Darwin vs Lamarck
Round 2

Biologia Evoluzionistica 2025/2026
Fabrizio Mafessoni
Universita' di Trieste

Today last lecture of the «evolutionary biology» part of the course

- We are going to try to wrap up and put everything together
  - Inheritance systems
  - Basic principles of evolution
  - Coevolutionary dynamics
  - Environmental change and plasticity
  - Modern takes on evolution (Extended Evolutionary Synthesis)

Lamarck

Learning and plasticity

Non-Genetic inheritance

Darwin

Natural selection

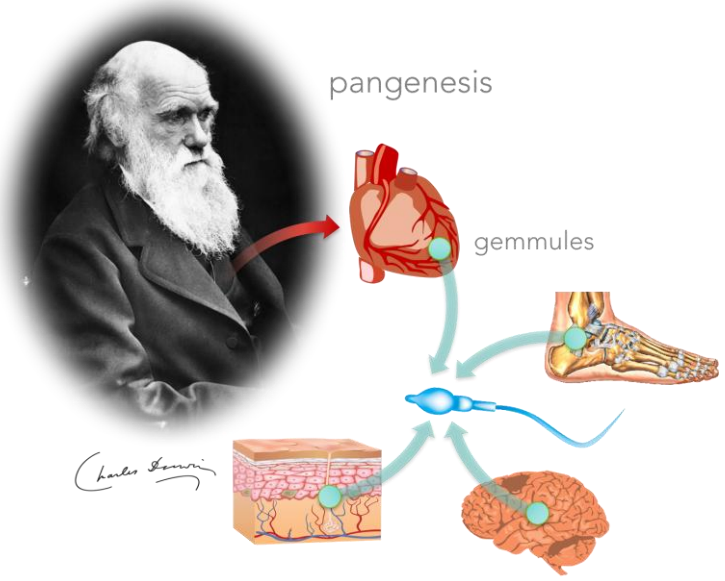
Genetic inheritance

Round 1: Darwin

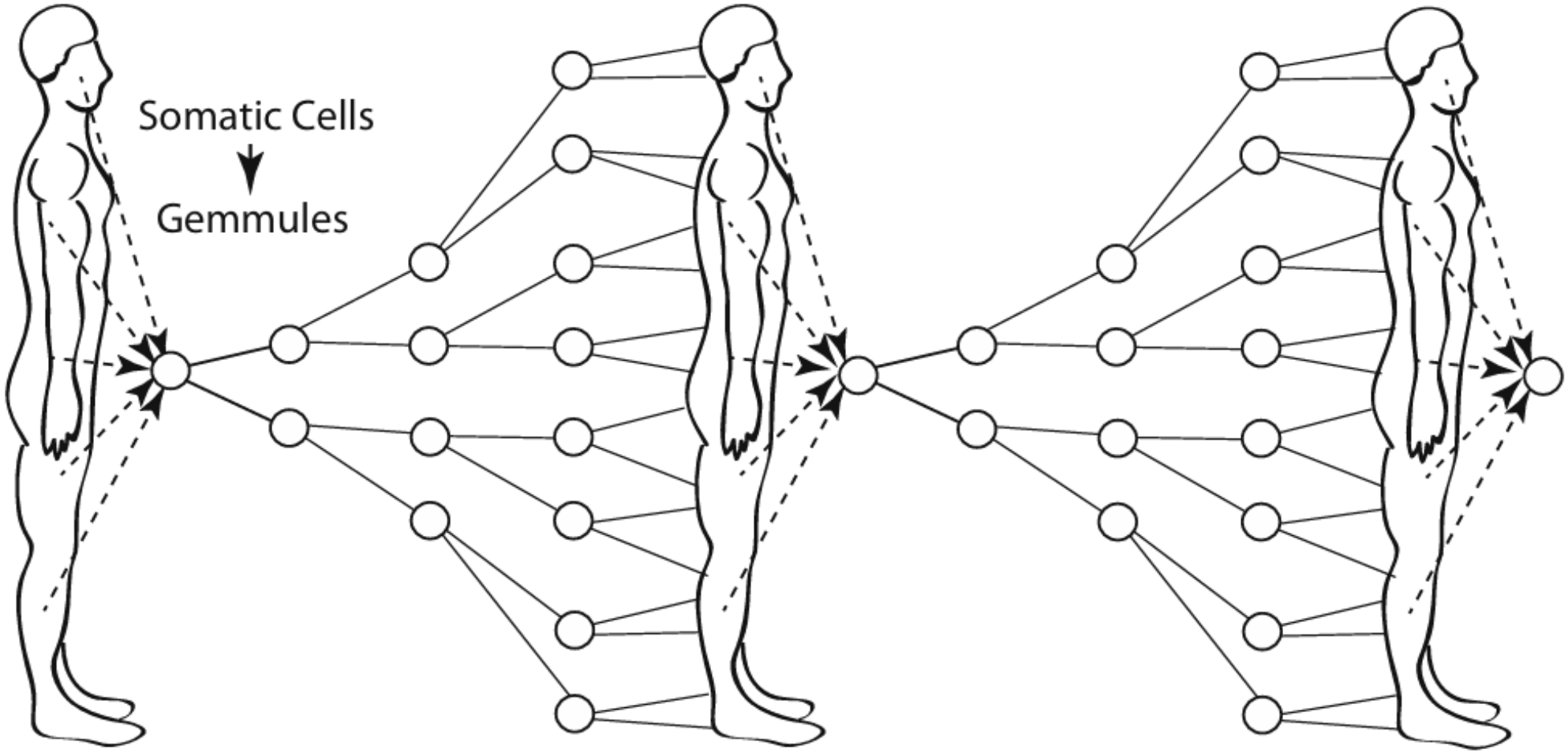
- «Darwinian evolution» works using three basic ingredients (variability, inheritance and natural selection), fitting empirical data and theoretical models
- Lamarck’s use and disuse fails to make good predictions (e.g. mutilations)

Round 1: Darwin

- However, Darwin's and Lamarck's inheritance model were not as different
- Darwin's pangenesis suffered of «blending inheritance»

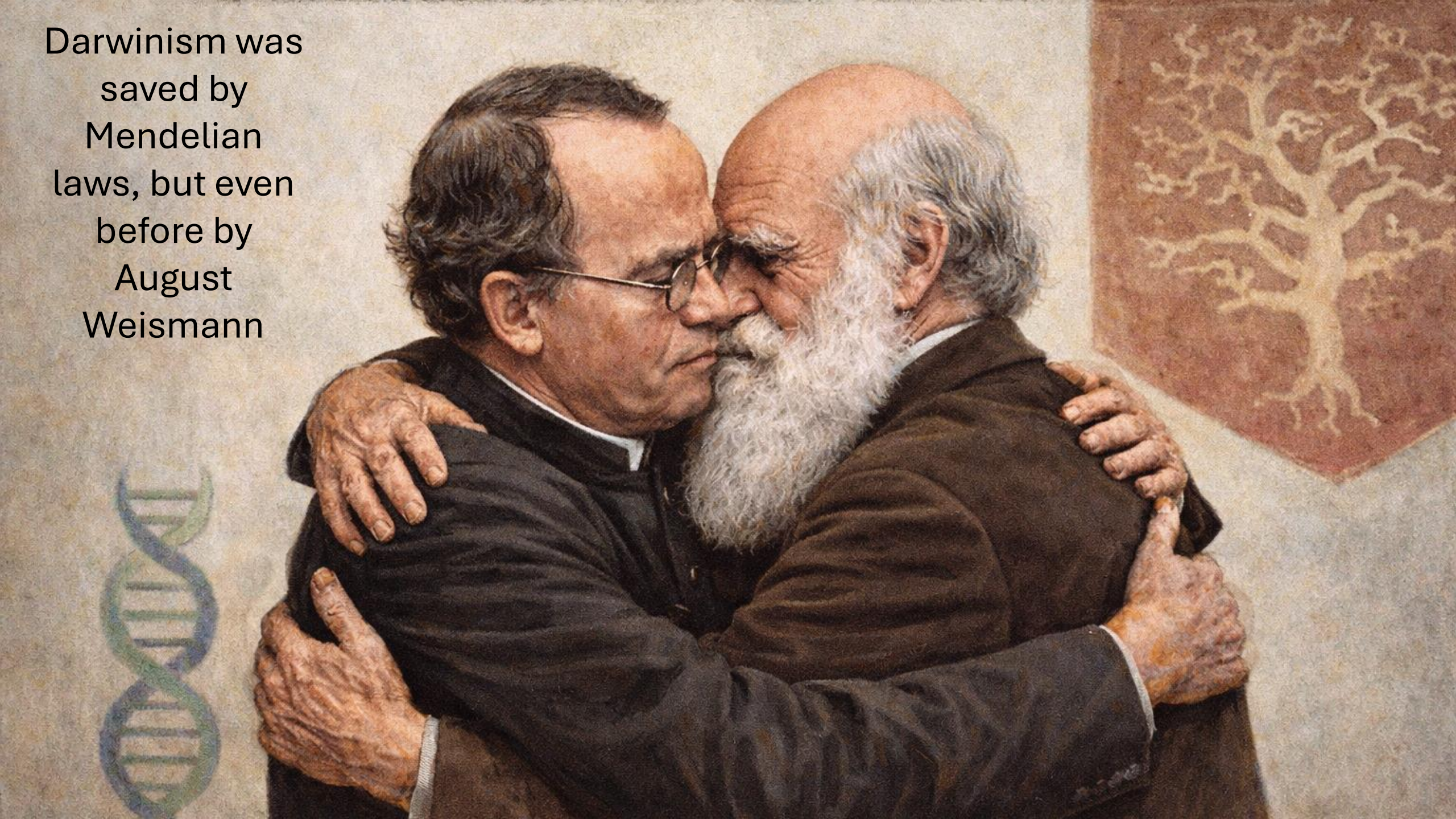


(a) Charles Darwin's Pangenesis Theory

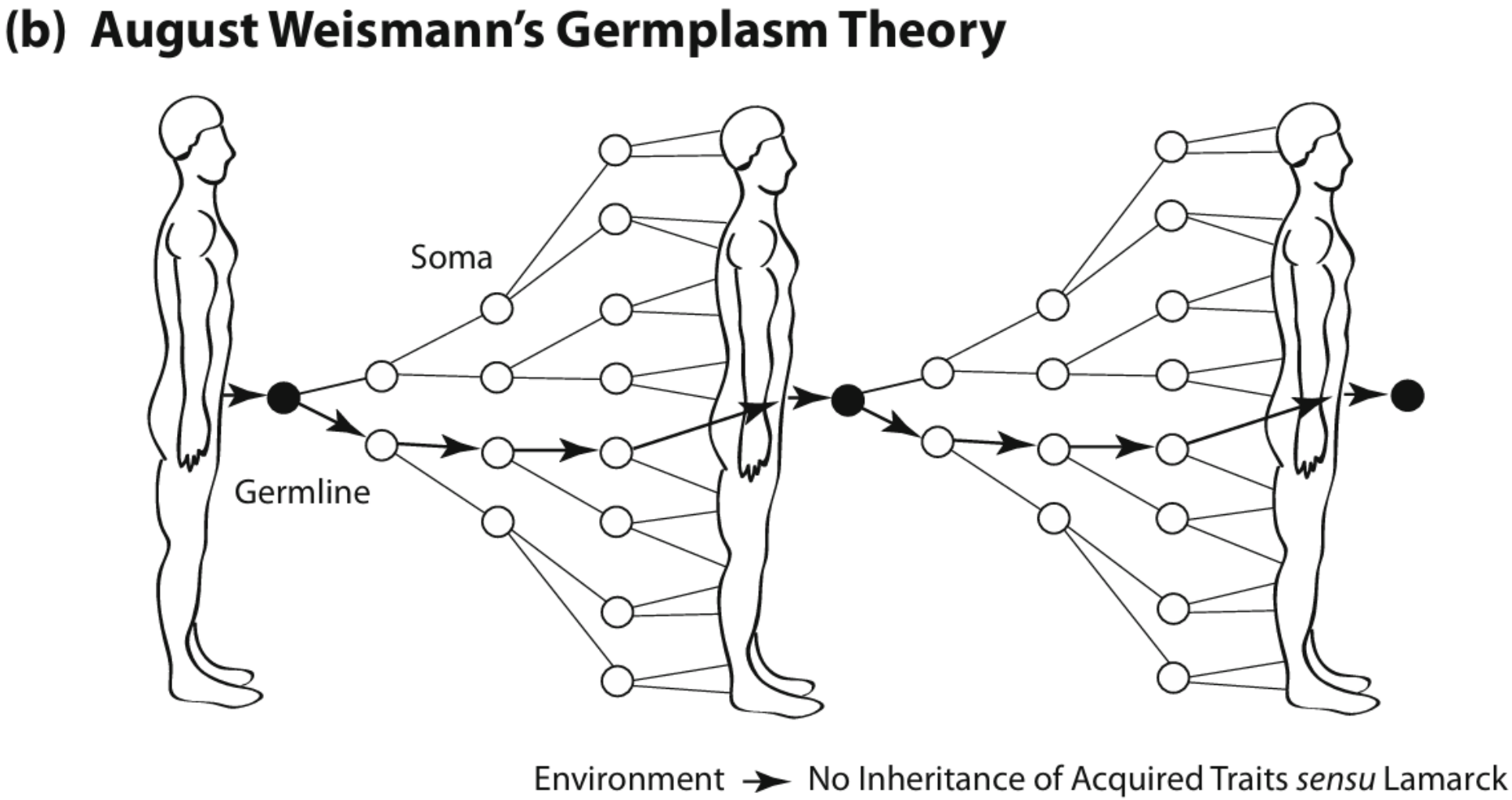


Environment → Modified Gemmules → Inheritance of Acquired Traits

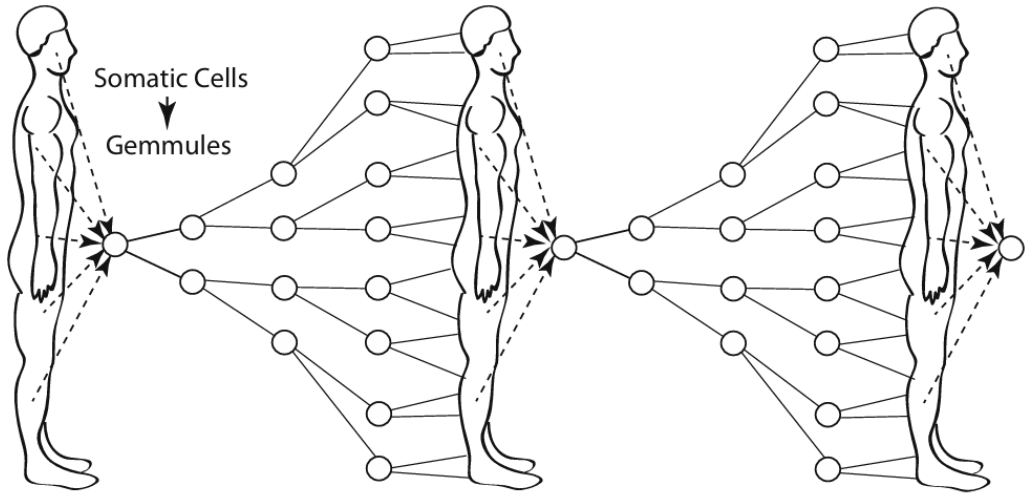
Darwinism was saved by Mendelian laws, but even before by August Weismann



Weismann's barrier (about 1880-1890)

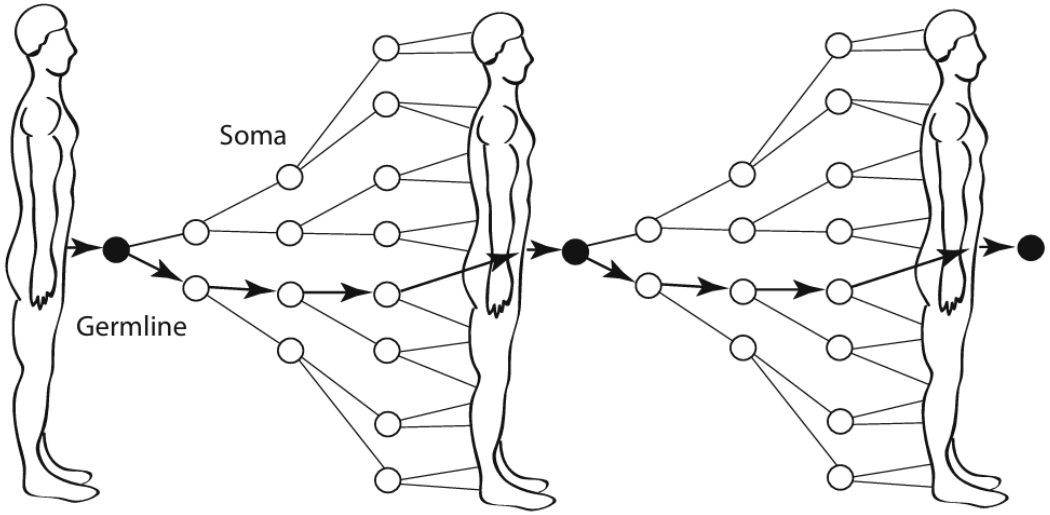


(a) Charles Darwin's Pangenesis Theory



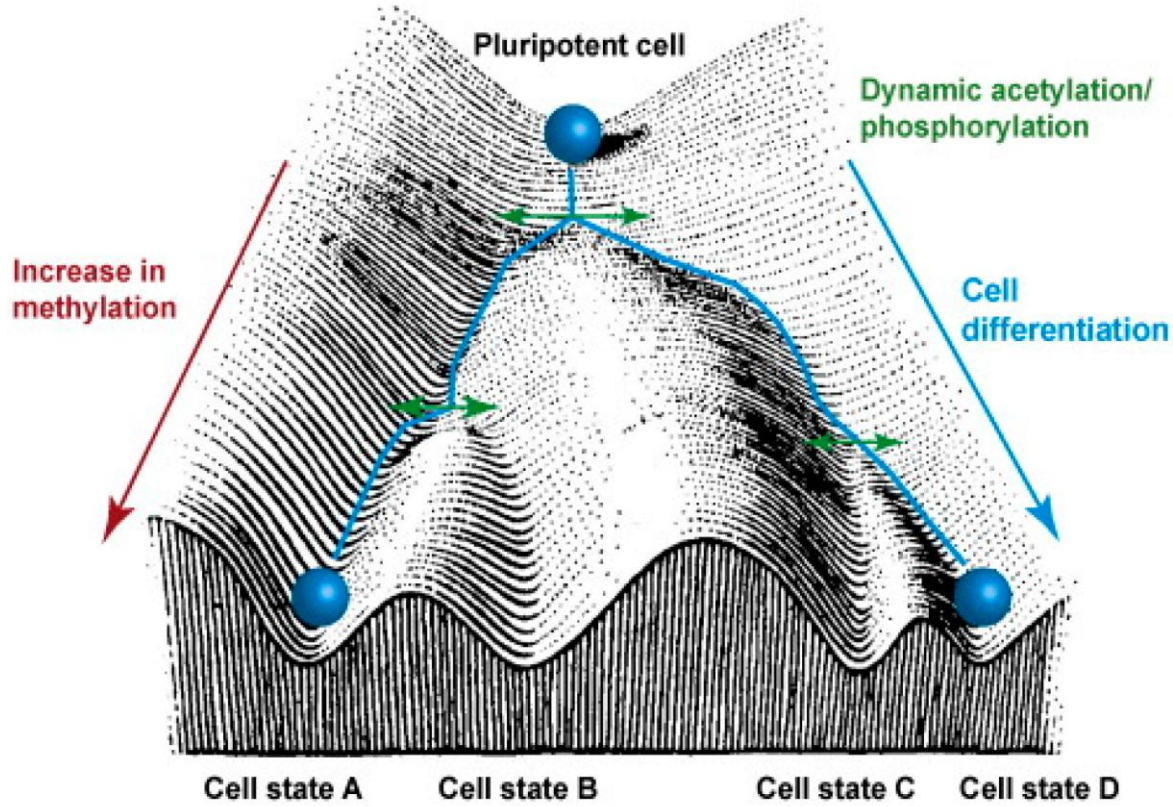
Environment → Modified Gemmules → Inheritance of Acquired Traits

(b) August Weismann's Germplasm Theory

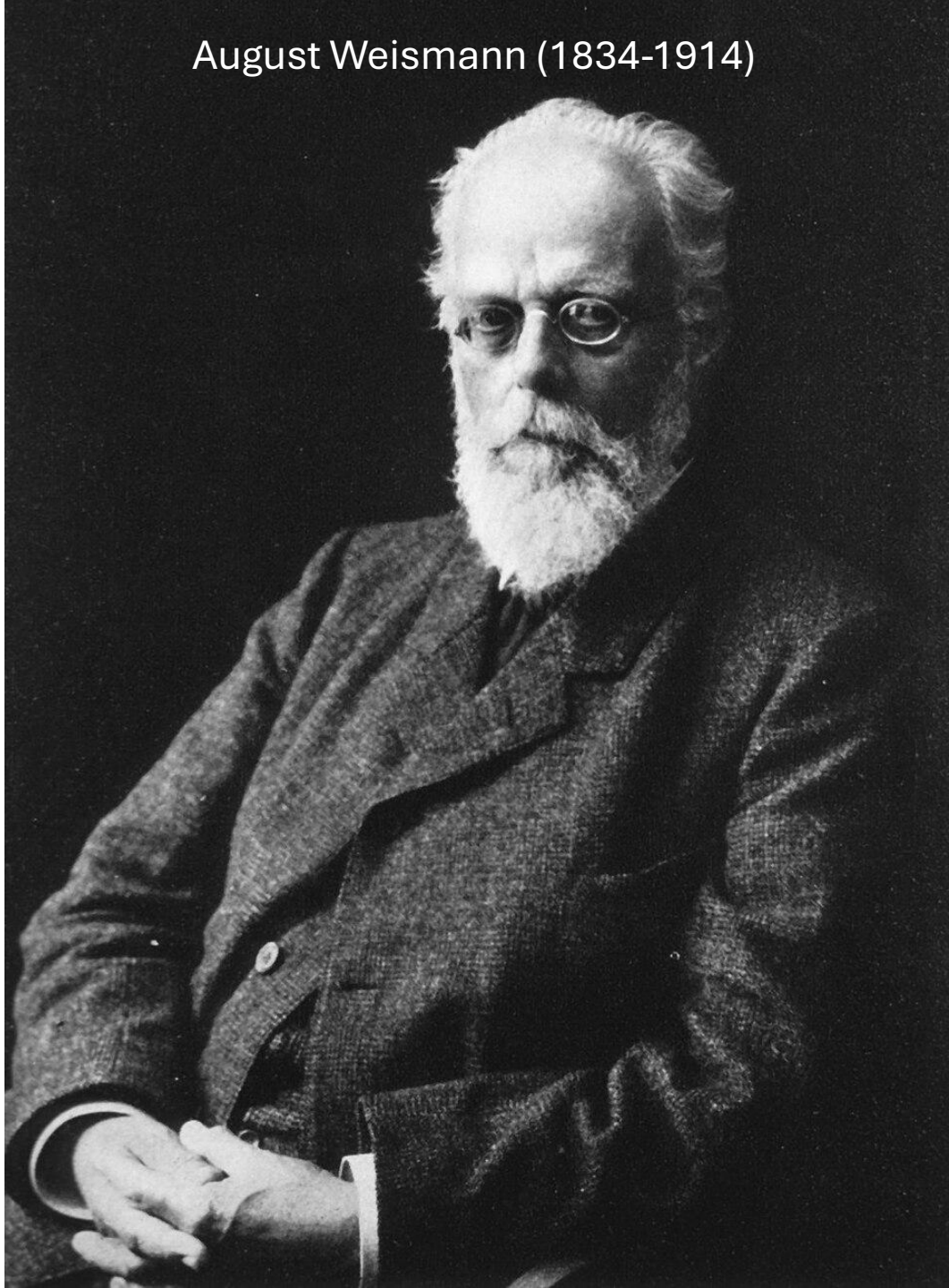


Environment → No Inheritance of Acquired Traits *sensu* Lamarck

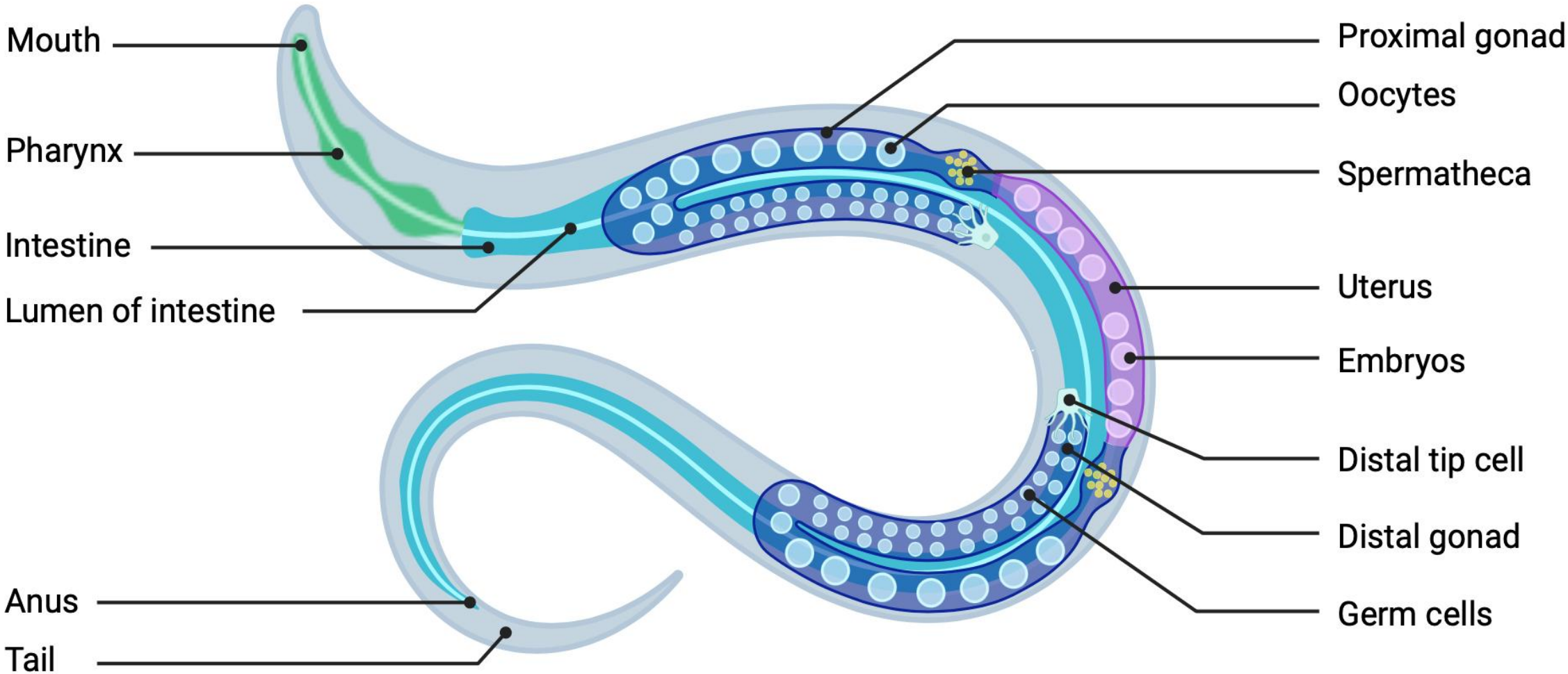
Cell differentiation is a key component of both Darwin's pangenesis and Weismann's germplasm theory, but in Weismann's there is no leakage of information from the soma/the "reset" is complete



Was Weismann (and our idea of Darwinian evolution) correct?

