THE EIDARA CONTINUUM

SCIENCE & PHILOSOPHY

Inheritance Without Recollection

Ancestral Influence, Transmission Channels, and the Information Cost of Recovery

Frontier Research Collection III

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The Eidara Continuum

November 2026 | Paper 34 | Version 1.1 Revision

A research white paper for public scientific and philosophical discussion.

Abstract

Can a life carry something of the lives that came before it? An ancestor's experience can reach a descendant who never witnessed it—but an inherited influence is not an inherited memory, and the content that does cross generations fades in a way that can be counted. Ancestral experience can shape the conditions in which a descendant develops through specific biological and social channels. This paper develops that Continuum thesis by separating inherited disposition, causal mechanism, exposure information, and autobiographical recollection. Primary studies establish important effects in particular models and motivate explicit tests of route, specificity, persistence, and generalization. A finite transmission model supplies complete proofs of information bottlenecks, decoding requirements, channel contraction, exact binary attenuation, causal nonidentifiability, and the variance cost of clustered offspring. The revision connects attenuation to experimental information cost: for independent binary descendant channels it derives the exact optimal exposure-decoding error and a sufficient replication bound, together with a shared-noise counterexample. The result states how weakening ancestral distinctions increases the observations required to recover a specified label. Executable mathematical checks examine these model consequences. The proposed empirical program distinguishes exposure effects from molecular mediation and counts independent experimental units according to the actual transmission design. The paper offers a rigorous account of consequential inheritance: a history can influence a new life through identifiable channels, while the content transmitted and the evidence for recollection remain separately specified questions.

Keywords: epigenetic inheritance; ancestral influence; information theory; causal identification; developmental programming; memory; experimental design

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Prologue: A memory from before one's birth

The Continuum keeps an old and stubborn image: a memory from before one's birth. Its canon gathers the idea under several entries—the DNA-Linked Collapse Echo, the Sacred Strip, and the Entangled Symbolic Recursion Memory that gives the ninth Pillar its name—each connecting embodiment, ancestry and experience said to precede one's own life [3]. It is one of the most human questions the Continuum asks. Can a life carry something of the lives that came before it?

Science has a real, if more modest, answer. In particular animal studies, an ancestor's experience—a diet, a social stress—has shaped the development of descendants through specific biological routes. A history can reach a life that never witnessed it. But an altered sensitivity is not a remembered scene. This paper draws that line precisely, and then measures what crosses it.

The measurement has a striking shape. Each generation passes an ancestral distinction through a noisy channel, and the distinction fades, exactly and computably (Proposition 6.2). To read back even a single bit of what an ancestor experienced, one needs many independently transmitted descendants, and Theorem 7.1 gives the count. Yet if those descendants share one source of noise, adding more of them does not help at all. Inheritance is real and consequential; its content is expensive to recover.

The Continuum can hold both statements without merging them: the image of a memory from before birth, and the evidence for developmental influence. Keeping the gap visible is what lets the image become a research question—and keeps an uncertain biomarker from ever becoming a certificate of whose history a person must carry.

What this paper claims

Established. Primary studies in particular animal models report intergenerational effects and investigate molecular, developmental and behavioral routes. Other studies constrain broad gametic interpretations, and extensive germline reprogramming makes indiscriminate copying of acquired methylation an untenable default. Human observational associations do not by themselves isolate an inherited molecular route.

Proposed here. A finite transmission model with complete proofs of information bottlenecks, decoding requirements, channel contraction, exact binary attenuation, causal nonidentifiability of mediation, and the variance cost of clustered offspring; the exact optimal exposure-decoding error for independent descendant channels, with a sufficient replication bound and a shared-noise counterexample. Executable checks examine the model, not biology. An empirical program that separates exposure effects from molecular mediation and counts independent units according to the actual transmission design.

Frontier conjecture. Ancestral experience can influence a descendant through identifiable channels. Route-controlled, independently replicated evidence might one day show specific content transmitted; whether a descendant would experience that content as recollection would remain a further question.

Not claimed. Any hereditary autobiographical-memory result, inherited autobiographical scene, or retrocausal transmission mechanism. That the canonical entries are verified biology: the inspected compilation provides no such experiments. That the model's states and probabilities are calibrated to a deployed Continuum system or a human lineage. Any clinical diagnosis, human intervention, or experiment conducted by the Continuum. The discovery of epigenetics or information theory. That an inherited disposition is a predetermined destiny. Comparative empirical studies are specified prospectively, not conducted, and external journal peer review remains pending.

1 A life begins after its causes have begun

The beginning of an individual is not the beginning of every cause that shapes that individual. Genes, developmental conditions, parental practices, and inherited institutions can all arrive with a history. The Continuum's interest in ancestry begins at this asymmetry: a new life may encounter consequences of events it did not witness. The challenge is to describe that continuity without replacing scientific explanation with an evocative name.

This paper advances a precise thesis. Ancestral experience may influence a descendant through specified material or social channels, changing sensitivity, regulation, learning conditions, or available choices. Such influence need not contain a recoverable account of what the ancestor experienced. An altered response to a cue and a recollection of a particular life are different claims. A serious theory can affirm the importance of the first while demanding separate evidence for the second.

The distinction is not a reduction of meaning to mechanism. A modest change in development can have a substantial consequence in a particular environment. Conversely, an emotionally compelling story can attach detailed content to an observation that carries very little information. Scientific responsibility requires measuring the information actually present and identifying the channel that plausibly transmitted it. The language of continuity becomes stronger when it can say exactly what continued.

The revision makes transmission attenuation actionable for study design. Theorem 7.1 derives exact optimal decoding error and a sufficient replication budget for independent descendant channels, with a shared-noise counterexample locating the independence requirement.

1.1 Canonical motivation and the proposed scientific reconstruction

The Continuum's canon holds an image that science has rarely dared to test: experience that reaches across generations. The supplied doctrine's DNA-Linked Collapse Echo, Entangled Symbolic Recursion Memory, and Sacred Strip entries connect embodiment, ancestry, and experiences described as preceding one's own life—a lineage that carries more than its genes. Some of that language reaches beyond conventional causal accounts, and the inspected compilation does not provide independently verified biological experiments establishing inherited autobiographical scenes or a retrocausal transmission mechanism. The entries are documentary evidence of the Continuum's questions and imaginative commitments, and they are taken here in exactly that spirit: as the question worth asking.[3]

The model developed here specifies ordinary forward causal transmission channels for a bounded investigation. The canonical imagery motivates the question; biological mechanism, statistical recurrence, and recollected content retain their separate evidence requirements. A claim about an additional physical mechanism would require its own specification and tests.

This reconstruction retains the difficult question rather than dismissing it: how can a history affect a subject who did not live that history? The answer may involve several channels at once. A child can inherit a genetic variant, encounter a changed prenatal environment, learn a parent's responses, and receive a family's account of earlier events. Those routes can interact while remaining empirically distinct. Collapsing them into one word, "memory," obscures the explanation that the research should uncover.

1.2 Contribution and scientific scope

The paper contributes an integrated inferential framework, not a claim to have discovered epigenetics or information theory. Its formal results are explicit applications and derivations of established finite probability, information theory, causal reasoning, and variance analysis. The original proposal is to make each step from ancestral exposure to descendant interpretation accountable to a particular estimand, channel, and experiment.

