 1 Where variation is attributed by the multi-response phylogenetic mixed model (MR-PMM) described here. Key parameters estimated are four standard deviations σ_A^{phy} , σ_B^{phy} , σ_A^{ind} , and σ_B^{ind} , and two correlations ρ_{AB}^{phy} and ρ_{AB}^{ind} . For both phylogenetically structured and phylogeny-independent components and for the total, $\Sigma_{AB} = \rho_{AB}\sigma_A\sigma_B$.

Components of variation	Expression
Total variation in A	$\sigma_A^2 = (\sigma_A^{\text{phy}})^2 + (\sigma_A^{\text{ind}})^2$
Phylogenetic signal in A	$\lambda_A = \frac{(\sigma_A^{\text{phy}})^2}{\sigma_A^2}$
Total variation in B	$\sigma_B^2 = (\sigma_B^{\text{phy}})^2 + (\sigma_B^{\text{ind}})^2$
Phylogenetic signal in B	$\lambda_B = \frac{(\sigma_B^{\text{phy}})^2}{\sigma_B^2}$
A-B correlation associated with phylogeny (called here conservative trait correlation CTC)	$\rho_{AB}^{\text{phy}} = \frac{\Sigma_{AB}^{\text{phy}}}{\sqrt{\Sigma_{AA}^{\text{phy}}\Sigma_{BB}^{\text{phy}}}}$
A-B correlation independent from phylogeny	$\rho_{AB}^{\text{ind}} = \frac{\Sigma_{AB}^{\text{ind}}}{\sqrt{\Sigma_{AA}^{\text{ind}}\Sigma_{BB}^{\text{ind}}}}$
Total A-B correlation (expression in terms of lambda as given by Housworth et al., 2004)	$\rho_{AB} = \frac{\Sigma_{AB}^{\text{phy}} + \Sigma_{AB}^{\text{ind}}}{\sigma_A\sigma_B} = \rho_{AB}^{\text{phy}}\sqrt{\lambda_A\lambda_B} + \rho_{AB}^{\text{ind}}\sqrt{(1 - \lambda_A)(1 - \lambda_B)}$

How often do you have a change in X and what's the timescale of causation?



A singular event

A few events

A bunch of events

Continuous feedback

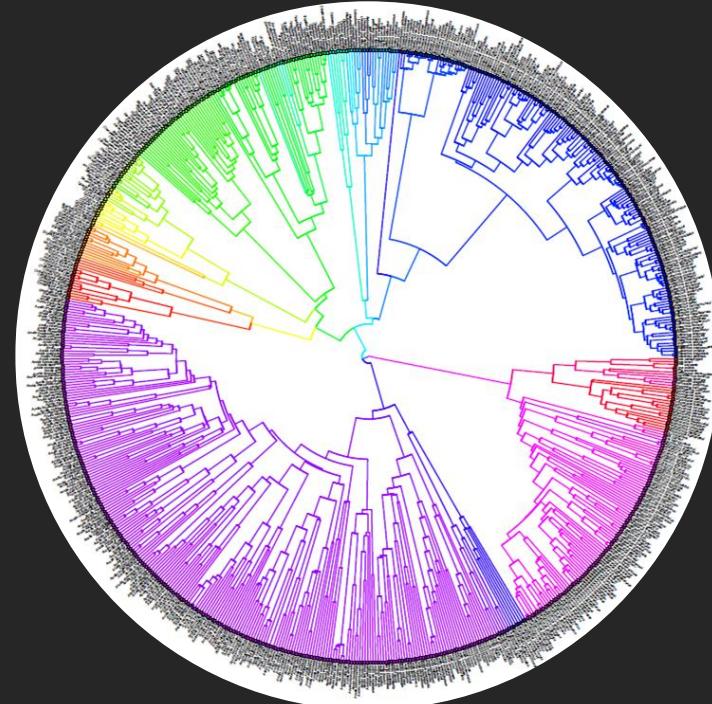


Alencar *et al.*: Diversification of Pleurodonta

- Diverse group of lizards in the Americas
- Incl. anoles, iguanas, spiny and horned lizards

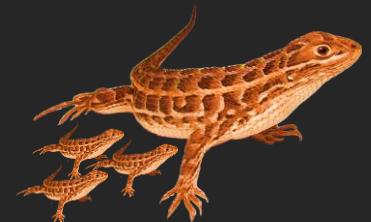
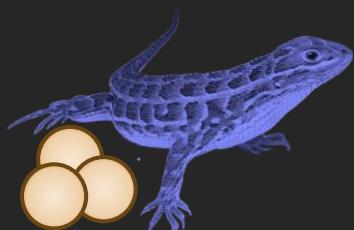
Dated phylogeny:

- 733/1111 species, based on Tonini *et al.* (2016)

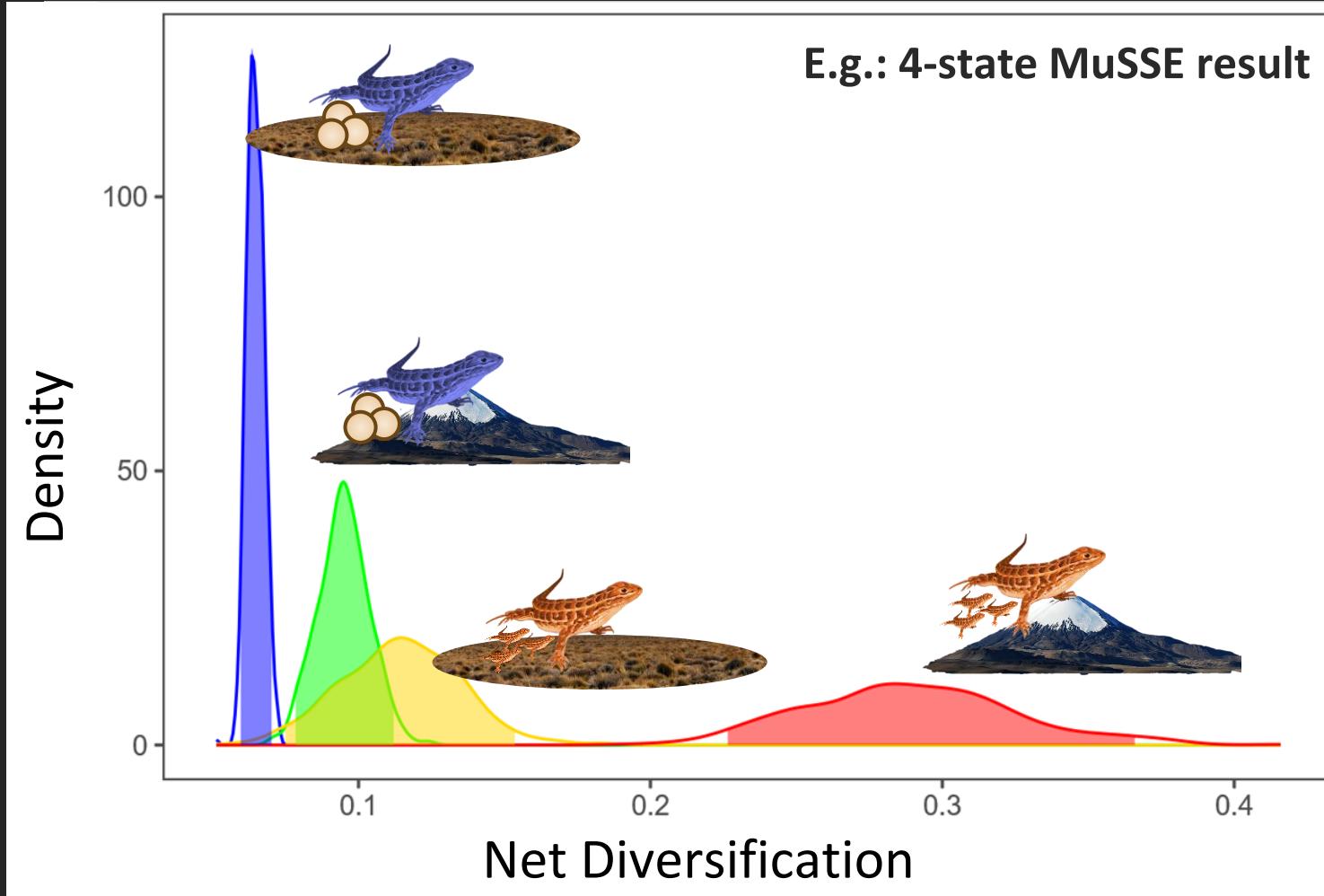
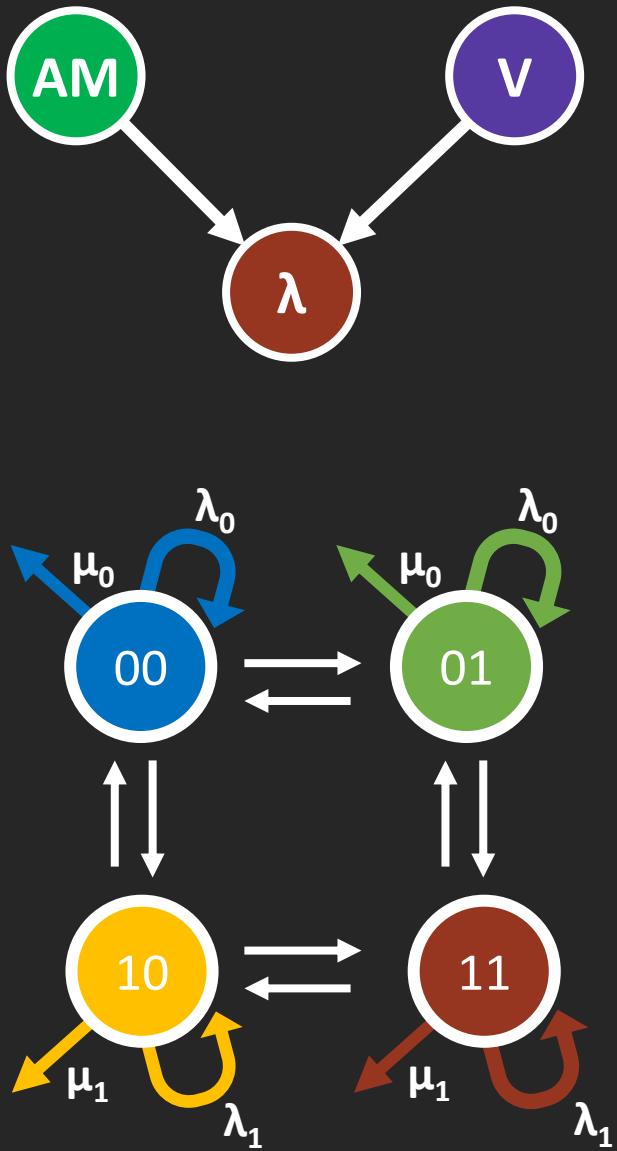


Focal traits:

- Reproductive mode (**oviparous**/**viviparous**)
- Occurrence in Andean mountains (or not)

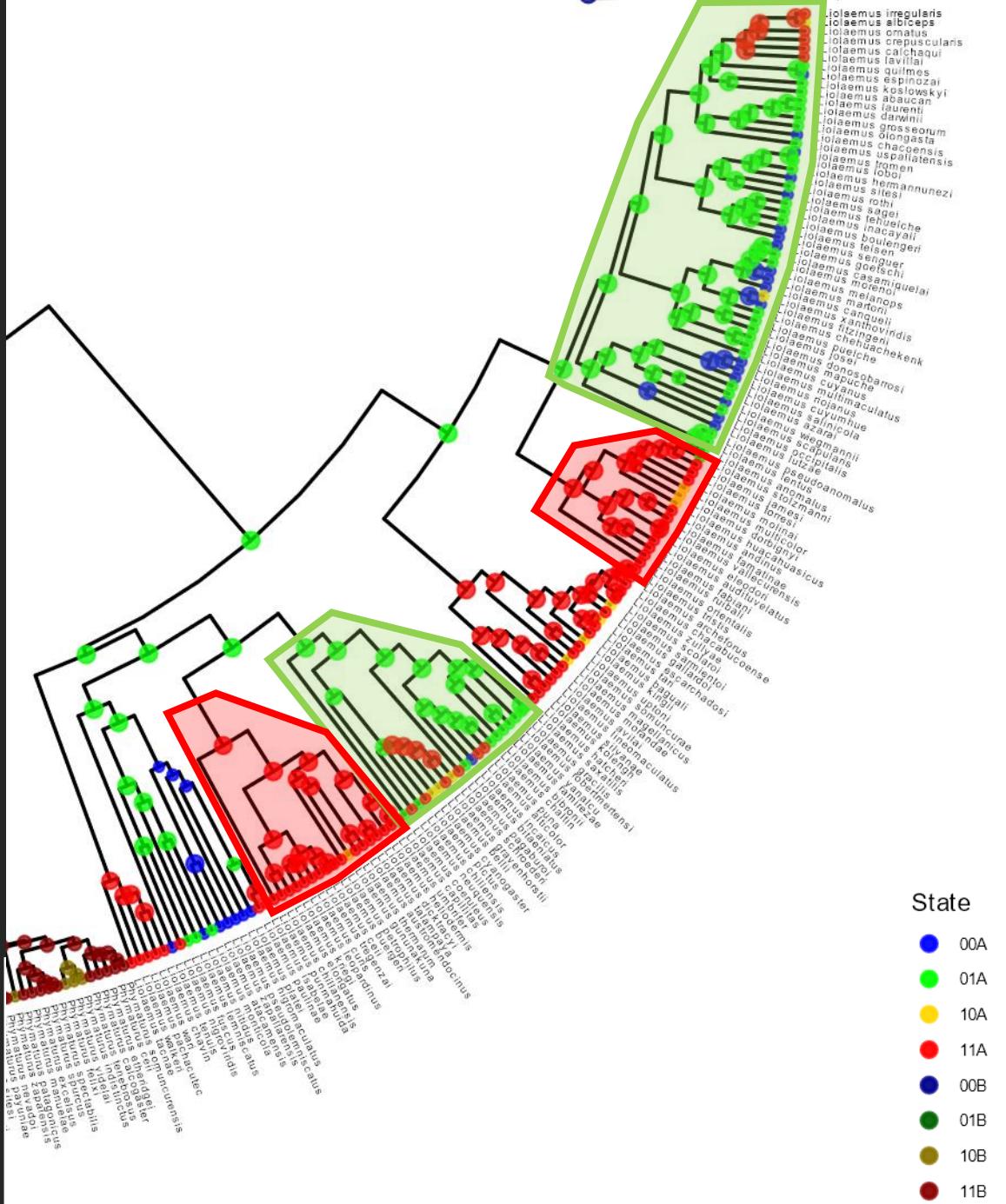
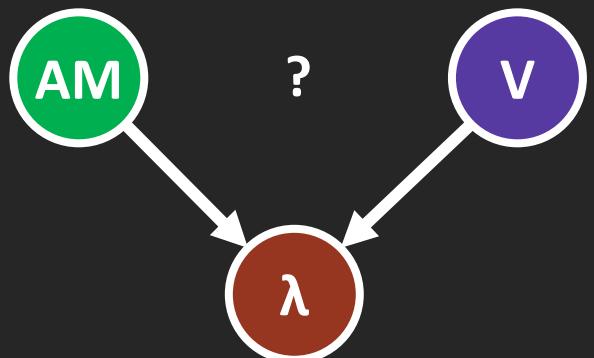


Combination of Viviparity & Andes drives Radiation?



Data-Disbelief

- Is it plausible that the predominantly Andean viviparous clades diversify fastest?
 - How do they compare to their Andean oviparous (and other) sister clades?