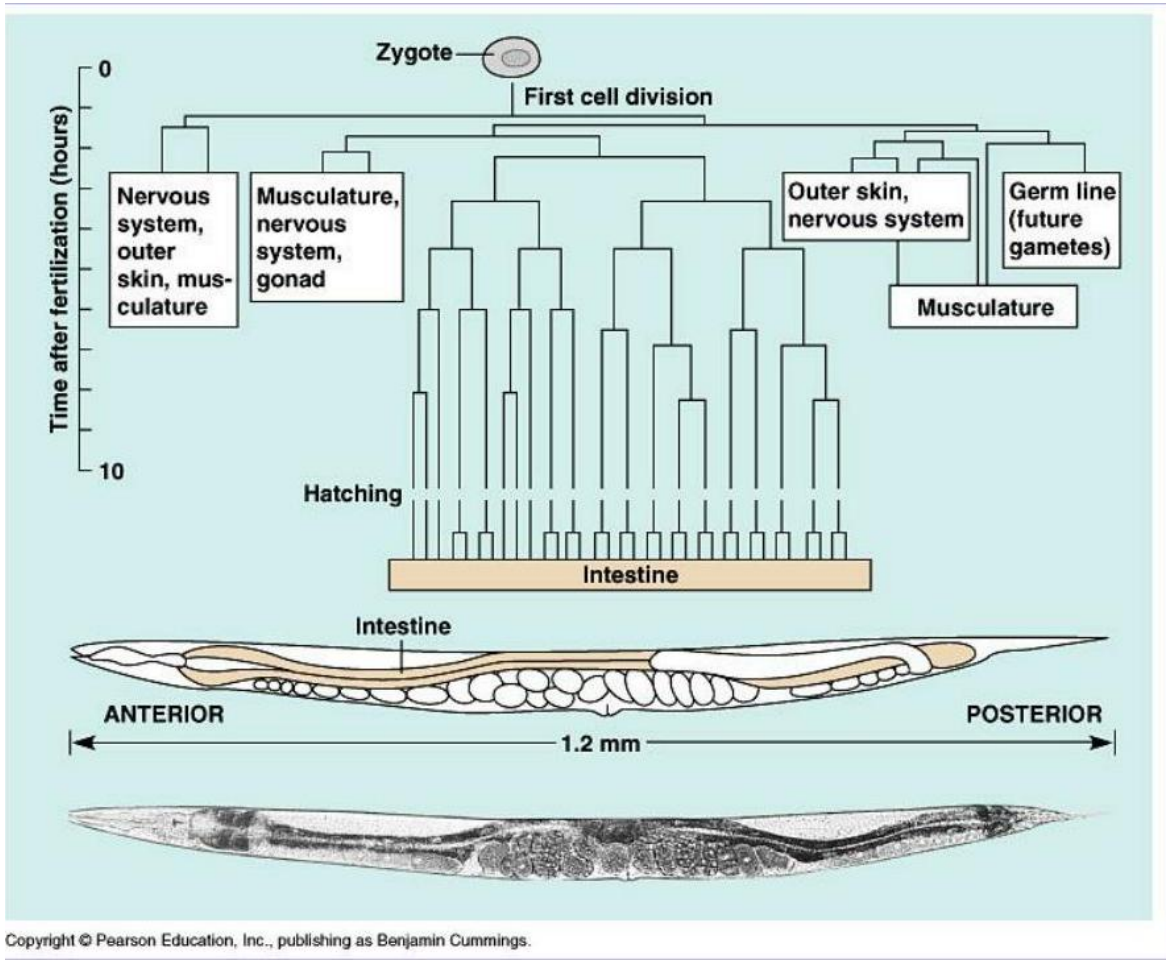


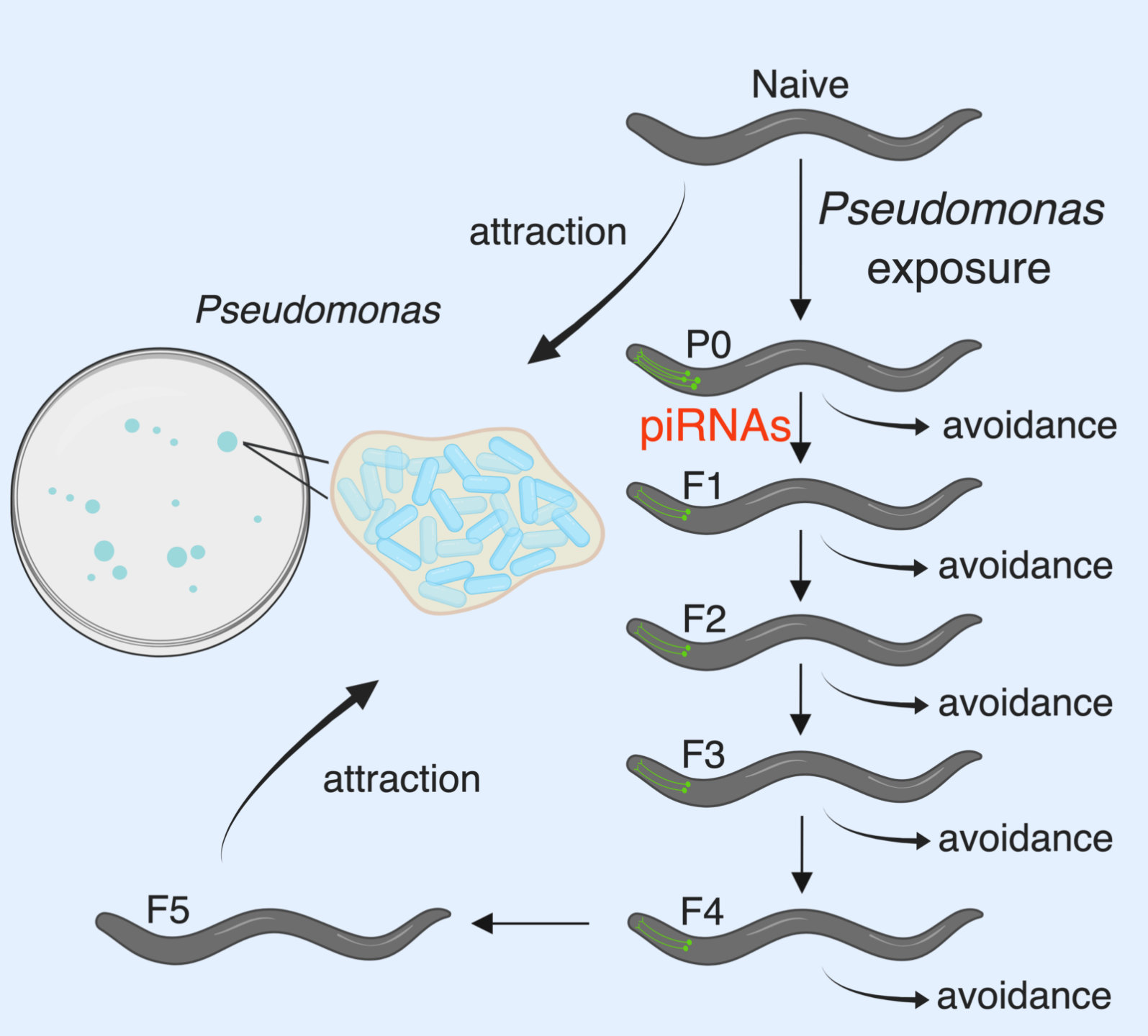
Anatomy of *Caenorhabditis elegans*



Caenorhabditis elegans

- Transparent body — every cell visible in vivo
- Fixed cell lineage — all 959 somatic cells fully mapped
- 3-day generation time, large broods — fast genetics
- Self-fertilizing hermaphrodite — easy maintenance of mutant lines
- ~20,000 genes, ~40% human orthologues
- First multicellular organism with fully sequenced genome (1998)
- First with complete connectome mapped (302 neurons, all wired)
- No DNA methylation — clean system for RNA-based epigenetics
- Transparent germline — small RNA pathways directly observable





C.elegans learns through exposure to avoid pathogens, and transmit "knowledge" to offspring

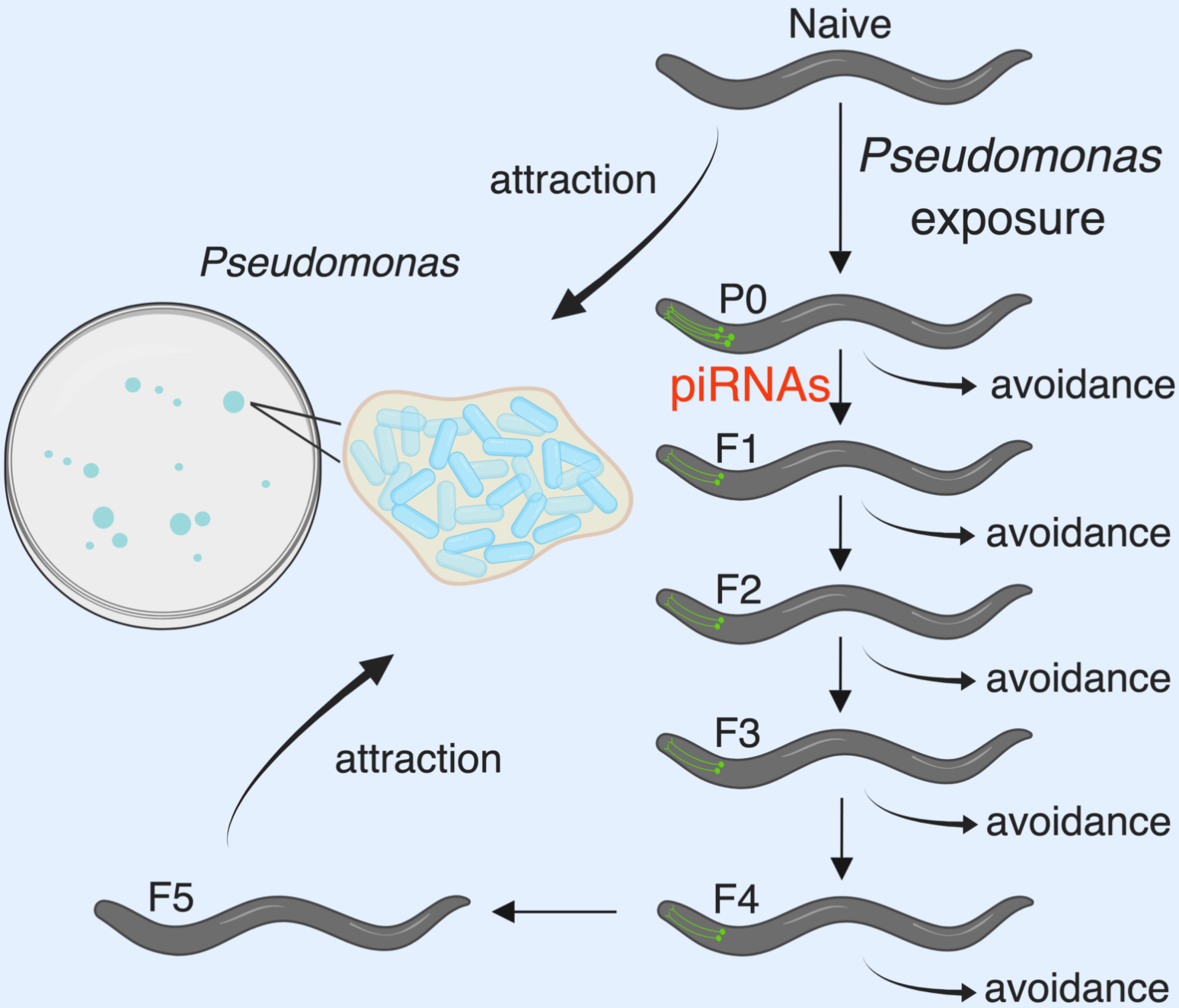
Worms trained to avoid pathogenic *Pseudomonas aeruginosa* transmit this avoidance behavior to offspring for 4 generations — without offspring ever encountering the pathogen.

The signal involves small RNAs (specifically 22G-RNAs targeting the *maco-1* neuronal gene) transmitted through the germline.

The behavior is inherited via RNA — a remarkable convergence of learning and epigenetic inheritance.

Moore et al. 2019, *Cell*; Kaletsky et al. 2020

Transgenerational epigenetic inheritance or TEI



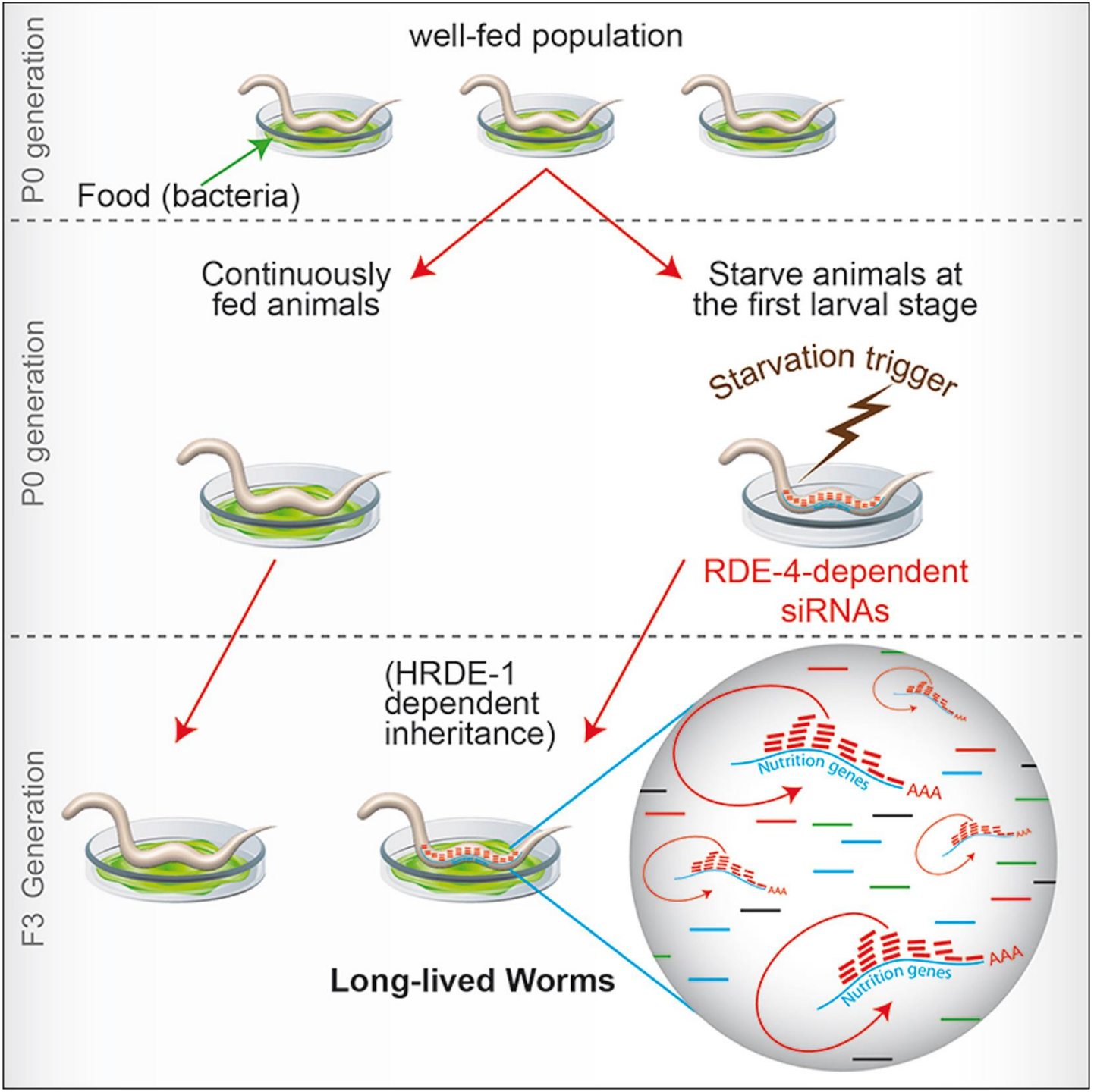
C.elegans learns through exposure to avoid pathogens, and transmit "knowledge" to offspring

Worms trained to avoid pathogenic *Pseudomonas aeruginosa* transmit this avoidance behavior to offspring for 4 generations — without offspring ever encountering the pathogen.

The signal involves small RNAs (specifically 22G-RNAs targeting the *maco-1* neuronal gene) transmitted through the germline.

The behavior is inherited via RNA — a remarkable convergence of learning and epigenetic inheritance.

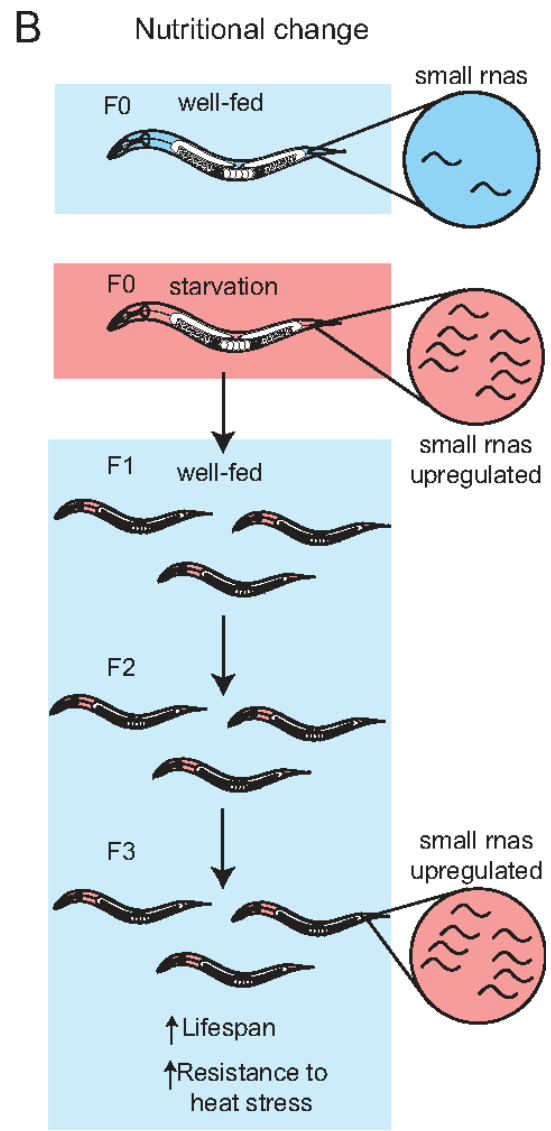
Moore et al. 2019, *Cell*; Kaletsky et al. 2020



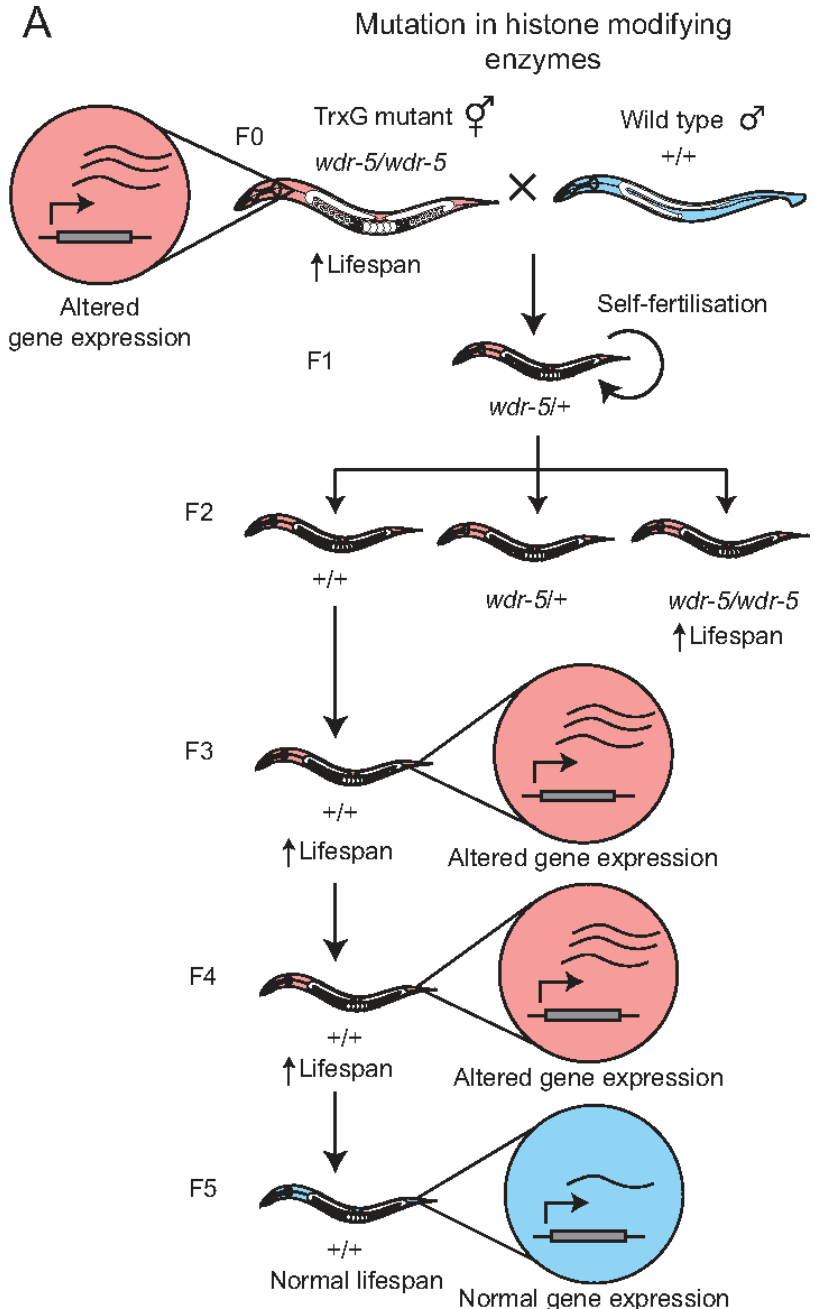
Transgenerational regulation of lifespan

Worms with extended lifespan due to reduced insulin/IGF-1 signaling transmit partial longevity to offspring via germline H3K4me3 patterns (Greer et al. 2011, *Nature*).

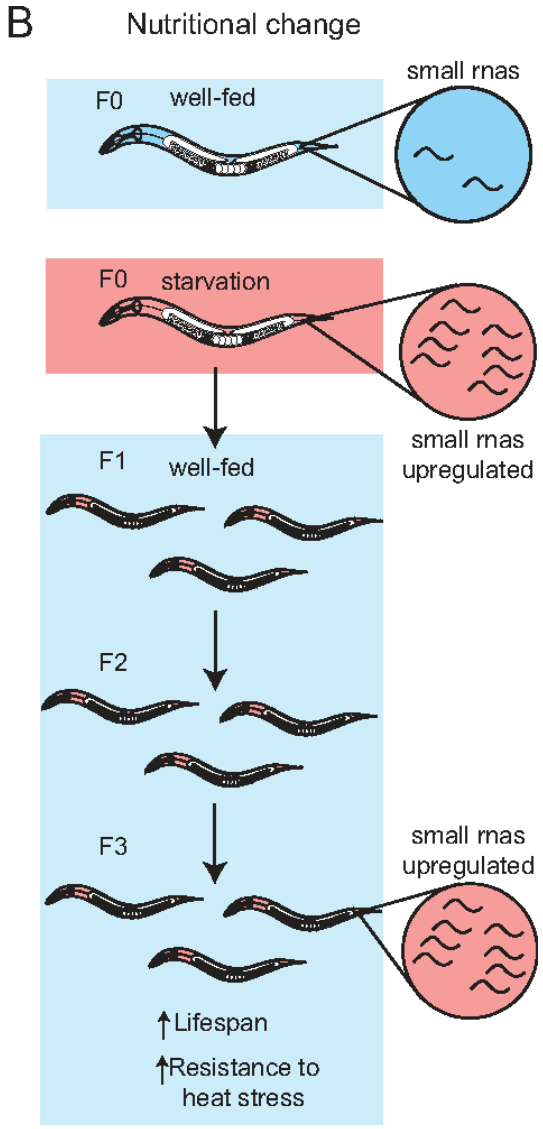
Experimentally induced via stress



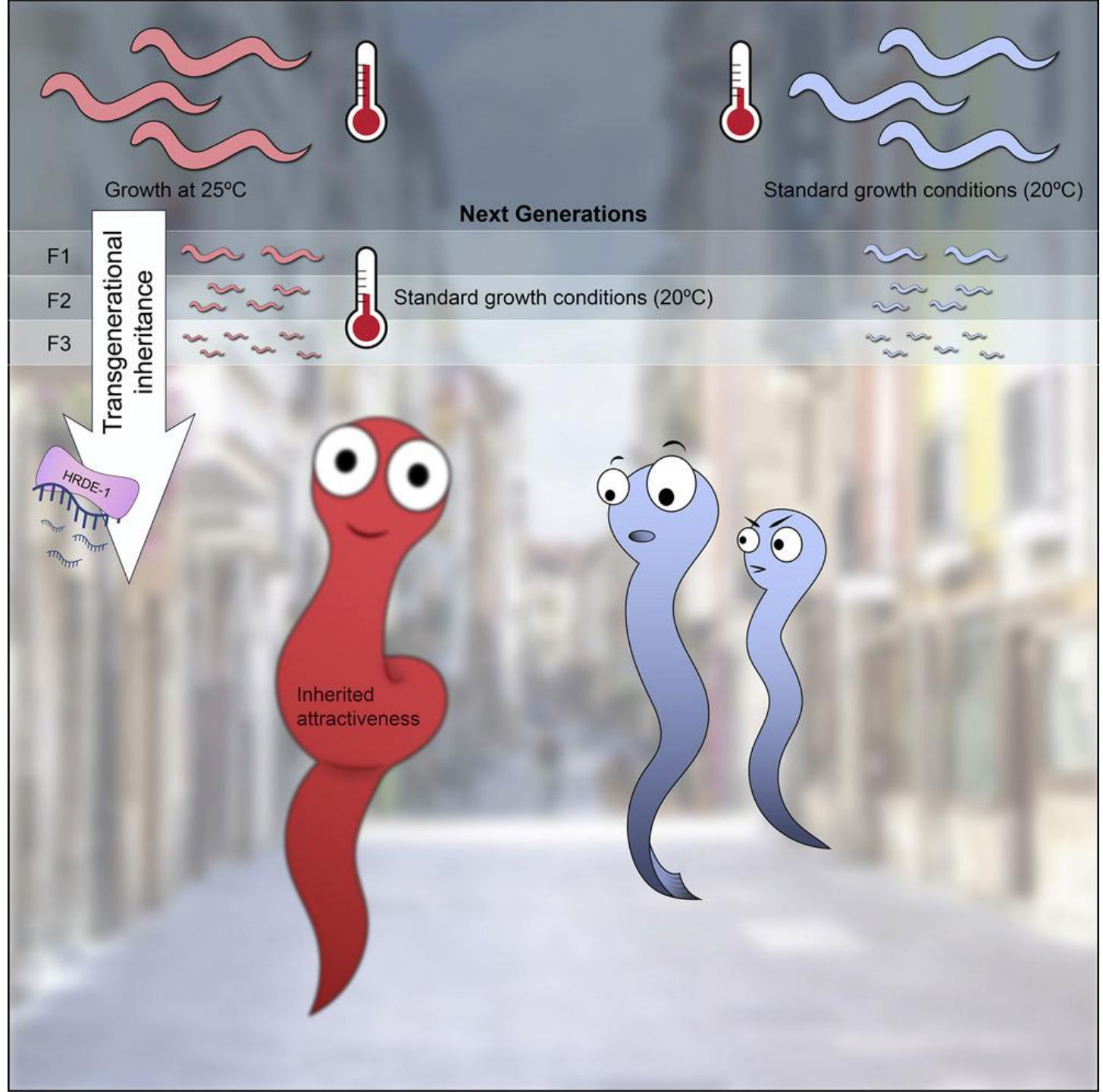
Experimentally induced via “transient mutation”



Experimentally induced via stress



Transgenerational epigenetic inheritance can be initiated via environmental factors (e.g. stress exposure) or presence (then “removed”) of triggering genetic variants


Growth at 25ºC
Standard growth conditions (20ºC)
Next Generations
F1
F2
F3
Standard growth conditions (20ºC)
Transgenerational inheritance
HRDE-1
Inherited attractiveness

Other experimental demonstrations in *C.elegans*

- **Osmotic stress TEI (Kishimoto et al. 2017, *Science*).** High-salt exposure induces expression of a reporter driven by the *osmosensitive* gene *tbb-6*. This induced expression persists for ~14 generations after removal of the stressor — one of the longest-lasting TEI effects documented in any animal. Requires *hrde-1* and chromatin factors. A compelling case for adaptive epigenetic memory far exceeding what "leakage" would predict.