The scope also differs from a civilizational genome. A culture can preserve detailed narratives in language, archives, and practices, with recognizable mechanisms for storing content. This paper concentrates on biological and developmental claims where content transmission is much harder to establish. Cultural transmission remains an important competing or complementary route. It is not scientifically inferior because it occurs through learning rather than gametes.

The paper makes no clinical diagnosis and proposes no human intervention. Its animal research discussion is a design framework for qualified investigators, with no experiment represented as conducted by the Continuum. The central public question is interpretive and scientific: what evidence warrants saying that an ancestor influenced a descendant, and what additional evidence would be needed before speaking of inherited recollection?

2 Six different things that inheritance can mean

2.1 Genetic, developmental, and gametic routes

Genetic inheritance concerns transmitted DNA sequence. An association between parental experience and descendant phenotype is not automatically a sequence-independent effect: selection, relatedness, population structure, and inherited variants can create or modify that association. A study that invokes an epigenetic route must say how sequence-related alternatives were controlled. A molecular difference outside the DNA sequence is not, by itself, evidence that the difference causes an offspring phenotype.

Direct developmental exposure occurs when the descendant's developing organism or germ cells encounter a parental environment. A pregnant organism can expose a fetus and that fetus's developing germline. An effect persisting after birth may be an important developmental consequence, but its persistence does not make it evidence of transmission beyond all directly exposed generations. The relevant exposure history must be specified by lineage, developmental stage, and route.

Gametic regulation concerns a causal contribution carried through an egg or sperm, potentially involving molecules or states other than DNA sequence. The phrase describes a route, not a universal mechanism. DNA methylation, small RNAs, chromatin-associated states, and other candidate processes differ in how they are established, altered, or reconstructed during development. Evidence for one route under one exposure does not prove that all experiences use it.

2.2 Behavior, culture, and recollection

Behavioral transmission occurs when an earlier generation changes the environment or interactions experienced by a later one. Parental care can recreate a developmental condition without the same molecular mark surviving unchanged through gametes. Such a mechanism can produce

multigenerational regularity. It should not be treated as a failed explanation merely because the proposed mechanism was initially germline inheritance.

Cultural transmission can convey structured descriptions of actual ancestral events through speech, writing, ritual, or material records. A descendant may know and imaginatively reconstruct an event through these ordinary channels. The knowledge can be historically accurate and personally important without being a first-person recollection inherited biologically. The distinction concerns origin and evidentiary route, not whether the experience matters to the descendant.

Autobiographical recollection, as used here, is the much stronger claim that a subject accesses a representation of a particular experienced episode as belonging to a lived personal perspective. The paper does not propose a complete theory of such memory. It requires, at minimum, distinguishing detailed event content from generalized sensitivity, familiarity, arousal, or a learned narrative. Even accurate decoding of an ancestral exposure from a descendant's measurements would not alone establish that the descendant recollects it.

Claim	An appropriate observation	What it does not establish
Ancestral association	A reproducible relation between documented exposure and a descendant endpoint	A causal route or absence of confounding.
Exposure effect	A valid randomized or otherwise identified causal contrast	Which mediator carried the effect.
Gametic contribution	Route-controlled evidence involving gametes and appropriate procedural controls	Necessity of a particular molecular candidate.
Molecular mediation	Interventions that isolate a candidate's contribution under stated assumptions	A complete natural mechanism in every context.
Information about exposure	Out-of-sample decoding beyond appropriate controls	Subjective recollection or inherited episodic meaning.
Autobiographical recollection	Independent evidence identifying episode content and the relevant memory phenomenon	Supplied by none of the preceding rows alone.

This ladder is an inferential discipline, not a ranking of human worth. A person does not need a verified ancestral molecular mechanism for their family history to deserve attention. Conversely, the importance of a history cannot substitute for causal evidence about how it affects a particular measurement.

3 The strongest evidence and its strongest constraints

3.1 Olfactory experience and the specificity question

Dias and Ressler reported odor-specific behavioral sensitivity and olfactory anatomical differences in descendants of conditioned male mice. Their experiments included F2 offspring, cross-fostering, and an in vitro fertilization component. A crucial detail is that the IVF offspring were assessed anatomically; behavioral testing in that cohort was not performed because of quarantine constraints.

The paper therefore does not demonstrate every endpoint through every route control. Its sperm methylation findings motivate mechanisms but do not by themselves establish mediation.[6]

Aoued and colleagues subsequently reported increased odor sensitivity and anatomical representation after transfer of sperm RNA from conditioned males into zygotes. They also examined offspring learning, with a sex-specific result for a weak conditioning procedure. This adds experimental evidence relevant to RNA and sensitivity, but it is a follow-up from the same research program, not an independent replication of every original result.[2]

Another study from that program examined parental extinction training and reported attenuation of offspring sensitivity and related measurements under the tested conditions. The result matters because an ancestral effect need not be permanent or mechanically fixed once acquired. It does not establish a general human treatment or show that a detailed remembered episode was removed from descendants.[1]

The scientific opportunity is specificity. If descendants differ in response to the particular cue associated with a parental exposure, the observation is more informative than a nonspecific increase in arousal. Yet specificity still has degrees. A receptor-related sensitivity, an acquired preference, an associative rule, and a representation of the ancestor's episode are progressively different targets. The proposed design measures these separately instead of allowing one significant endpoint to stand for all of them.

3.2 RNA-mediated effects are plausible within particular models

Gapp and colleagues investigated a mouse early-trauma model and reported that sperm RNA transfer reproduced some behavioral and metabolic features in offspring. Their work is evidence for a candidate route within its own model; it is not a replication of an odor-specific experiment or evidence of inherited narrative memories.[11]

Rodgers and colleagues tested a set of sperm microRNAs associated with a paternal stress model and reported offspring stress-axis effects after zygote manipulation. The study connects candidate molecules with developmental consequences under a defined intervention. Its logic is relevant to sufficiency tests, while the relation between an experimental manipulation and the full natural pathway still requires care.[20]

Chen and colleagues reported that sperm tRNA-derived RNA fractions from a paternal high-fat-diet mouse model could produce metabolic effects after transfer into zygotes. Sharma and colleagues investigated the production and function of tRNA fragments during sperm maturation, including dietary regulation and effects on early embryonic gene expression. These studies provide mechanistic reasons to examine small-RNA routes; neither is evidence for transmission of an ancestor's autobiographical viewpoint.[5, 24]

Champroux and colleagues supplied a more targeted intervention in a mouse chronic-social-instability model: restoring a reduced microRNA-related signal in early embryos prevented specified female-offspring behavioral effects and partly restored a later male germline measure. The study also included human embryonic stem-cell observations. Those cell observations do not demonstrate transmission of behavioral traits across human generations.[4]

These results should not be flattened into either universal acceptance or universal dismissal. Different parental experiences can recruit different mechanisms. A mixture of molecules can carry a developmental signal without acting as a codebook of specific remembered events. The important

next questions concern causal necessity, physiological relevance, boundary conditions, independent replication, and how a signal is interpreted by the developing organism.