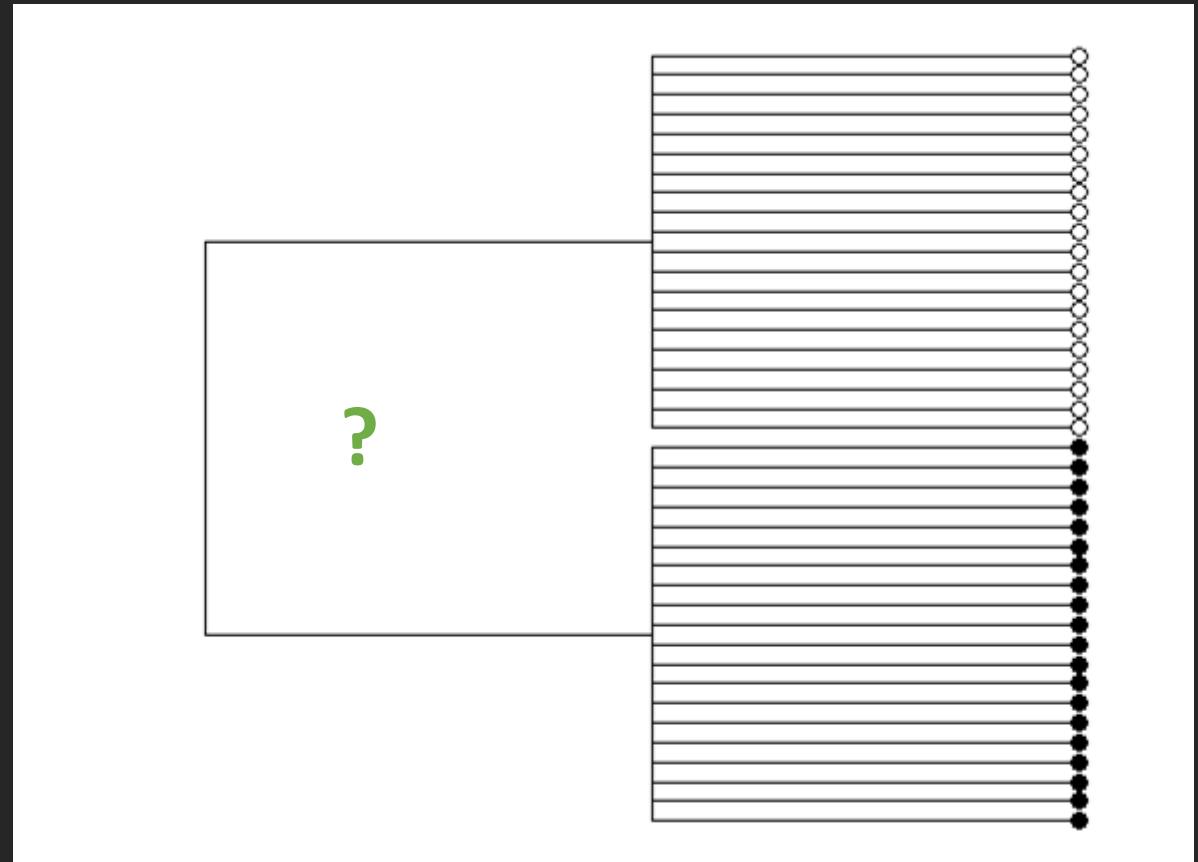
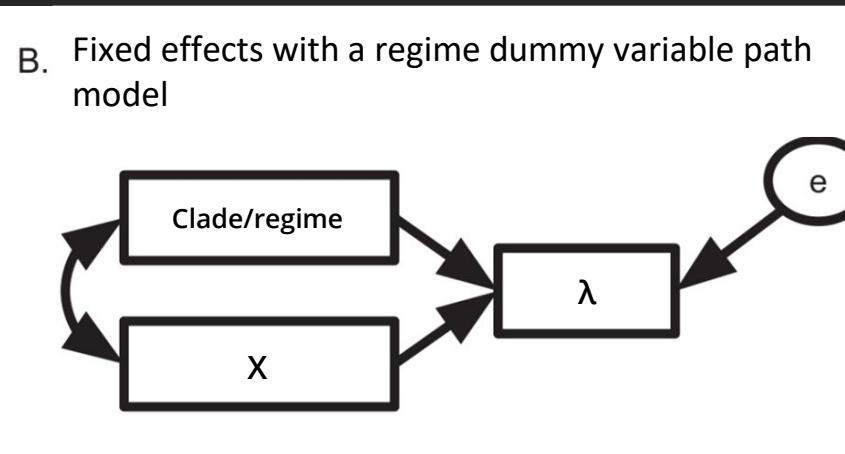
Causal Diversification: Conditioning on ‘known’ rate shifts

Diversification of Pleurodonta:

- What are *actual* effects of viviparity and Andean mountains?

Re: Felsenstein:

- What are the 'latent' clades we need to condition on?



Causal Diversification: Conditioning on ‘known’ rate shifts

Diversification of Pleurodonta:

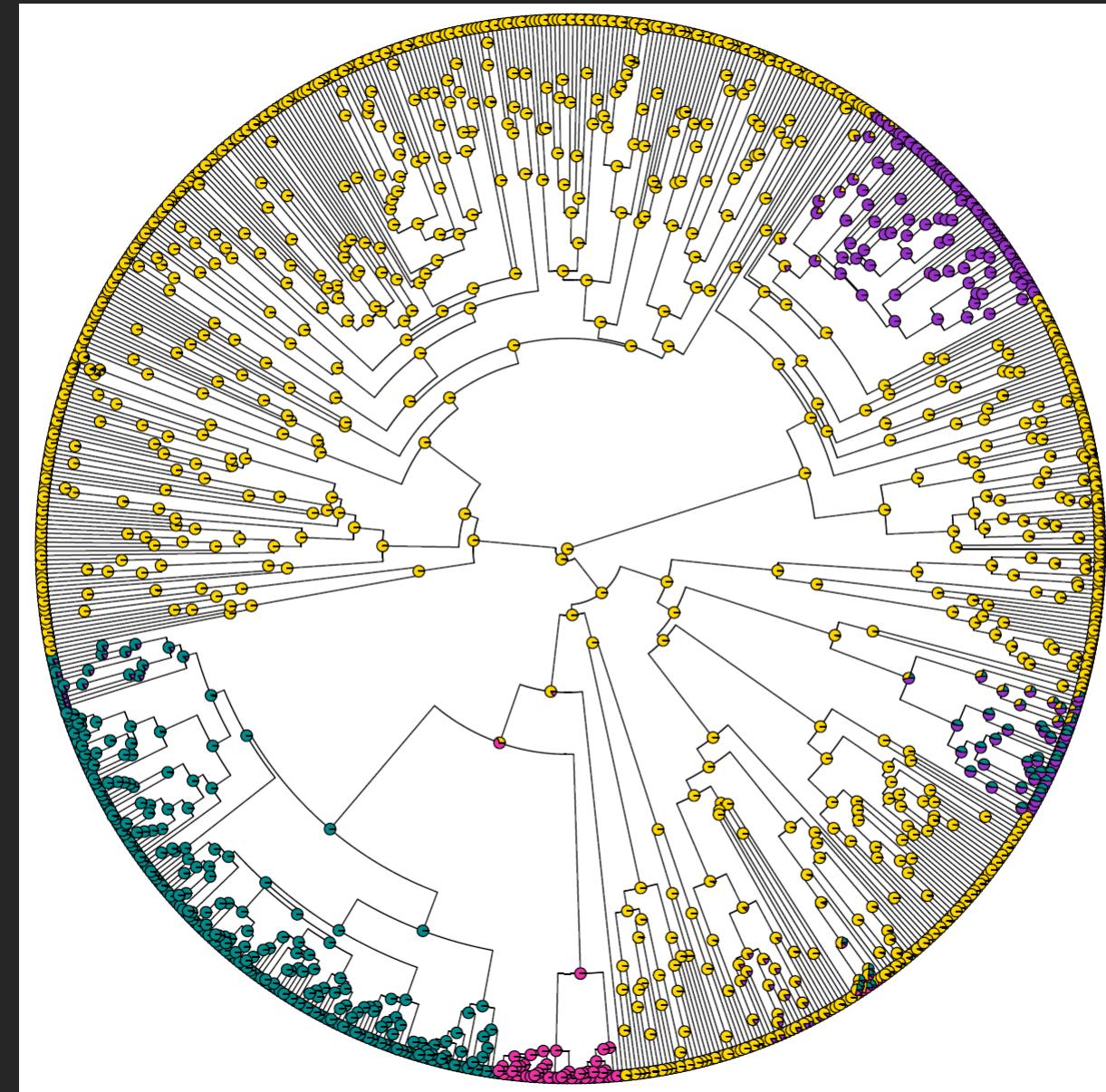
- What are *actual* effects of viviparity and Andean mountains?

Re: Felsenstein:

- What are the 'latent' clades we need to condition on?

Infer overall rate heterogeneity/shifts, e.g.:

- Shifts between rate categories
- MiSSE model (SSE w/o traits)

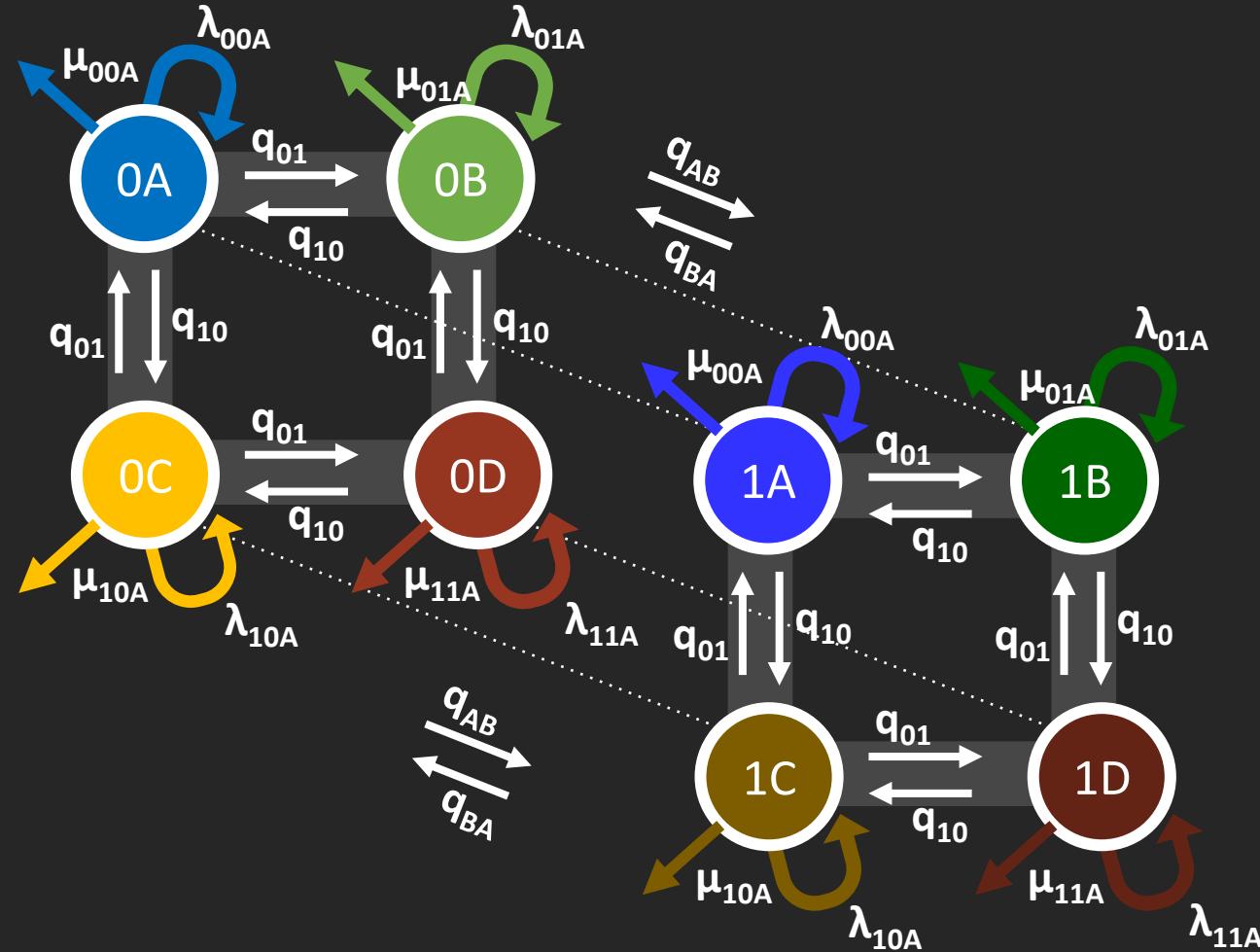


Causal Diversification: Conditioning on Shift Regimes

How to test traits while conditioning on the observed rate shifts?

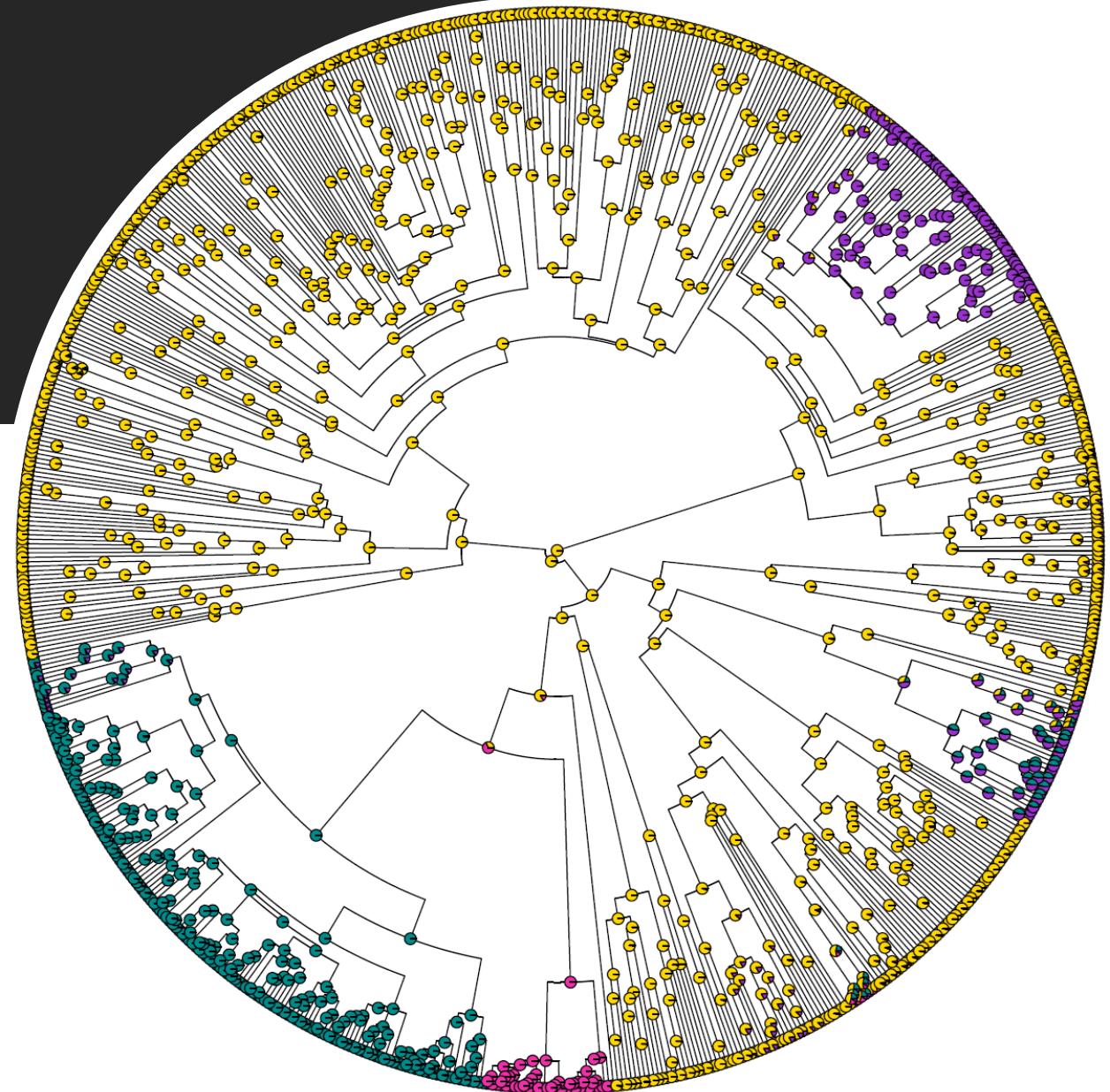
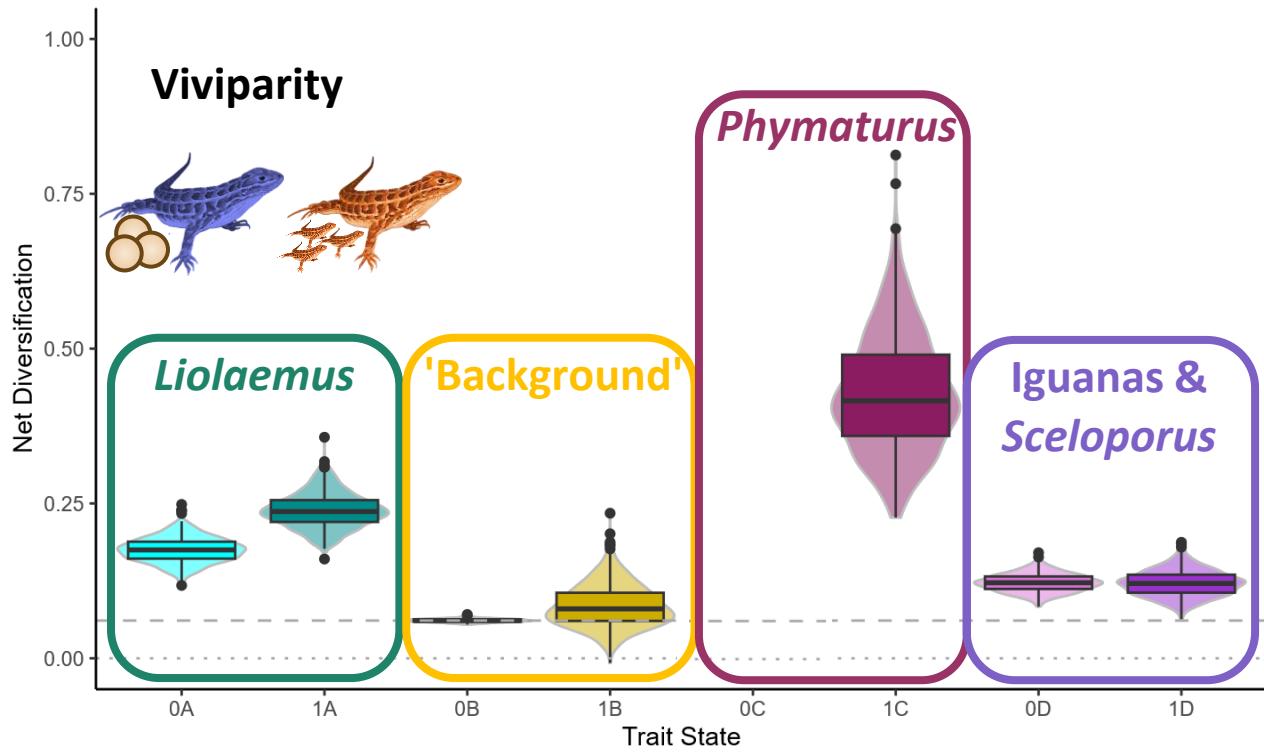
→ Create Pseudo-trait:

- Merge MiSSE-states with traits
- Analyse as 8-state MuSSE



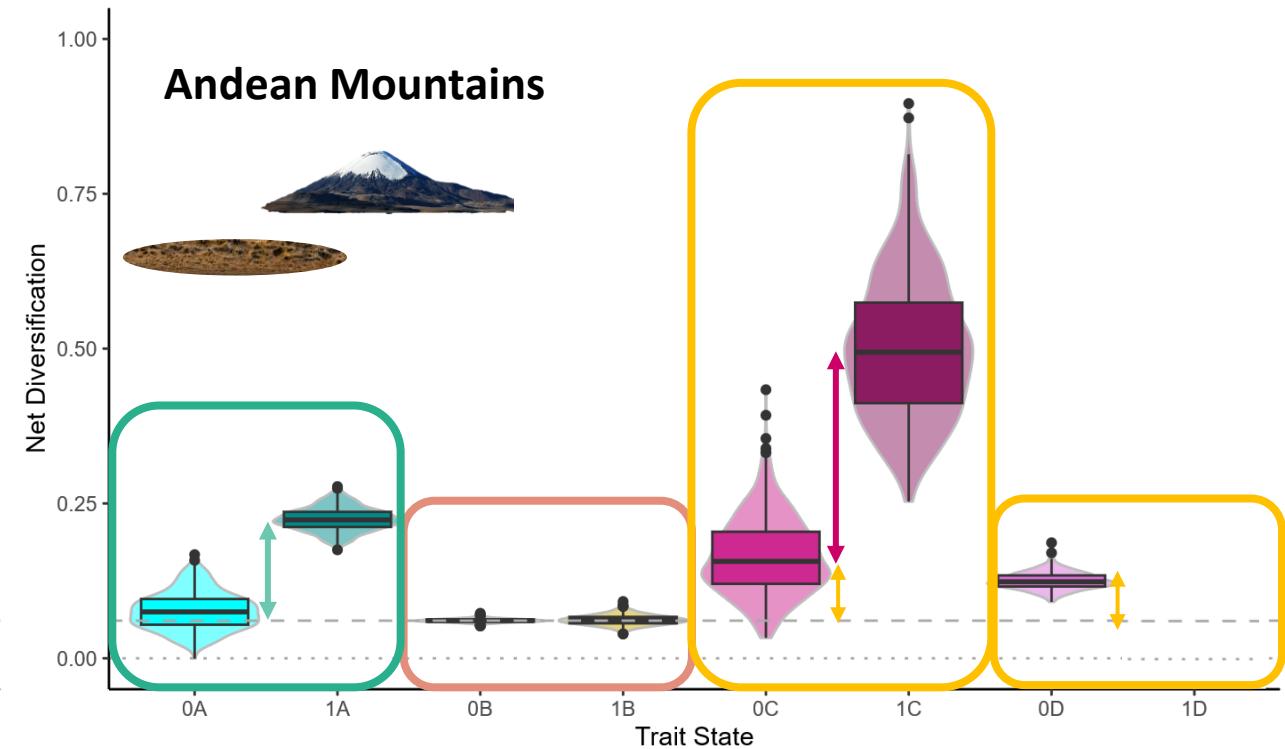
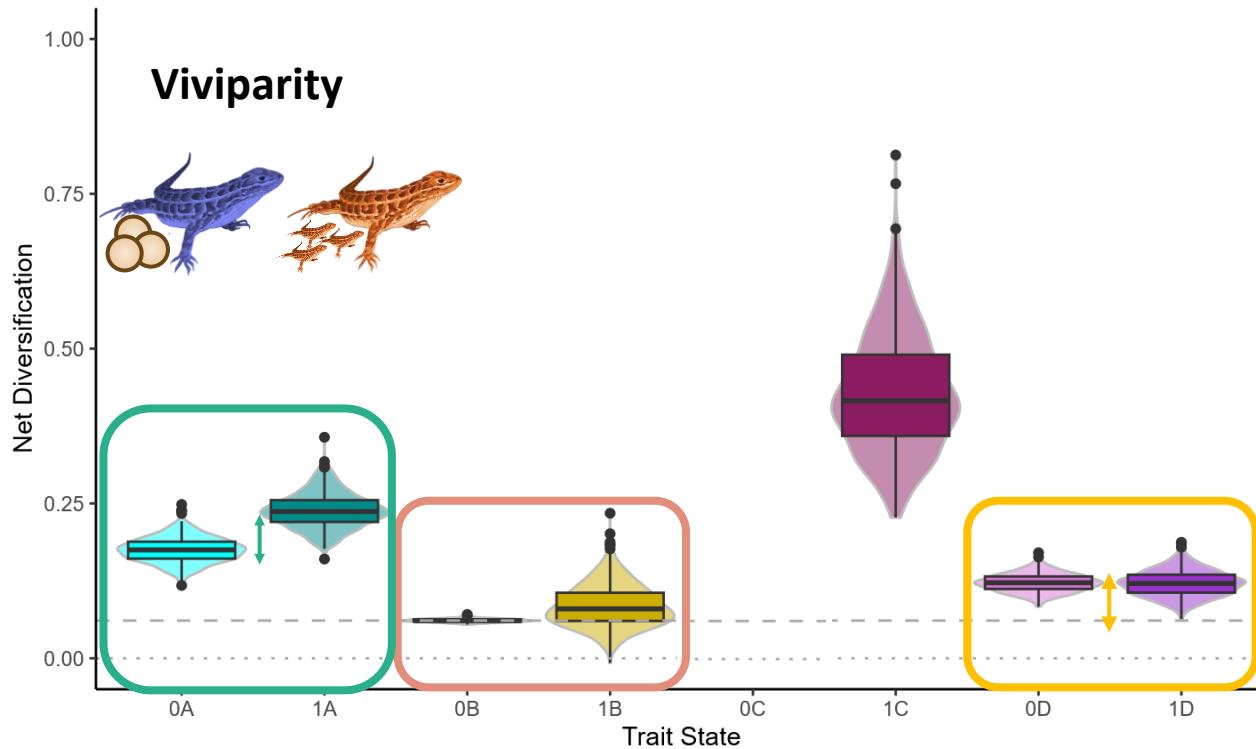
Causal Diversification: Overall Result

Net Diversification rates
for combinations of
rate shift clades & trait states



Causal Diversification: Insights to be gained

1. In some clades, traits have no effect
2. In *Liolaemus*
 1. Andes could explain the whole shift
 2. Viviparity explains little at best
3. In some rate shifts, the traits explain...
 1. ... **only part** of the shift
 2. ... **none** of the shift



Causal Diversification: Insights to be gained

1. In some clades, traits
2. In *Liolaemus*
 1. Andes could explain
 2. Viviparity explained

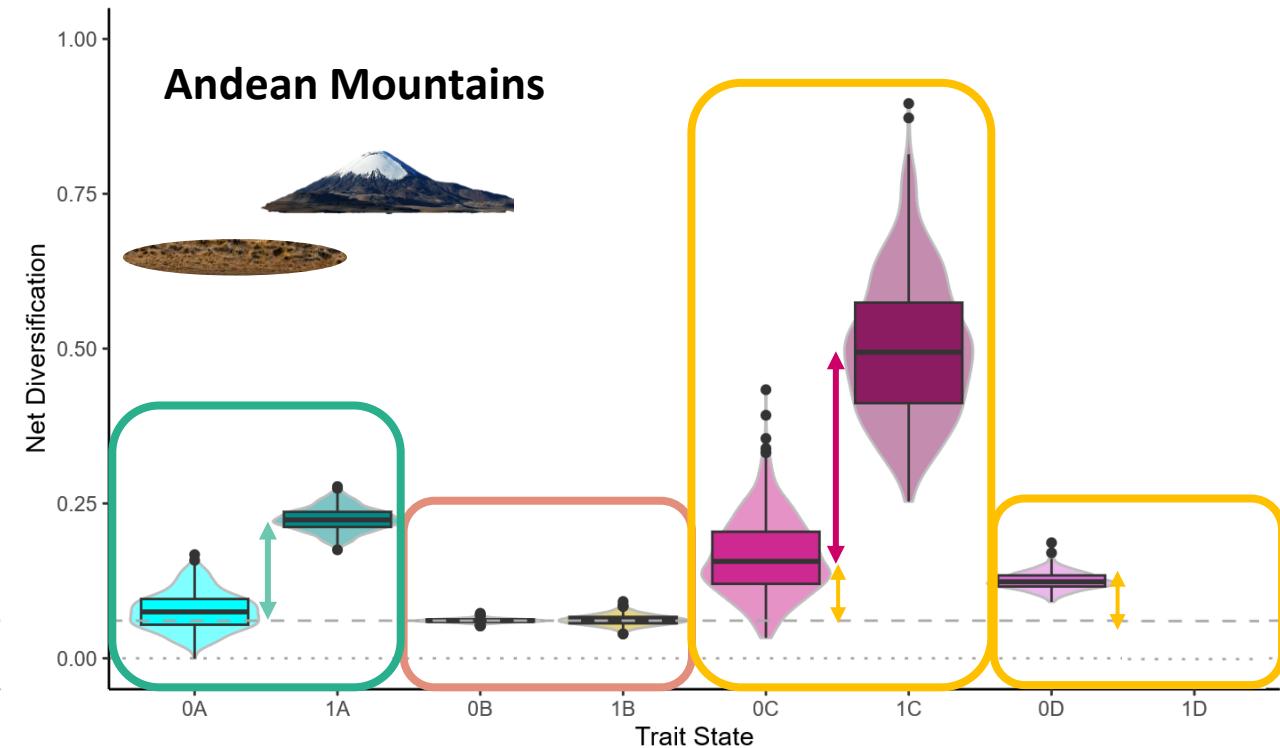
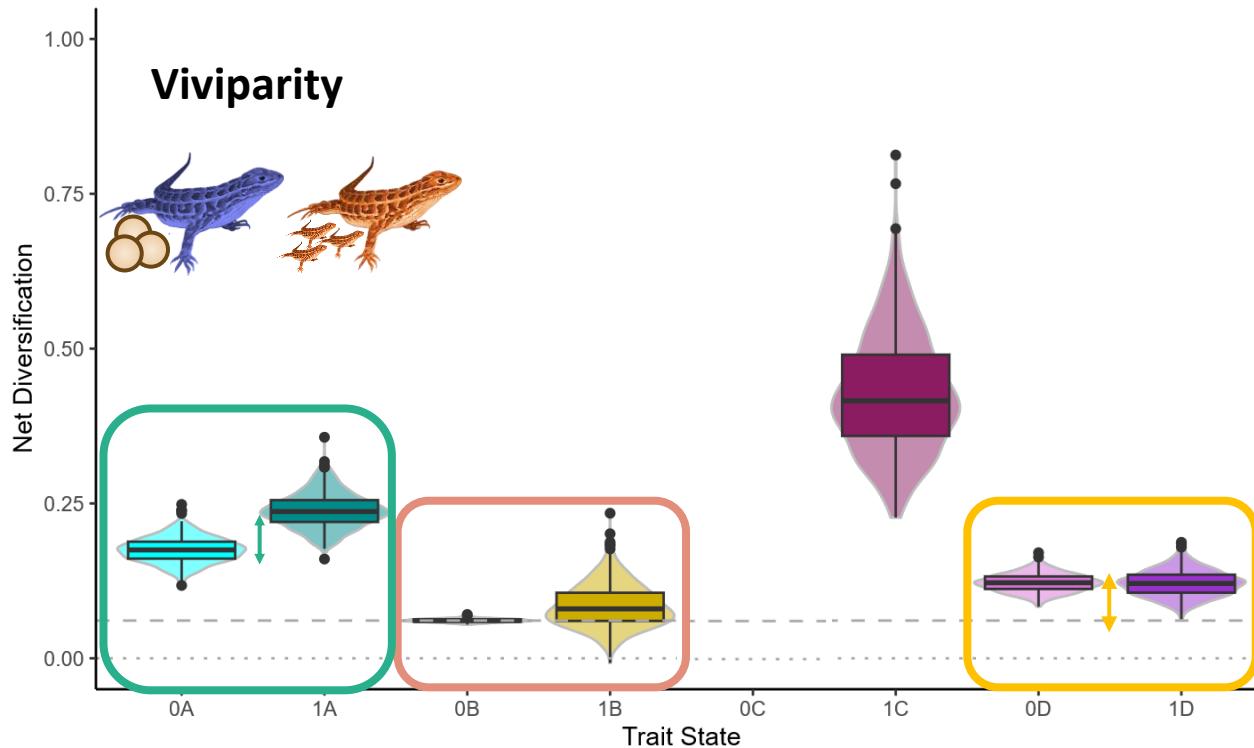
We learn about our rate shifts:

1. Mountains explain some
2. Viviparity barely any
3. Some remain unexplained

traits, the traits

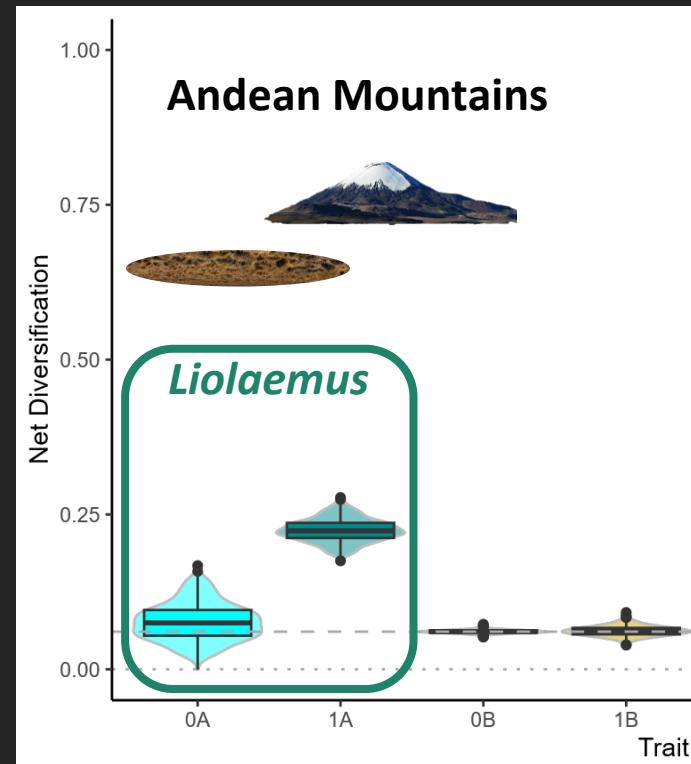
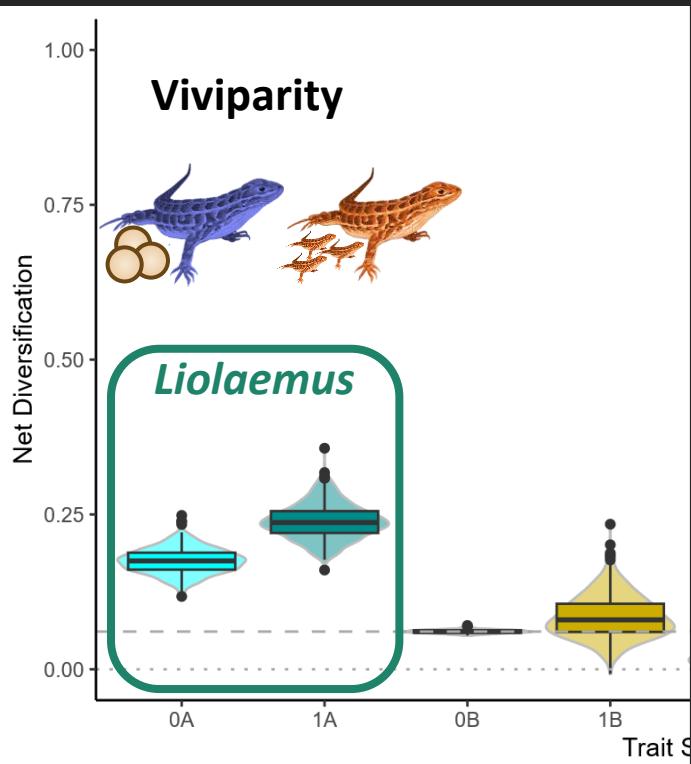
of the shift