- **piRNA surveillance as germline immune memory (Ashe et al. 2012; Shirayama et al. 2012).** When a new "foreign" sequence enters the germline (experimentally introduced transgene), piRNAs initiate its silencing, which is then maintained by 22G-RNAs and HRDE-1 indefinitely — even after the piRNA trigger is gone. Conversely, sequences recognized as "self" by CSR-1 are protected. This is a **self/non-self discrimination system with heritable memory** — essentially an epigenetic immune system.

Is this happening only in *C.elegans*?

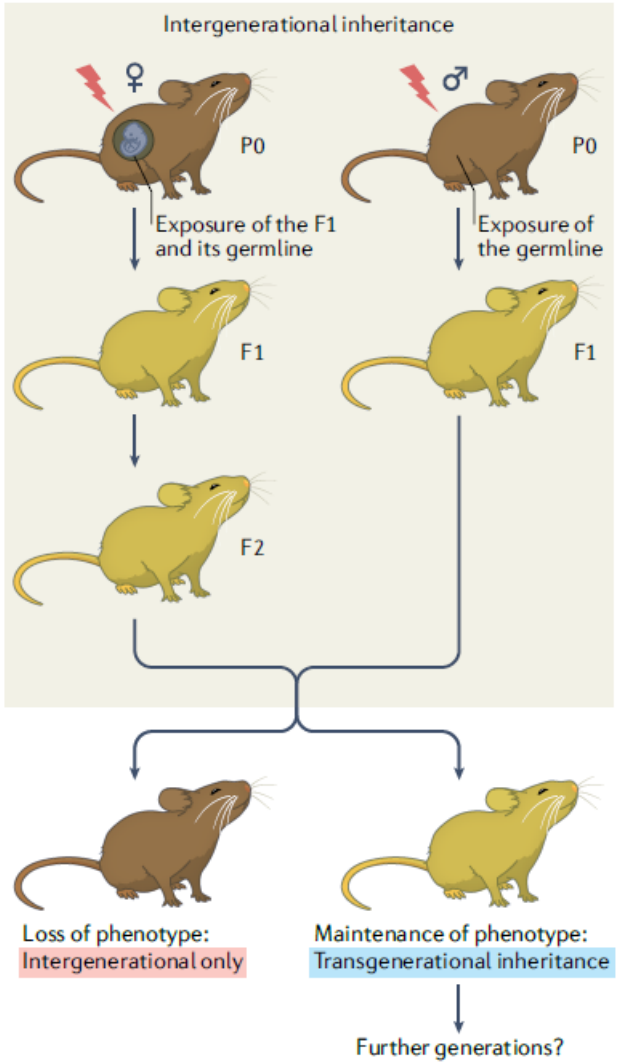
Why *C. elegans* is special for TEI — structural features

- No DNA methylation. *C. elegans* lacks cytosine methylation entirely — so all TEI operates through RNA-based and histone-based mechanisms.

- Extremely well-characterized small RNA pathways. Three partially overlapping systems:
  - piRNA pathway (21U-RNAs) — surveils the germline for non-self sequences, initiates silencing
  - siRNA/RNAi pathway (22G-RNAs, produced by RNA-dependent RNA polymerases, RdRPs) — amplifies and maintains silencing
  - CSR-1 pathway — licenses "self" genes, protects them from piRNA-initiated silencing
  - These interact in what is essentially a germline immune system with memory

- Mortal germline phenotype. Worms lacking key small RNA components (e.g. *mutator* genes, *hrde-1*) show progressive germline deterioration over generations at elevated temperature — demonstrating that active epigenetic maintenance is required for germline immortality. TEI is not a quirk!

Intergenerational vs Transgenerational inheritance

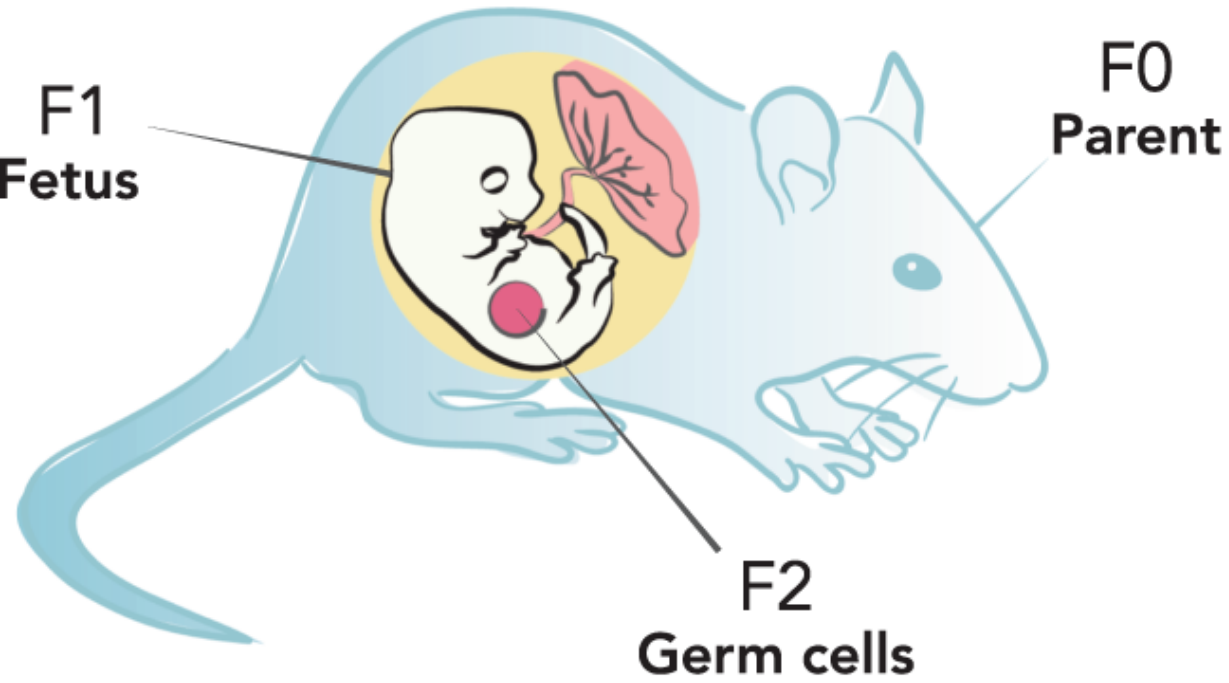


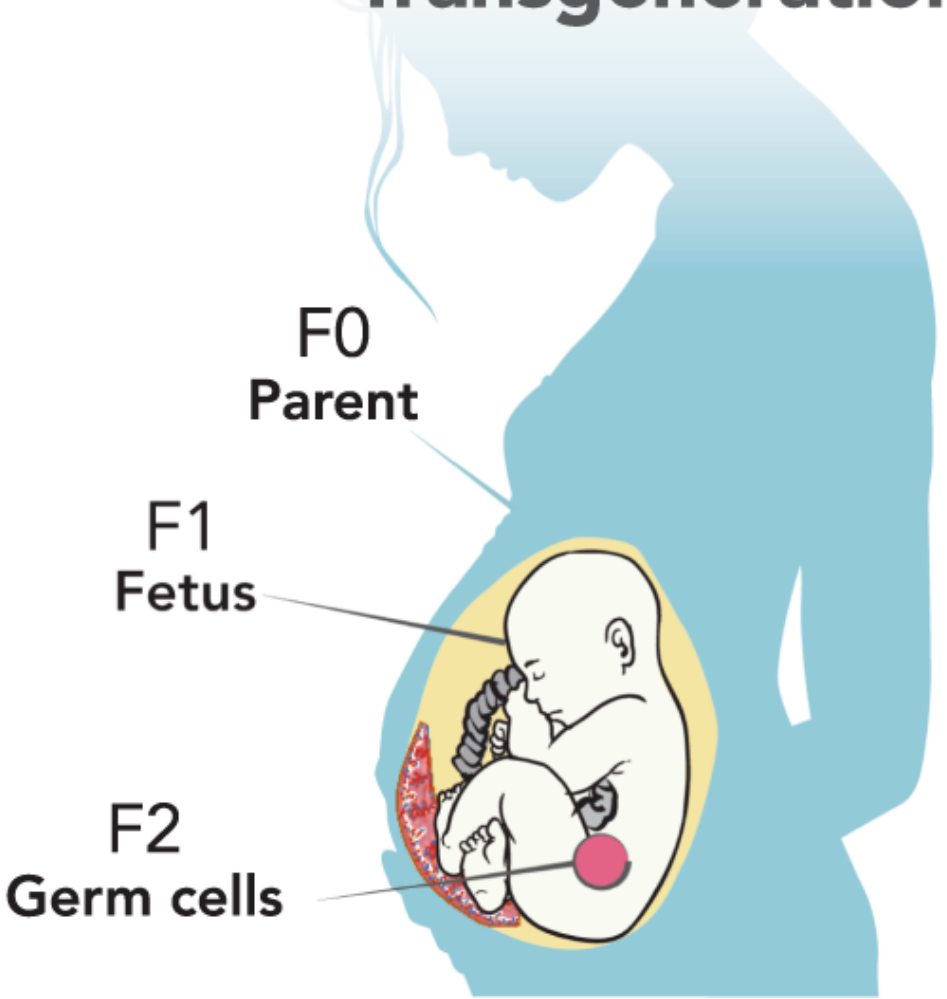
- Intergenerational inheritance refer to that to the F1 – since the germline is already present in F0.
- In pregnant females, the term intergenerational is extended to F2 as well as the germline of the F1 could be in principle already present at the time of stress.

F0 Parent → **F1** Child → **F2** Grandchild → **F3** Great-Grandchild

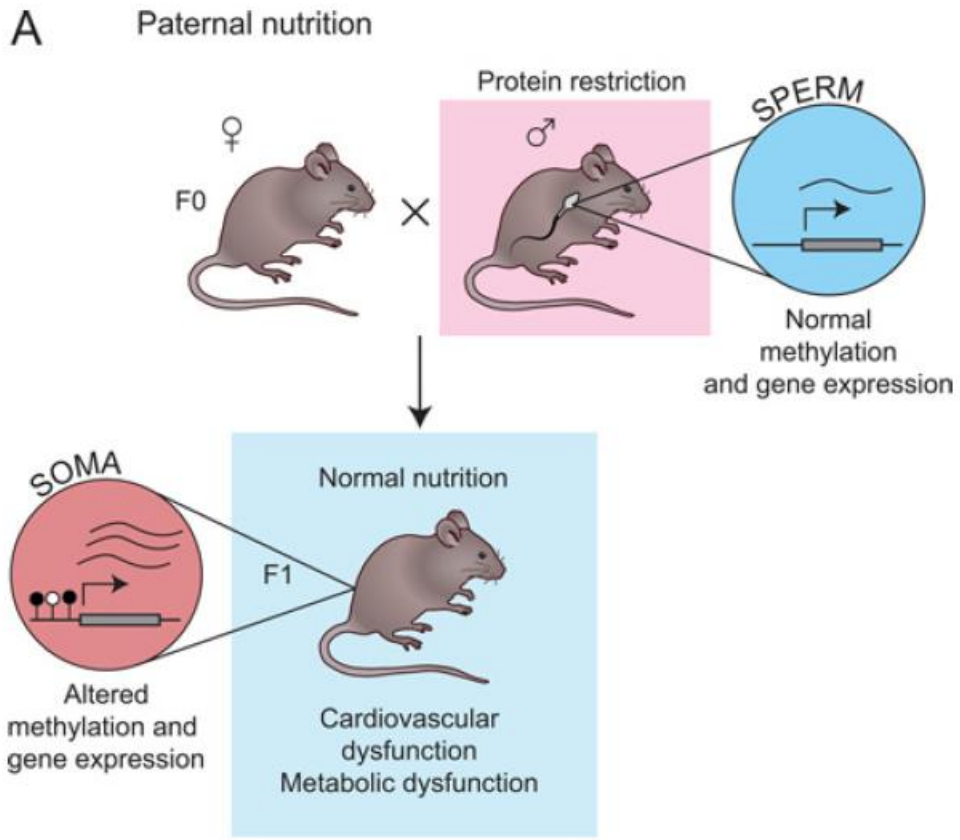
Intergenerational

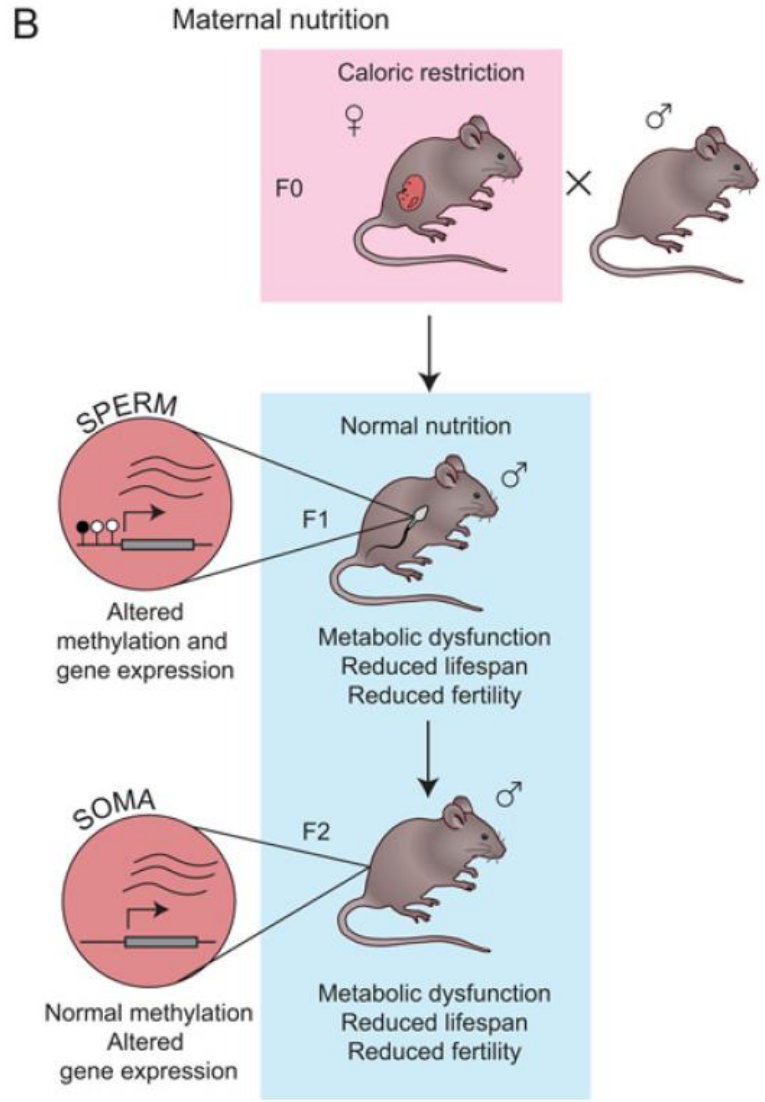
Transgenerational





Intergenerational transmission in mammals

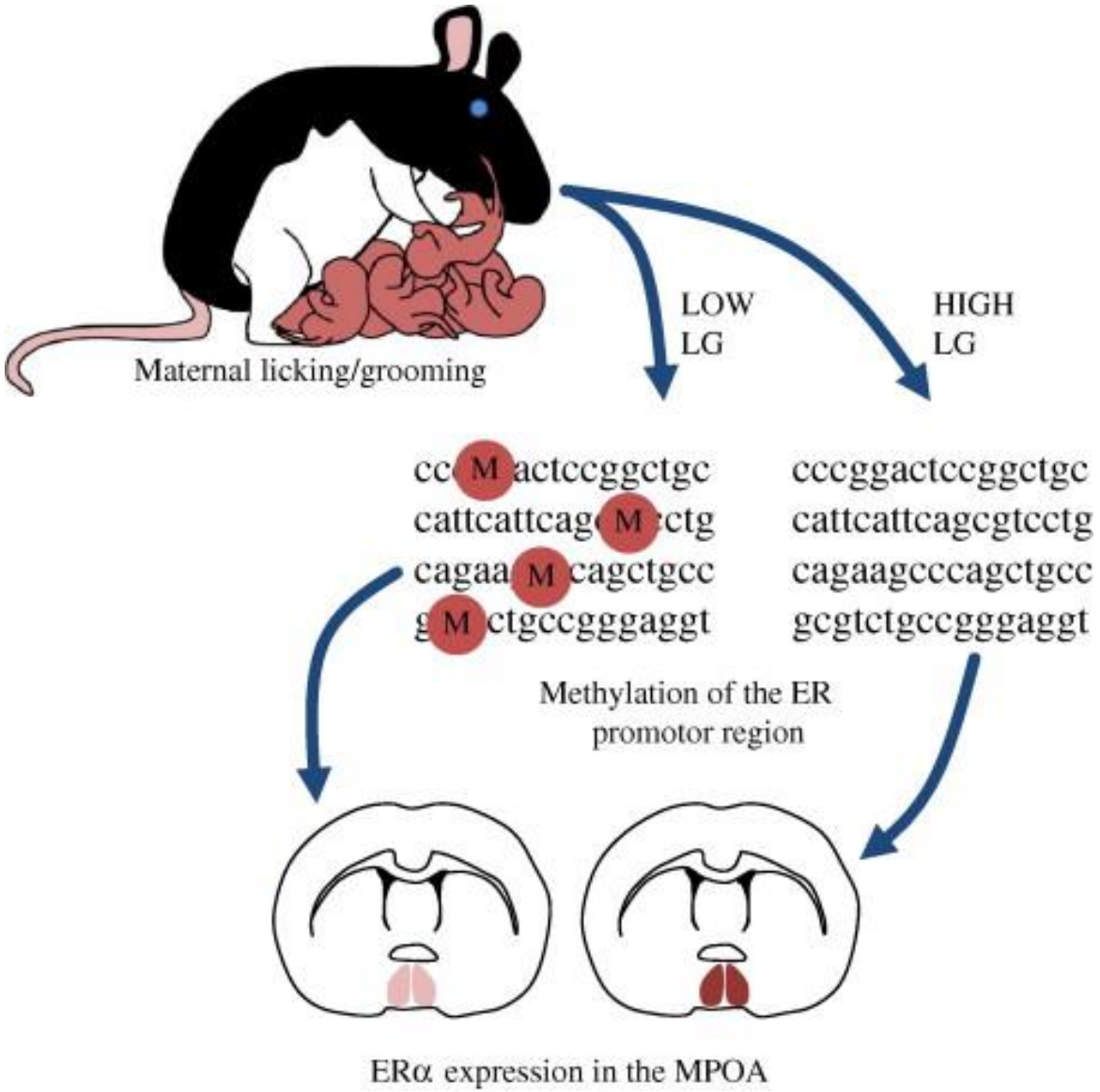




Vast evidence of the long-term effects of prenatal exposure to bacteria components, tobacco, nicotine, fungi, air pollutants, diet, maternal stress, and allergens in models of allergic asthma, ...

But is there transgenerational epigenetic transmission in mammals?

Responsivity to stress has a strong epigenetic component

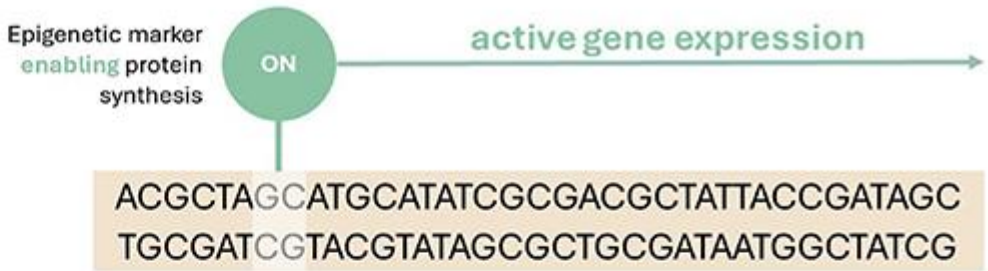


- High Licking and Grooming mothers (LG)→ **hypomethylation** of the exon 17 promoter of the GR gene in hippocampus → increased NGFI-A transcription factor binding → **increased GR expression**
- Low LG mothers → **hypermethylation** of the same CpG site → reduced NGFI-A binding → **reduced GR expression**
- Functional consequence: offspring of high LG mothers have more hippocampal GRs → stronger glucocorticoid negative feedback → **dampened HPA axis response to stress** — they are calmer, less reactive
- The methylation difference emerges in the **first week of life**, persists into adulthood, and is reversed by cross-fostering — proving it is experience-driven not genetic
- Pharmacological reversal with HDAC inhibitor TSA normalizes both the chromatin state and the HPA response — proving causality

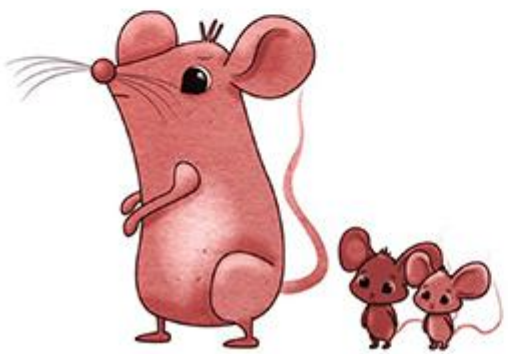
Weaver et al. (2004) *Nature Neuroscience, Champagne et al., works 2008-2014*

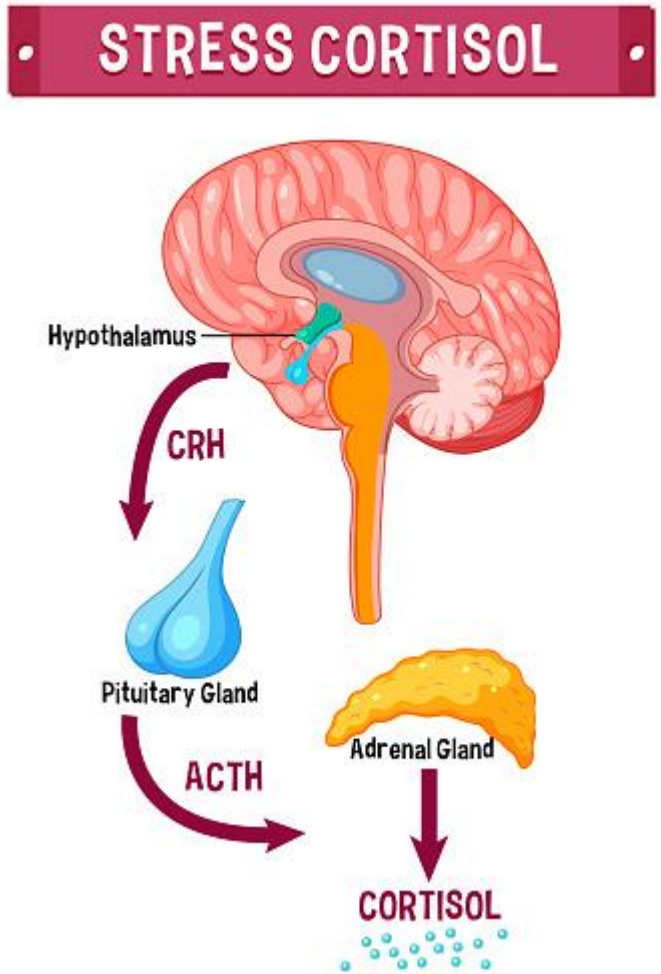
Parental care in early childhood shapes the offspring's stress responses for life