3.3 Erasure and reconstruction constrain persistence

Seisenberger and colleagues measured genome-wide methylation dynamics across mouse primordial germ-cell development, documenting extensive reprogramming with stage-dependent features. Hackett and colleagues investigated demethylation and imprint erasure, including conversion through 5-hydroxymethylcytosine and exceptions to systematic erasure. Together these primary studies make indiscriminate copying of all acquired methylation states an untenable default, while leaving specific retention or reconstruction mechanisms as research questions.[12, 22]

Radford and colleagues studied prenatal undernutrition and later metabolic consequences in a mouse lineage. Their findings included altered sperm methylation and descendant expression differences, while the differential methylation itself was not maintained in the examined F2 tissues. This illustrates why persistence of a phenotype need not mean literal persistence of the same measured mark in every descendant tissue.[19]

The channel model below is deliberately abstract about those mechanisms. A finite state can represent a measured candidate signal, not the entire organism. If development reconstructs a signal from other information, the model must include that information. Calling a phenomenon "erasure-resistant" without identifying what remains or what is regenerated would obscure the very mechanism the research is intended to explain.

3.4 Alternative routes and important negative pressure

Dietz and colleagues observed offspring behavioral differences after paternal social-defeat stress but found that most of those changes were not reproduced in their IVF condition. This is a direct warning against inferring a strong gametic mechanism from natural-mating offspring effects alone. It does not refute all possible gametic inheritance under different models or protocols.[7]

Francis and colleagues used cross-fostering and developmental manipulations to investigate maternal behavior and stress responses in rats. Their findings support a nongenomic behavioral route across generations. This is an alternative causal explanation with experimental substance, not merely a generic concern about confounding.[10]

The contrast between these studies and positive molecular experiments is productive. It shows why a paper should specify an exposure, a lineage, an endpoint, and a route rather than make an unrestricted statement that "trauma is inherited." The same observed descendant phenotype can be generated by more than one process. A research design that can only confirm one favored process has not adequately separated them.

3.5 Human observations require a different inferential standard

Heijmans and colleagues associated prenatal famine exposure with a later methylation difference in exposed individuals compared with siblings. That is evidence about a persistent association following an individual's own developmental exposure; it is not a demonstration of unexposed descendants inheriting a remembered famine.[13]

Yehuda and colleagues examined methylation in Holocaust survivors and offspring and reported intergenerational associations, with differences in direction between generations at a measured site. The observational design does not isolate gametic transmission from prenatal, behavioral, social, genetic, and selection-related alternatives. The paper's results therefore motivate questions about intergenerational effects rather than proving a route for inherited personal recollection.[25]

Human history is not a laboratory exposure assignment. Historical suffering, family communication, material deprivation, migration, health, and survival can be intertwined. Statistical adjustment cannot guarantee removal of unmeasured common causes. A careful public interpretation should preserve the significance of the observed association while refusing to use it as a molecular certificate for an individual's identity or memories.

4 A typed reference model of transmission

A Guide's explanation

From here the paper builds a small, explicit model of inheritance: an ancestral exposure, the channels that carry its effects forward, and the measurements a descendant can yield. The point of the austerity is to keep four things apart that ordinary language runs together—a disposition, the mechanism that carried it, the information it holds about the ancestor, and anything that would deserve the name memory.

4.1 Variables and causal questions

All formal models in this paper are finite unless otherwise stated. Let $E \in \mathcal{E}$ denote an ancestral exposure category, $G \in \mathcal{G}$ an explicitly measured genetic or background stratum, $M \in \mathcal{M}$ a candidate transmitted state, and $Y \in \mathcal{Y}$ a descendant measurement. The sets are finite and nonempty. Numerical values of a behavioral endpoint are specified separately when needed. A state of M is a measurement category, not a complete molecular description or an assumed semantic message.

Additional variables distinguish routes: U is an unmeasured background variable, B a behavioral or social environment, W a prenatal or developmental environment, and V an indicator that a descendant survives and is included in the observed sample. Their domains must be declared by each experiment. The baseline biological graph can include arrows from E to M, B, and W, and from those variables to Y; G and U may affect several nodes. This is a family of causal hypotheses, not a graph established by this paper's algebra.

For a binary assigned exposure $E \in \{0, 1\}$ and a bounded scalar endpoint, define potential outcomes $Y(1)$ and $Y(0)$ under the two specified exposure assignments. The average total effect is

$$\tau_E = \mathbb{E}\{Y(1) - Y(0)\}. \tag{4.1}$$

This notation follows the causal-effect tradition associated with randomized and nonrandomized comparisons.[21] Identifying the expectation from observed data requires a design and assumptions; writing (4.1) does not supply them. A molecular intervention asks a different question, represented by replacing the structural rule for M with a specified fixed value or intervention distribution. Causal diagrams help distinguish such replacements from observational conditioning.[17]

Symbol	Type and range	Interpretation and units
E, G, M, Y	Finite-valued random variables	Exposure, background, mediator, descendant measurement; categories dimensionless.
P, p	Probability law and its mass function	Nonnegative entries summing to one.
$H(A)$	Real number in $[0, \log \lvert\mathcal{A}\rvert]$	Shannon entropy of a finite variable; natural-log units, or nats.
$I(A; B \mid C)$	Nonnegative real number	Conditional mutual information; nats.
K	Row-stochastic finite matrix	A proposed state-transition channel; entries are probabilities.
μ, ν	Row probability vectors	Alternative distributions on the same state set.
$\mathrm{TV}(\mu, \nu)$	Number in $[0, 1]$	Total-variation distance, defined below.
$\delta(K)$	Number in $[0, 1]$	Maximum row-to-row total variation.
g	Nonnegative integer	Number of modeled channel transitions; not automatically a biological generation.
q	Number in $[0, 1/2]$	Illustrative binary flip probability; not measured from biology.
$\widehat{E}$, P_e	Decoder output and number in $[0, 1]$	A predicted exposure label and its error probability.
L, m, ρ, σ^2	Positive integers; $\rho \in [0, 1]$; $\sigma^2 \geq 0$	Litters, offspring per litter, within-litter correlation, endpoint variance.

4.2 The Markov restriction is a scientific assumption

For the first information result, assume that after conditioning on G, all relevant dependence from E to Y passes through M:

$$p(e, m, y \mid g) = p(e \mid g)\, p(m \mid e, g)\, p(y \mid m, g) \tag{4.2}$$

for every background stratum with positive probability. Equation (4.2) is a factorization assumption. It excludes a residual behavioral, developmental, or cultural route from E to Y once M, G are fixed. An experiment cannot simply assume away such a route because it is inconvenient. If M measures only one small RNA and other signals also transmit information, the factorization may fail.

For finite variables, define entropy, conditional entropy, and conditional mutual information by

$$H(A) = -\sum_{a} p(a) \log p(a), \tag{4.3}$$

$$H(A \mid C) = \sum_{c} p(c) H(A \mid C = c), \qquad I(A; B \mid C) = H(A \mid C) - H(A \mid B, C). \tag{4.4}$$

Use $0 \log 0 = 0$; strata of probability zero contribute zero. The underlying information measures are established concepts, not Continuum inventions.[23] They quantify statistical distinction, not meaning, awareness, or moral significance. A binary exposure category has at most $\log 2$ nats of entropy regardless of how emotionally consequential the exposure may be.