2. ... none of the shift



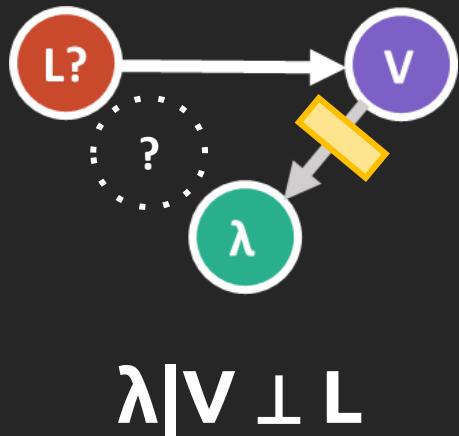
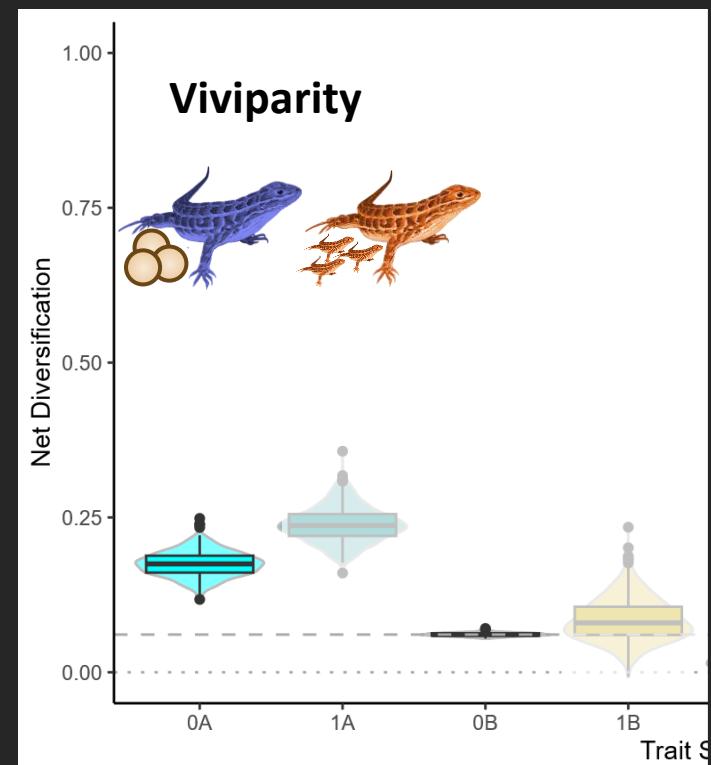
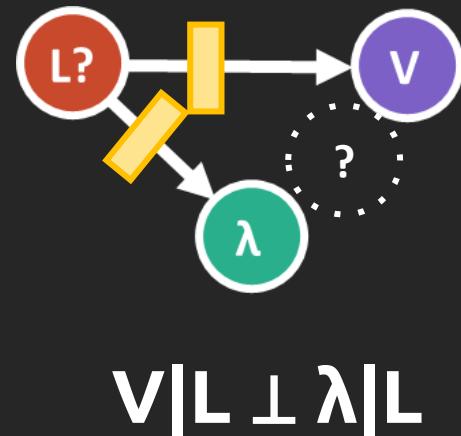
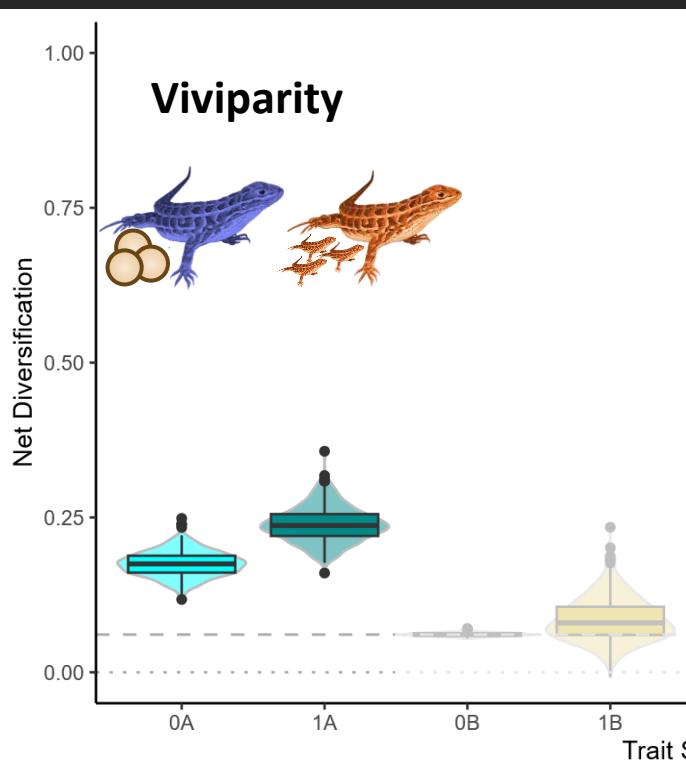
Causal Diversification: Conditional Effect Sizes

→ Focus on *Liolaemus*:



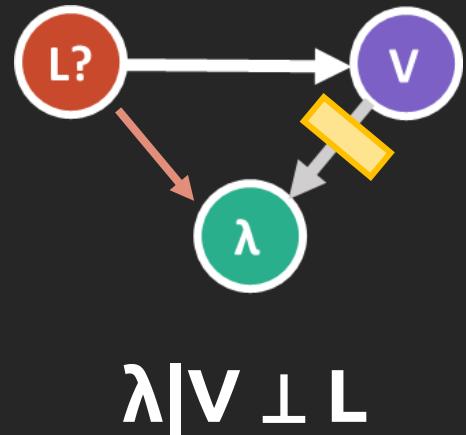
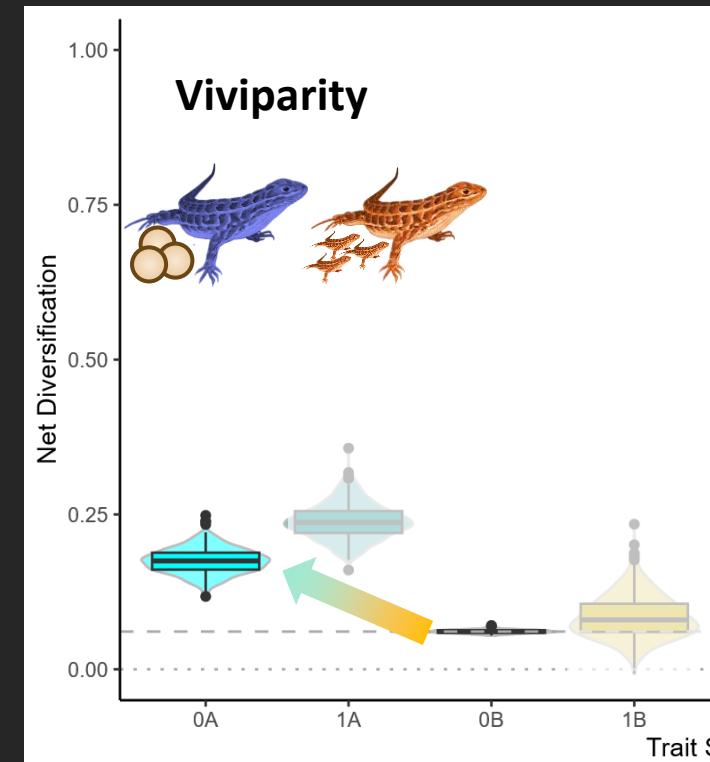
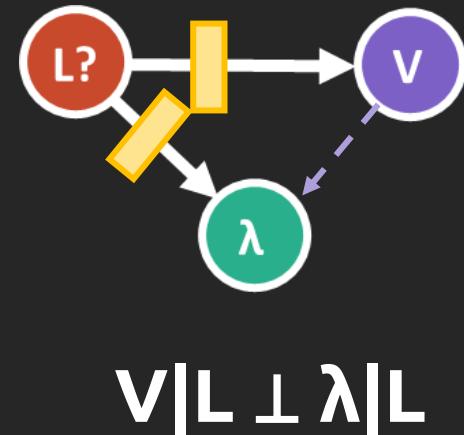
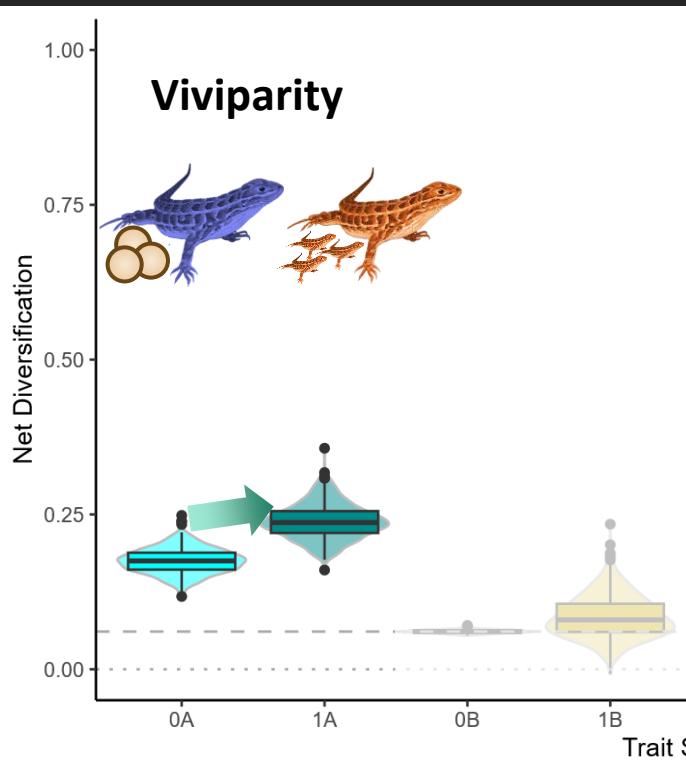
Causal Diversification: Conditional Effect Sizes

- Does conditioning on one factor reveal a remaining effect of the other?
[i.e., is there still a correlation?]
- Does **viviparity** show an effect on rate after **conditioning** on **clade**?
- Does **clade** show an effect on rate after **conditioning** on **viviparity**?



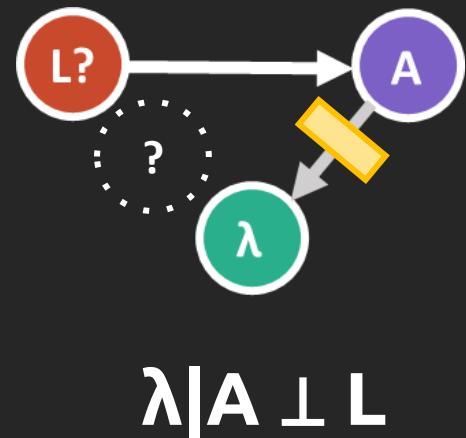
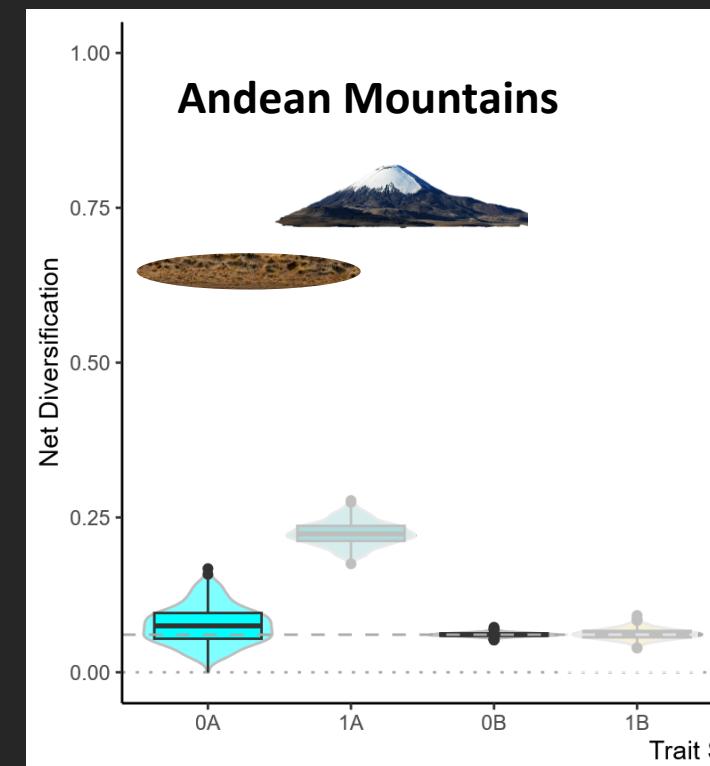
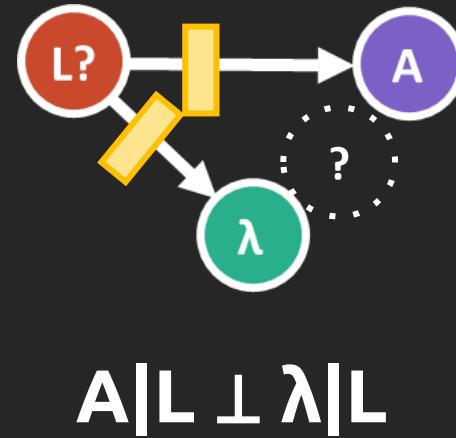
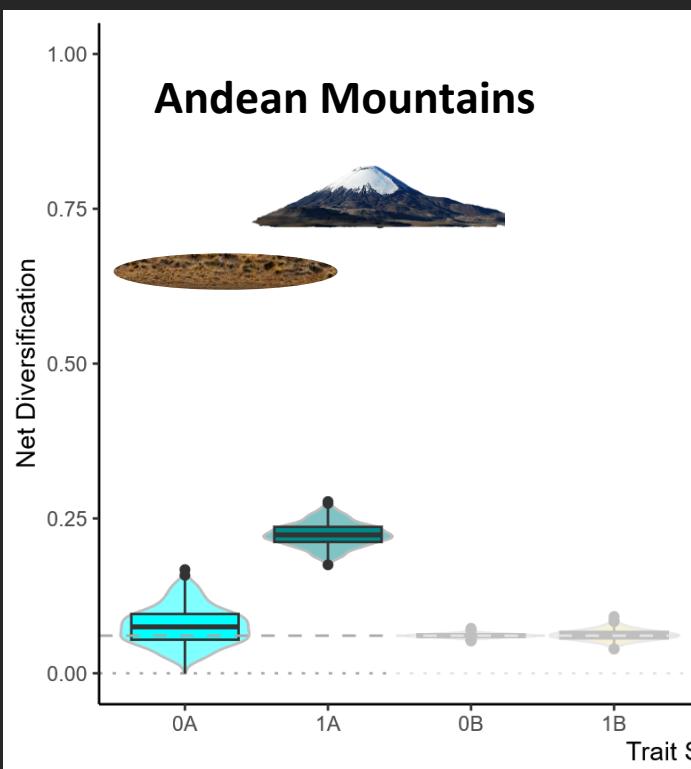
Causal Diversification: Conditional Effect Sizes

- Does conditioning on one factor reveal a remaining effect of the other?
[i.e., is there still a correlation?]
- Correlation weak!
→ Viviparity has little effect within *Liolaemus*
- Correlation persists!
→ Viviparity alone *can't explain* rate shift



Causal Diversification: Conditional Effect Sizes

- Does conditioning on one factor reveal a remaining effect of the other?
[i.e., is there still a correlation?]
- Do the Andes show an effect on rate after conditioning on clade?
- Does clade show an effect on rate after conditioning on the Andes?