A

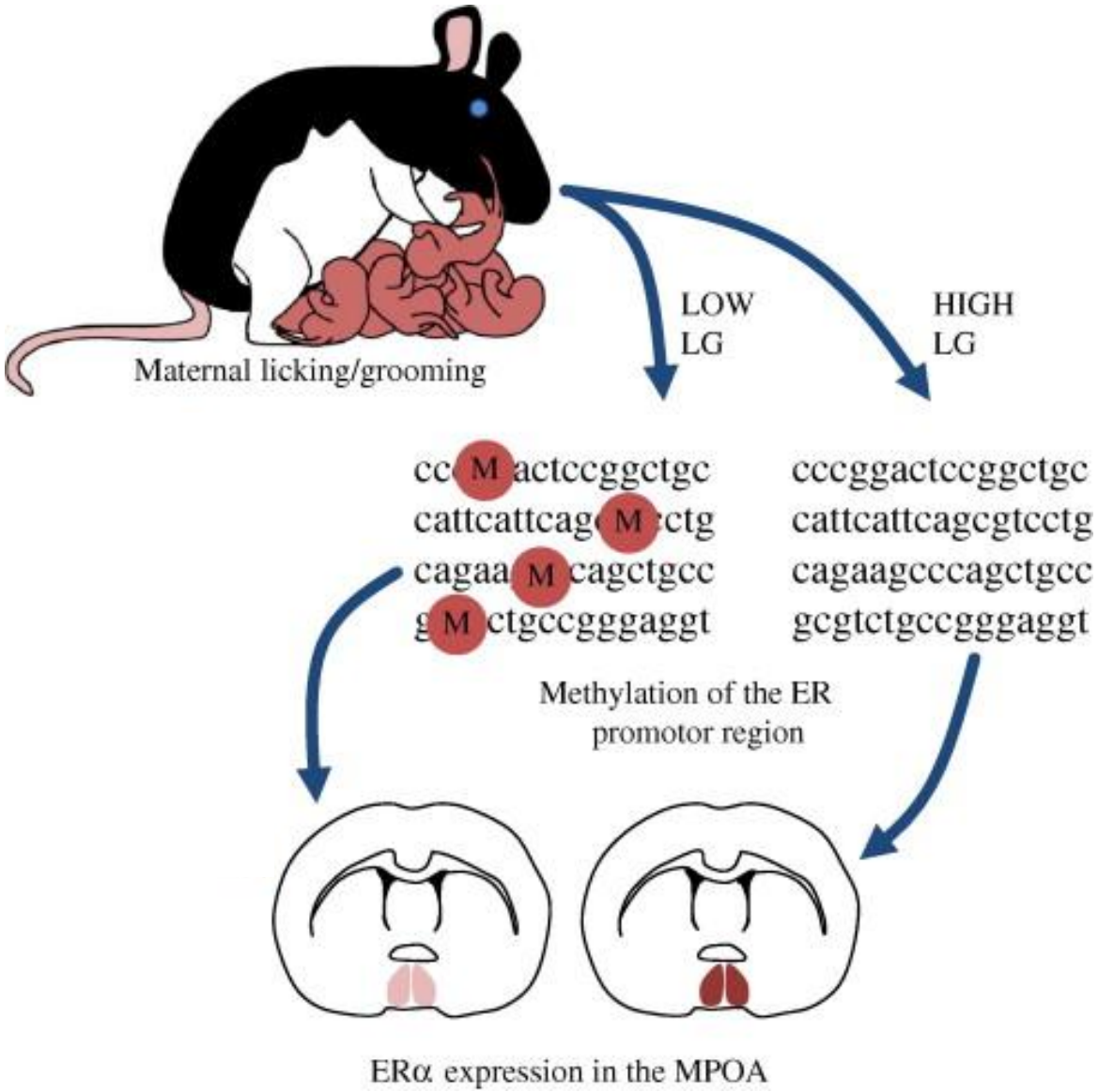


B



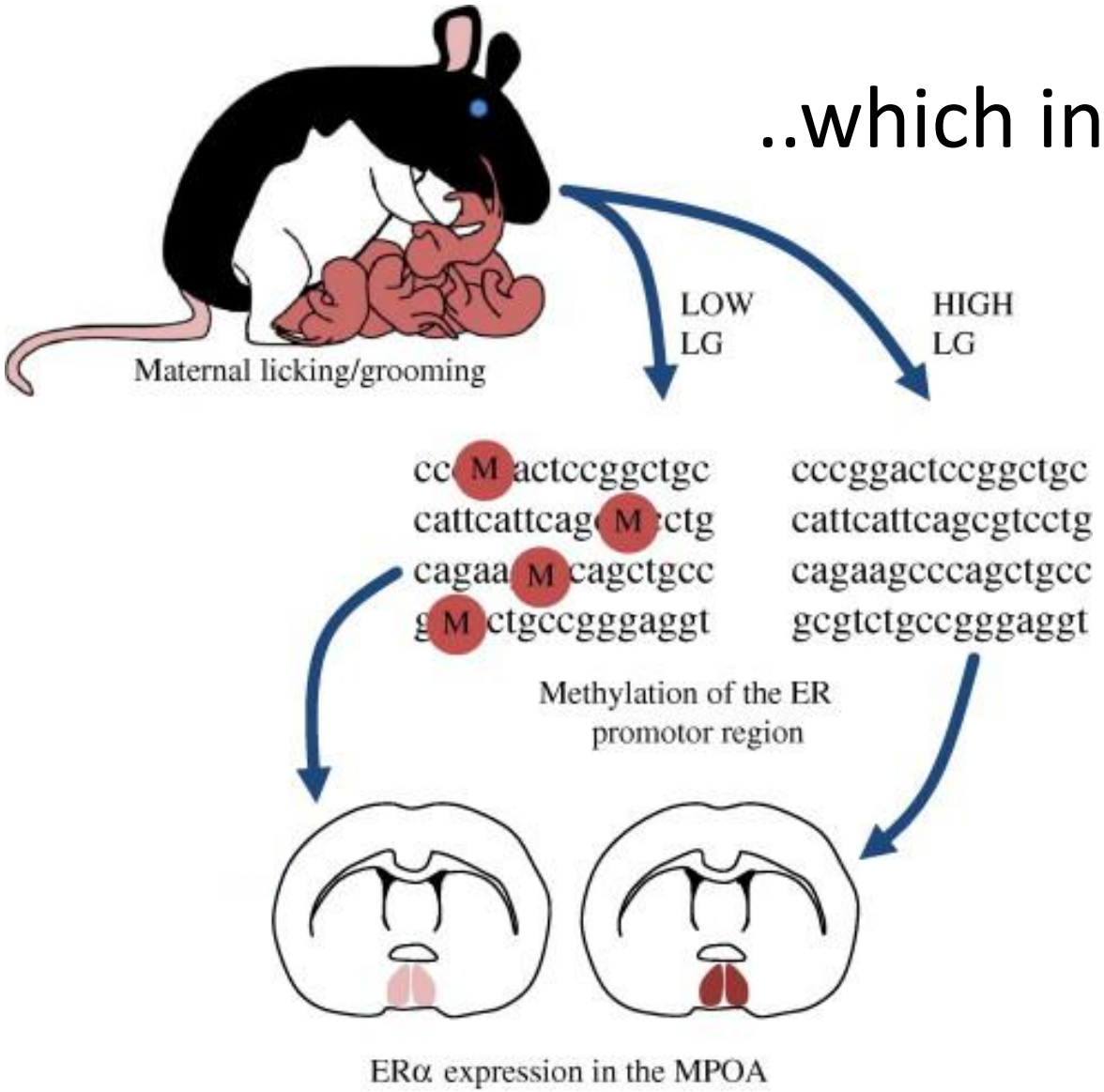


Maternal care the offspring's stress responses..

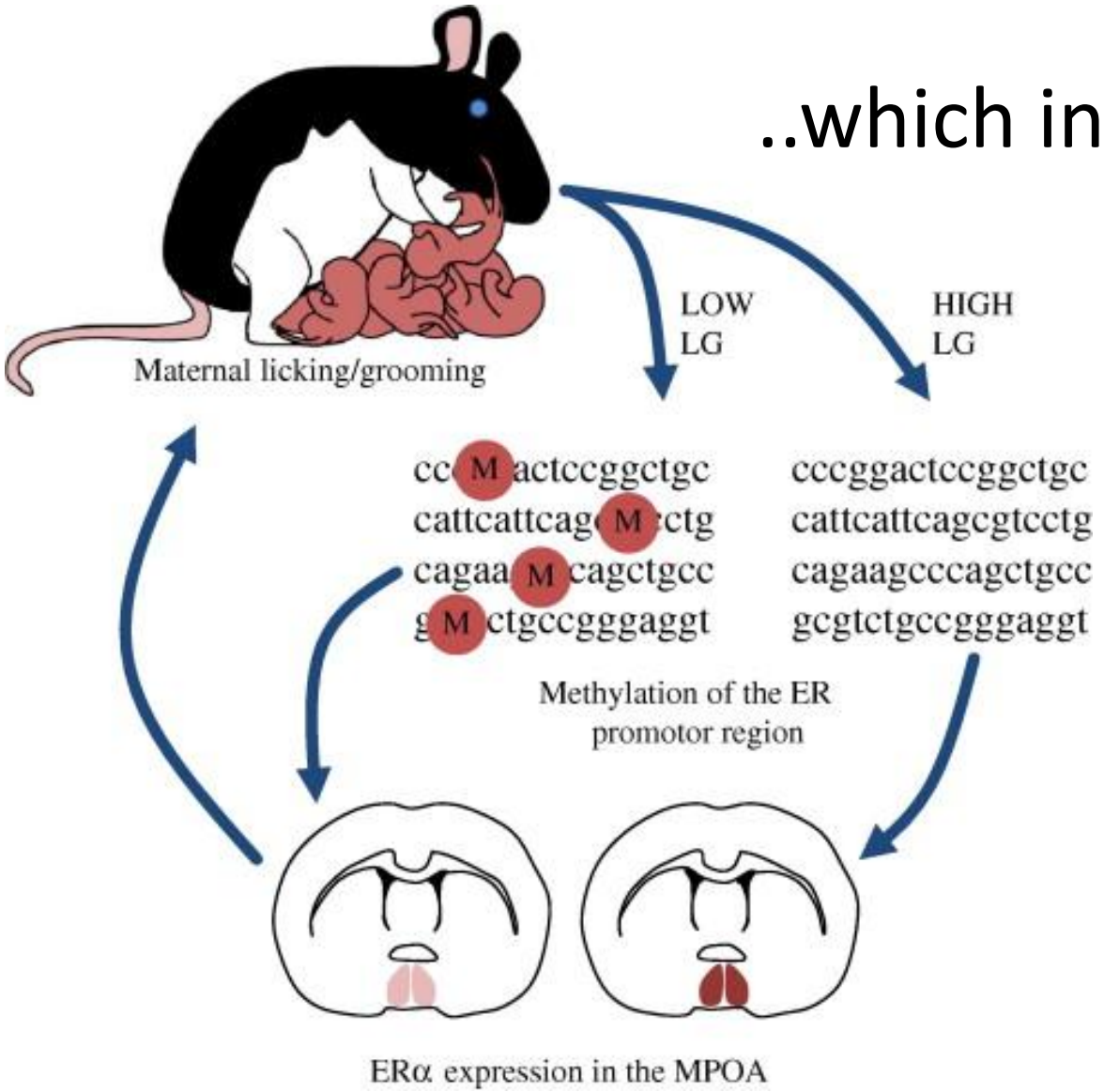


Maternal care the offspring's stress responses..

..which influences F1 offspring's behavior..



Maternal care the offspring's stress responses..

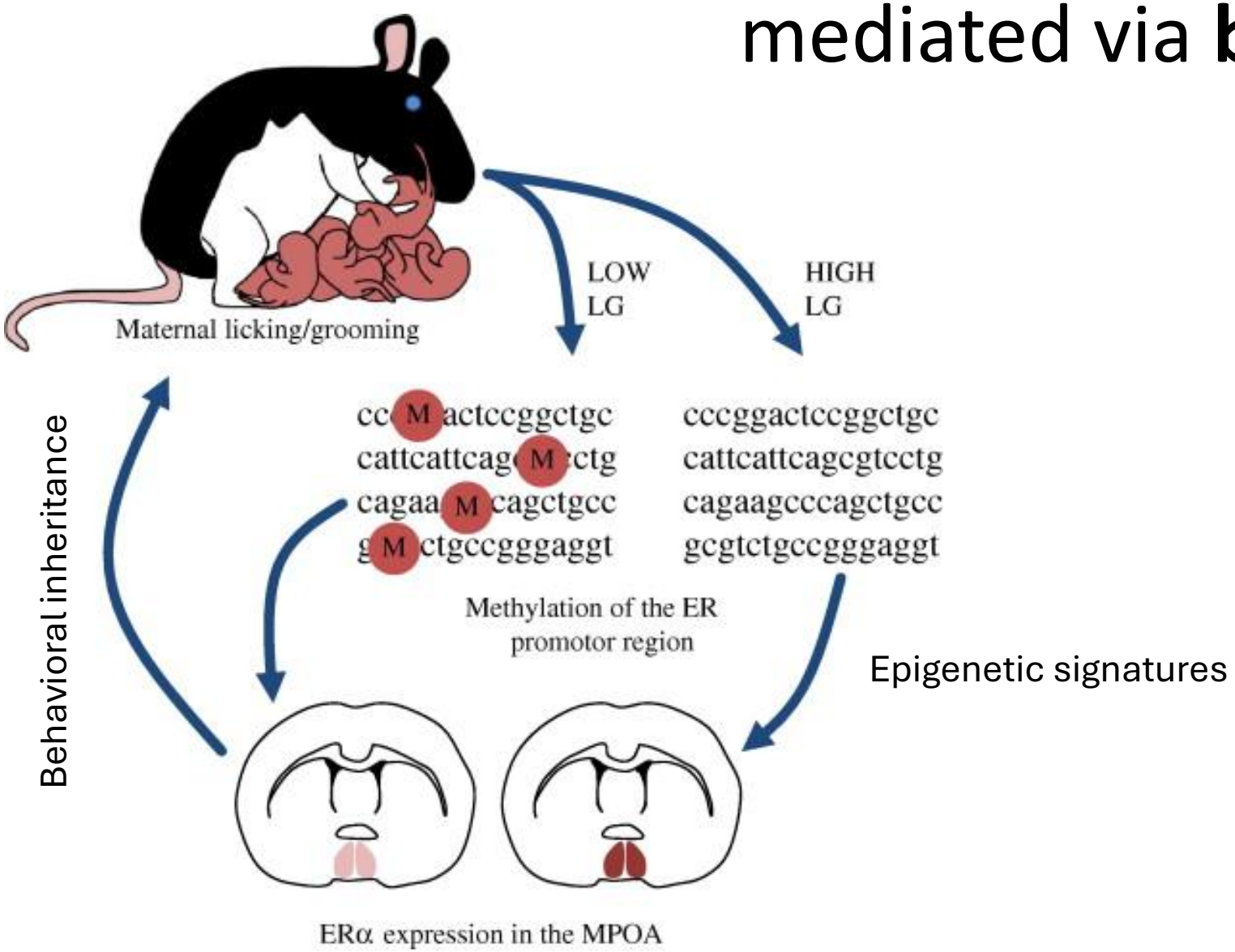


..which influences F1 offspring's behavior..

..which influences F1 maternal behavior in offspring..

..which influences the F2's stress responses..

The **transgenerational epigenetic inheritance** of stress is mediated via **behavioral inheritance**

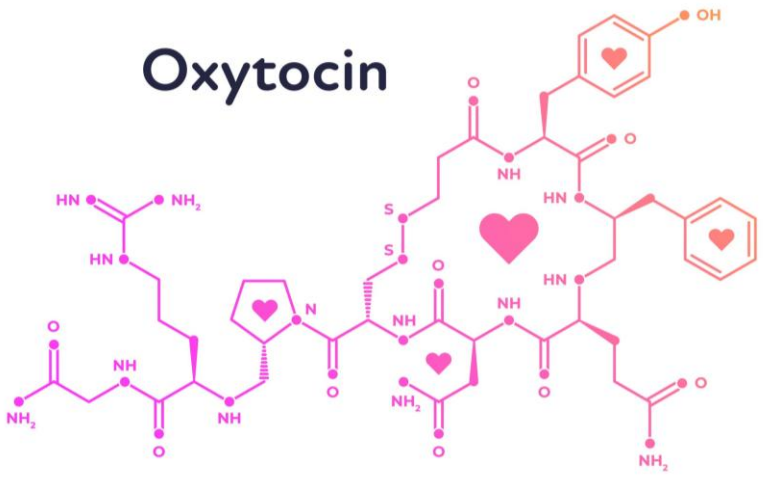


Maternal behavior is influenced even more directly than just “stress”

- **Estrogen receptor alpha (*Esr1*) — the maternal behavior transmission circuit**
- **Champagne et al. (2003, 2006)**
- Low LG received as a pup → **hypermethylation** of the *ERα* promoter in the **medial preoptic area (MPOA)** — the brain region governing maternal motivation
- Reduced ERα expression → blunted sensitivity to estrogen priming of oxytocin receptors before parturition → **reduced maternal motivation**
- This is the mechanism by which low-LG daughters become low-LG mothers — the epigenetic cycle Champagne described in her 2008 *Frontiers in Neuroendocrinology* paper
- The key molecular chain: low LG received → ERα methylation in MPOA → low ERα → low oxytocin receptor induction → low LG given

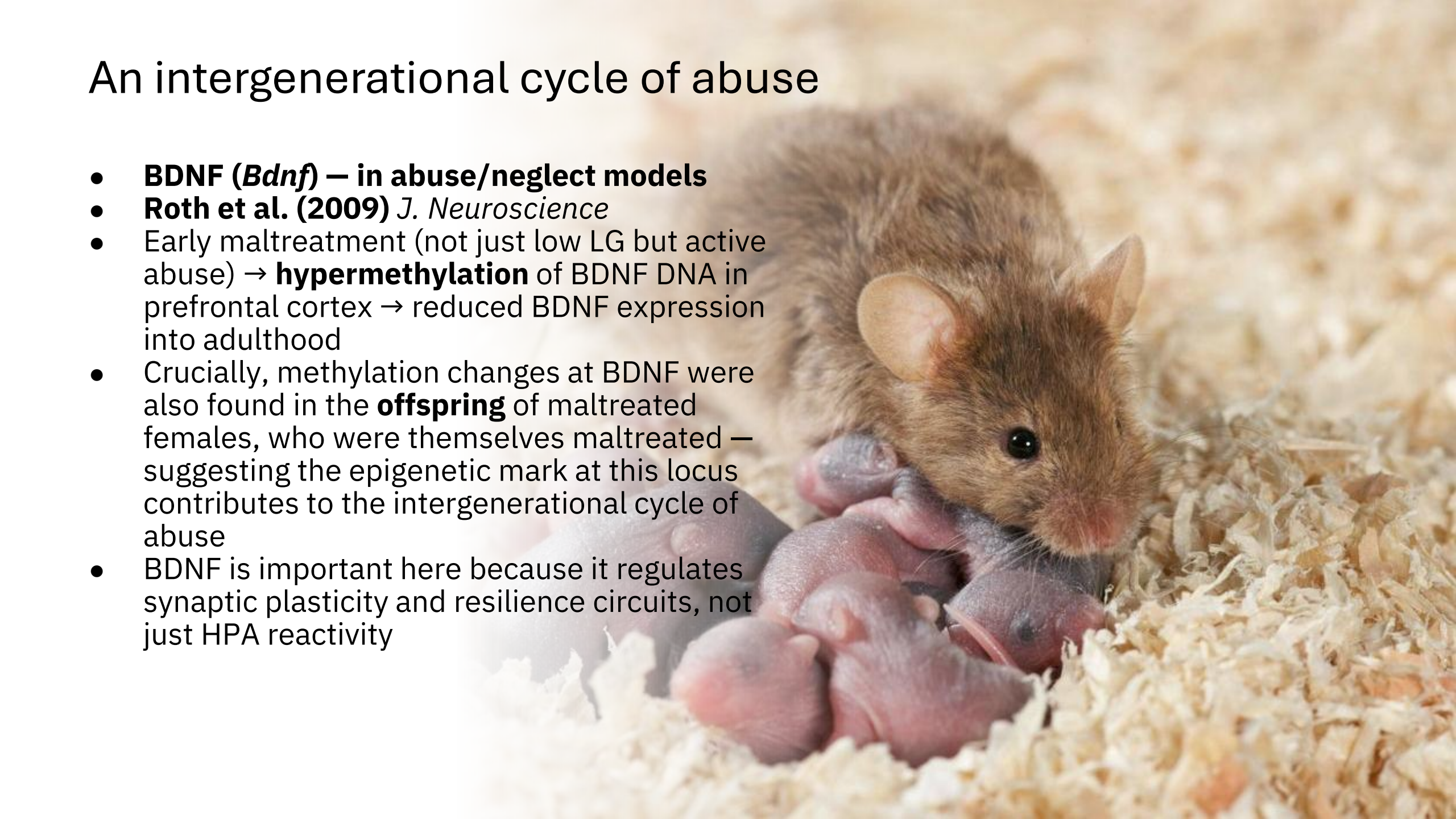
Maternal behavior is influenced even more directly than just “stress”

- **Oxytocin receptor (*Oxtr*)**
- Downstream of the ERα story — MPOA oxytocin receptor levels are reduced in low-LG females, contributing to impaired maternal motivation
- The oxytocin receptor promoter has CpG-rich regions and is a candidate for direct methylation programming, though the evidence is less complete than for GR and ERα



An intergenerational cycle of abuse

- **BDNF (*Bdnf*) — in abuse/neglect models**
- **Roth et al. (2009)** *J. Neuroscience*
- Early maltreatment (not just low LG but active abuse) → **hypermethylation** of BDNF DNA in prefrontal cortex → reduced BDNF expression into adulthood
- Crucially, methylation changes at BDNF were also found in the **offspring** of maltreated females, who were themselves maltreated — suggesting the epigenetic mark at this locus contributes to the intergenerational cycle of abuse
- BDNF is important here because it regulates synaptic plasticity and resilience circuits, not just HPA reactivity



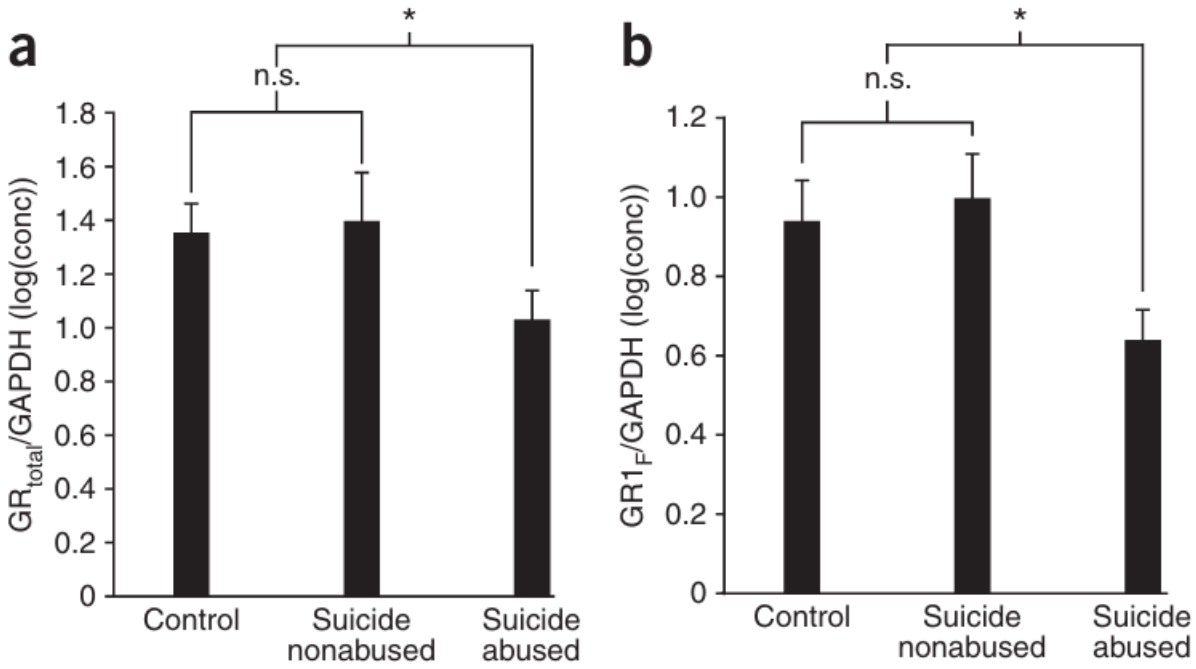
A summary of stress epigenetic inheritance associated to maternal behavior in mice

The overall picture

Gene	Brain region	Direction in low-LG offspring	Functional consequence
Nr3c1 (GR)	Hippocampus	Hypermethylated → less GR	Weaker HPA feedback → more stress reactive
Esr1 (ERα)	MPOA	Hypermethylated → less ERα	Less maternal motivation → become low-LG mothers
Bdnf	Prefrontal cortex	Hypermethylated → less BDNF	Reduced resilience, transmitted to offspring
GABA-A subunits	Amygdala/LC	Reduced expression	Less inhibitory tone on fear
CRF receptor	Amygdala/LC	Increased expression	Amplified fear/stress response

Behaviorally-mediated epigenetic programming in humans

- Postmortem hippocampus of suicide victims with childhood abuse history shows hypermethylation of *NR3C1* exon 1F promoter and reduced GR expression — same locus, same mechanism as in rats
- Replicated in living adults across multiple studies: childhood maltreatment consistently associates with *NR3C1* hypermethylation and blunted cortisol stress responses
- **Critical caveat:** no cross-fostering is possible in humans — genetic confounds, prenatal hormonal effects, and socioeconomic factors cannot be fully excluded; evidence is epidemiological, not experimental



McGowan et al. (2009) *Nature Neuroscience* 12: 342–348

Not only genetic inheritance: different interacting channels of inheritance

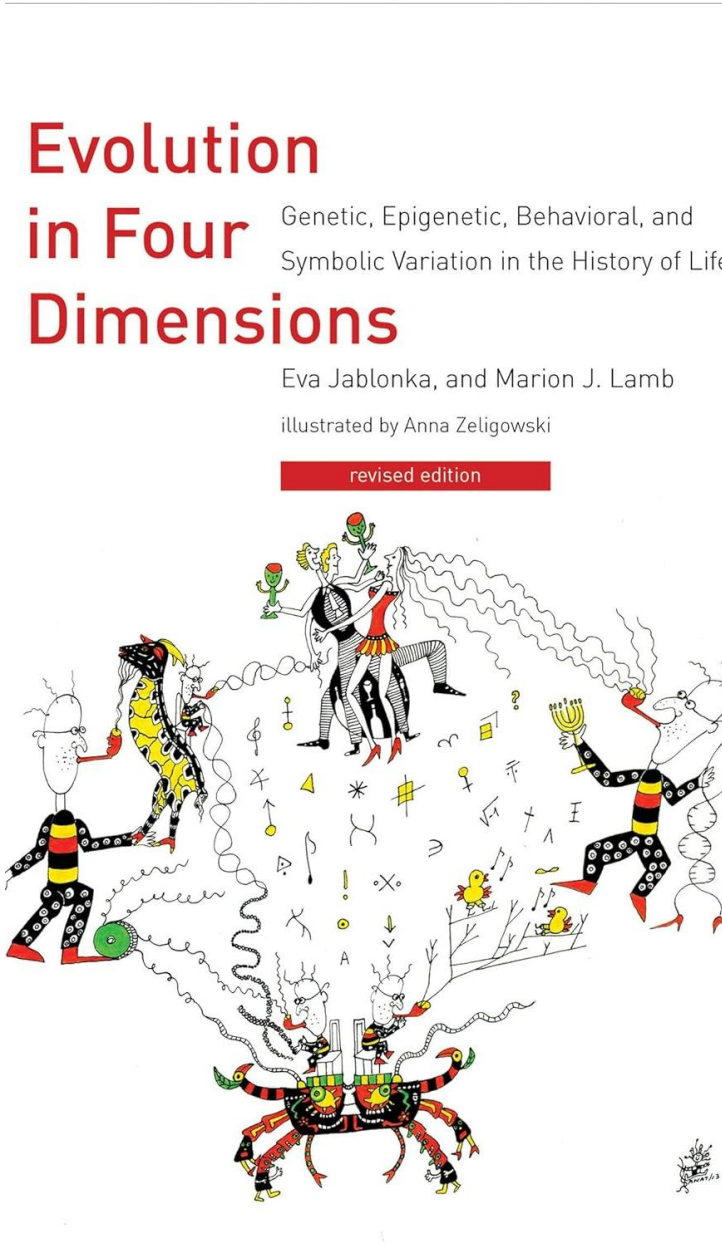
Genetic inheritance

Behavioral inheritance

Cultural (symbolic) inheritance

Epigenetic inheritance

Environmental inheritance (niche construction)