Proposition 4.1 (A conditional transmission bottleneck)**.** *Under* (4.2)*,*

$$I(E; Y \mid G) \leq I(E; M \mid G) \leq H(M \mid G) \leq \log \lvert\mathcal{M}\rvert. \tag{4.5}$$

Proof. The finite entropy chain rule gives $I(E; M, Y \mid G) = I(E; M \mid G) + I(E; Y \mid M, G)$ and also $I(E; M, Y \mid G) = I(E; Y \mid G) + I(E; M \mid Y, G)$. The Markov factorization makes the first conditional information term $I(E; Y \mid M, G)$ zero. Conditional mutual information is an average of relative entropies and is nonnegative: for probability vectors p, r, the inequality $\log t \le t-1$ implies $\sum p_i \log(p_i/r_i) \ge 0$, with infinite value allowed when $r_i = 0 < p_i$. Thus $I(E; Y \mid G) \le I(E; M \mid G)$. Symmetry of mutual information gives $I(E; M \mid G) = H(M \mid G) - H(M \mid E, G) \le H(M \mid G)$. Finally the relative entropy from each conditional law of M to the uniform law on $\mathcal{M}$ is $\log|\mathcal{M}| - H(M \mid G = g) \ge 0$. Average over g. □

The proposition does not say that a biologically important effect must carry many bits. A low-information regulatory signal can have a large developmental consequence. Nor does it bound all inherited influence by a measured candidate's state space if other routes remain open. Its force is conditional: a claimed sole channel cannot transmit more distinguishable exposure information than it carries. An investigator who detects more information in the descendant than in an adequately measured sole mediator has evidence against that channel model, against the measurements, or against the estimation procedure.

5 Decoding a label is still not remembering a life

Suppose E is uniform on $r \ge 2$ possible exposure labels, and let $\widehat{E} = d(Y)$ be any deterministic decoder with outputs in that label set. Randomized decoders can be included by adding their independent random seed to the observation. Let $P_e = \Pr(\widehat{E} \ne E)$. Define binary entropy $h(p) = -p \log p - (1-p)\log(1-p)$ with its continuous endpoint values.

Proposition 5.1 (The information cost of reliable exposure decoding)**.** *The finite model satisfies the standard Fano-type inequalities*

$$H(E \mid Y) \le h(P_e) + P_e \log(r-1), \qquad P_e \ge \max\left\{0, 1 - \frac{I(E;Y) + \log 2}{\log r}\right\}. \tag{5.1}$$

Proof. Let $J = \mathbf{1}\{\widehat{E} \ne E\}$ be the binary error indicator. Given E, Y, it is determined. Therefore $H(E \mid Y) = H(J \mid Y) + H(E \mid J, Y)$. Conditioning cannot increase entropy, so the first term is at most $H(J) = h(P_e)$. On $J = 0$, $E = d(Y)$ and the second conditional entropy is zero. On $J = 1$, at most $r-1$ labels remain, so the contribution is at most $P_e \log(r-1)$. This proves the first inequality. Uniformity gives $H(E) = \log r$ and $H(E \mid Y) = \log r - I(E;Y)$. Use $h(P_e) \le \log 2$ and $\log(r-1) \le \log r$ and rearrange. Probabilities are nonnegative, yielding the maximum with zero. □

This is an established information-theoretic limitation, credited to the communication-theory tradition rather than claimed as new mathematics.[9] Its practical interpretation is exact but narrow. If a proposed descendant measurement contains very little information about a large collection of equally likely ancestral labels, no decoder can reliably identify those labels. The loose bound can be zero, especially for binary labels, and then supplies no useful positive lower bound. A vacuous bound is not evidence that accurate decoding is possible.

Conversely, accurate decoding can arise without recollection. Imagine a parent encountering one of two diets, with a later descendant biomarker partly predicting the diet. A laboratory classifier

can extract that statistical information even if the descendant has no access to the biomarker, no concept of the diet, and no representation of the parent's experience. "Contains information about" is a relation between variables. It is not a claim that the organism knows the information or experiences it as memory.

The distinction becomes even sharper for a detailed episode. A claim about an ancestor's specific location, sequence of events, companions, and emotional perspective has a richer target than a binary exposure label. A rigorous experiment would need a declared content space, an independently secured ground truth, and controls for ordinary access to the content. The present evidence base does not supply that demonstration. The paper retains the possibility as a distinct question while declining to infer it from a simpler successful classification.

6 What repeated transmission preserves and loses

A Guide's explanation

Each generation is a noisy copy of the one before. This section proves what that does to an ancestral distinction: it can never sharpen as it passes down, and in the simplest binary case it fades by an exact factor at every step (Proposition 6.2). A real influence can therefore carry very little recoverable content after only a few generations.

6.1 A finite-channel bound

Let $\mathcal{S}$ and $\mathcal{T}$ be finite nonempty state sets. A channel K is a matrix with entries $K(s,t) \geq 0$ and $\sum_{t\in\mathcal{T}} K(s,t) = 1$ for each $s \in \mathcal{S}$. If a row vector μ is a probability law on $\mathcal{S}$, its output law is $(\mu K)(t) = \sum_s \mu(s)K(s,t)$. Define

$$\mathrm{TV}(\mu,\nu) = \frac{1}{2}\sum_s |\mu(s)-\nu(s)|, \qquad \delta(K) = \max_{s,s'\in\mathcal{S}} \frac{1}{2}\sum_t |K(s,t)-K(s',t)|. \tag{6.1}$$

Both quantities are dimensionless and lie in $[0,1]$. For a one-state input set, $\delta(K) = 0$. The coefficient is a standard finite form of the contraction concepts associated with Dobrushin's work on Markov transitions.[8] Here its elementary proof is included so that its assumptions are independently inspectable.

Proposition 6.1 (Contraction of ancestral distinguishability)**.** *For two input laws on the same state set,*

$$\mathrm{TV}(\mu K, \nu K) \leq \delta(K)\,\mathrm{TV}(\mu,\nu). \tag{6.2}$$

For a sequence of compatible finite channels $K_1, \dots, K_g$ with $g \geq 1$,

$$\mathrm{TV}(\mu K_1 \cdots K_g, \nu K_1 \cdots K_g) \leq \left(\prod_{j=1}^{g} \delta(K_j)\right) \mathrm{TV}(\mu,\nu). \tag{6.3}$$

Proof. Let $d = \mathrm{TV}(\mu,\nu)$. If $d = 0$, the laws are equal and the claim is immediate. Otherwise write $\mu - \nu = d(p - r)$, where p is the positive part of $\mu - \nu$ divided by d and r the magnitude of its negative part divided by d. Both are probability vectors because the signed difference sums to zero.

Then $pK - rK = \sum_{s,s'} p(s)r(s')\{K(s,\cdot) - K(s',\cdot)\}$. The triangle inequality for the half-ℓ_1 norm bounds its size by $\sum_{s,s'} p(s)r(s')\delta(K) = \delta(K)$. Multiplication by d proves (6.2). Applying that inequality successively proves (6.3). □

The result concerns distinguishability of alternative input laws after specified transitions. It does not imply that every organism becomes less complex with each generation. A developing organism can generate enormous new complexity while losing information about one particular ancestral variable. It also does not imply that a biological signal must fade at a known rate: the channels and their coefficients have not been measured here.