Causal Diversification: Conditional Effect Sizes

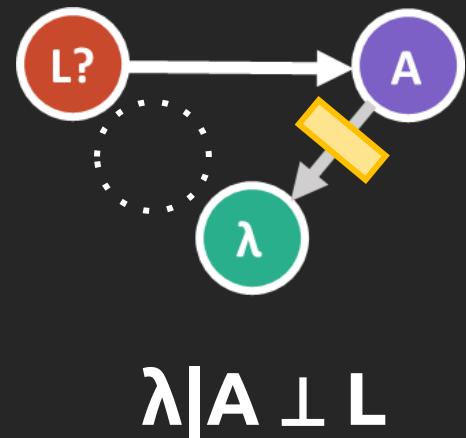
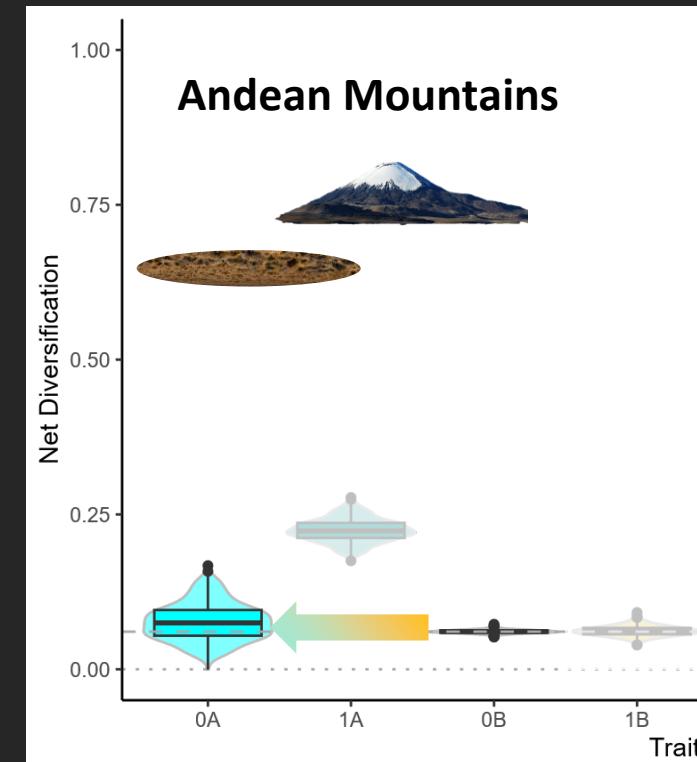
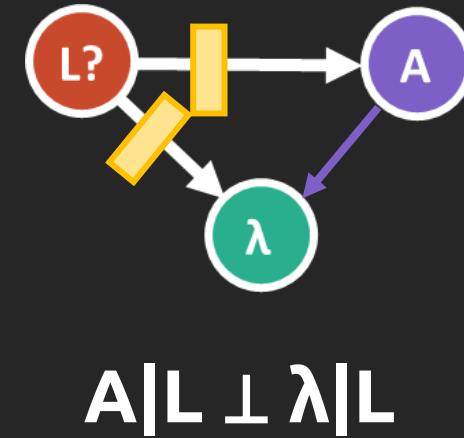
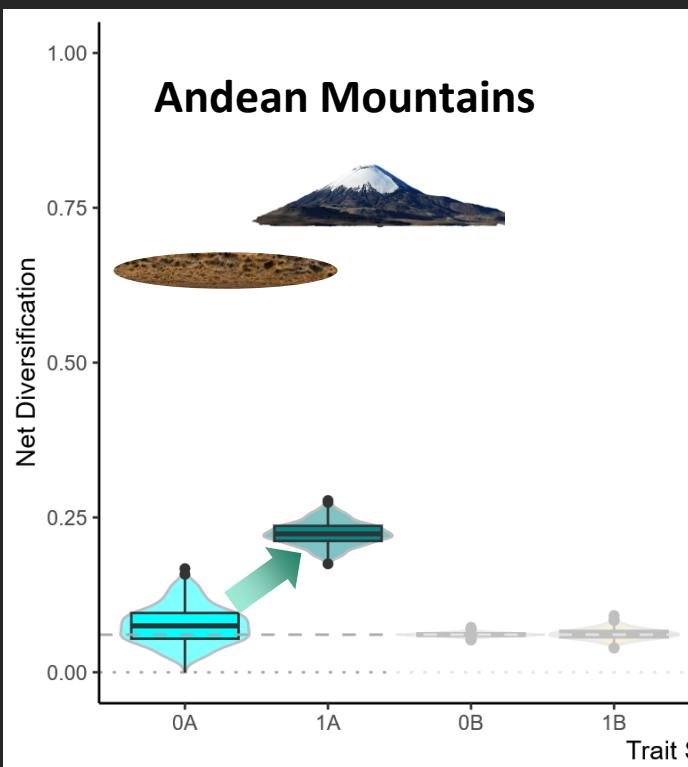
- Does conditioning on one factor reveal a remaining effect of the other?
[i.e., is there still a correlation?]

- Correlation strong!

→ Andes have substantial effect in *Liolaemus*

- Correlation disappears!

→ Andes alone *could explain* this rate shift



How often do you have a change in X and what's the timescale of causation?



A singular event

A few events

A bunch of events

Continuous feedback

Granger causality

Causes precede effects

Past values of X improve prediction of Y
(beyond past values of Y alone)

Temporal predictive power, not true causation

Does a PCM model precedence?

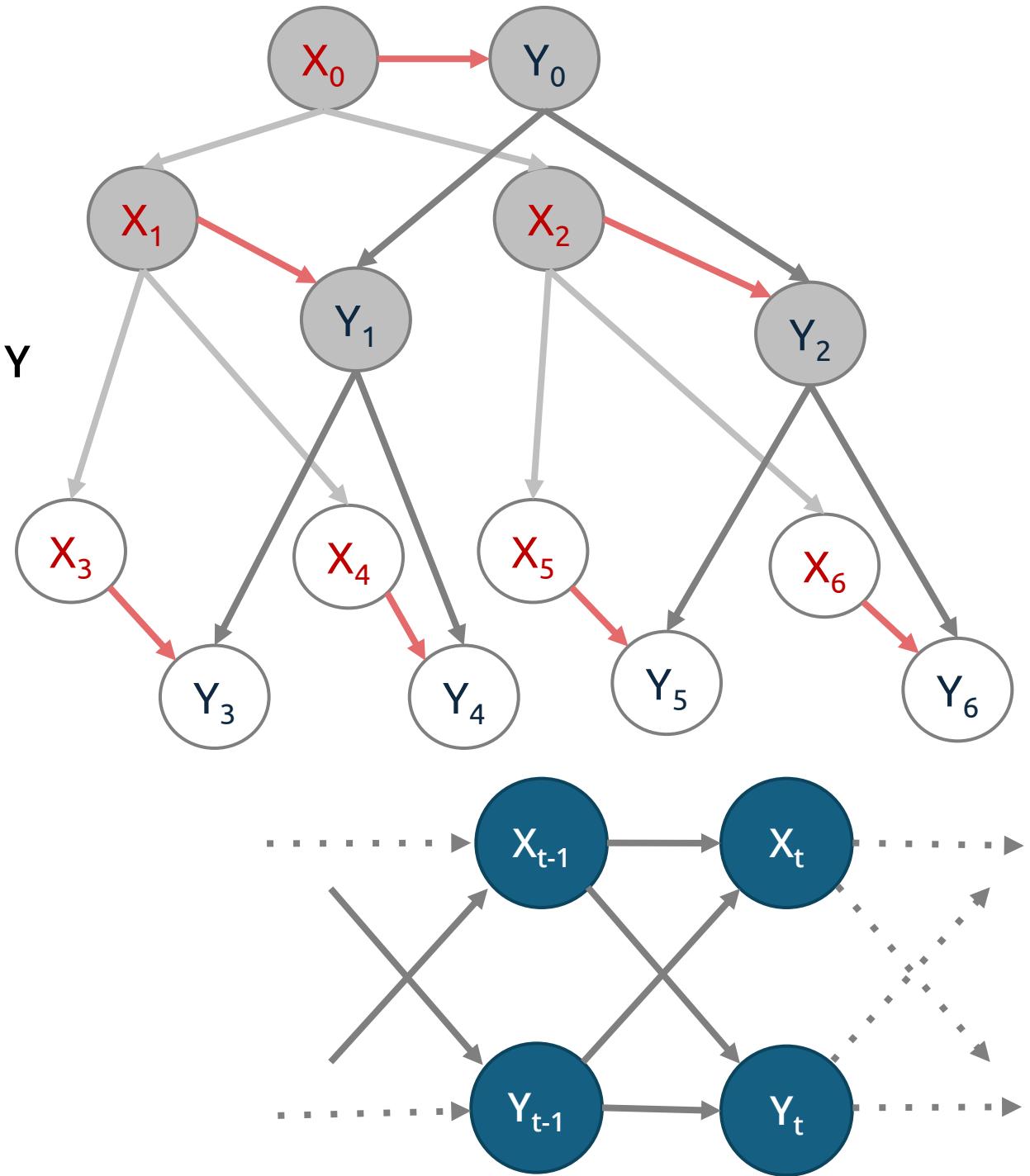
Many PCMs do not test:
PGLS, PICs, mvBM etc.

Discrete traits:

Continuous time Markov models
(e.g. Pagel's correlation test)

Continuous traits:

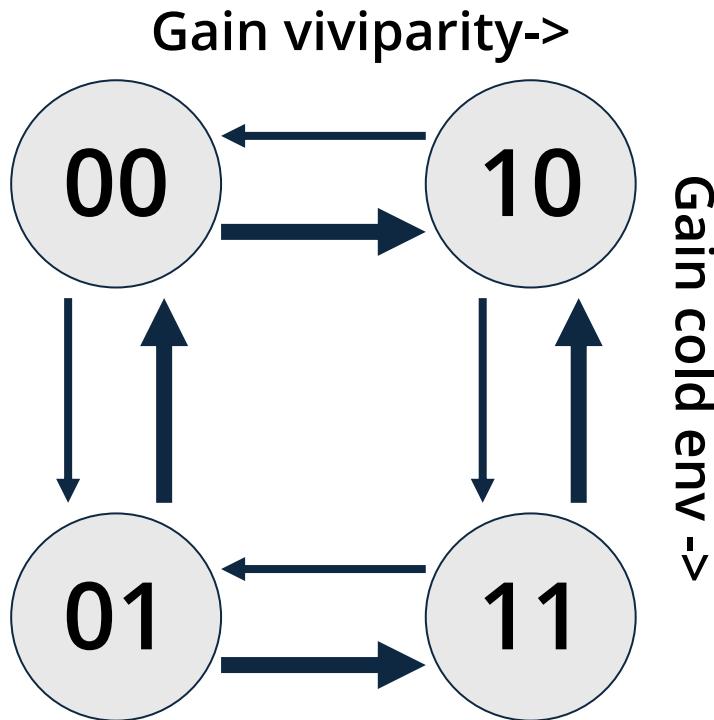
Some OU & mvOU models



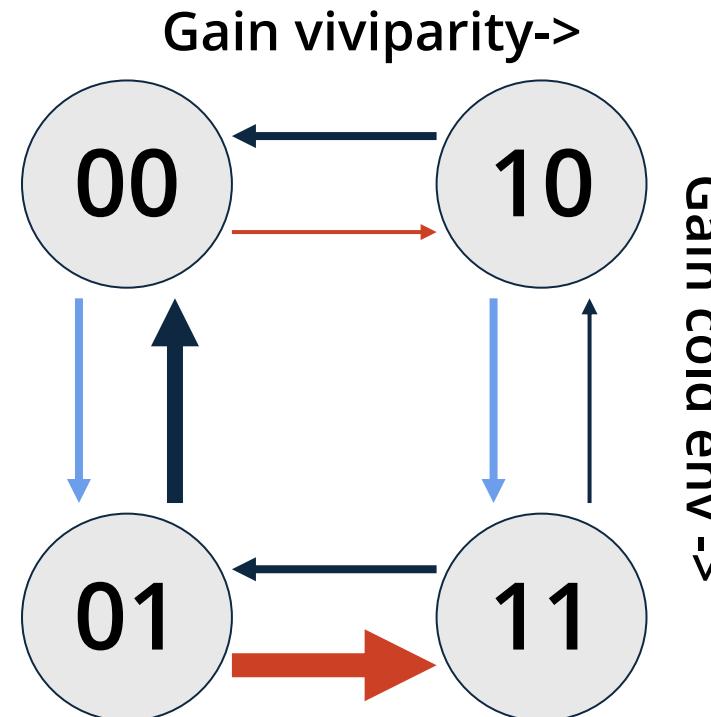
Markov models (e.g. Pagel's correlation)

Consider two binary characters (e.g. viviparity & cold env)

Independent model



Correlated model



Unequal rates depending on background state means correlation.

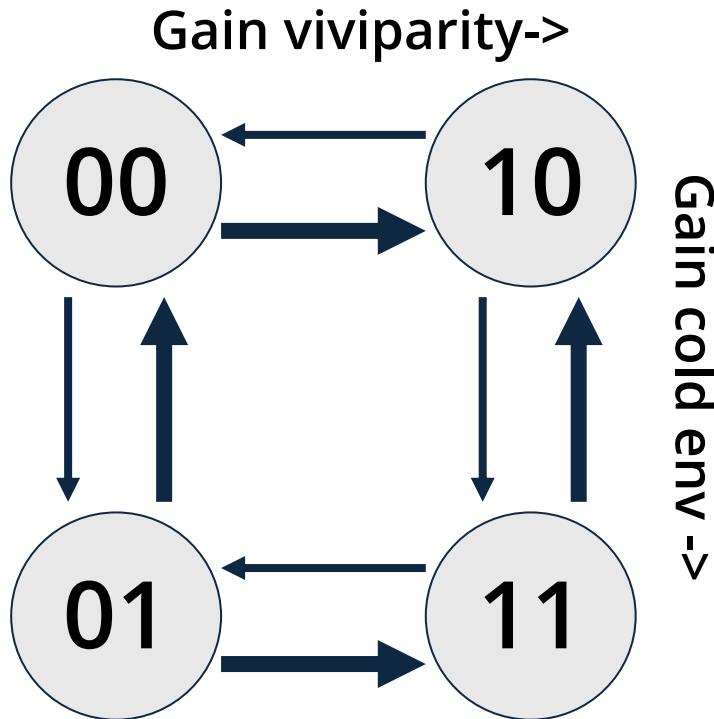
In the hypothetical example,
 $q_{[01 \rightarrow 11]} > q_{[00 \rightarrow 10]}$
 $q_{[10 \rightarrow 11]} = q_{[00 \rightarrow 11]}$

Cold environment is a Granger cause of viviparity (e.g. trait adaptation)

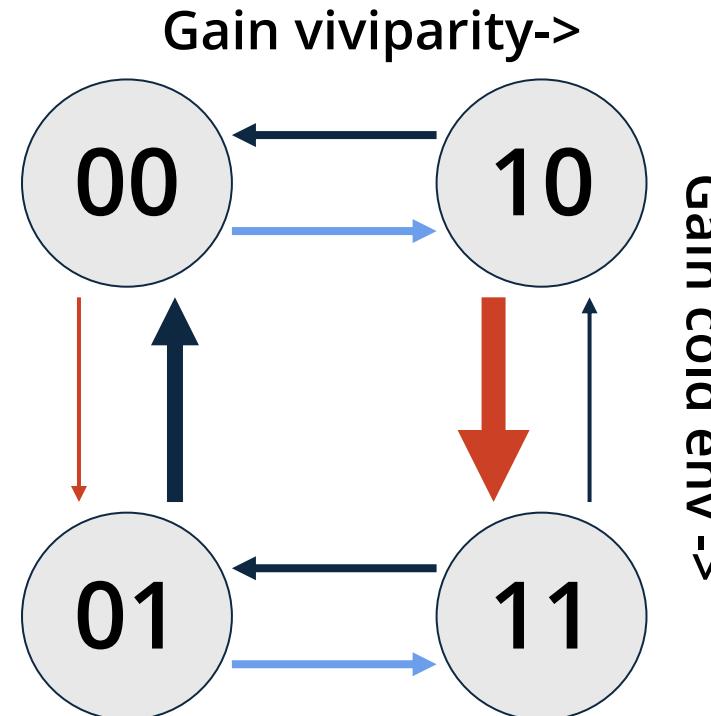
Markov models (e.g. Pagel's correlation)