Epigenetic reprogramming erases epigenetic marks

Essays in Biochemistry (2016) **60** 191–202
DOI: 10.1042/EBC20160025

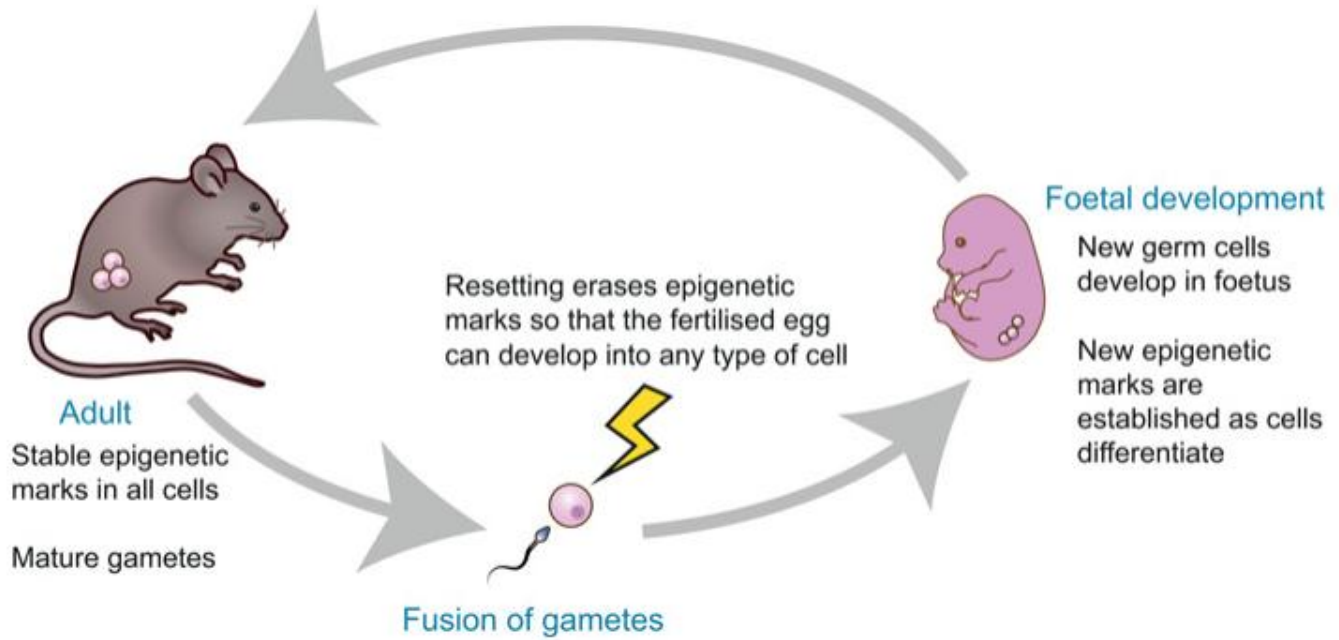


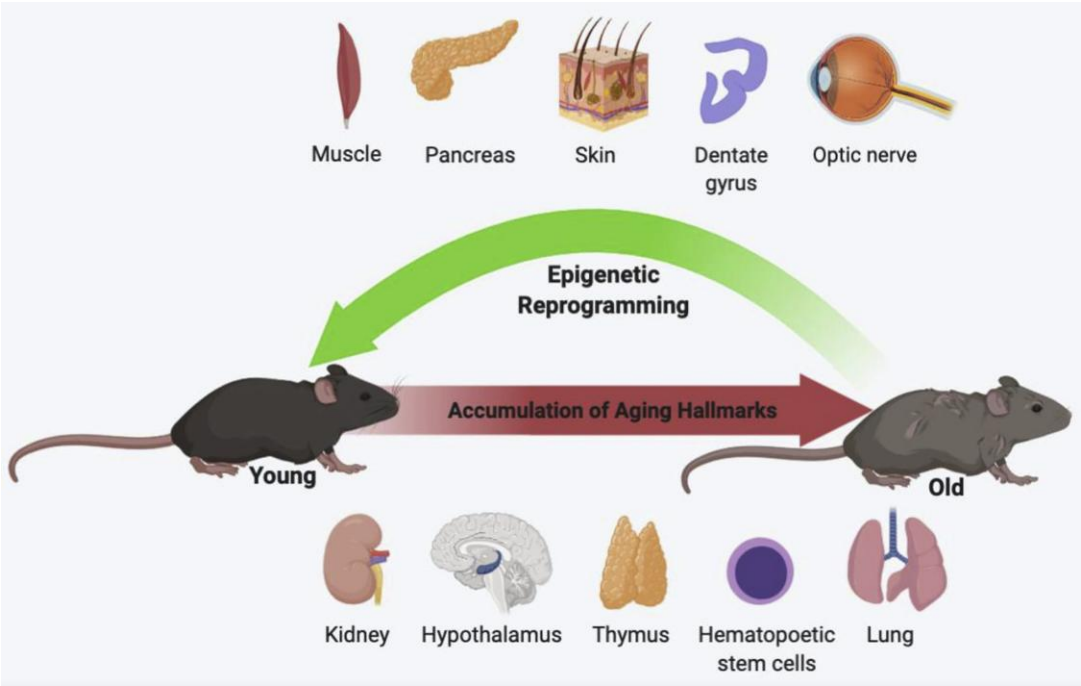
Figure 2. Reprogramming erases epigenetic marks

During gametogenesis and early development, a process called germline resetting takes place, where most epigenetic marks in chromatin are removed. This allows the reacquisition of pluripotency in the early embryo and proper differentiation of tissues during development.

Epigenetic reprogramming erases epigenetic marks

•**Two waves of global erasure** in mammals: at fertilization, and during primordial germ cell (PGC) specification. Most methylation marks are stripped and re-laid de novo.
•**Why forget?** Reprogramming solves two problems:

- Restores **totipotency** — the zygote must be able to become any cell type; somatic epigenetic identities must be erased
- Prevents **accumulation of epigenetic errors** — methylation drift and environmentally-induced marks must not compound across generations
- Try to limit the "selfish genome" problem. Transposable elements exploit epigenetic states; reprogramming + re-silencing of TEs in the germline is a major function of the PGC wave. Piwi-interacting RNA(piRNA)-directed re-methylation of TEs happens during this window.

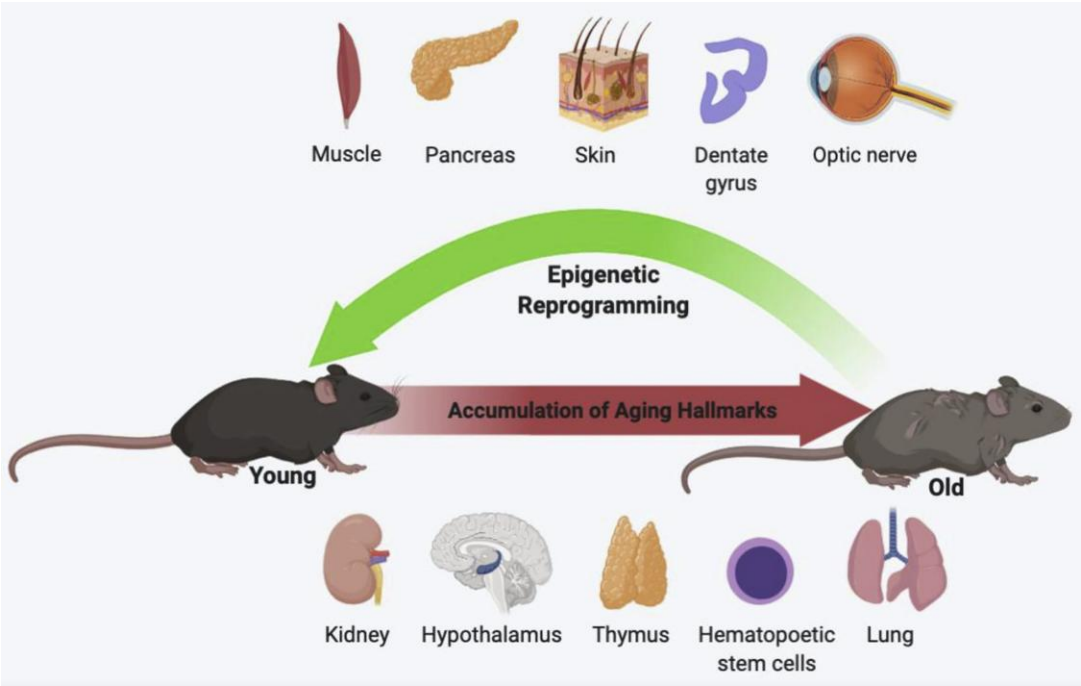


Epigenetic reprogramming erases epigenetic marks

•**Two waves of global erasure** in mammals: at fertilization, and during primordial germ cell (PGC) specification. Most methylation marks are stripped and re-laid de novo.
•**Why forget?** Reprogramming solves two problems:

- Restores **totipotency** — the zygote must be able to become any cell type; somatic epigenetic identities must be erased
- Prevents **accumulation of epigenetic errors** — methylation drift and environmentally-induced marks must not compound across generations
- Try to limit the "selfish genome" problem. Transposable elements exploit epigenetic states; reprogramming + re-silencing of TEs in the germline is a major function of the PGC wave. Piwi-interacting RNA(piRNA)-directed re-methylation of TEs happens during this window.

•**The cost of forgetting.** Reprogramming means each generation must rebuild its epigenome from scratch using genetic sequence information + developmental signals. Slow, energetically costly, and occasionally imperfect — which is where TEI leaks through.



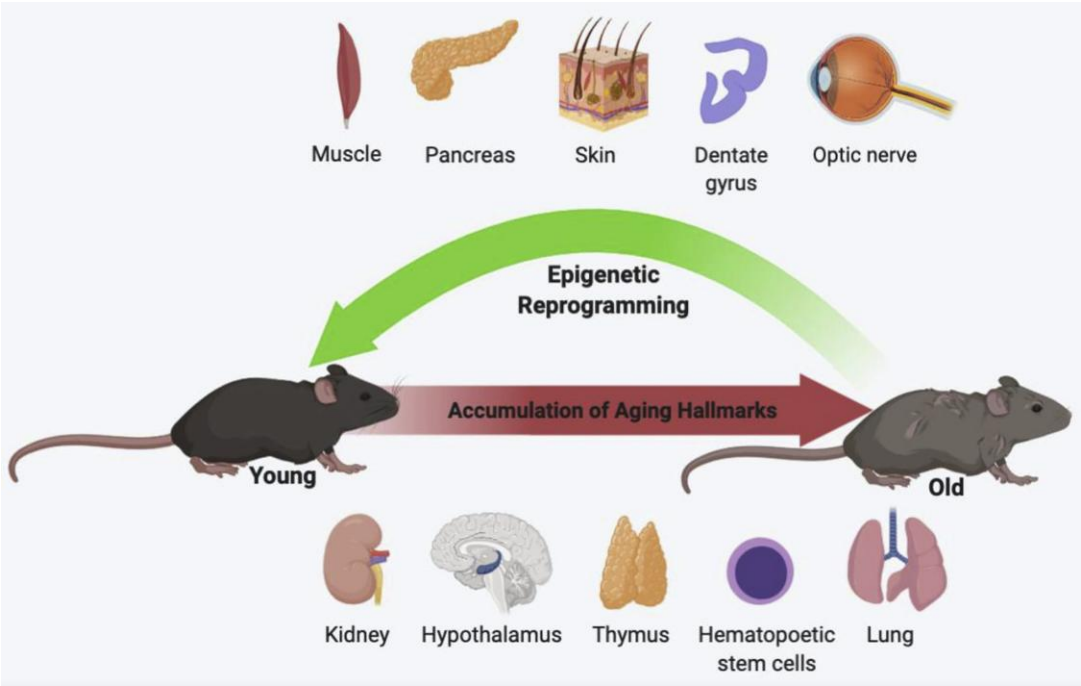
Epigenetic reprogramming erases epigenetic marks

•**Two waves of global erasure** in mammals: at fertilization, and during primordial germ cell (PGC) specification. Most methylation marks are stripped and re-laid de novo.
•**Why forget?** Reprogramming solves two problems:

- Restores **totipotency** — the zygote must be able to become any cell type; somatic epigenetic identities must be erased
- Prevents **accumulation of epigenetic errors** — methylation drift and environmentally-induced marks must not compound across generations
- Try to limit the "selfish genome" problem. Transposable elements exploit epigenetic states; reprogramming + re-silencing of TEs in the germline is a major function of the PGC wave. Piwi-interacting RNA(piRNA)-directed re-methylation of TEs happens during this window.

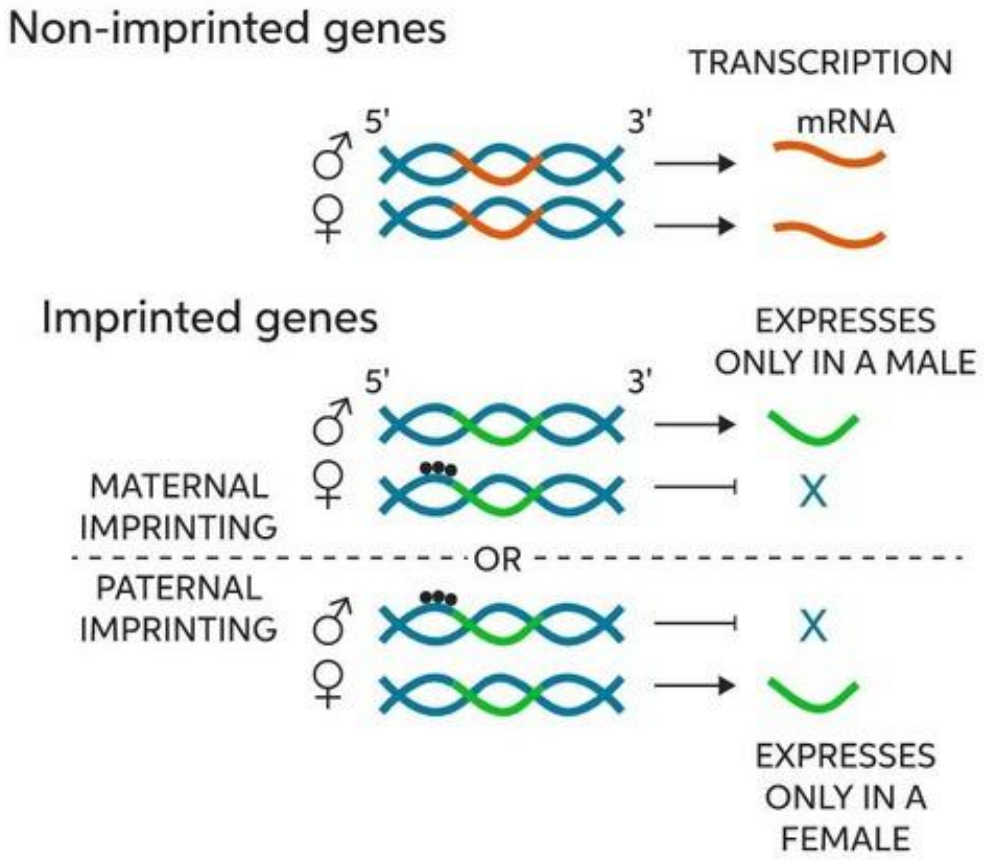
•**The cost of forgetting.** Reprogramming means each generation must rebuild its epigenome from scratch using genetic sequence information + developmental signals. Slow, energetically costly, and occasionally imperfect — which is where TEI leaks through.

Despite this we saw that sometimes reprogramming does not erase everything…



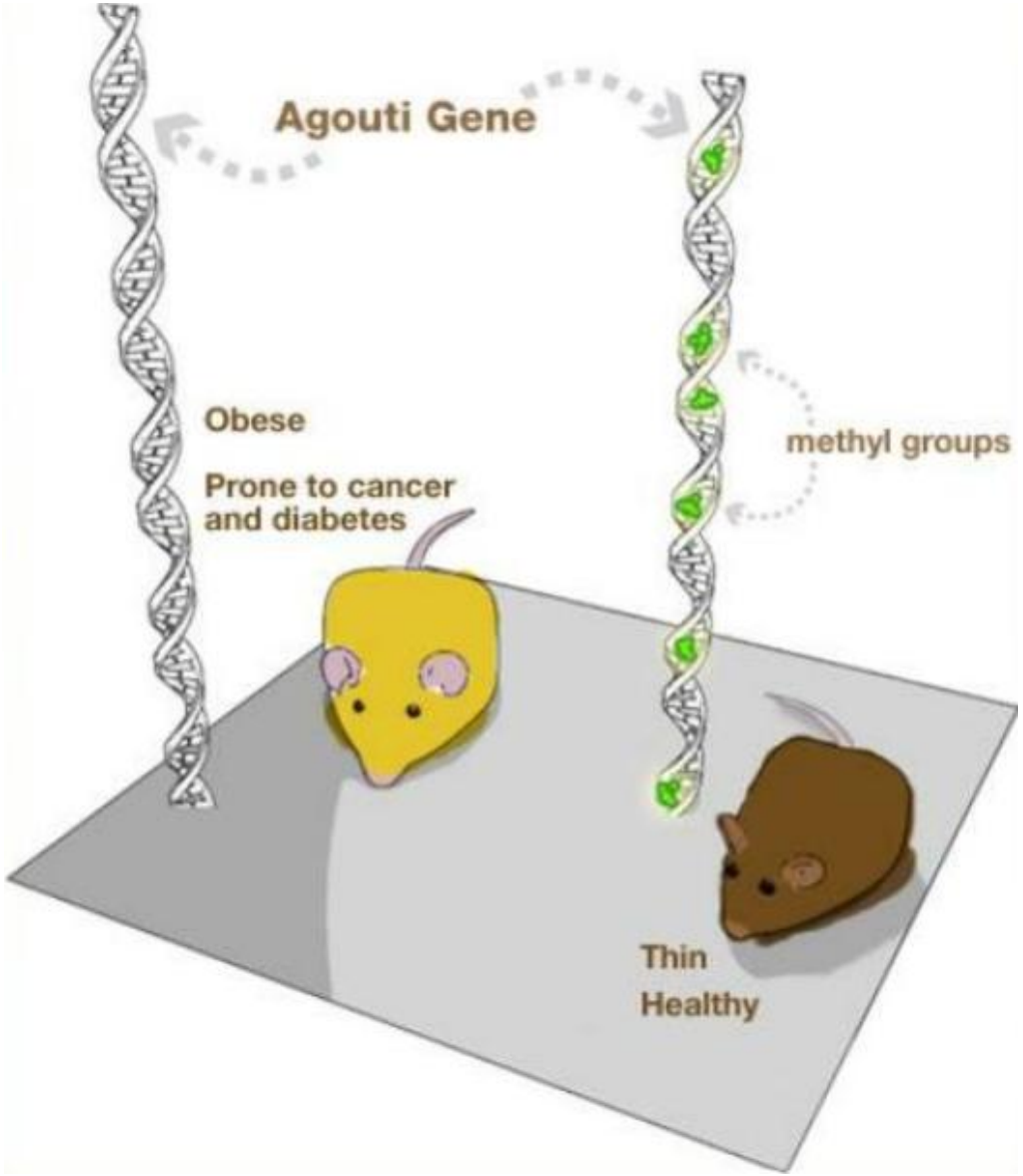
Occasional escape from epigenetic reprogramming

- **Imprinting as deliberate non-erasure.** ~100 mammalian genes escape reprogramming by design — their parent-of-origin marks are protected. Proof that the machinery can selectively spare loci when there is adaptive reason to do so.
- **Escape from reprogramming** is the mechanistic bottleneck for mammalian TEI. Candidates: certain retrotransposon-associated loci, regions with incomplete demethylation in PGCs, and germline-expressed small RNAs (piRNAs, tRNA fragments) which are packaged into sperm/oocyte independently of chromatin.



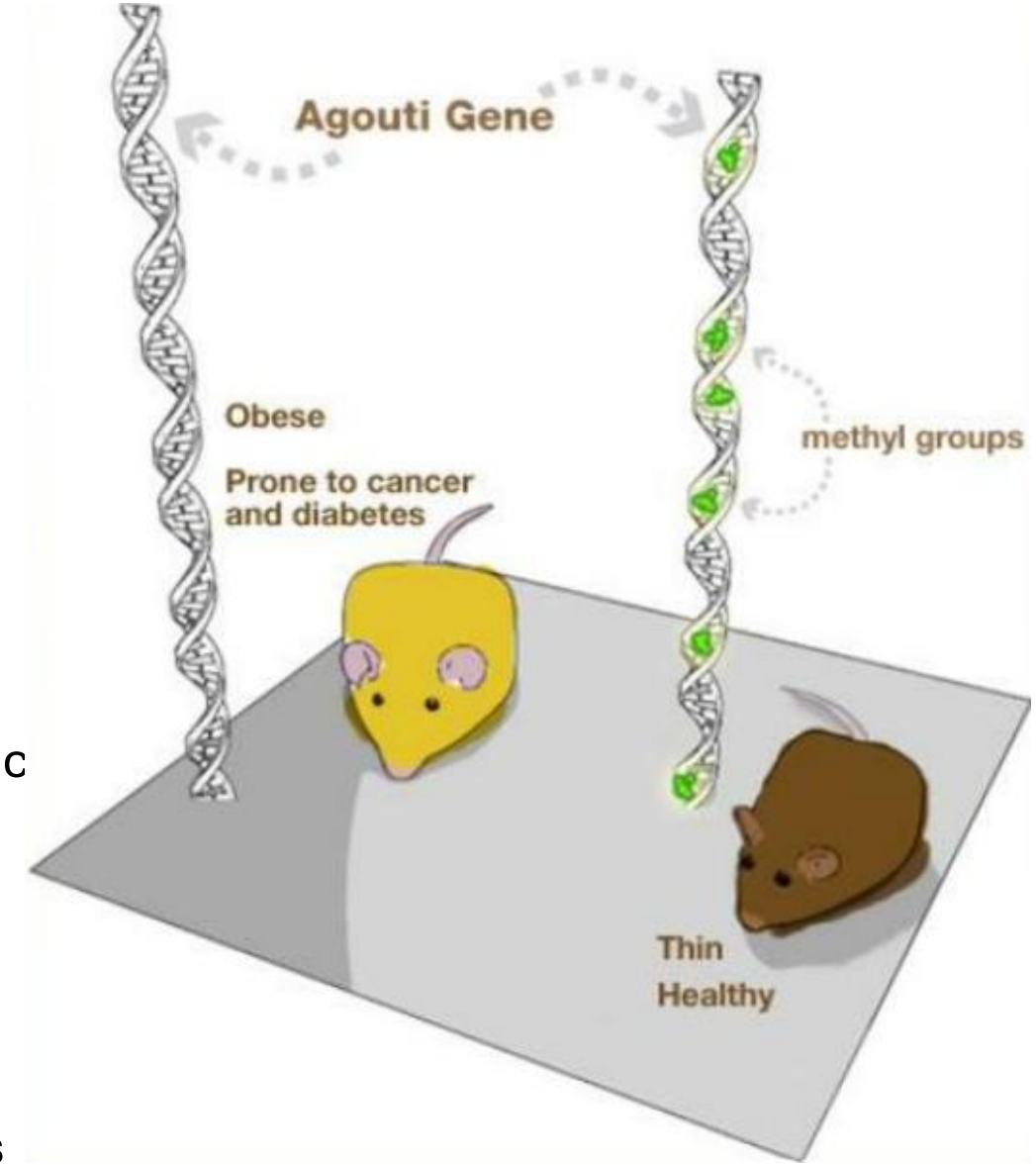
Best evidence for germline TEI in mammals

- ***Avy* agouti mouse** — retrotransposon-driven metastable epiallele; methylation state inherited through both germlines; maternal diet shifts offspring coat colour distribution across generations — cleanest mammalian example, but an unusual locus



Best evidence for germline TEI in mammals

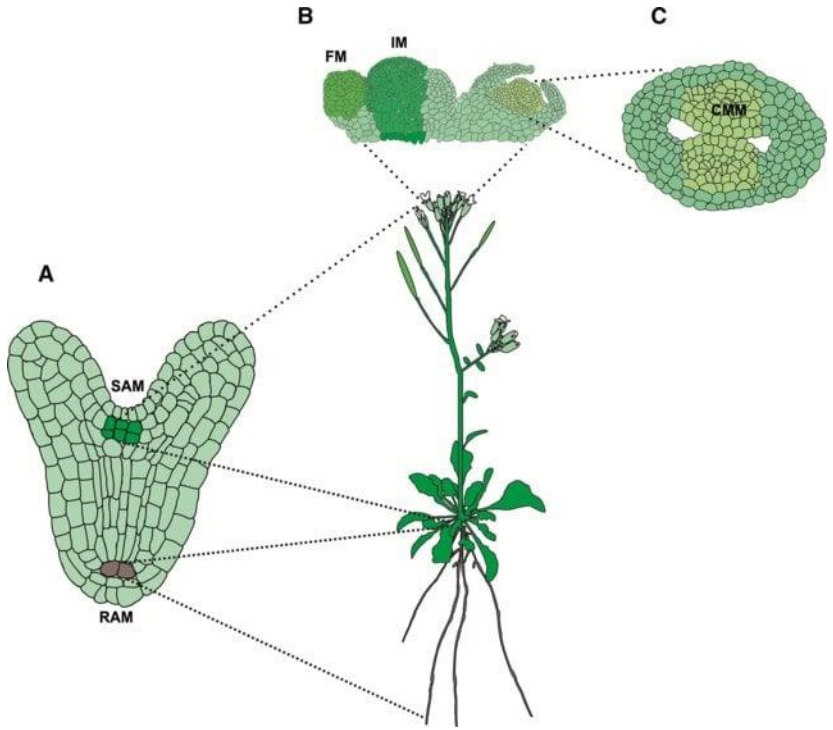
- ***Avy* agouti mouse** — retrotransposon-driven metastable epiallele; methylation state inherited through both germlines; maternal diet shifts offspring coat colour distribution across generations — cleanest mammalian example, but an unusual locus
- **Paternal trauma → sperm RNAs (Gapp et al. 2014, *Nat. Neurosci.*)** — injecting sperm RNAs from traumatised males into naive zygotes reproduces behavioural/metabolic phenotype in offspring — one of very few causal demonstrations in mammals
- **Paternal diet → tRNA fragments (Chen et al. 2016, *Science*)** — low-protein diet alters sperm tRF profile; injecting these tRFs into naive zygotes reproduces metabolic phenotype — clean mechanism, sperm RNA as vector
- **Vinclozolin (Skinner lab)** — endocrine disruptor produces male fertility defects to F4 with sperm methylation changes — widely cited but inconsistently replicated



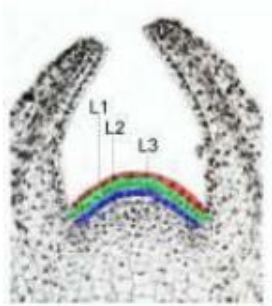
What about organisms other than vertebrate then?