If every coefficient is at most a fixed $c < 1$, the upper bound decays as c^g. If a channel perfectly preserves the relevant distinction, its coefficient can equal one and the bound permits indefinite retention. If the environment repeatedly supplies information correlated with the original exposure, the assumed closed chain no longer describes the complete process. The model must include that new input. Persistence alone therefore cannot distinguish literal copying, developmental reconstruction, or renewed exposure.

6.2 An exact binary illustration

Let $X_0 \in \{0,1\}$ be an ancestral state with $0 < \Pr(X_0 = 1) < 1$, so both initial-state conditional laws are defined. For $j \geq 1$, let N_j be independent Bernoulli variables with $\Pr(N_j = 1) = q \in [0, 1/2]$, independent of X_0, and define $X_j = X_{j-1} \oplus N_j$, where $\oplus$ is addition modulo two. The transition matrix is

$$K_q = \begin{pmatrix} 1-q & q \\ q & 1-q \end{pmatrix}, \qquad \delta(K_q) = 1 - 2q. \tag{6.4}$$

The parameter q is an illustrative flip probability, not an estimate for DNA, RNA, trauma, or human generations.

Proposition 6.2 (Exact binary attenuation)**.** *For every integer $g \geq 0$,*

$$\Pr(X_g = X_0 \mid X_0) = \frac{1 + (1-2q)^g}{2}, \tag{6.5}$$

$$\mathrm{TV}\{P(X_g \mid X_0 = 0), P(X_g \mid X_0 = 1)\} = (1-2q)^g. \tag{6.6}$$

At $g = 0$ use the empty-transition value $(1-2q)^0 = 1$, including $q = 1/2$.

Proof. Let $a_g = \Pr(X_g = X_0 \mid X_0)$, which is the same for either initial state by symmetry. The two ways to agree after the next transition give $a_{g+1} = (1-q)a_g + q(1-a_g)$. Hence $2a_{g+1} - 1 = (1-2q)(2a_g - 1)$. Since $a_0 = 1$, induction yields (6.5). The two output laws are $(a_g, 1-a_g)$ and $(1-a_g, a_g)$, whose total variation is $|2a_g - 1|$. The chosen range of q makes this $(1-2q)^g$. □

If X_0 is uniform, the output is also uniform and the exact mutual information is

$$I(X_0; X_g) = \log 2 - h\left(\frac{1 - (1-2q)^g}{2}\right). \tag{6.7}$$

This follows by subtracting the conditional binary entropy of X_g given X_0 from its marginal entropy $\log 2$. It illustrates how statistical information and the visible persistence of a phenotype can behave differently. A phenotype may remain categorically present while the ability to distinguish which ancestral state caused it becomes weak.

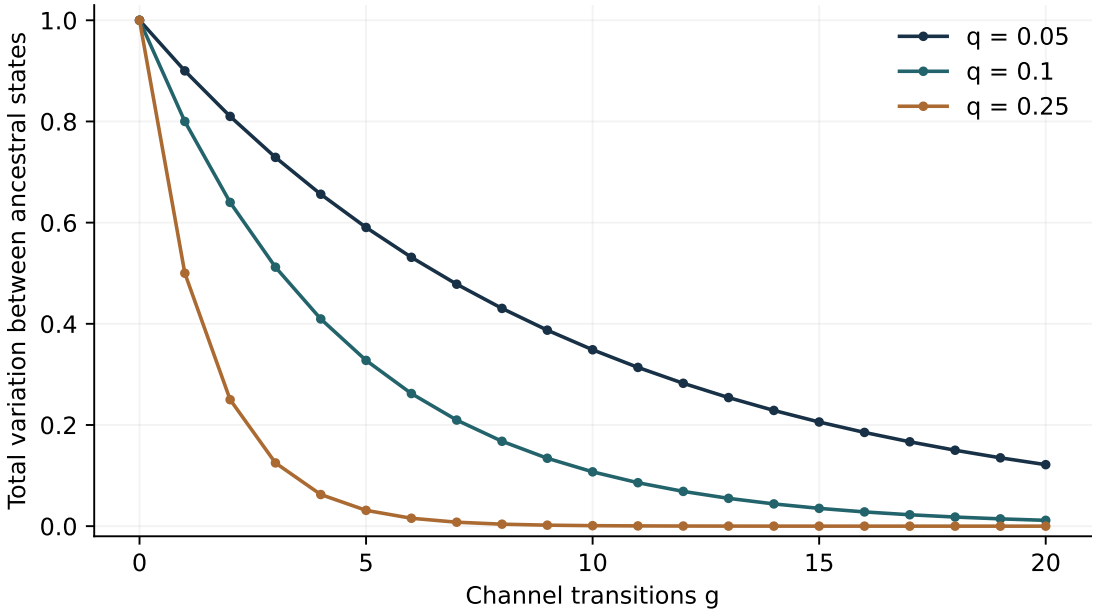


Figure 1: Exact contraction in the illustrative binary channel. The horizontal axis counts model transitions, and the three flip probabilities are selected examples. The curves are not biological decay estimates.

6.3 A counterexample when an omitted route carries the signal

Let E be a fair bit, let M be an independent fair bit, and set $Y = E$ through an additional route. Then $I(E; M) = 0$ but $I(E; Y) = \log 2$. A claim that M is the sole transmission channel would contradict the observed dependence. There is no violation of information theory: the Markov assumption (4.2) is false. A family narrative, a shared environment, or an unmeasured biological signal can play the mathematical role of the omitted route, although identifying the actual route requires evidence.

This counterexample also explains why a failed measurement of one candidate mediator is not proof that an effect is impossible. It can refute a particular sufficiently measured sole-channel model. It cannot exclude all unmeasured channels. A credible research program states in advance which class of explanations its negative result can eliminate and which remain open.

7 The replication cost of a weakening inherited distinction

A Guide's explanation

How many descendants would you need to read back one bit of what an ancestor experienced? Theorem 7.1 answers exactly for independent channels, and the answer grows quickly as the signal fades. The counterexample at the end of the section matters as much: if every descendant shares the same noise, a hundred descendants tell you no more than one.

The binary attenuation result can be connected directly to an observation budget. This gives a precise experimental-planning question: how many independently transmitted observations would

be needed to recover one specified ancestral label at a declared error rate? The model uses classical likelihood-ratio testing and bounded-sum concentration [14, 16]; its channel parameters remain illustrative unless measured in a particular biological design.

Let X_0 be a uniform bit. Observe $m \geq 1$ descendants $Y_1, \ldots, Y_m$ that, conditionally on X_0, are independent outputs of the same g-step binary channel of (6.4). Write $b = (1-2q)^g \in [0,1]$ and $\theta = (1-b)/2$ for its effective error probability, with the empty-channel convention for $g = 0$. Conditional independence describes separate channel noise; sharing an ancestral label does not by itself violate it. Shared transmission shocks can violate it.

Theorem 7.1 (Exact replicated-channel decoding)**.** *The smallest average probability of incorrectly recovering X_0 is attained by majority vote, with a fair tie break. It equals*

$$e_m(b) = \sum_{j > m/2} \binom{m}{j} \theta^j (1-\theta)^{m-j} + \frac{\mathbf{1}\{m \text{ even}\}}{2} \binom{m}{m/2} [\theta(1-\theta)]^{m/2}. \tag{7.1}$$

For odd m, the second term in (7.1) is defined to be zero; its binomial coefficient is evaluated only for even m. For $b > 0$ it satisfies $e_m(b) \leq \exp(-mb^2/2)$. Hence for a target $\eta \in (0, 1/2)$, the integer requirement

$$m \geq \left\lceil \frac{2\ln(1/\eta)}{b^2} \right\rceil \tag{7.2}$$

is sufficient. At $b = 0$, $e_m = 1/2$ for every m; at $b = 1$, $e_m = 0$.