Consider two binary characters (e.g. viviparity & cold env)

Independent model



Correlated model



In the hypothetical example,
 $q_{[01 \rightarrow 11]} = q_{[00 \rightarrow 10]}$
 $q_{[10 \rightarrow 11]} > q_{[00 \rightarrow 11]}$

Viviparity is a Granger cause
of cold environment
(e.g. environmental filtering)

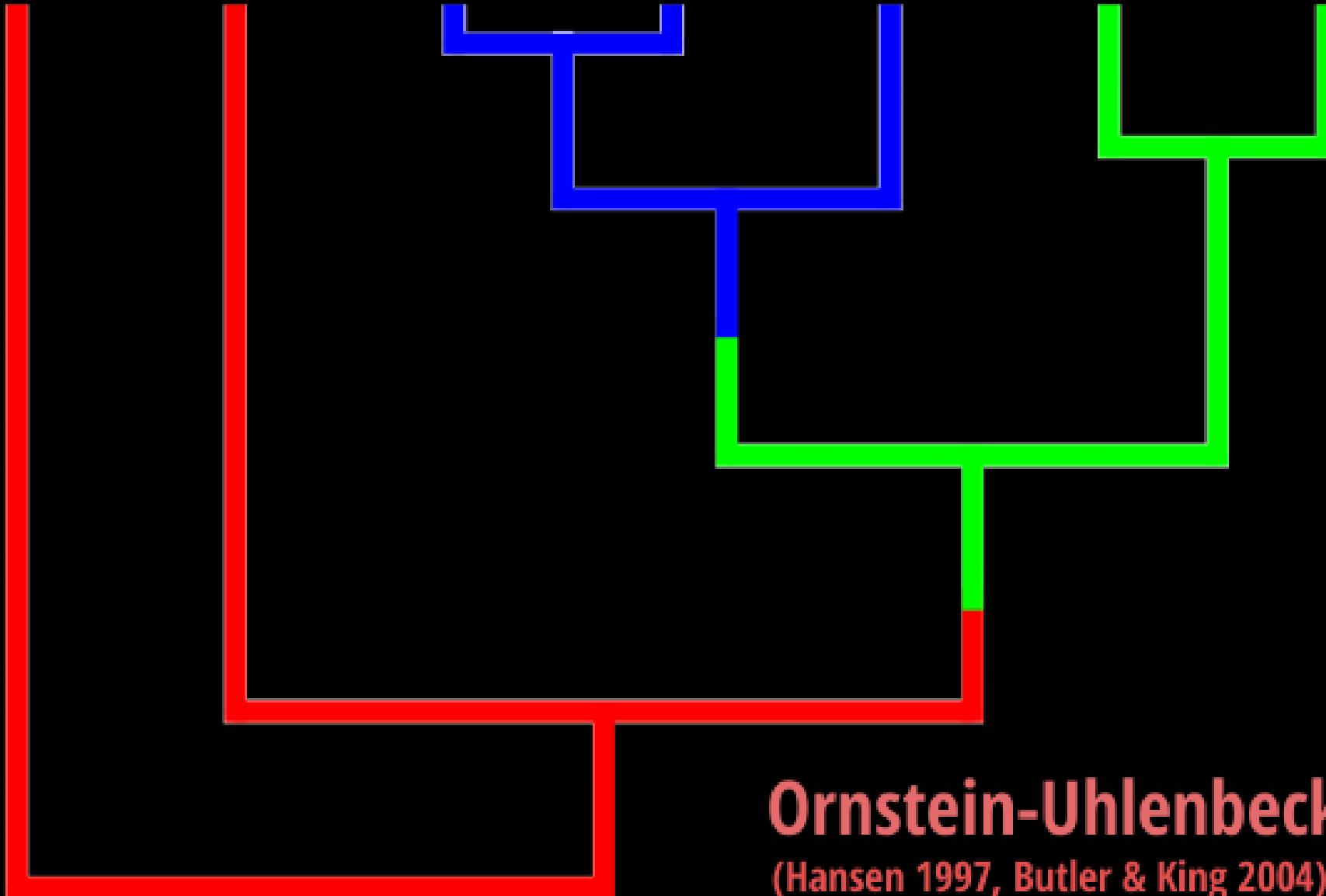
Ornstein-Uhlenbeck models

Univariate OU models - *ouch, OUwie, etc.*

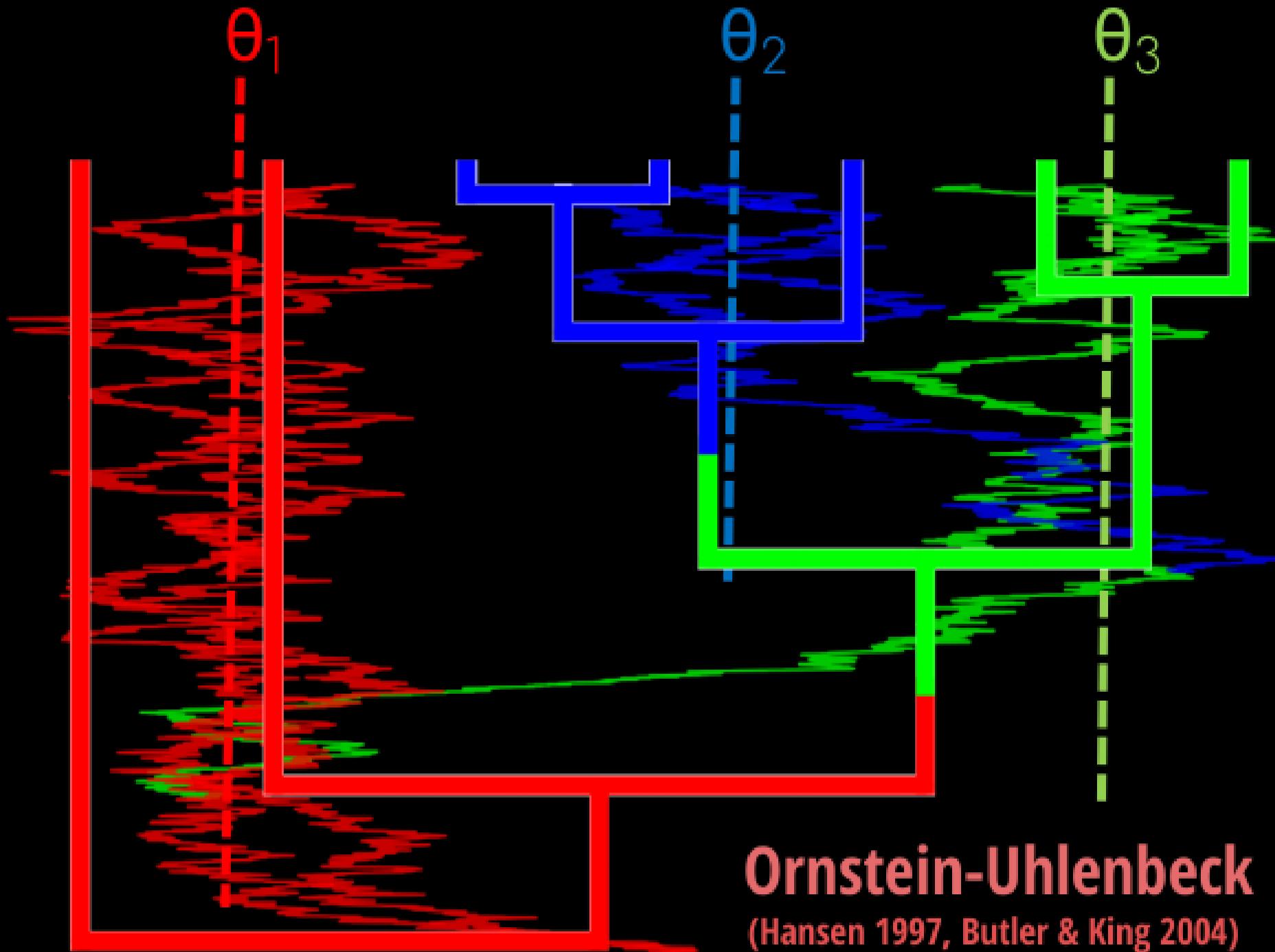
OUBM model - *slouch* (Hansen et al. 2008)

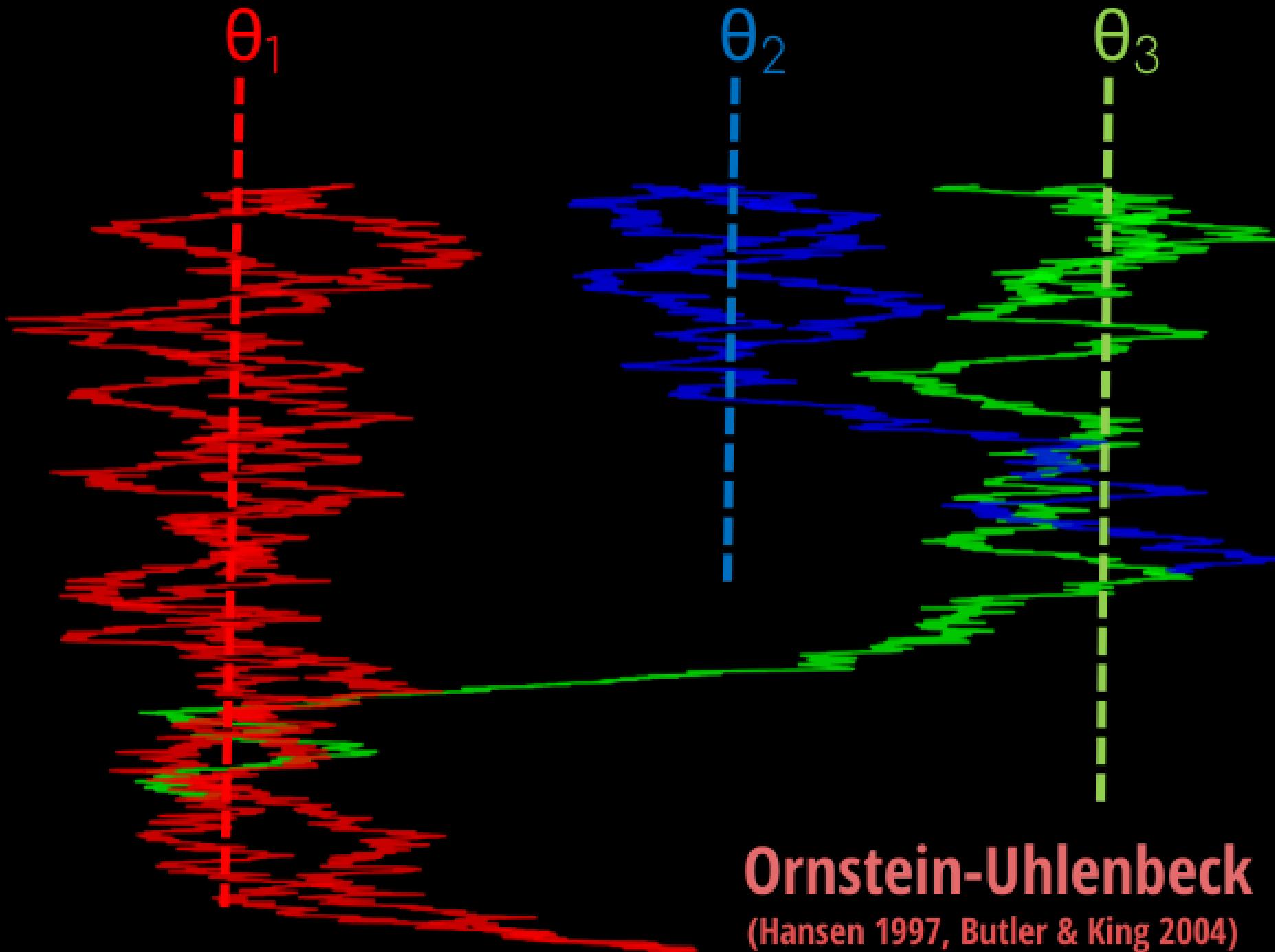
Assumes X → Y;

estimates strength of causal effect (adaptation) in units
proportional to time: α

θ_1 θ_2 θ_3 

Ornstein-Uhlenbeck
(Hansen 1997, Butler & King 2004)





Ornstein-Uhlenbeck models

OUOU model - *mvSLOUCH, layeranalyzer*

Allows effects in both directions, can be compared using model selection

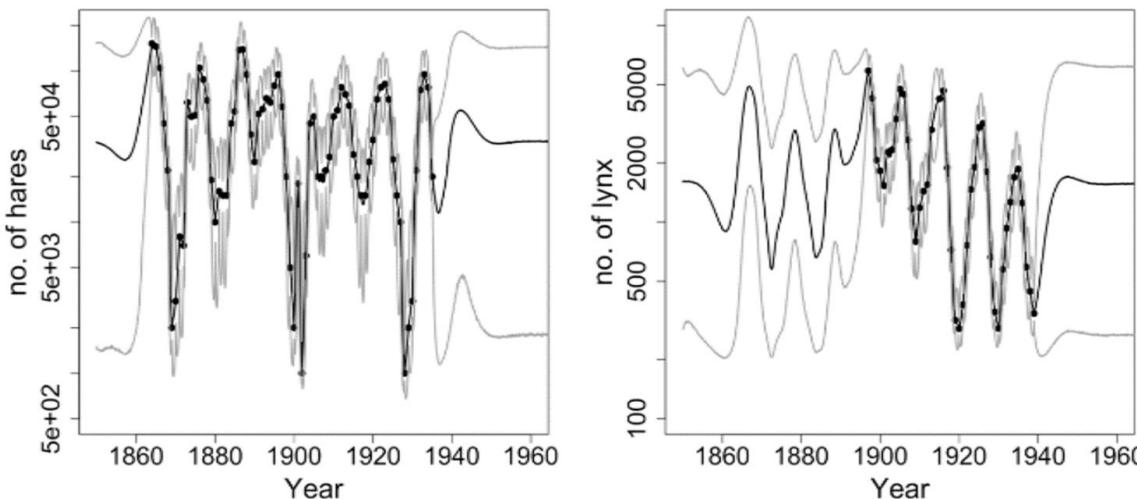
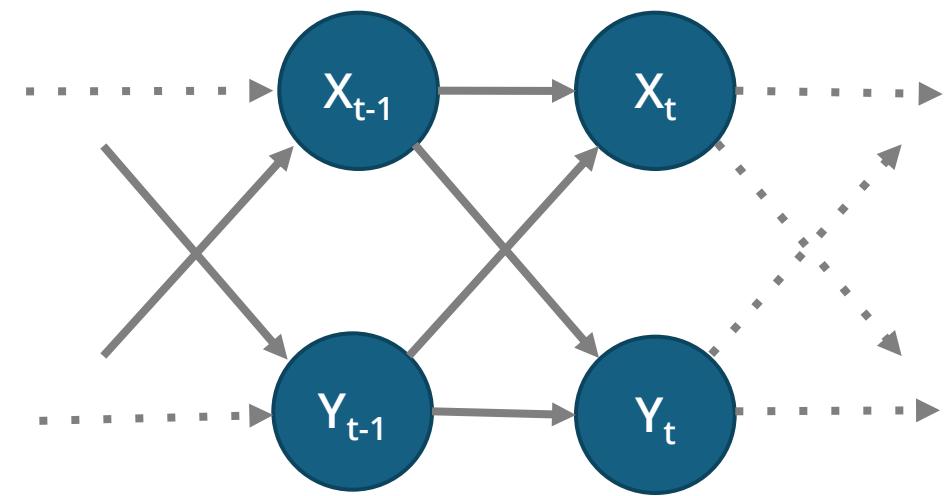


Figure 2: Lynx and hare process state inference. The plotted points are empirical data while black lines are estimated means and grey lines are 95% credibility bands. Lynx data start in 1897 and end in 1939 (with 1914 missing), while hare data start in 1864 and end in 1935. Note that the uncertainty bands make small "bubbles" between each years as the models are in continuous time and the uncertainty increases between measurements.