Plants

- Two main meristems: the root apical meristem (RAM) and the shoot apical meristem (SAM).
- Both meristems contain a niche environment with the stem cells.
- During the reproductive stage, the SAM gives rise to the inflorescence meristem (IM), which in turn produces the floral meristem (FM)



Lack of a Weismann's barriers in plants



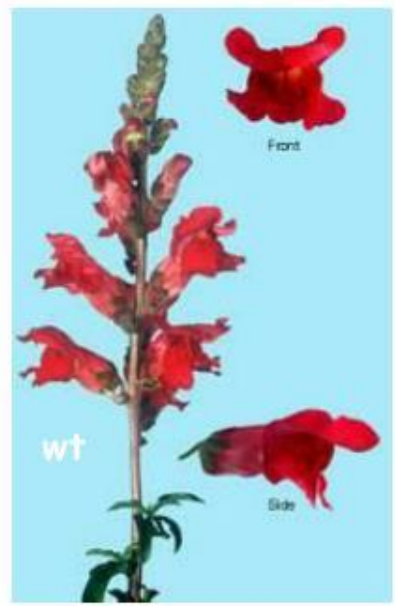
Vegetative meristem

CO
FLC
AGL20
AGL24

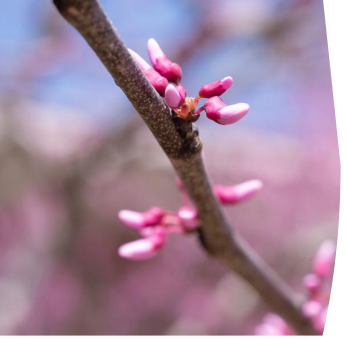
Inflorescence meristem

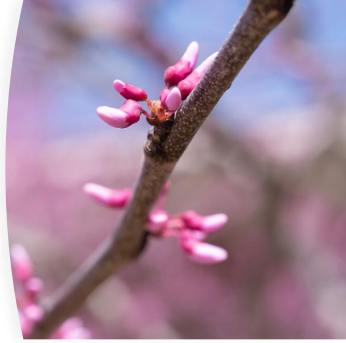
LFY/FLO

Flower meristem

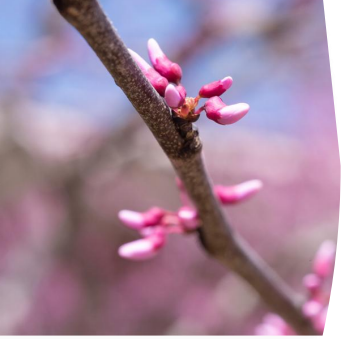


Lack of a Weismann’s barriers in plants

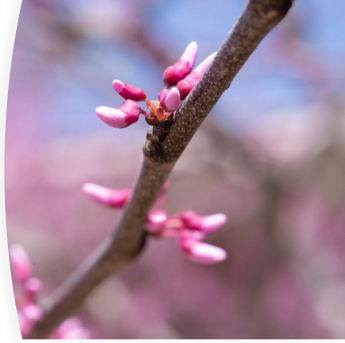




•The germline derives from floral meristems, which in turns derives from the shoot apical meristem (SAM)
•The SAM is purely vegetative for most of the plant's life — making leaves, stems, branches
•At flowering, environmental signals (photoperiod, vernalization) trigger the SAM to convert into an **inflorescence meristem**, then **floral meristems**
•Floral meristems are still somatic tissue — they generate sepals, petals, stamens, carpels
•Only inside stamens and carpels do a handful of cells then differentiate *de novo* into meiocytes (PMCs and MMCs)



Lack of a Weismann’s barriers in plants



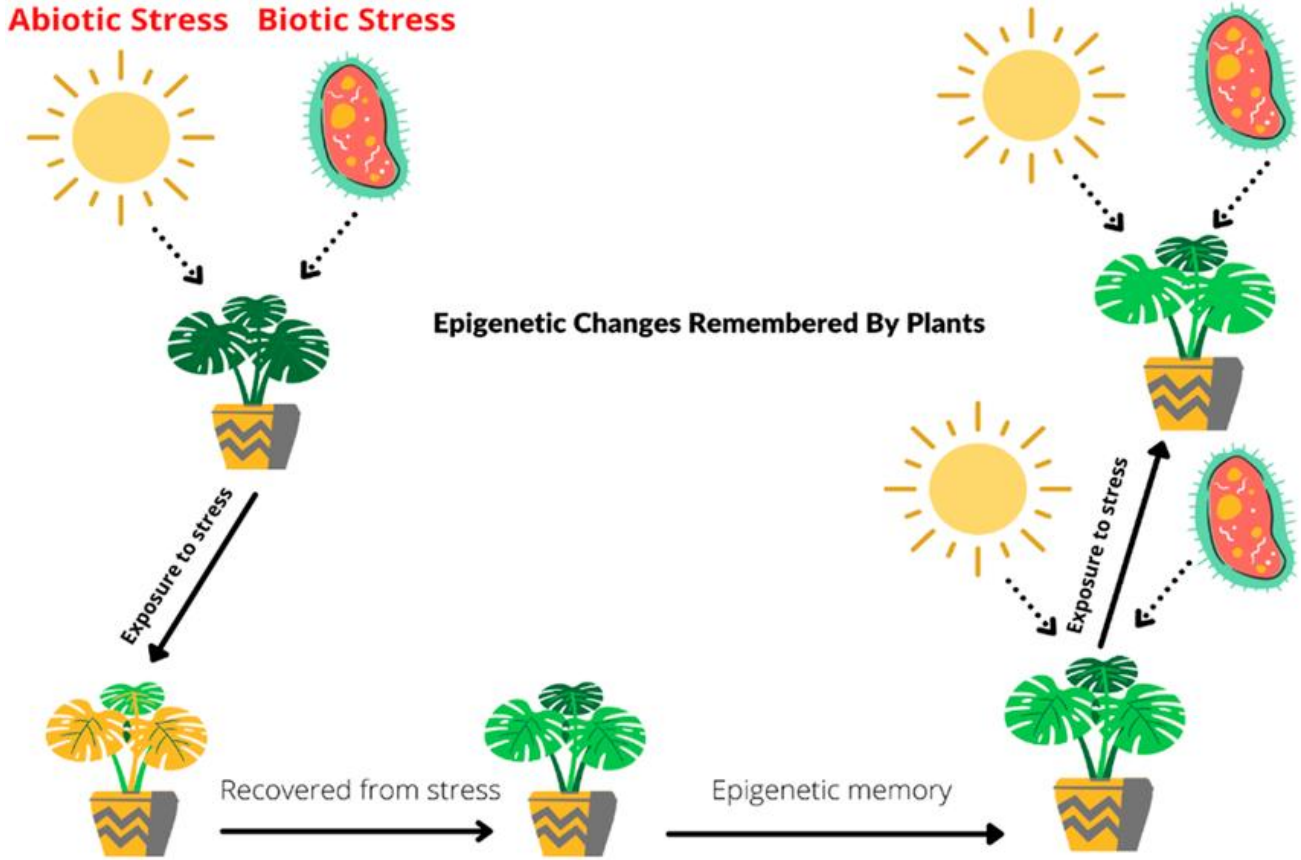
•The germline derives from floral meristems, which in turns derives from the shoot apical meristem (SAM)
•The SAM is purely vegetative for most of the plant's life — making leaves, stems, branches
•At flowering, environmental signals (photoperiod, vernalization) trigger the SAM to convert into an **inflorescence meristem**, then **floral meristems**
•Floral meristems are still somatic tissue — they generate sepals, petals, stamens, carpels
•Only inside stamens and carpels do a handful of cells then differentiate *de novo* into meiocytes (PMCs and MMCs)
•These meiocyte precursors are **ordinary somatic cells that acquire reproductive fate late** — they were never set aside, never protected, never isolated from environmental experience
•The entire vegetative life of the plant — all its responses to stress, drought, pathogens, temperature — runs through the same meristematic lineage that will eventually produce gametes
•**There is no moment of sequestration.** In animals, the germline is walled off in the embryo. In plants, the germline is recruited from soma at the last possible moment, flower by flower, throughout reproductive life

Lack of a Weismann’s barriers in plants

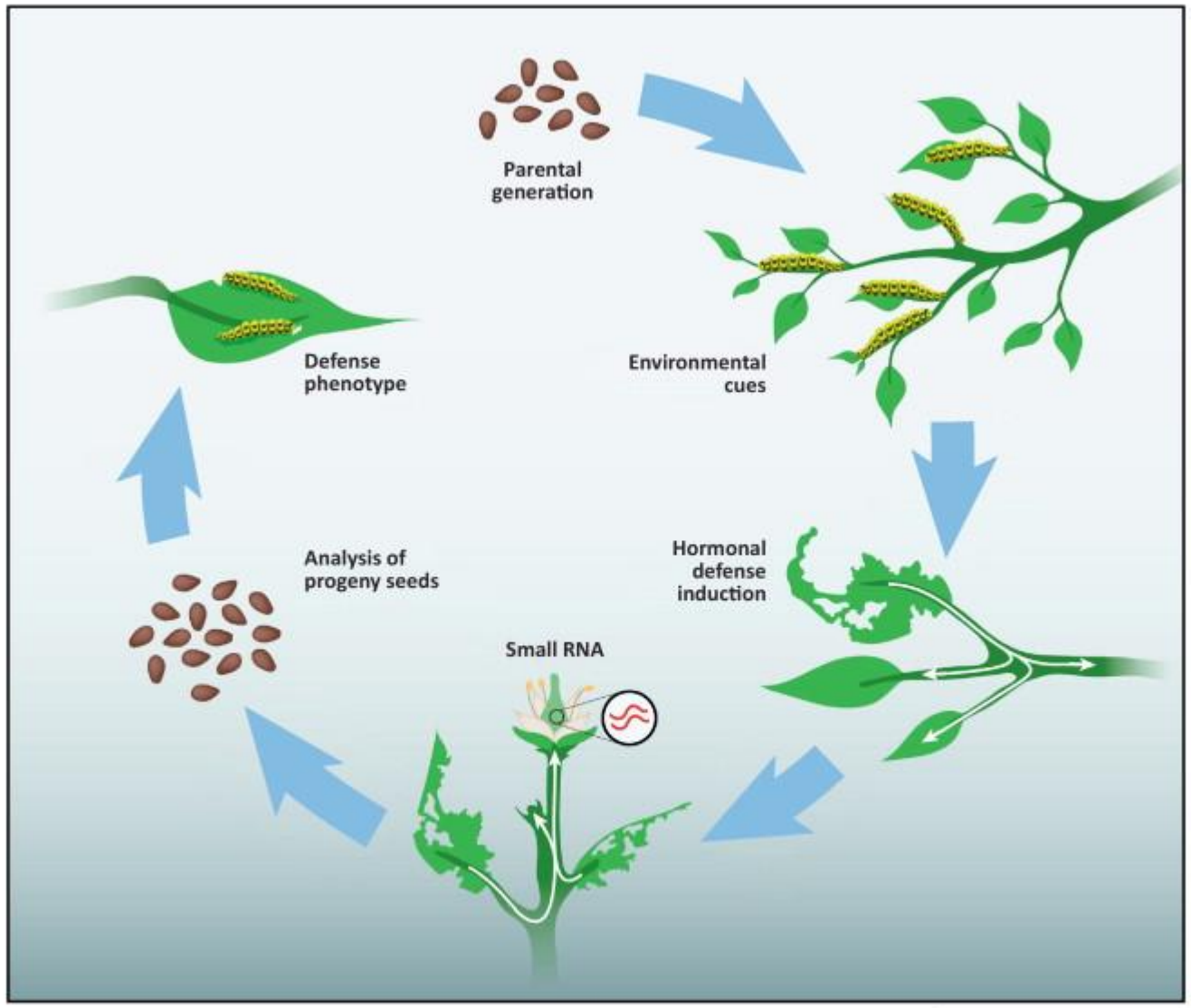
(at the molecular level)

- **Limited reprogramming scope.** Plant reprogramming during gametogenesis is partial. It primarily targets transposable elements via small RNA pathways (24-nt siRNAs) in companion cells (tapetum, endosperm), not necessarily gene-body methylation or stress-induced marks at coding loci.

- **Chromatin memory systems** (often using Polycomb) are conserved in plants and can maintain repressed or active transcriptional states through multiple mitotic divisions, providing a cellular memory mechanism that survives into the germline lineage.



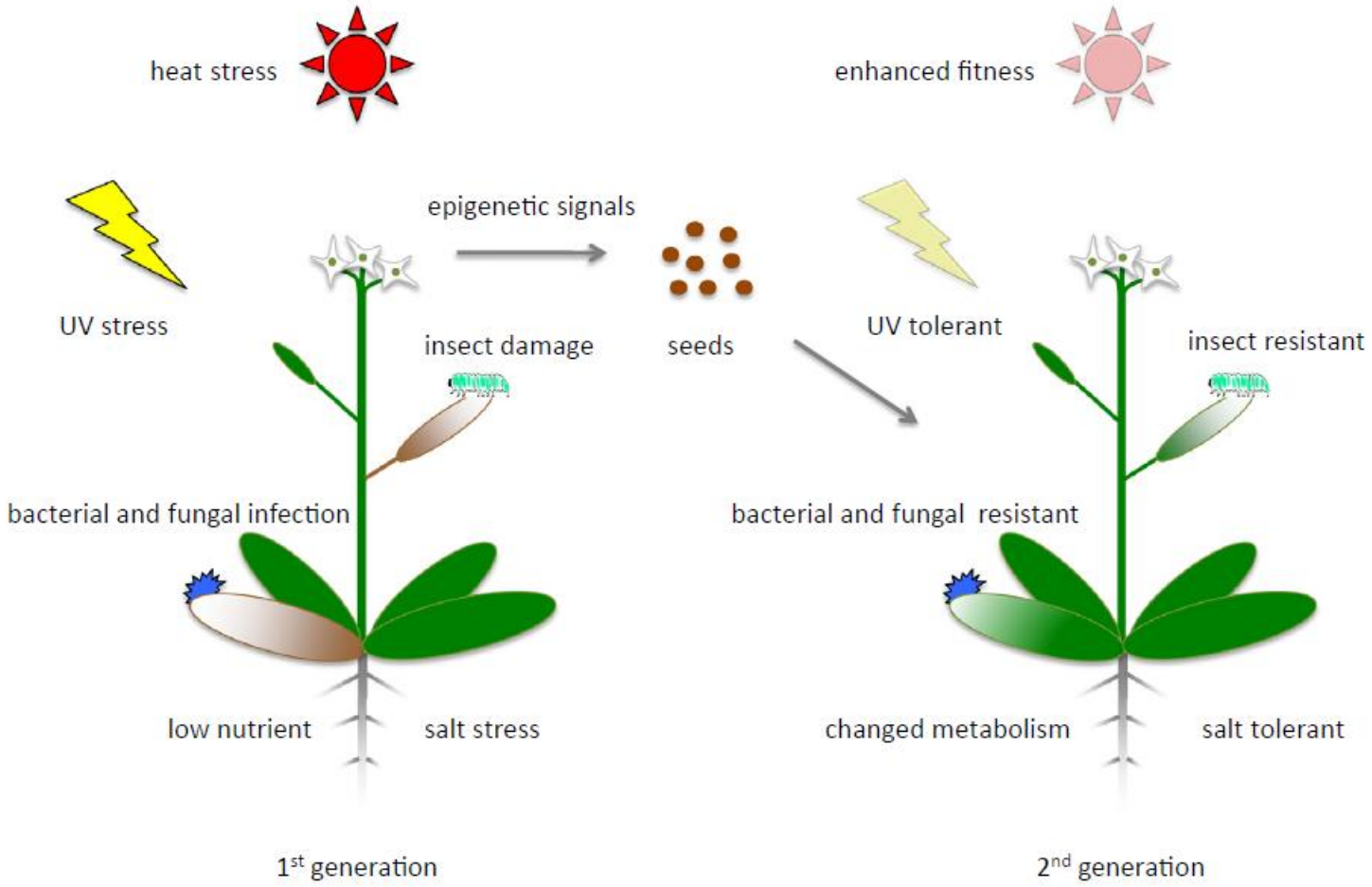
Transgenerational pathogen priming



Infection with bacterial pathogens primes offspring plants (F1 and sometimes F2) for faster and stronger immune responses (systemic acquired resistance, SAR). Associated with hypomethylation at defense gene loci and primed H3 acetylation.

Slaughter et al. 2012, *PNAS*; Luna et al. 2012

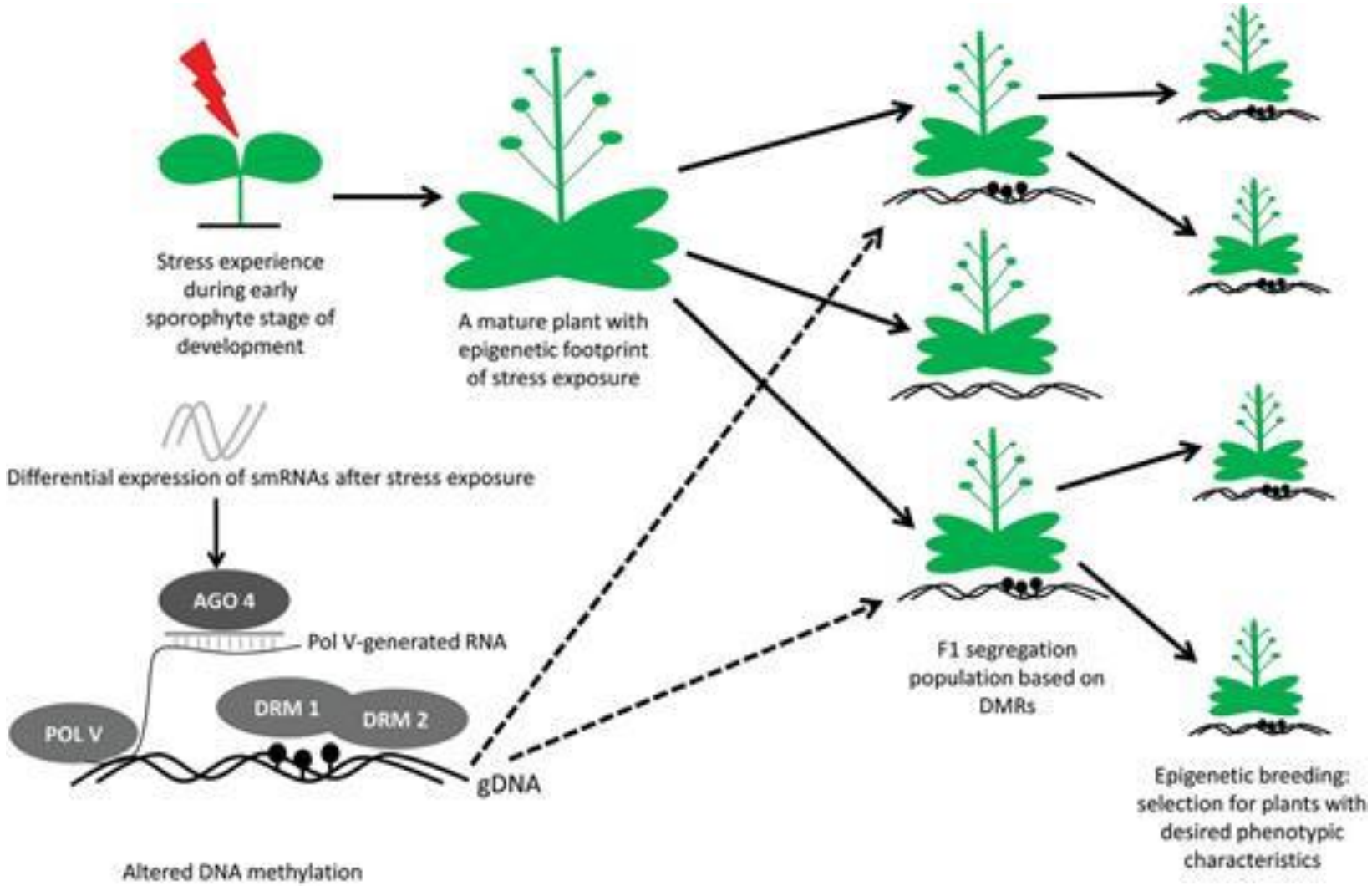
Stress-induced transgenerational inheritance



Stress-induced methylation changes
Exposure of *Arabidopsis* to UV, pathogen infection, or salinity induces changes in global methylation patterns (Boyko & Kovalchuk, 2010–2011 and others) and increased homologous recombination frequency.

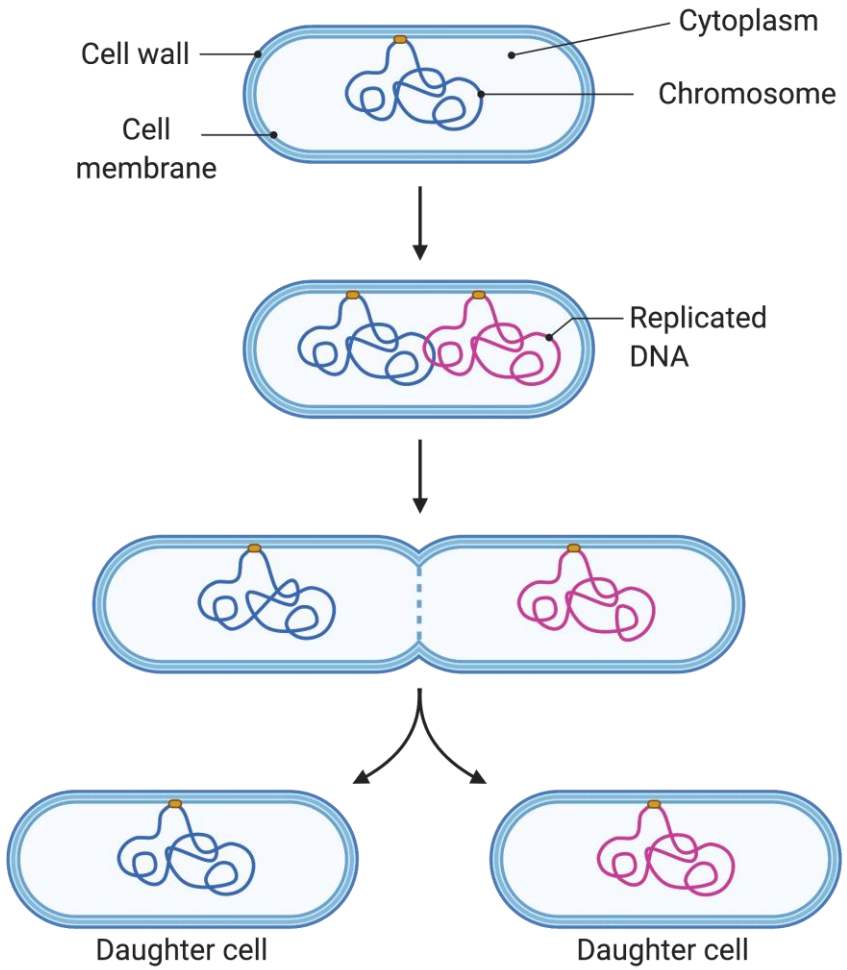
These changes persist in offspring that were never exposed to the stress, and are associated with increased stress tolerance — suggesting adaptive TEI.