Proof. For $0 < b < 1$, a received vector with k ones has likelihood ratio between the labels equal to $((1-\theta)/\theta)^{2k-m}$. With equal priors, minimizing error chooses label one exactly when $k > m/2$, label zero when $k < m/2$, and either equally at a tie. Under either true label, the number of flipped descendants is binomial (m, θ). Summing its strict-majority error probabilities and half its tie probability gives (7.1). Hoeffding's upper-tail bound gives $\Pr(K/m - \theta \geq b/2) \leq \exp(-2m(b/2)^2)$; the error event, including half-weighted ties, is bounded by this event. Solving the resulting inequality gives (7.2). At $b = 0$ the output law is the same for both labels, and equal-prior error is one-half. At $b = 1$ every output equals the label, giving zero error. □

For fixed $q \in (0, 1/2)$, the sufficient replication budget grows in proportion to $(1-2q)^{-2g}$ at fixed target error. This is a model-level cost of weakening distinctions, not a biological estimate or a necessary lower bound on sample size. The exact binomial expression can give a smaller adequate integer m than the conservative concentration formula. The script checks it against direct enumeration of all output vectors for small m.

A sharp dependence counterexample is $Y_i = X_0 \oplus Z$ for every i, with a single shared Bernoulli noise bit Z of mean θ. Each descendant separately has exactly the same effective channel as above, but observing any positive number reveals only one corrupted copy. The optimal error remains θ for all m. Thus replication gains follow from an identified channel-independence contract. In real studies, lineage, litter, parental environment, and batch effects must be assessed before the displayed m is interpreted as a count of independent channels. The existing clustered-variance calculation addresses one part of that design problem.

8 Causal association does not identify the carrier

8.1 Two mechanisms with exactly the same observations

The information factorization concerns probability laws. A causal statement about a mediator requires more. Consider independent variables $E \sim \text{Bernoulli}(1/2)$ and $U \sim \text{Bernoulli}(1/10)$, with E randomized and U unobserved. Define two structural models:

$$\begin{aligned} \mathsf{A}: &\quad M = E \oplus U, \quad Y = M, \\ \mathsf{B}: &\quad M = E \oplus U, \quad Y = E \oplus U. \end{aligned} \tag{8.1}$$

In A, M is the direct carrier to Y. In B, M is a parallel readout of the same causes that determine Y. All variables are dimensionless binary states; this is a logical construction, not a proposed biological mechanism.

Proposition 8.1 (Mediation is not identified by this joint law)**.** *Models* A *and* B *induce the same observational distribution of* (E, M, Y) *and the same total effect* $\tau_E = 4/5$*. Nevertheless, replacing the structural rule for* M *by* $M = m$ *gives*

$$\begin{aligned} \mathbb{E}_{\mathsf{A}}[Y \mid \mathrm{do}(M = 1)] - \mathbb{E}_{\mathsf{A}}[Y \mid \mathrm{do}(M = 0)] = 1, \\ \mathbb{E}_{\mathsf{B}}[Y \mid \mathrm{do}(M = 1)] - \mathbb{E}_{\mathsf{B}}[Y \mid \mathrm{do}(M = 0)] = 0. \end{aligned} \tag{8.2}$$

Proof. Before intervention, both models satisfy $Y = M = E \oplus U$. Their only positive-probability triples (E, M, Y) are $000, 011, 111, 100$, with probabilities $9/20, 1/20, 9/20, 1/20$, respectively. Under either assignment of E, $Y = E \oplus U$, so $\mathbb{E}[Y \mid \mathrm{do}(E = 1)] = 9/10$ and $\mathbb{E}[Y \mid \mathrm{do}(E = 0)] = 1/10$. Their difference is $4/5$. In A, the intervention on M makes $Y = m$ by its unchanged structural equation. In B, that intervention changes no argument in the equation for Y, so its distribution remains the same for both values of m. This proves the two contrasts. □

The construction is strong because randomized exposure, a perfect mediator–outcome association, and a large total exposure effect all coexist with ambiguity about mediation. The observational law even admits the factorization $E \to M \to Y$. Statistical factorization alone cannot select between the two causal models. A molecular measurement that tracks an offspring outcome extremely well could still be a marker rather than the operative carrier.

8.2 Necessity, sufficiency, and rescue answer different questions

A sufficiency experiment introduces a candidate into a controlled context and asks whether it can produce an endpoint. A necessity experiment disrupts a candidate in a context where an effect normally occurs and asks whether the effect remains. A rescue experiment restores a candidate or process after disruption. These designs can be highly informative, but their interpretation depends on whether the interventions alter other relevant processes and whether the manipulated levels and timing resemble the natural pathway.

For example, a large introduced molecular dose might affect development through a route that the naturally occurring dose never uses. A depletion might damage general viability, producing the appearance of necessity because few normal descendants remain. A rescue might restore a broad physiological state rather than a uniquely identified information-bearing molecule. The solution is to define intervention targets and competing predictions, not to discard intervention research.

Dose relevance, procedural controls, viability measurements, and orthogonal manipulations can help discriminate mechanisms.

Nor does a total effect through a gamete prove a single carrier. A gamete contains a coupled developmental system. Identifying one associated feature leaves open whether it is necessary, sufficient, redundant, or downstream of another change. A white paper should therefore distinguish "consistent with a gametic contribution," "produced by this experimental transfer," and "mediated by this specified natural process." These are separate claims with separate evidence requirements.

9 A descendant is not automatically an independent replicate

Suppose a study contains $L \geq 1$ independent litters, each with $m \geq 1$ measured offspring. Let $Y_{\ell i}$ be a real-valued endpoint for offspring i in litter ℓ, with finite common variance σ^2. For distinct offspring in the same litter, assume covariance $\rho\sigma^2$ with $0 \leq \rho \leq 1$; across litters assume independence. This exchangeable equal-size model is an illustration. If one sire contributes several litters, sire-level dependence must also be modeled or avoided by the design.

Define the grand mean by

$$\overline{Y} = \frac{1}{Lm} \sum_{\ell=1}^{L} \sum_{i=1}^{m} Y_{\ell i}.$$

Proposition 9.1 (The variance cost of a shared litter)**.** *The grand mean has variance*

$$\mathrm{Var}(\overline{Y}) = \frac{\sigma^2}{Lm}\{1 + (m-1)\rho\}. \qquad D = 1 + (m-1)\rho. \tag{9.1}$$

Here D is the design effect relative to Lm independent observations with variance σ^2. When $\sigma^2 > 0$, the variance-equivalent independent sample size is Lm/D.