Ornstein-Uhlenbeck models

OUOU model - *mvSLOUCH, layeranalyzer*

Allows effects in both directions, can be compared using model selection

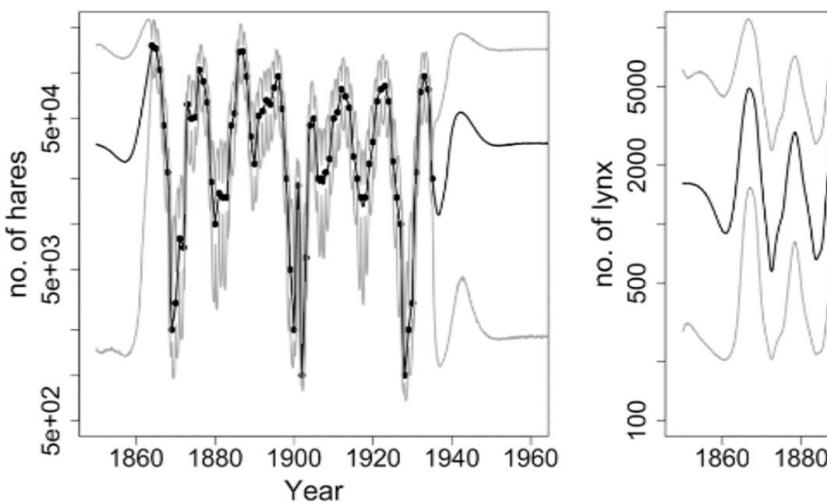


Figure 2: Lynx and hare process state inference. The plotted points are empirical data with means and grey lines are 95% credibility bands. Lynx data start in 1897 and end in 1939 (with data start in 1864 and end in 1935). Note that the uncertainty bands make small "bubbles" because the models are in continuous time and the uncertainty increases between measurements.

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<https://doi.org/10.1093/sysbio/syac079>
Advance Access Publication December 28, 2022

Model Selection Performance in Phylogenetic Comparative Methods Under Multivariate Ornstein–Uhlenbeck Models of Trait Evolution

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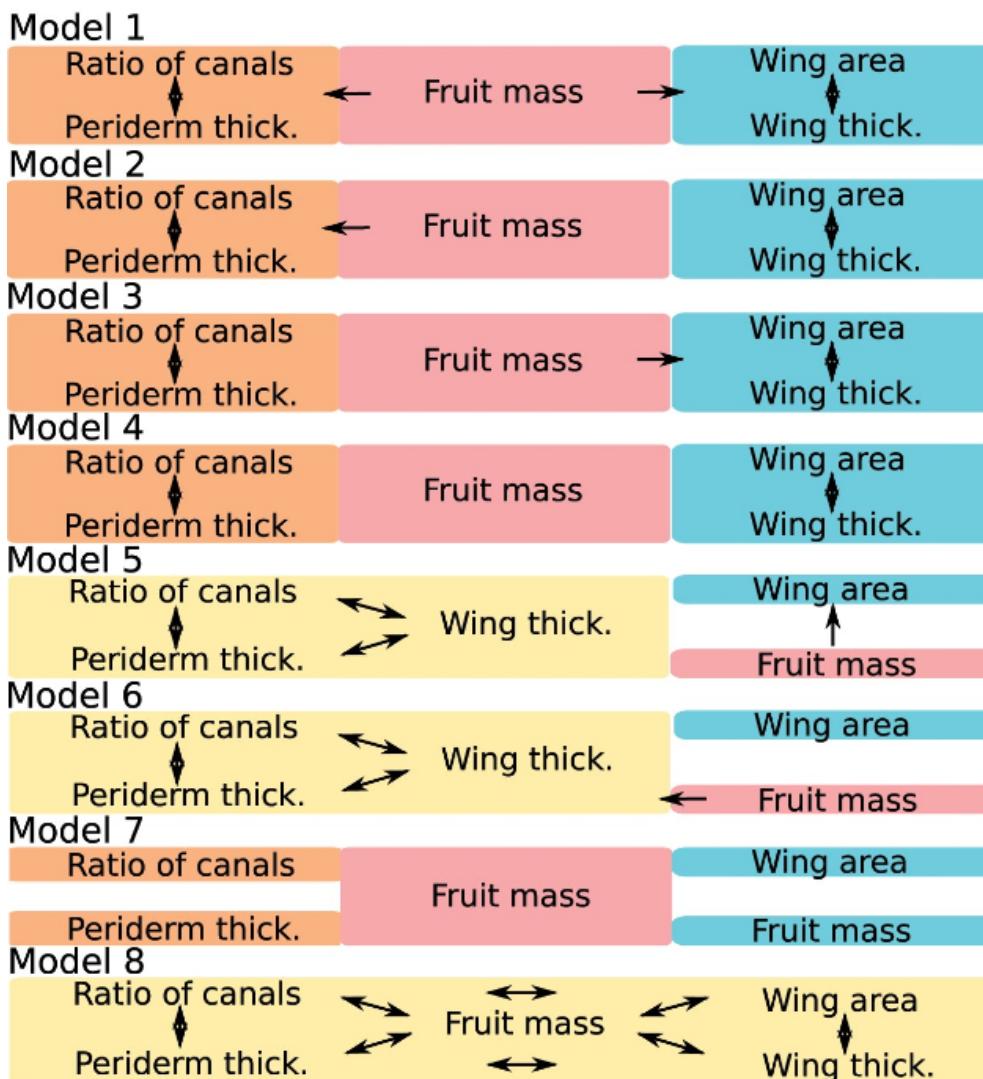
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Fig 3. Bartoszek et al. 2023. Different causal models evaluated by different structures of the A matrix



Model	$A_{Eq. (1)}$	Model	$A_{Eq. (1)}$
1	+ ? 0 0 ? ? + 0 0 ? 0 0 + ? ? 0 0 ? + ? 0 0 0 0 +	2	+ ? 0 0 ? ? + 0 0 ? 0 0 + ? ? 0 0 ? + 0 0 0 0 0 +
3	+ ? 0 0 0 ? + 0 0 0 0 0 + ? ? 0 0 ? + ? 0 0 0 0 +	4	+ ? 0 0 0 ? + 0 0 0 0 0 + ? ? 0 0 ? + 0 0 0 0 0 +
5	+ ? 0 ? 0 ? + 0 ? 0 0 0 + 0 ? ? ? 0 + 0 0 0 0 0 +	6	+ ? 0 ? ? ? + 0 ? ? 0 0 + 0 0 ? ? 0 + ? 0 0 0 0 +
7	+ 0 0 0 0 0 + 0 0 0 0 0 + 0 0 0 0 0 + 0 0 0 0 0 +	8	? ?

Advanced methods in Causal Inference

Interventions & counterfactuals can be evaluated from DAGs

What would have happened, what will happen w/do()

What would the diversification rate for *Liolaemus* been if the Andes didn't uplift?

What proportion of the elevated diversification rate of *Liolaemus* can be attributed to viviparity?

Evaluate the DAG with the estimated causal effects, but with values of causal factors set to specified values.

Do we have PCM “Amnesia”?

Once you commit to one causal explanation, you restrict all others.

PCM practice - We share trees, same clades

Different studies foreground different focal predictors

Background everything else (e.g. BM of residuals or hidden states) often despite previous studies

You can't explain the same thing twice unless:

- 1) they are part of the same causal chain
- 2) one is spurious or misestimated.

A plausible DAG may be wrong, but alternative DAGs are likely restricted to a similar distribution of explanatory factors.

Committing to one causal scenario (e.g. the Andes causes diversification of lizards) can necessitate new explanations for other clades (why didn't this Andean clade diversify?)

Can we build & synthesize, rather than background previous findings?

Case Study I

Annals of Botany **134**: 1131–1138, 2024

<https://doi.org/10.1093/aob/mcae107>, available online at www.academic.oup.com/aob

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ORIGINAL ARTICLE

The smallest angiosperm genomes may be the price for effective traps of bladderworts

František Zedek^{1,*}, Jakub Šmerda¹, Aneta Halasová¹, Lubomír Adamec², Adam Veleba¹, Klára Plačková¹ and Petr Bureš^{1,*}

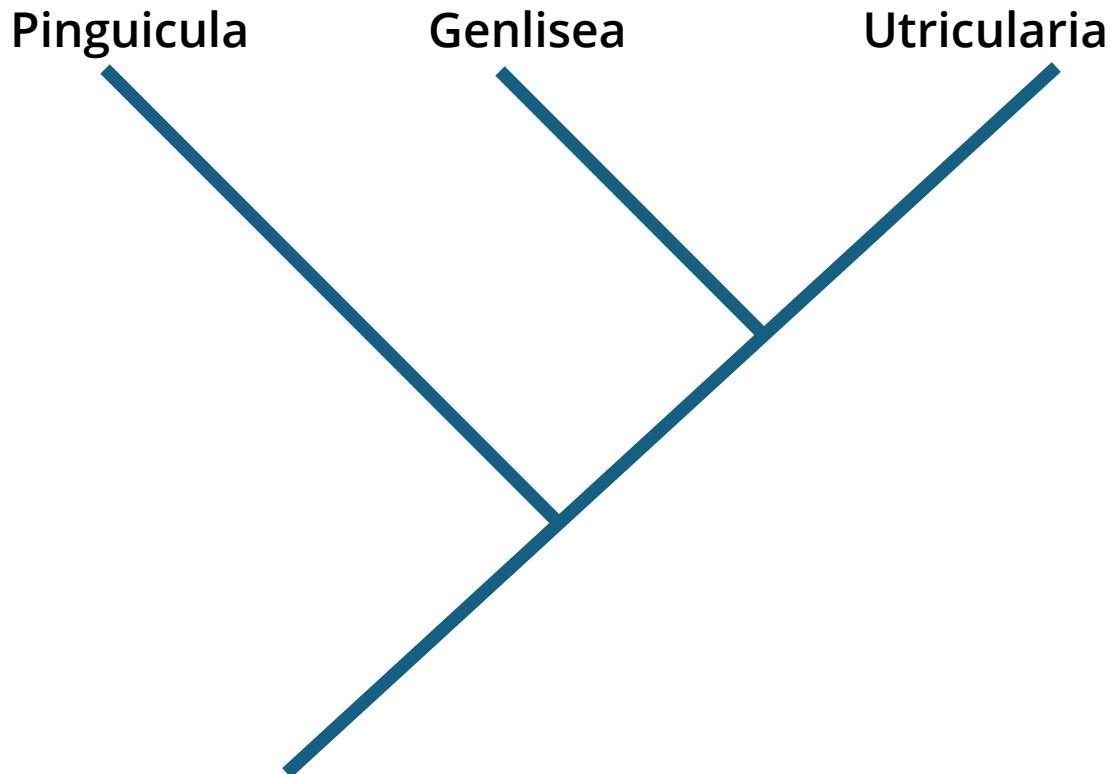
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²Department of Experimental and Functional Morphology, Institute of Botany of the Czech Academy of Sciences, Dukelská 135, 37901, Třeboň, Czech Republic

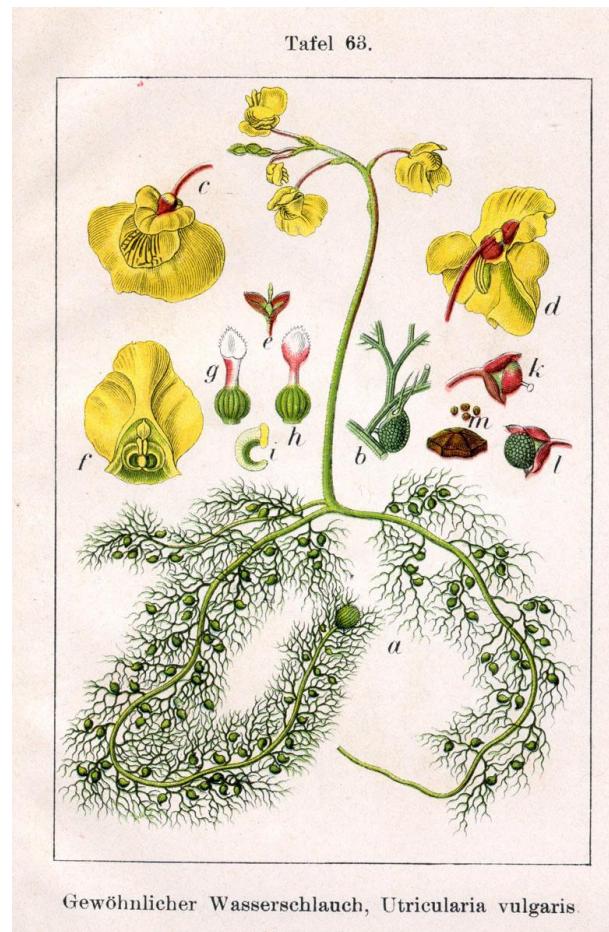
*For correspondence. E-mail fzedek@gmail.com or bures@sci.muni.cz

Miniature genomes in the Lentibulariaceae

Smallest plant genomes ~61Mbp
(*Genlisea tuberosa*) Fleischmann et al.
2014.



Bladderworts (*Utricularia*)
Aquatic/terrestrial/epiphytic
Suction traps, no true roots
All genomes tiny



Corkscrew plants (*Genlisea*)
Terrestrial
Passive “lobster traps”
Some tiny genomes



Butterworts (*Pinguicula*)
Terrestrial, outgroup
Passive flypaper traps
Normal genome sizes

Case Study II

ORIGINAL ARTICLE



doi:10.1111/evo.13734



Adaptive radiation and the evolution of nectarivory in a large songbird clade

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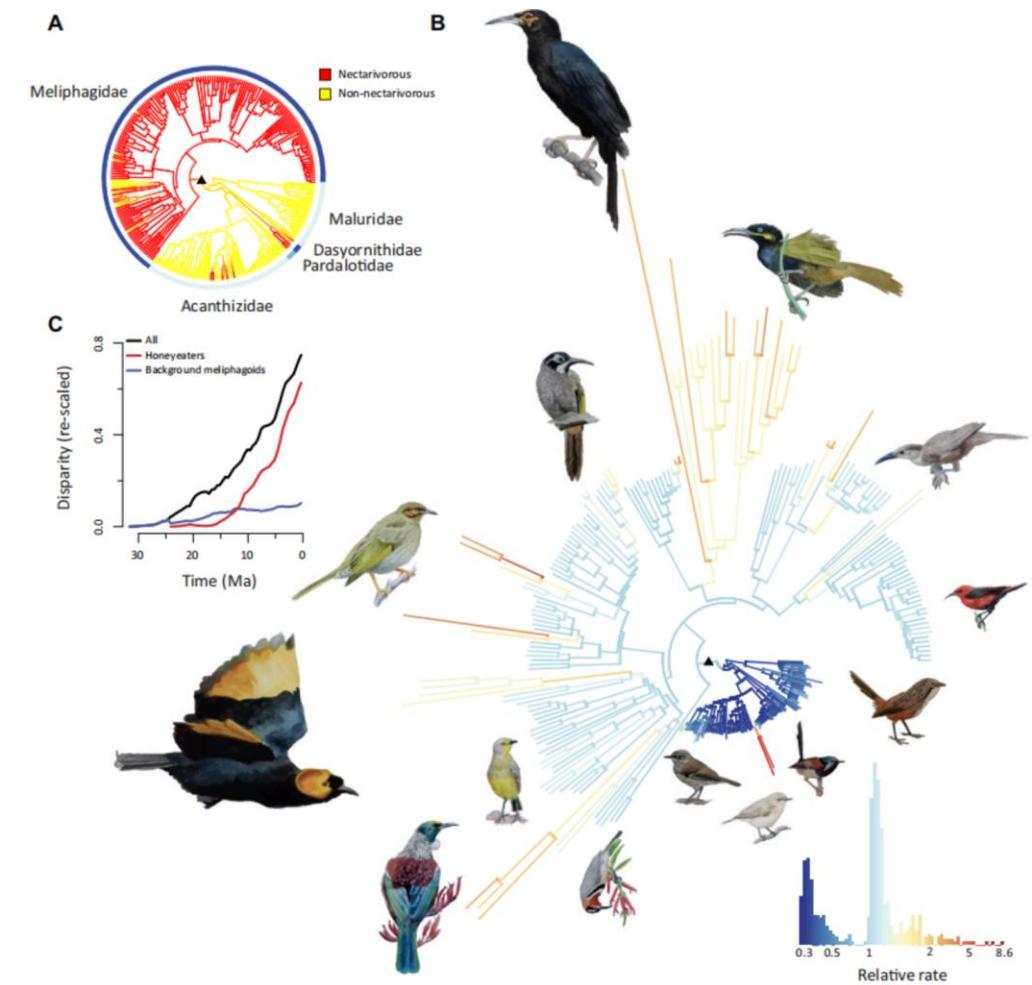
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Divergence & Diversification of the Honeyeaters (Meliphagidae)

~185 species mostly in Australia & New Guinea

Either nectarivorous, insectivorous, frugivorous, or a combination.