Epigenetic breeding



Weismann's barrier in unicellular organisms

- The barrier is **meaningless by definition** — there is no soma/germline distinction; every cell *is* the germline
- Any epigenetic change acquired during the cell's lifetime is directly transmitted to daughter cells at division
- DNA methylation changes, chromatin states, prion-like protein conformations — all inherited mitotically
- Environmentally induced changes (stress, nutrient shifts) can propagate indefinitely through clonal lineages

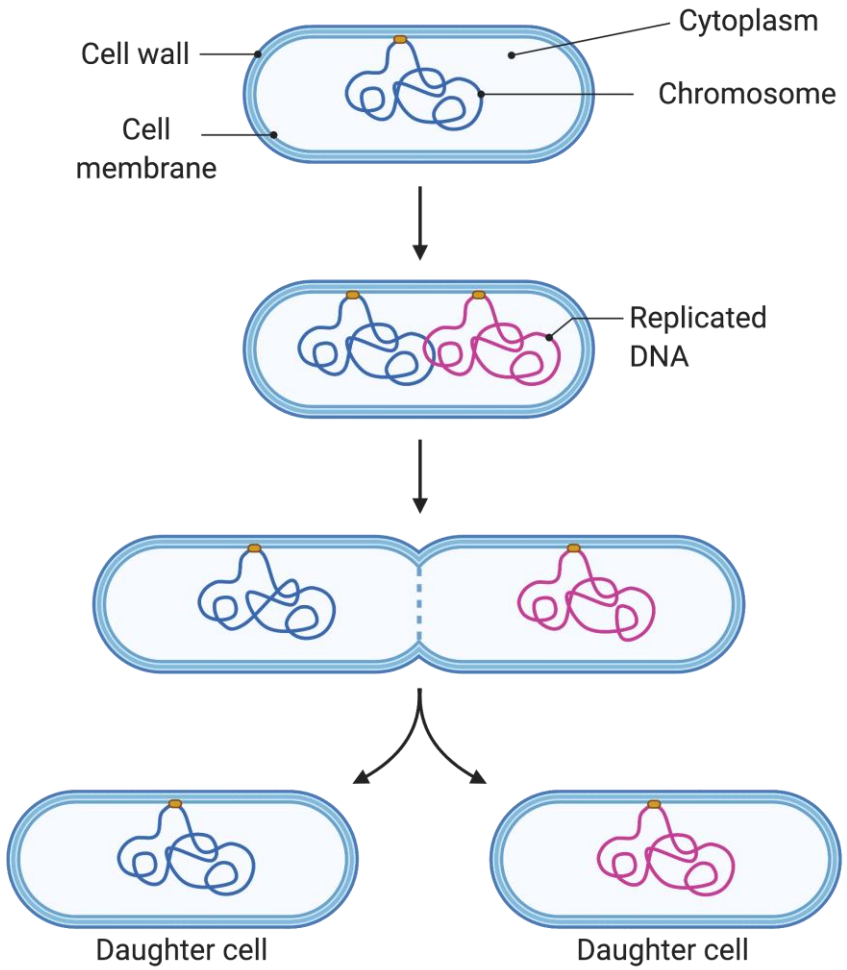


Weismann's barrier in unicellular organisms

- The barrier is **meaningless by definition** — there is no soma/germline distinction; every cell *is* the germline
- Any epigenetic change acquired during the cell's lifetime is directly transmitted to daughter cells at division
- DNA methylation changes, chromatin states, prion-like protein conformations — all inherited mitotically
- Environmentally induced changes (stress, nutrient shifts) can propagate indefinitely through clonal lineages

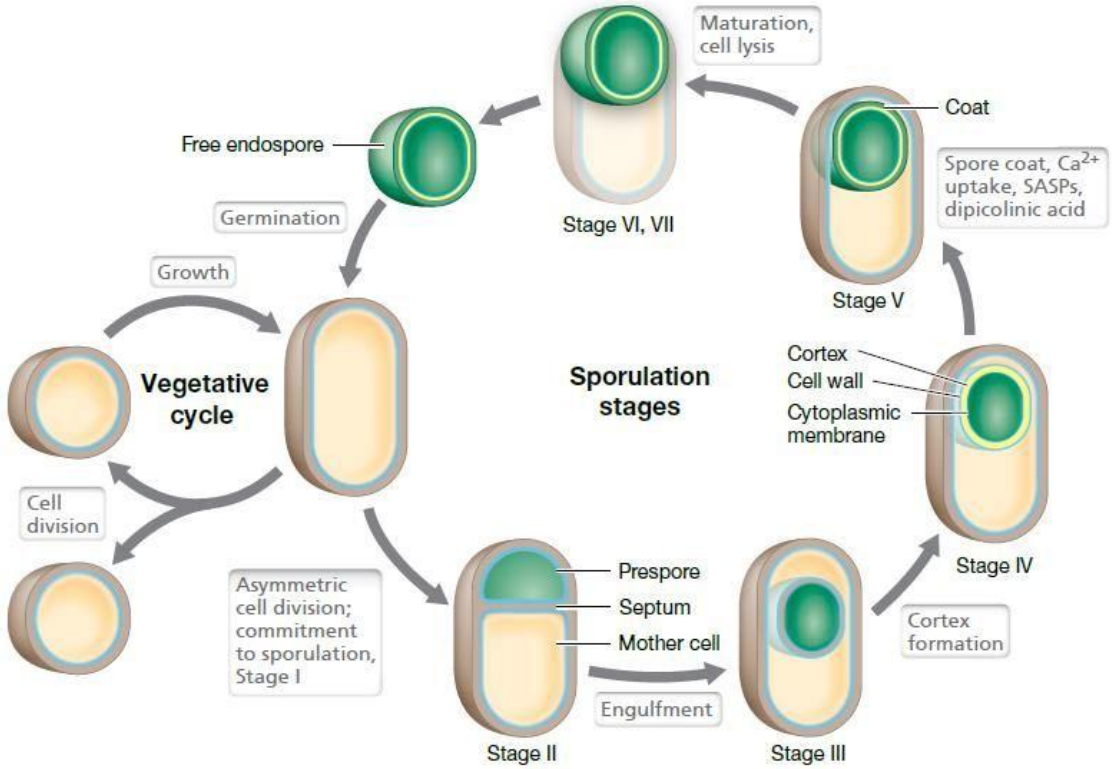
- Paradox: the organism with the *least* barrier has the *most* reprogramming pressure — selection rapidly purges maladaptive epigenetic states because generation times are so short

- Weismann's barrier can be seen as evolution's solution to a problem unicellulars don't have: protecting the germline from somatic noise accumulated over a long multicellular lifetime



Bet-hedging in *B. subtilis* sporulation (Elowitz lab and others)

- Genetically identical cells in the same environment stochastically switch between sporulating and non-sporulating states
- The switch is mediated by a bistable circuit involving Spo0A phosphorylation — an epigenetic toggle, not a mutation
- The "decision" is inherited by daughter cells for several generations before resetting
- Classic demonstration that heritable phenotypic variation without genetic change is adaptive in fluctuating environments

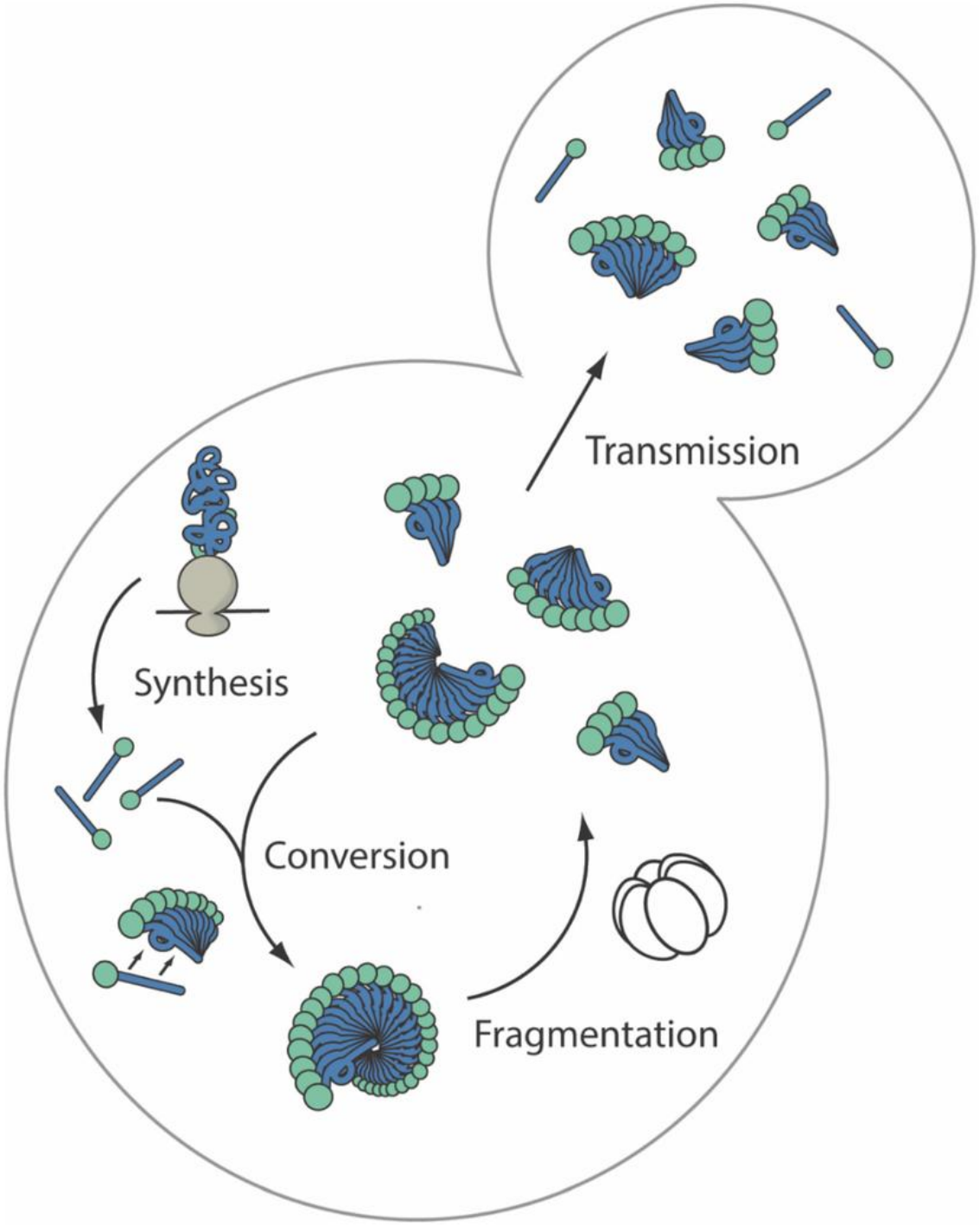


Prions in Yeast

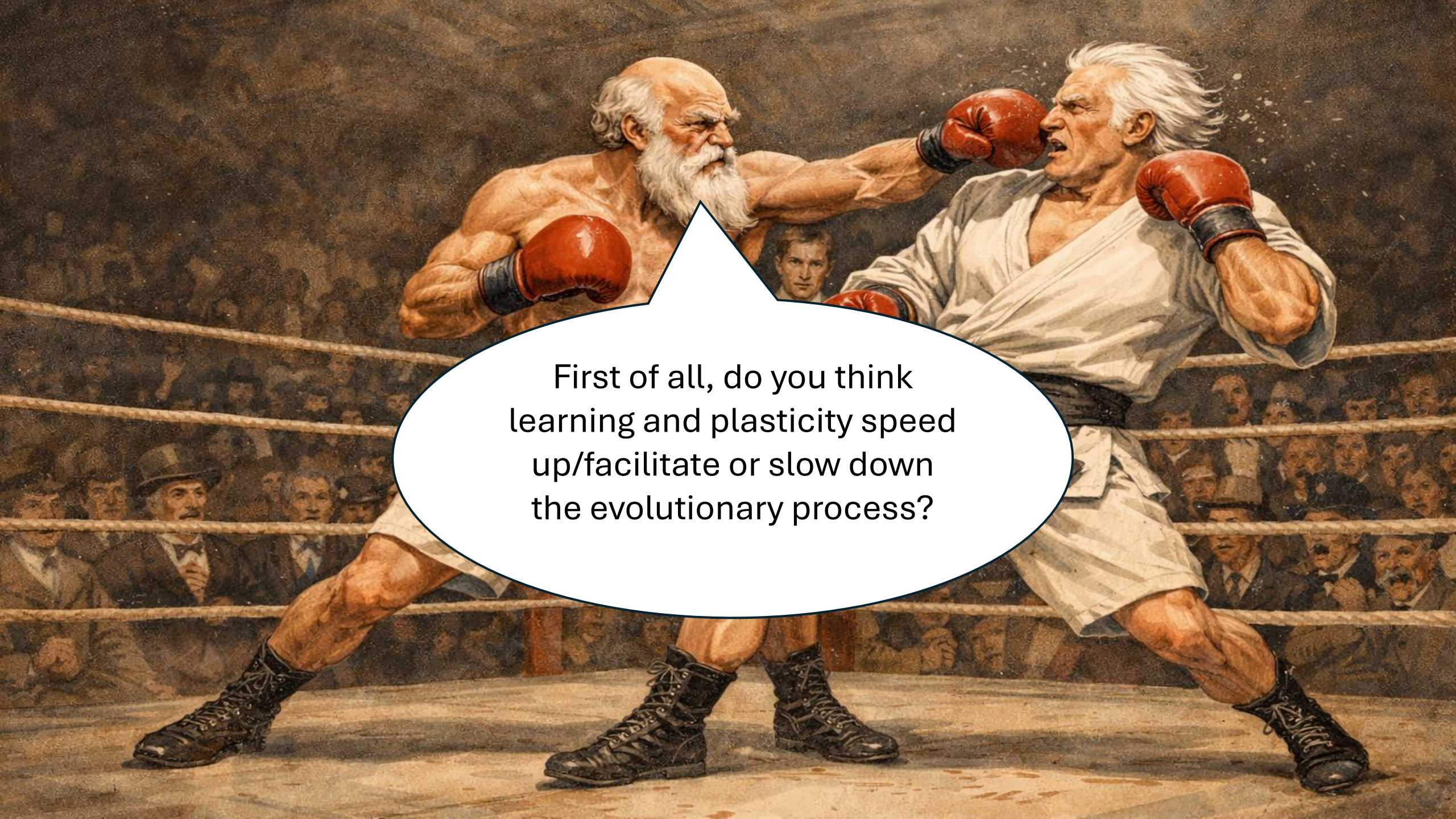
•The prion conformation is self-templating and inherited indefinitely through cell division — purely protein-based epigenetic inheritance

•An example is the Sup35 protein can adopt two conformations: normal (translation terminator) and prion ([PSI+]), which causes read-through of stop codons.

•[PSI+] has been suggested to uncover cryptic genetic variation by suppressing stop codons, potentially revealing new phenotypes for selection to act on



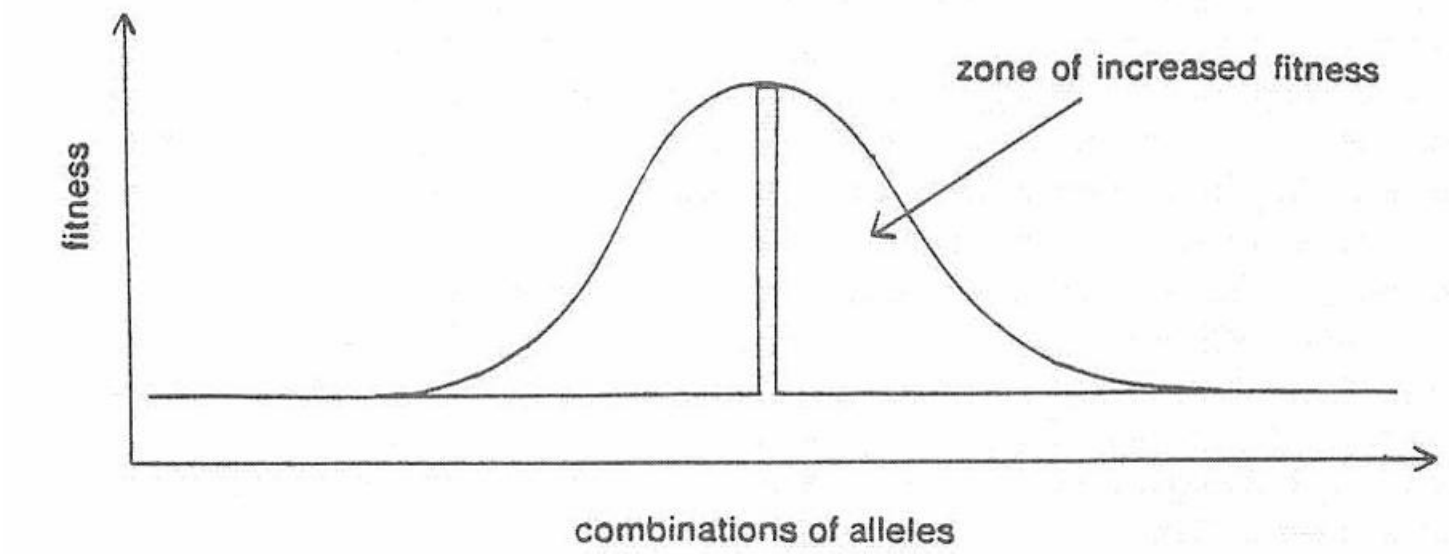

Will Lamarck win the match?
At least some rounds?
Which rounds?
When does one win and when the other?

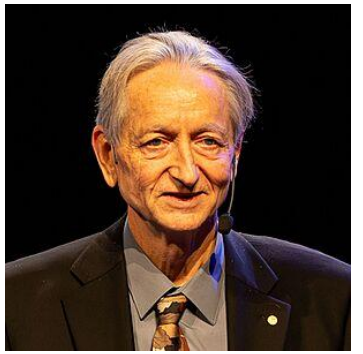

First of all, do you think learning and plasticity speed up/facilitate or slow down the evolutionary process?

Complex Systems **1** (1987) 495–502

How Learning Can Guide Evolution

Geoffrey E. Hinton
Steven J. Nowlan
Computer Science Department, Carnegie-Mellon University,
Pittsburgh, PA 15213, USA



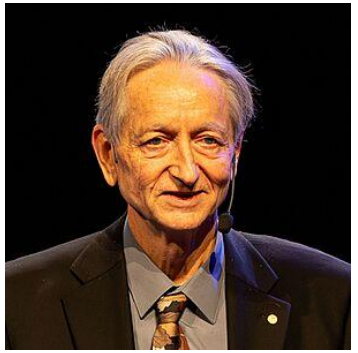


Geoffrey Hinton, Nobel in Physics 2024

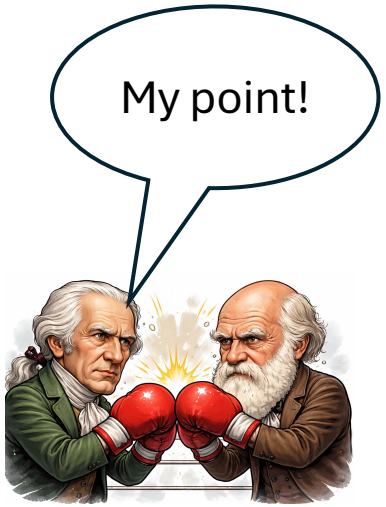
Complex Systems **1** (1987) 495–502

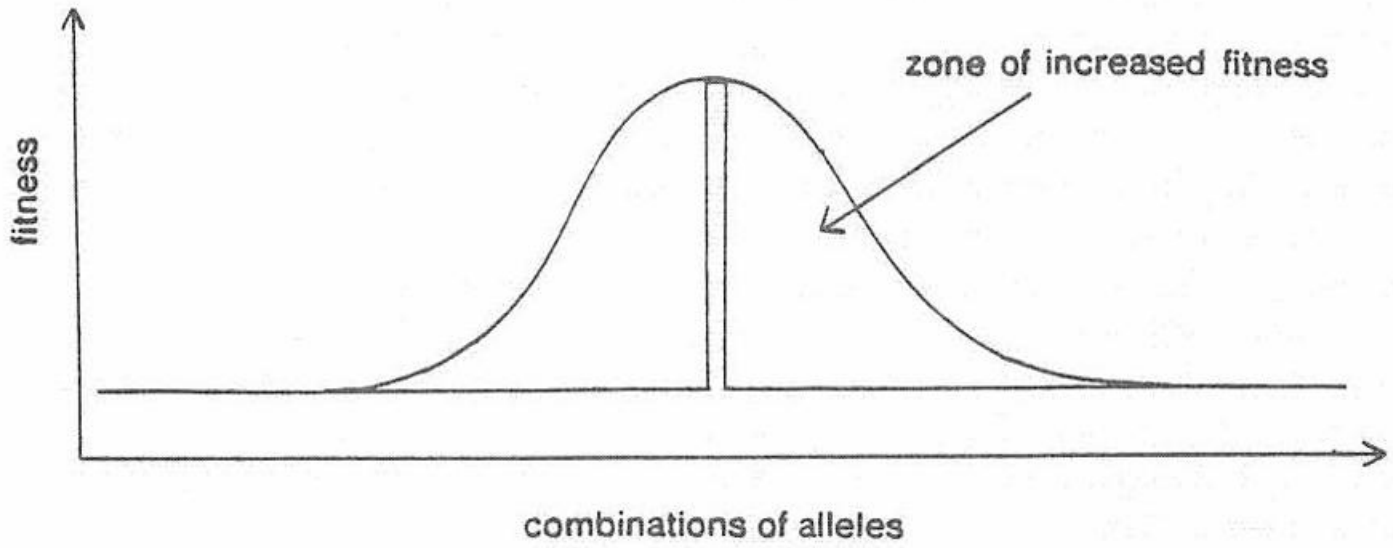
How Learning Can Guide Evolution

Geoffrey E. Hinton
Steven J. Nowlan
Computer Science Department, Carnegie-Mellon University,
Pittsburgh, PA 15213, USA



Geoffrey Hinton, Nobel in Physics 2024

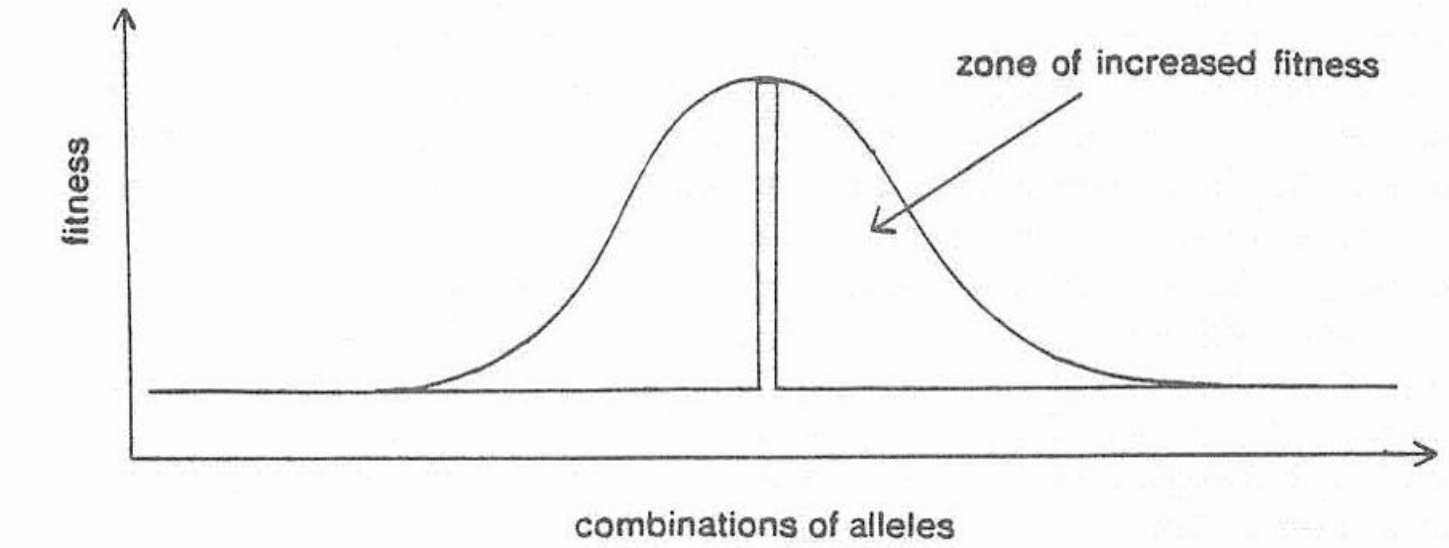




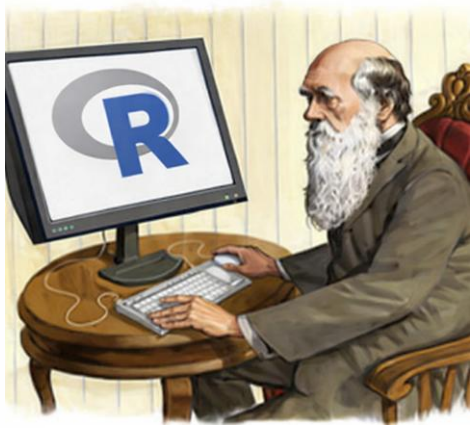
Complex Systems **1** (1987) 495–502

How Learning Can Guide Evolution

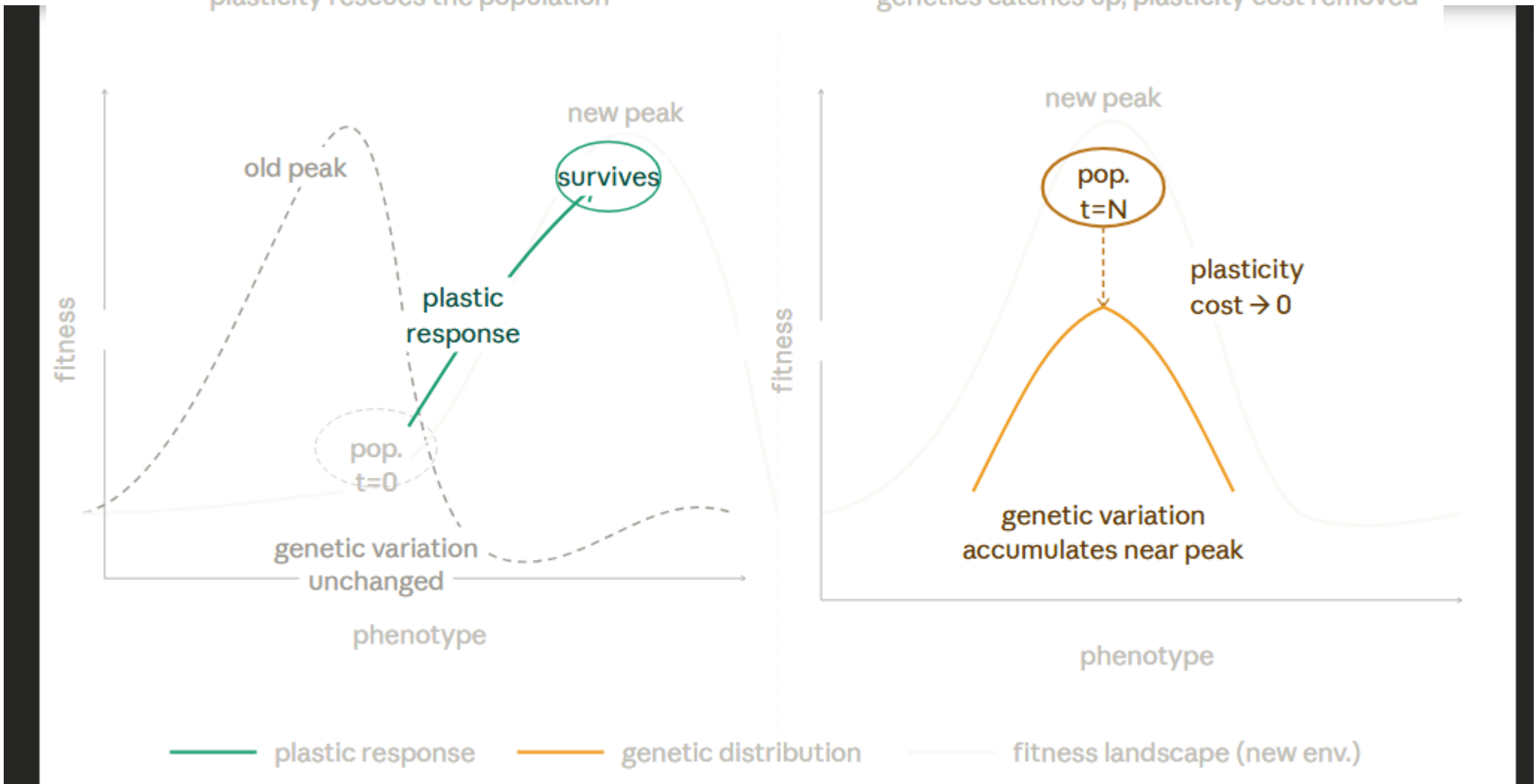
Geoffrey E. Hinton
Steven J. Nowlan
Computer Science Department, Carnegie-Mellon University,
Pittsburgh, PA 15213, USA



Geoffrey Hinton, Nobel in Physics 2024



This leads to what was theorized as Baldwin's effect



James Mark Baldwin
(1861-1934)

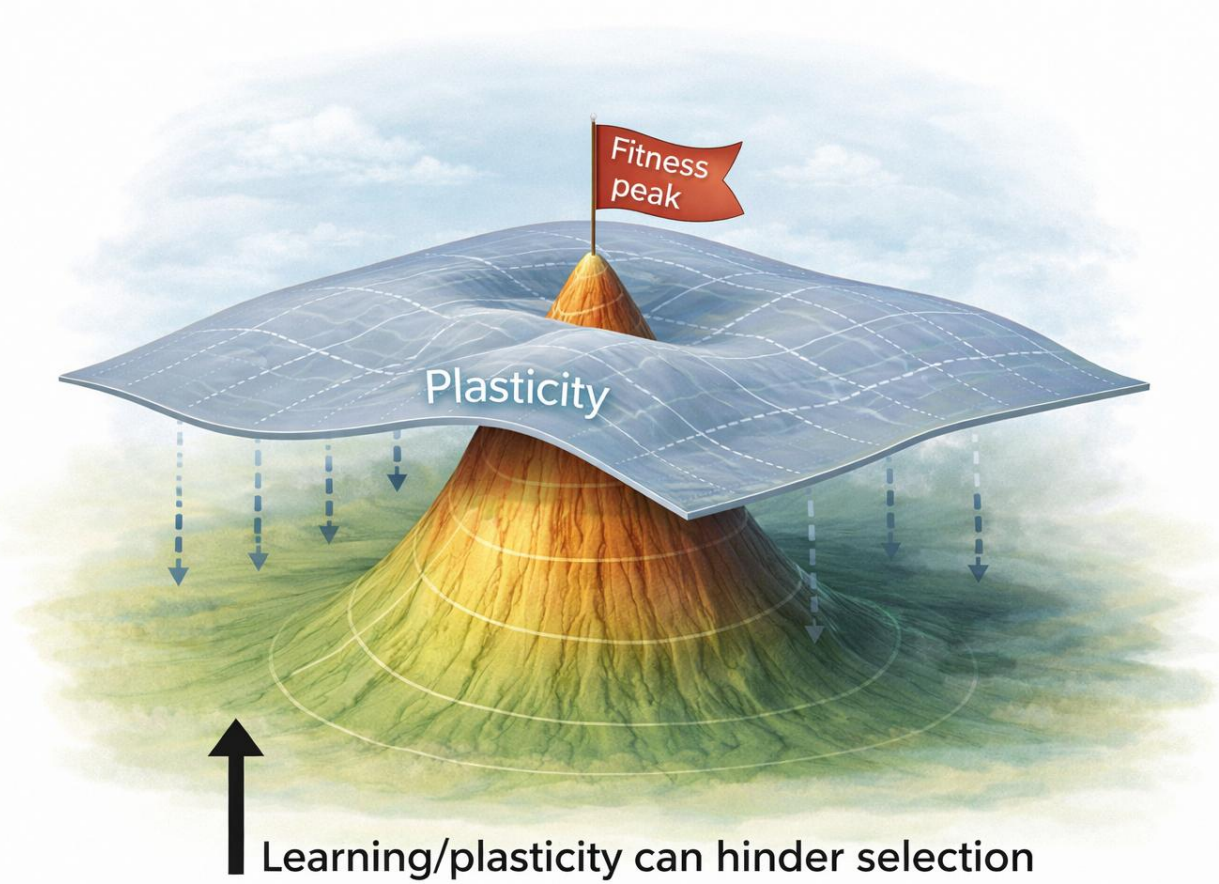
The Baldwin effect

- Plasticity allows individuals to survive in a new environment before genetic adaptation can occur
- Survivors reproduce → genetic variants closer to the plastic phenotype are now under selection
- Over generations the plastic response becomes genetically encoded — **canalization**
- Net effect: plasticity converts a fitness spike into a slope — **evolution can now climb it**
- Learning/behavior/epigenetics can all play the role of plasticity

However learning/plasticity can even hinder evolution!