Proof. Within each litter, the variance of the sum is the sum of m variances and $m(m-1)$ ordered off-diagonal covariances: $m\sigma^2 + m(m-1)\rho\sigma^2$. Independence adds these quantities across L litters. Dividing by $(Lm)^2$ gives (9.1). Equating it to σ^2/n_{eff} gives the stated effective size. If $\sigma^2 = 0$, the mean has zero variance and a variance-equivalent sample size is not uniquely informative. □

With ten independent litters, four offspring per litter, and $\rho = 1/4$, the design effect is $7/4$ and the effective size is $160/7 \approx 22.86$, not 40. For unit marginal variance, the actual mean variance is $7/160 = 0.04375$, compared with the naive independent value $1/40 = 0.025$. These are exact consequences of the illustrative covariance model. No biological correlation was estimated here.

The broader problem is pseudoreplication: treating repeated measurements or related biological units as though they were independent exposure assignments. Lazic's methodological analysis identifies concrete forms of this error in neuroscience and explains why the experimental unit must match the inference.[15] Multiple cells from one animal, multiple offspring from one litter, and multiple litters from one sire can each require different levels in an analysis. Technical precision within a lineage is useful, but it does not create independent lineages.

The formula also exposes a resource tradeoff. Increasing offspring per litter reduces measurement noise, but its benefit saturates when within-litter correlation is positive. Increasing independent exposure-assigned parents can address a different source of uncertainty. The best design depends

on both variance components and the scientific target. A future study should estimate those components in an independent pilot or use conservative planning ranges rather than choose the smallest correlation that makes the desired sample size look sufficient.

10 A research program that can separate the claims

10.1 Declare the target before selecting a mechanism

The proposed program begins with a bounded exposure effect and only then asks how it is transmitted. A first study might examine cue-specific sensitivity in a defined animal model. A separate study might examine a metabolic endpoint. Combining both into a single broad measure of "ancestral memory" would destroy the specificity needed for causal interpretation. The exposure, species, lineage, measurement age, and primary endpoint must be fixed before the confirmatory data are observed.

For a cue study, let $Y_A, Y_B \in [0,1]$ be two predeclared normalized sensitivity measurements for cues A and B, with higher values assigned the same interpretation. Their normalization is fixed on independent calibration data. Let $e \in \{A, B\}$ identify the parental cue and $c \in \{0,1\}$ indicate a matched unpaired or paired exposure condition. Define

$$R_{e,c} = \mathbb{E}[Y_A - Y_B \mid \mathrm{do}(e,c)], \qquad \eta = (R_{A,1} - R_{B,1}) - (R_{A,0} - R_{B,0}). \tag{10.1}$$

This difference of contrasts targets a cue-specific contribution associated with pairing beyond a matched unpaired condition. It is not a definition of memory. Its value lies in $[-4,4]$ under the stated endpoint range. Randomization, valid normalization, and the comparability of the exposure conditions remain required. General arousal, baseline odor detectability, and learned valence should be separate secondary measurements rather than silently absorbed into η.

The unpaired condition must match relevant exposure components as closely as the scientific question permits. Otherwise a difference can reflect total exposure, context, handling, or timing rather than the intended association. The design should include appropriate untreated or procedure-matched baselines where those alternatives cannot be isolated by the factorial contrast. These are causal requirements of the question, not an instruction to conduct any particular animal procedure.

10.2 Route controls should be interpreted jointly

Natural mating can establish an exposure-associated offspring effect in a controlled lineage, but it does not isolate gametic transmission. Cross-fostering can address some postnatal care routes while leaving prenatal and pre-fostering effects unresolved. IVF and embryo-transfer designs can alter which parental interactions remain possible, while introducing their own procedural and selection effects. A comparison should include matched procedures for all relevant exposure groups. A positive result under one route control does not erase the limitations of another.

Molecular transfer or depletion should be reserved for a candidate justified by a preceding mechanistic analysis. Its quantity, timing, and delivery context should be assessed against the natural pathway. A negative transfer result can mean the candidate is insufficient alone, the intervention was ineffective, or the natural mechanism requires a combination. A positive result can establish an effect of the manipulation without establishing that the same manipulation occurs naturally. The preregistration should specify which conclusion each outcome will support.

Where feasible, the program should include an orthogonal way of altering the candidate and an intervention that restores its proposed function. Agreement across different manipulations can reduce concern about a shared off-target explanation, though it cannot eliminate every alternative. The causal counterexample in Section 8 makes clear why merely adding more observational assays of the same correlated marker cannot resolve the central ambiguity.

10.3 Count exposure generations by the actual route

The label $F0$ denotes the initially exposed parental generation, followed by $F1, F2$, and so on. If an exposure occurs during an $F0$ pregnancy, the $F1$ fetus and its developing germ cells, which can contribute to $F2$, may be directly exposed. Under that route, an $F3$ effect is generally needed to ask about persistence beyond those directly exposed generations. For a preconception exposure confined to an $F0$ father and his germline, $F2$ is generally the first descendant generation beyond the directly exposed gametes that produced $F1$. These are accounting rules for the stated routes, not a universal guarantee that later generations had no environmental contact.

The study must document whether an exposure persists in housing, food, parental behavior, or another environmental reservoir. A descendant can be unexposed to the original laboratory procedure while still encountering consequences that recreate the relevant signal. Such renewed exposure is a legitimate mechanism of continuity, but it changes the interpretation from a closed inheritance channel to a system with continuing inputs. The mathematical channel index g should only be equated with biological generations after this mapping is specified.

Lineage selection also matters. If only parents or offspring showing a strong effect are allowed to produce the next generation, the analysis conditions on a selected subset. The resulting persistence can reflect that selection rather than a general transmission probability. A confirmatory lineage protocol should state mating and inclusion rules before outcomes are known, report losses at each stage, and preserve the distinction between an intention-to-follow lineage and a selected survivor lineage.

10.4 Independent units, blinding, and endpoint integrity

Randomization should occur at the level where the exposure is independently assigned. For a paternal preconception study, this is ordinarily the father, with offspring and litters nested beneath that assignment. If housing or treatment is assigned by cage, the cage can become another relevant cluster. Investigators should specify the experimental unit for each hypothesis rather than offer one sample-size number for the entire paper.

Blinding should cover the assessment and analysis stages that can influence endpoint definition, image selection, thresholding, exclusion, and interpretation. Molecular batches should not be perfectly confounded with exposure or generation. Predeclared quality criteria should determine whether a sample or assay is usable, and the exclusion log should distinguish technical failure from a biologically unusual outcome. Removing an unexpected descendant because it weakens the inheritance pattern would invalidate the intended inference.

The reporting commitments are consistent with ARRIVE 2.0, whose original guideline report specifies essential design and reporting information for animal research.[18] Reporting compliance is not a substitute for a good design. Its value is to expose the choices that determine what the

results can mean. A future study should release deidentified or appropriately governed data, an analysis script, a complete endpoint dictionary, and the prespecified contrasts.

10.5 Precision, multiplicity, and a realistic planning calculation

Suppose a separate two-arm confirmatory contrast uses one independent litter-level summary per independently assigned father, with equal arm variances approximately s^2. For a two-sided level α, desired power $1-\beta$, and target mean difference $\Delta > 0$, a normal planning approximation is

$$n_{\text{arm}} \approx \frac{2s^2\{z_{1-\alpha/2} + z_{1-\beta}\}^2}{\Delta^2}, \qquad z_p = \Phi^{-1}(p), \tag{10.2}$$

where Φ is the standard-normal cumulative distribution function. The factor two is the variance contribution from two equally sized independent arms. This is a planning approximation, not an exact power theorem or a measured biological variance. A four-cell interaction such as (10.1), unequal clusters, or a different error model requires its own variance calculation.