Case Study I

Group discussion 1.1 & 1.2	10 minutes
Full class discussion 1	5 minutes
Group discussion 1.3	5 minutes
Full class discussion 2	5 minutes
Group discussion 1.4 & 1.5	5 minutes
Full class discussion 3	10 minutes

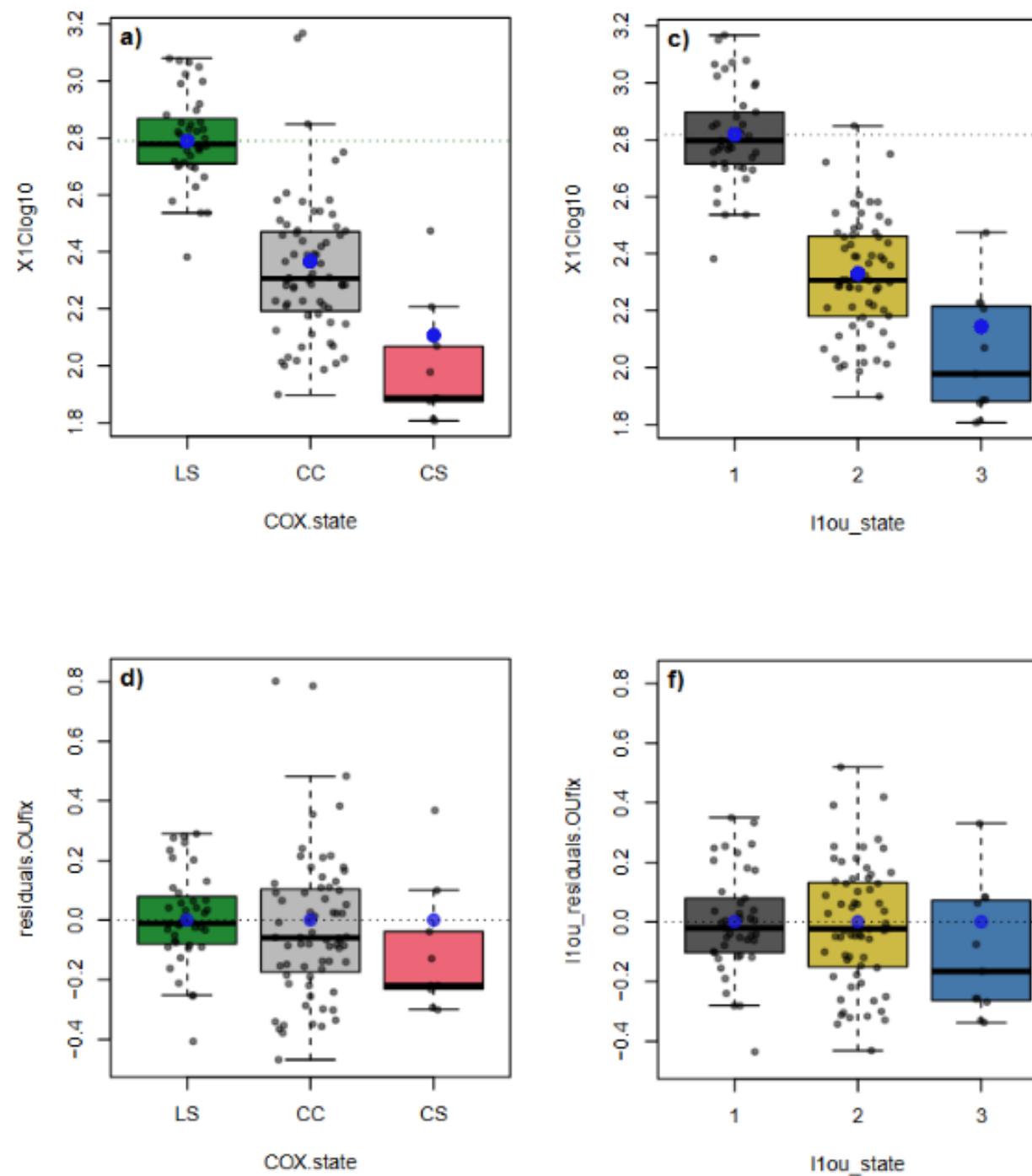
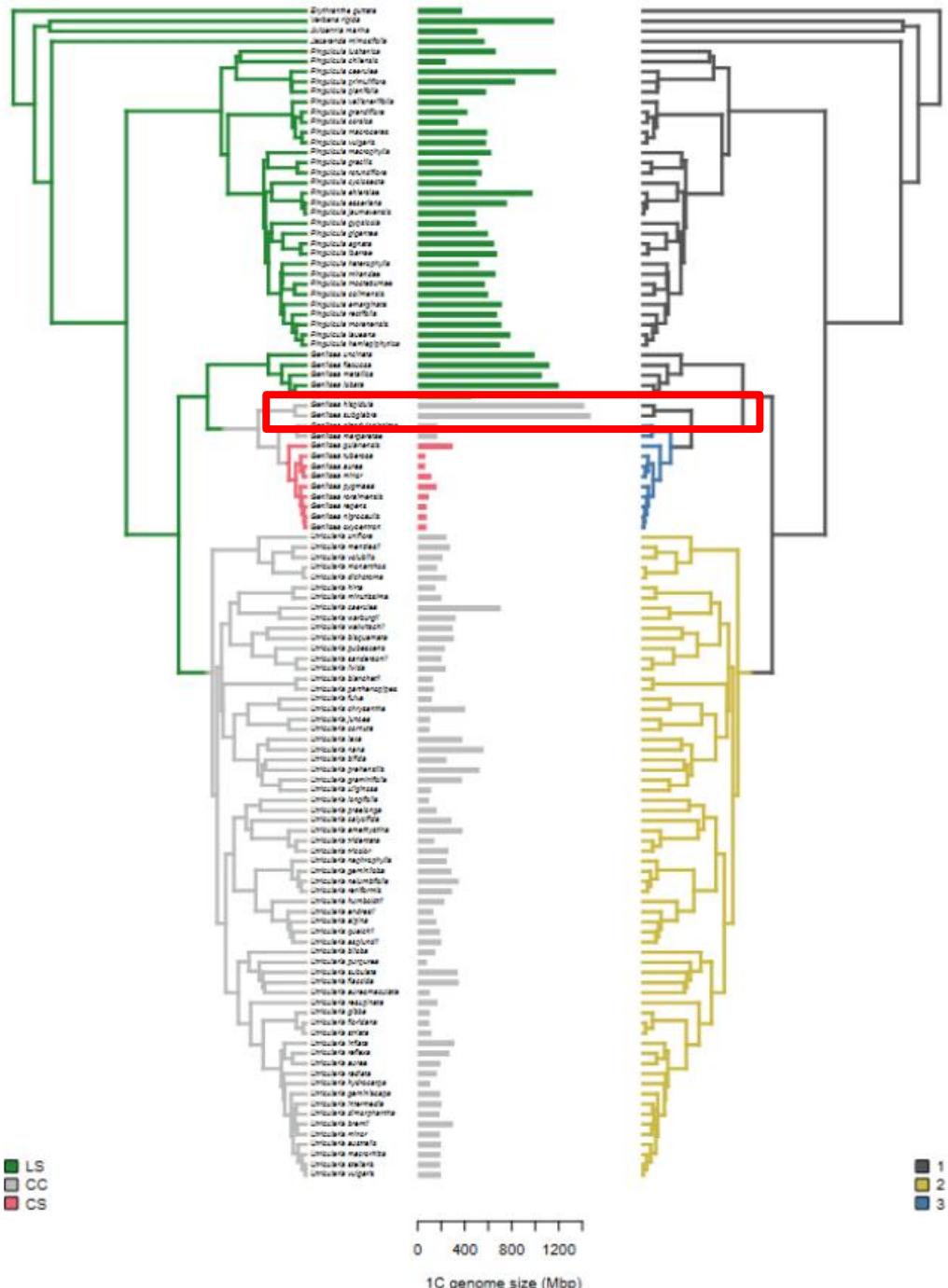


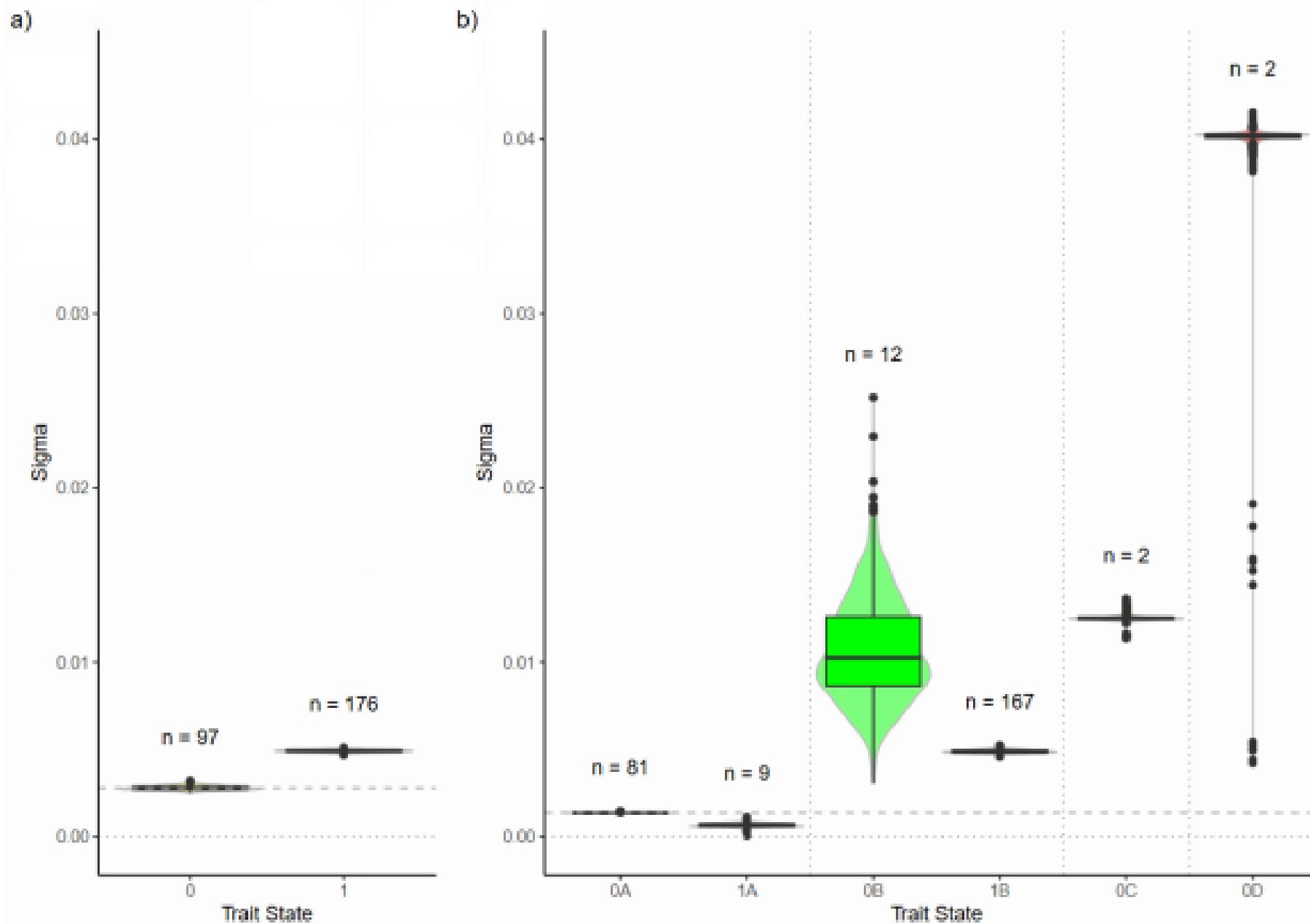
Figure 4: Distribution of \log_{10} genome sizes (a, c) and phylogenetic linear model residuals (d, f) between species carrying different COX mutations (a, d), or which are part of different OU-regimes (c, f). Gray points are genome sizes with added jitter, blue points are inferred expected values per group under the phylogenetic li

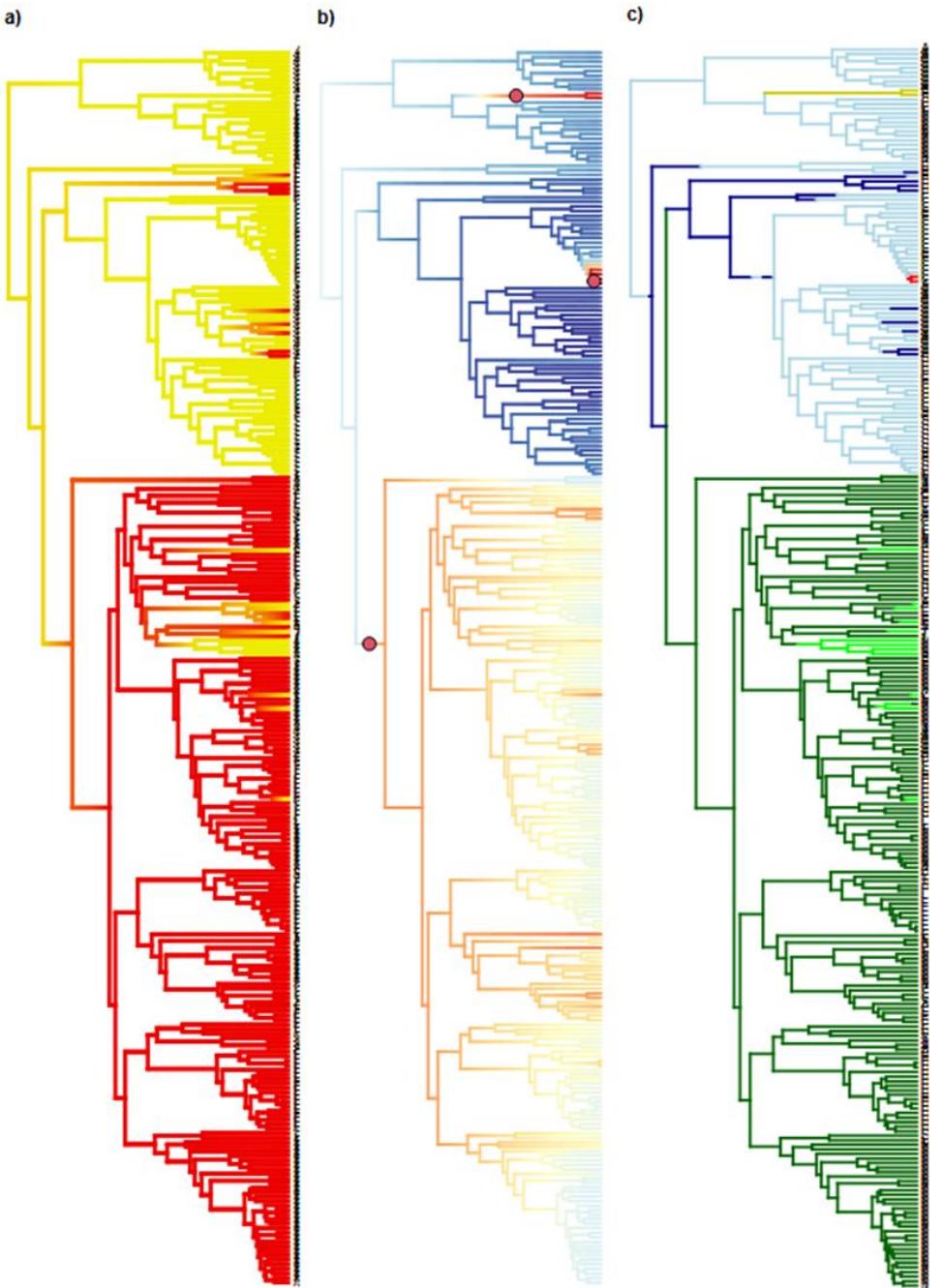


Case Study II

Group discussion 2.1 & 2.2	10 minutes
Full class discussion 1	5 minutes
Group discussion 2.3	10 minutes
Full class discussion 2	10 minutes
Group discussion 2.4 & 2.5	5 minutes

Figure 7: State dependent rates of Brownian motion for Meliphagidae body size (pPC1); a) rates for non-nectarivorous (0) and nectarivorous (1) lineages; b) rates for amalgamated states of nectarivity (0, 1) and BAMM rate regimes (A-D). Both analyses are based on 1000 stochastic mappings each,





Marki *et al.* analyses: TL;DR

1. Reconstruct nectarivory using ER model,
1000 stochastic mappings
→ origin around MRCA of honeyeaters
and other clades
2. pPCA on four traits
→ PC1 explains 65.3% variation, reflecting
overall size
3. mvBM model: rates of evolution of PC1,
separate for nectarivorous vs not
→ higher rates for nectarivorous
4. BayesTraits/BAMM on PC1 to find shifts in
rates of evolution
→ rate shift at base of honeyeaters, plus
1-2 smaller clades

Schedule

8:30 - 9:30	Lecture
9:30 - 9:45	Questions/Discussion
9:45 -10:00	Group building/introductions
10:00 - 10:30	Coffee break
10:30 - 11:00	Case Study I
11:00 - 11:30	Case Study II
11:30 - 12:00	Questions/Discussion

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