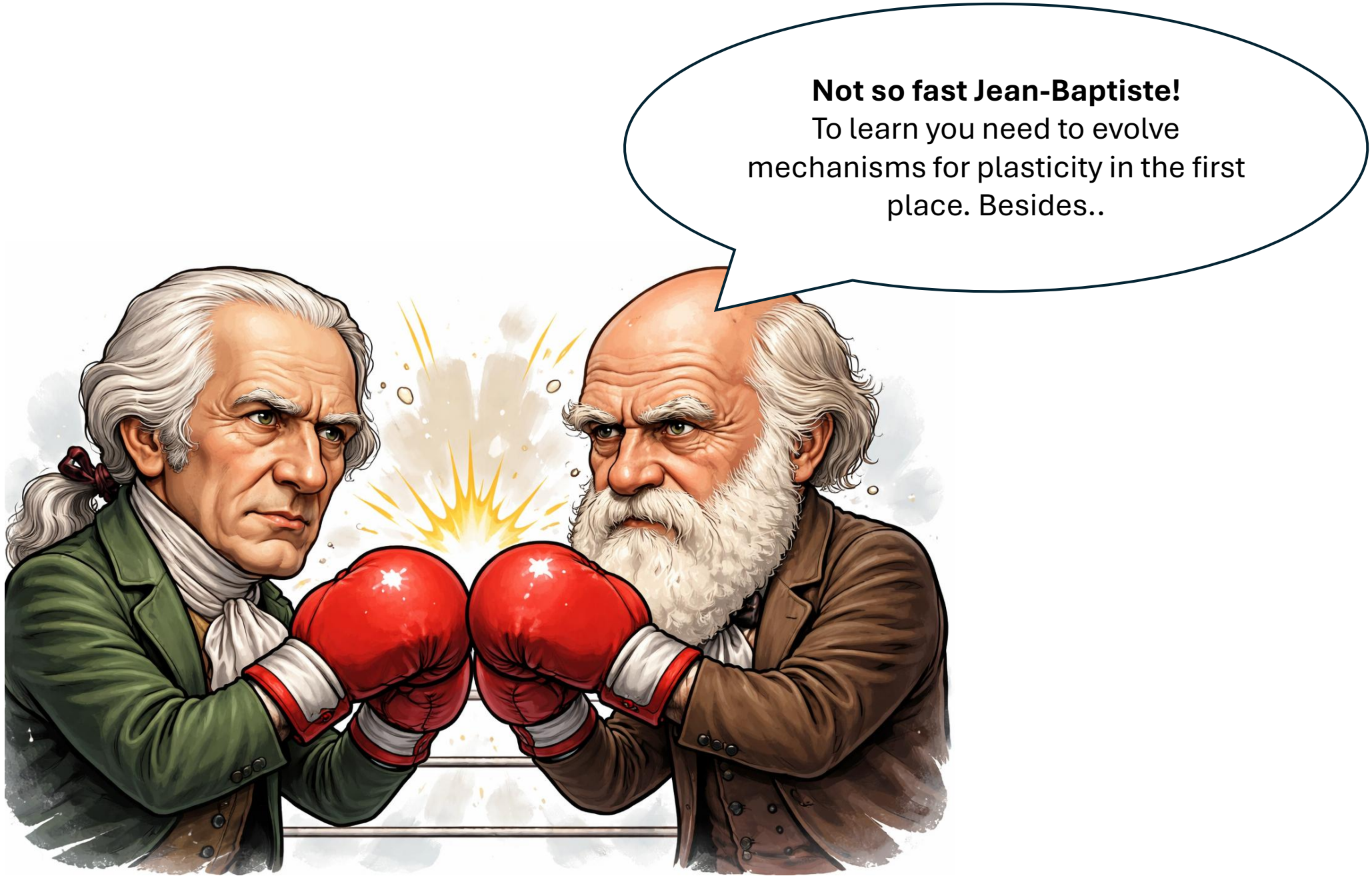
Genetic assimilation stalls!

- If plasticity buffers phenotype against environmental change, selection pressure on the underlying genotype is reduced the population stays genetically variable longer, slowing fixation (Waddington; West-Eberhard)
- This is what is often suggested f relaxed natural selection in spe with high learning/high cultural transmission

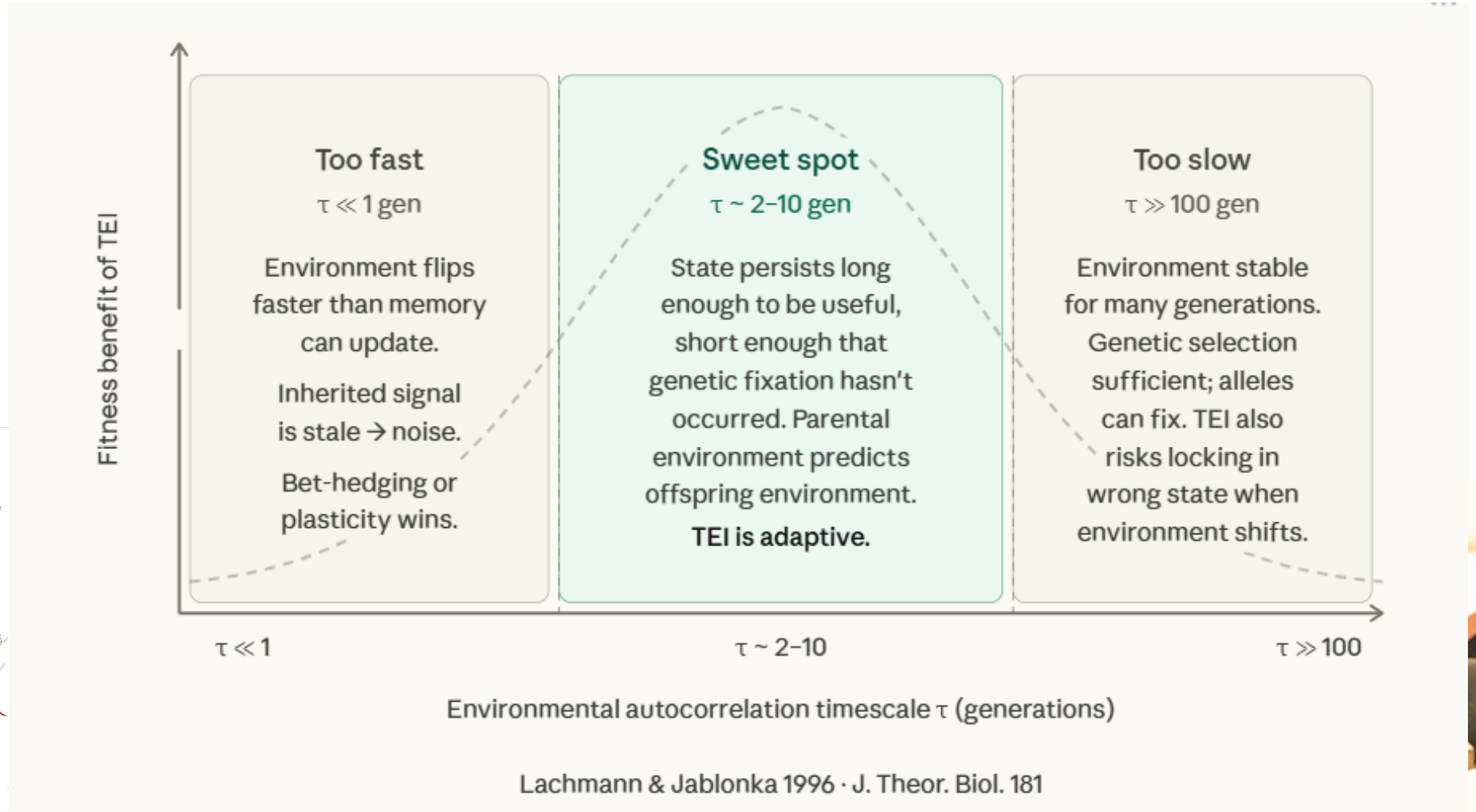


Why then TEI is not more frequent? Are we just blind to it?

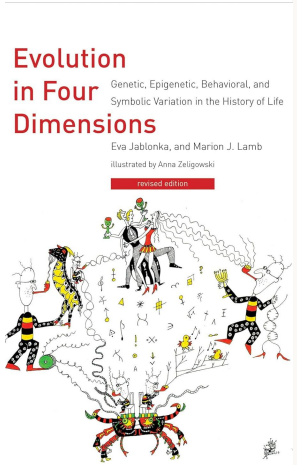
Learning and TEI are great!
If you can learn from the environment and teach it to your kids, why having to wait for your slow mutations and individuals die for natural selection?


Not so fast Jean-Baptiste!
To learn you need to evolve mechanisms for plasticity in the first place. Besides..

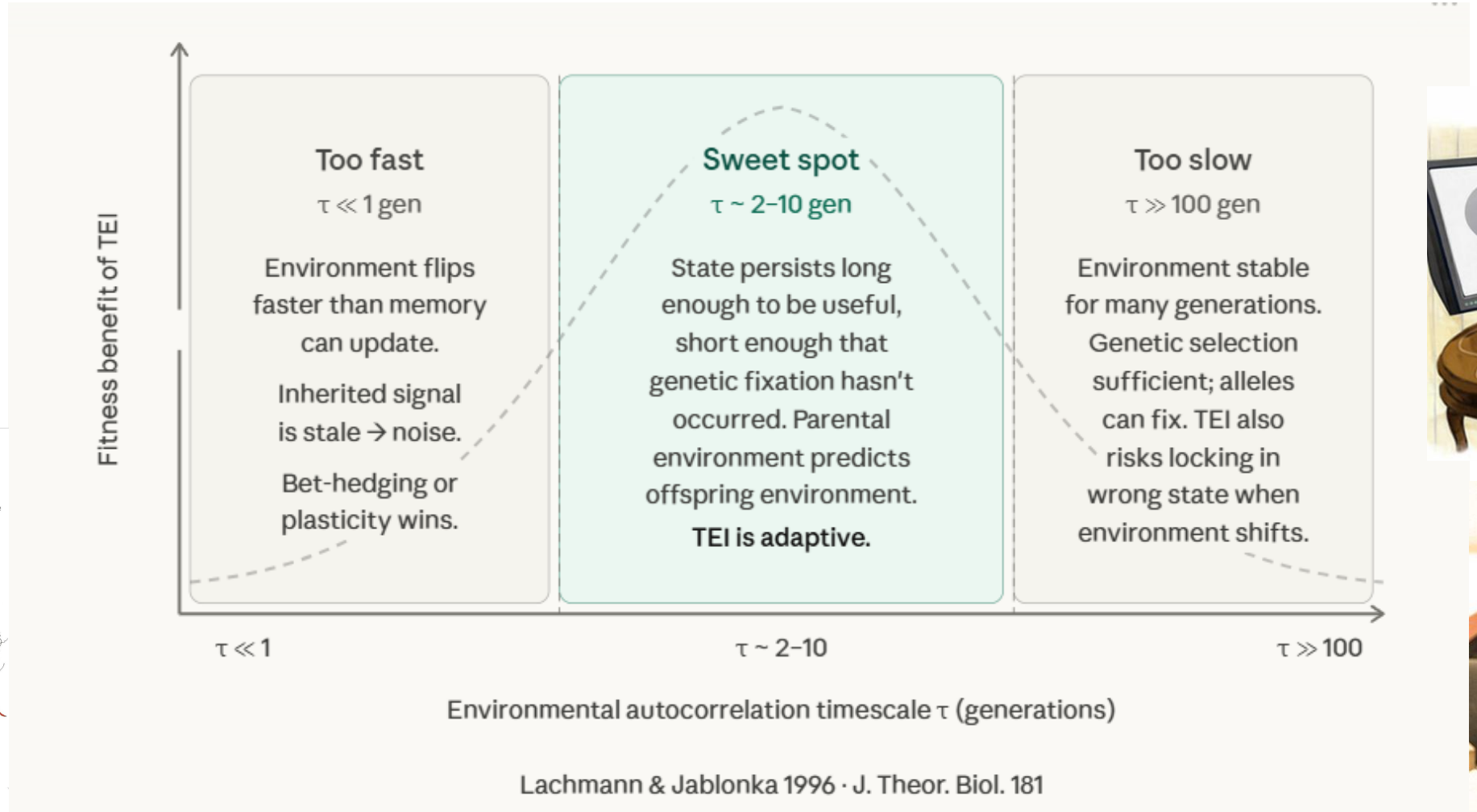
TEI is only advantageous under “narrow” conditions



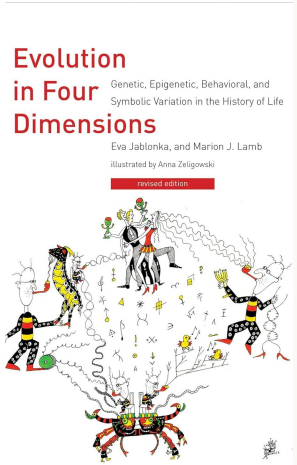
Lachmann & Jablonka 1996 · J. Theor. Biol. 181

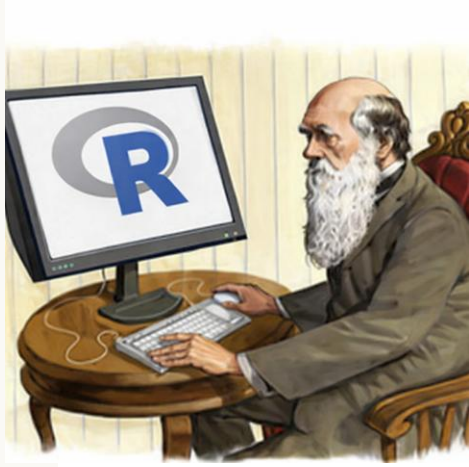


TEI is only advantageous under “narrow” conditions



Lachmann & Jablonka 1996 · J. Theor. Biol. 181






So who won?

EDITED BY
MASSIMO PIGLIUCCI
AND
GERD B. MÜLLER

EVOLUTION
THE EXTENDED
SYNTHESIS

CHALLENGING TH
MODERN SYNTHES

Adaptation, Development, and Inherita

Edited by PHILIPPE HUNEMAN
and DENIS WALSH

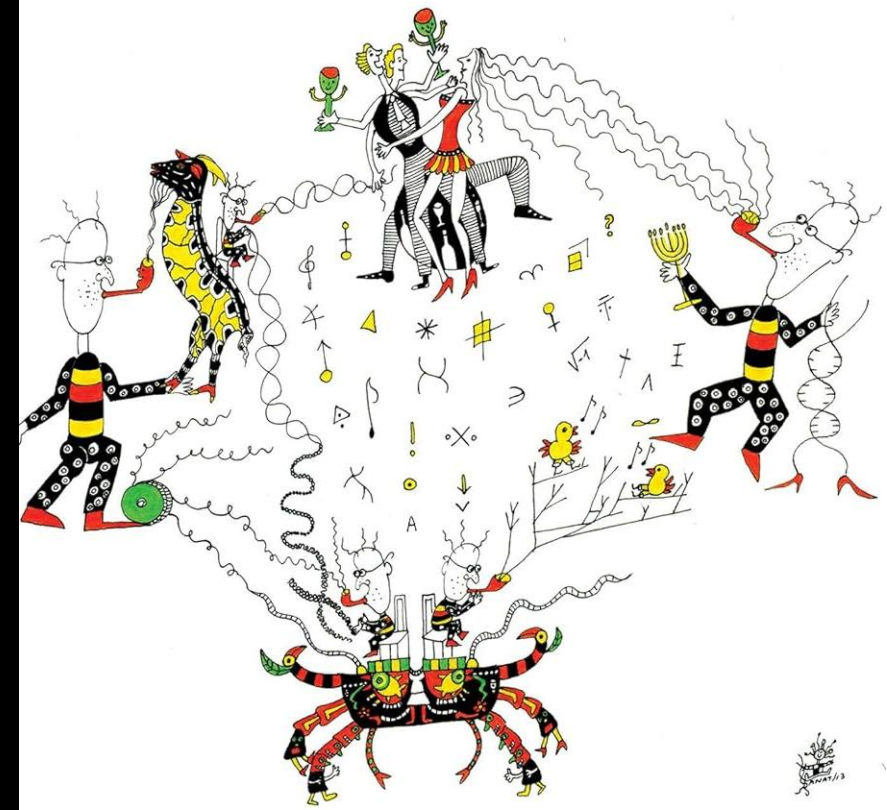
Evolution in Four Dimensions

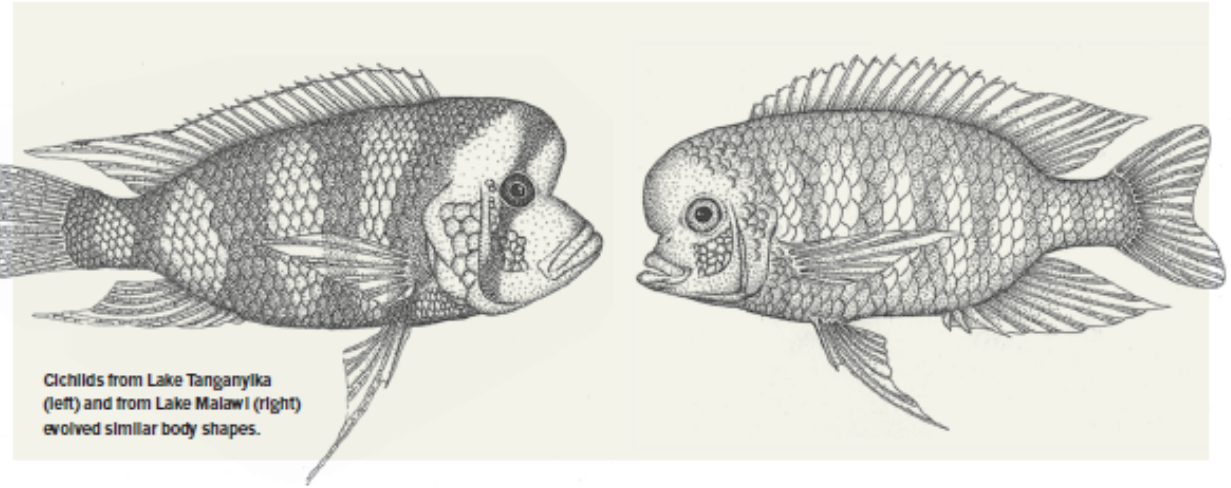
Genetic, Epigenetic, Behavioral, and Symbolic Variation in the History of Life

Eva Jablonka, and Marion J. Lamb

illustrated by Anna Zeligowski

revised edition





Cichlids from Lake Tanganyika (left) and from Lake Malawi (right) evolved similar body shapes.

Does evolutionary theory need a rethink?

Researchers are divided over what processes should be considered fundamental.

POINT

Yes, urgently

Without an extended evolutionary framework, the theory neglects key processes, say Kevin Laland *and colleagues.*

Charles Darwin conceived of evolution by natural selection without knowing that genes exist. Now mainstream evolutionary theory has come to focus almost exclusively on genetic inheritance and processes that change gene frequencies.

Yet new data pouring out of adjacent fields are starting to undermine this narrow stance. An alternative vision of evolution is beginning to crystallize, in which the processes by which organisms grow and develop are recognized as causes of evolution.

Some of us first met to discuss these advances six years ago. In the time since, as members of an interdisciplinary team, we have worked intensively to develop a broader framework, termed the extended evolutionary synthesis[1] (EES), and to flesh out its structure, assumptions and predictions. In essence, this synthesis maintains that important drivers of evolution, ones that cannot be reduced to genes, must be woven into the very fabric of evolutionary theory.

We believe that the EES will shed new light on how PAGE 162 ▶

COUNTERPOINT

No, all is well

Theory accommodates evidence through relentless synthesis, say Gregory A. Wray, Hopi E. Hoekstra *and colleagues.*

In October 1881, just six months before he died, Charles Darwin published his final book. *The Formation of Vegetable Mould, Through the Actions of Worms*[11] sold briskly: Darwin's earlier publications had secured his reputation. He devoted an entire book to these humble creatures in part because they exemplify an interesting feedback process: earthworms are adapted to thrive in an environment that they modify through their own activities.

Darwin learned about earthworms from conversations with gardeners and his own simple experiments. He had a genius for distilling penetrating insights about evolutionary processes — often after amassing years of observational and experimental data — and he drew on such disparate topics as agriculture, geology, embryology and behaviour. Evolutionary thinking ever since has followed Darwin's lead in its emphasis on evidence and in synthesizing information from other fields.

A profound shift in evolutionary thinking began PAGE 163 ▶

NICK VEASEY/GARDEN PHOTOS.COM

Genetic inheritance and the modern synthesis are still the core of evolutionary theory

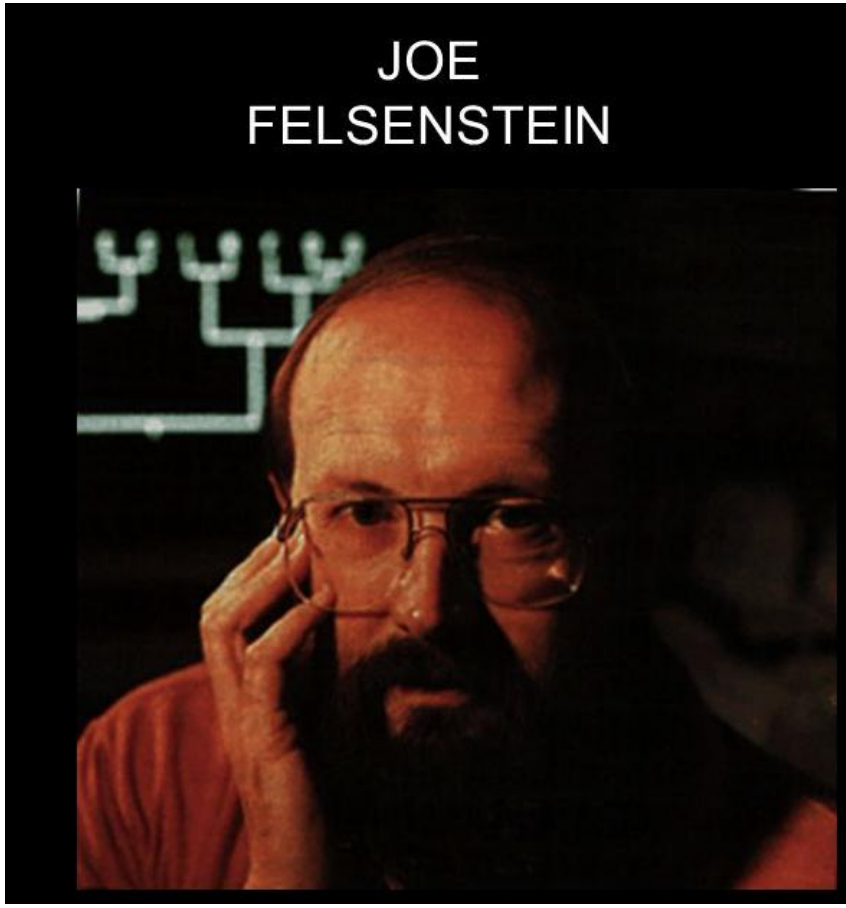
- …and well represent the vast majority of evolutionary processes regarding biological evolution (Darwin still wins 150+ years later the On the Origin of Species.
- But:
  - In certain relatively rare instances transgenerational epigenetic transmission is real
  - Other channels of inheritance are very important in some cases (e.g. cultural evolution in humans)
  - Other phenomena have been “appropriated” by the Extended Evolutionary Synthesis (e.g. role of development in evolution, niche construction, role of stochasticity in evolution with Punctuated Equilibria and “multi-level selection”) but – at least in my opinion – they fit already well in the “classical Darwinian framework”.

Some rants and diatribes by Joe (Felsenstein)

*Rant #5: **The Modern Synthesis has not been replaced.** Sure, all sorts of new phenomena have come along since the 1940s: neutral mutation, lateral gene transfer, symbiosis, evo-devo, epigenetics, etc. And we could declare the death of the current Synthesis each time one came along. But here's why we shouldn't do that:*

- *Otherwise every time John Blotz pointed out a new phenomenon he could strut around publicizing the fact that he, the great Blotz, had invalidated the evolutionary synthesis, and now we had (ta-da!) the Blotzian Synthesis. But he would be shocked a year or two later when Jane Schmerz came along and invalidated the Blotzian Synthesis in favor of the new Schmerzian Synthesis. And so it would go, synthesis after synthesis, until everyone was totally confused, and most people were several syntheses behind.*
- *Meanwhile the public would be continually told that all that stuff they learned in secondary school, about mutation and natural selection and some other evolutionary forces, was all wrong, because now we had the Blotzian (er, oops, actually the Schmerzian) Synthesis instead.*

It would be (temporarily) great for Blotz's and Schmerz's careers and egos, but a disaster for everyone else.



https://felsenst.github.io/rants.html

And now full on “Modern Synthesis”!!