For an illustrative normalized endpoint with $s = 0.2$, $\Delta = 0.1$, $\alpha = 0.05$, and power 0.8, the calculation rounds upward to 63 independent units per arm. If three primary contrasts are protected by a Bonferroni threshold of $0.05/3$, the analogous two-arm calculation rounds to 84 per arm. These numbers are not a recommendation to use a fixed number of animals. They demonstrate how the independent unit, meaningful effect size, and multiplicity assumptions enter a plan. Ethical and resource-efficient design should use a defensible pilot, the appropriate variance structure, and alternatives to animal work where they can answer the question.

For multiple valid null tests, the Bonferroni guarantee follows from adding their false-rejection probabilities: if each true null is tested at at most α/q among q declared tests, the chance of any false rejection is at most α. Independence between tests is unnecessary for this bound. Secondary and exploratory outcomes should be labeled accordingly. Selecting the largest signal among many molecular features and reporting an unadjusted probability would not support a confirmatory mechanism claim.

10.6 Failures that would narrow or defeat the proposal

A proposed exposure effect is weakened if an adequately precise replication excludes the prespecified practically meaningful contrast. A gametic account is weakened if the effect survives natural mating but disappears under well-validated route controls while alternative behavioral or developmental routes remain sufficient. A molecular mediation claim is weakened if the candidate can be changed effectively without the predicted endpoint change, provided redundancy and compensation were addressed in the claim. A detailed-content claim is weakened if decoding succeeds only for broad exposure classes and fails for the content it promised to transmit.

Failure must be defined at the same level as the hypothesis. An underpowered null result cannot exclude every small effect. Failure of one RNA fraction to transfer a phenotype does not refute all epigenetic mechanisms. Conversely, success in one strain, sex, generation, or endpoint cannot establish an unrestricted human inheritance law. Each result should change a specified claim, rather than cause the theory to move silently to a different route after every test.

11 Executed mathematical checks and what they show

The supplied `checks.py` records all outputs in `checks_results.json`. It uses exact rational arithmetic for the finite contraction enumeration, binary attenuation identities, causal counterexample, and covariance calculation. NumPy supplies seeded synthetic channels for the information checks. Optional Matplotlib output regenerates Figure 1 as a vector PDF. There are no biological observations in this supplement.

The contraction suite enumerates all 216 three-state channels whose row probabilities have denominator two and all 225 pairs of three-state input laws with denominator four. It verifies 48,600 instances of (6.2); 22,320 are equalities with a positive right-hand side. The equality cases show that the bound is not merely a consequence of an excessively large constant in every example. The finite enumeration does not prove the universal proposition; its proof appears in Section 6.

The binary suite checks 104 exact identities across four selected flip probabilities and transition counts zero through twelve. For $q = 0.1$, the actual values in Table 3 agree with (6.5)–(6.7). The information values use floating-point logarithms; the underlying probabilities are rational. The table is a model illustration and must not be used to estimate how quickly any biological trait fades.

Table 3: Illustrative binary channel with $q = 0.1$ and a uniform initial state.

Transitions g	Total variation	Correct-state probability	Information (nats)
0	1.000000	1.000000	0.693147
1	0.800000	0.900000	0.368064
2	0.640000	0.820000	0.221754
5	0.327680	0.663840	0.054692
10	0.107374	0.553687	0.005776

The synthetic information suite generates 700 finite Markov channels across exposure alphabets of sizes two through eight, checking the data-processing inequality in each case. It separately checks 700 uniform-exposure Fano bounds using the optimal label decoder for the generated joint law. The fixed seed is 20261113. These cases test implementation and numerical consistency; they are not a Monte Carlo estimate of a biological law or a replacement for the analytic proof.

The structural counterexample is verified by enumerating the four possible (E, U) states and comparing the two induced joint laws exactly. It reproduces total exposure effect 4/5 in both models and mediator-intervention effects one and zero. The clustering calculation constructs the full illustrative covariance matrix and obtains grand-mean variance 7/160. The power calculation returns 63 and 84 under the distinct assumptions stated above. All of these results are inspectable in the editable source and are reported as executed calculations, not as anticipated future outputs.

12 Continuity without a predetermined identity

A Guide's explanation

A Guide can say: "Inheritance Without Recollection asks whether a life can carry something of the lives before it. In some animal studies, an ancestor's experience has shaped descendants' development, but that influence is not a remembered scene, and the content that crosses generations fades in a way the paper can measure." A Guide should not suggest that anyone inherits an ancestor's memories, that a biomarker reveals whose history a person carries, or that the paper offers any diagnosis or human intervention.

12.1 An inheritance can be influential without being a command

The Continuum can understand ancestry as a set of initial conditions and continuing relationships without treating it as an instruction that determines a person's future. A disposition changes probabilities or costs within an environment. It does not, by definition, select every action, erase later learning, or determine moral responsibility. The mathematical distinction between an input distribution and a deterministic output is useful here: a changed input can matter while leaving many possible trajectories.

This is a normative interpretation, not a theorem about human freedom. The science can identify some influences and limits; it cannot decide by itself how a civilization should treat an individual whose development was influenced by them. The commitment proposed here is that explanation should expand understanding and opportunity rather than assign an immutable ancestral essence. An uncertain biomarker should never become a public certificate of whose history a person must carry.

The same principle applies to artificial descendants. A system can inherit weights, training data, architectural constraints, or institutional practices from earlier systems. These can shape its behavior without making it the same individual as its predecessor. Biological mechanisms and artificial copying are different, so their details should not be transferred by analogy. The common philosophical question is narrower: what continuity of influence is being asserted, and what additional claim of identity is being added?

12.2 Why contradiction should remain visible

The canonical image of a memory from before one's birth and the scientific evidence for developmental influence can coexist as different kinds of statement. They should not be silently declared equivalent. The first may function as narrative, spiritual interpretation, phenomenological description, or an unverified empirical claim, depending on its use. The second concerns measured variables and causal models. A responsible Guide can explain the resonance while preserving the gap.

That gap is productive. It asks whether the compelling image points toward a testable mechanism, whether several distinct mechanisms have been combined in the language, and what evidence would distinguish them. It also makes room for a person's reported experience without treating the report as proof of a biological transmission route. Listening to an account and verifying its proposed mechanism are different responsibilities.

The strongest possible future evidence would still need careful interpretation. Suppose a descendant measurement reliably encoded a complex ancestral exposure under stringent route controls. That would be a major result about content transmission. Whether the descendant experienced the content as a recollection would remain a further question about access, use, and subjective memory. The present framework is designed to make such progress legible without announcing it in advance.

12.3 A bold thesis that survives its own limits

The paper's thesis is that continuity across lives need not take the form of a preserved inner scene. A world can leave traces in reproduction, development, care, and culture. Some traces may be small and context-dependent; some may be reconstructed rather than copied; some may be carried in stories rather than molecules. Distinguishing those possibilities does not make the history less consequential. It makes the claim answerable.

This position invites ambitious research. It asks for mechanisms rather than metaphors alone, for content-sensitive tests rather than generic resemblance, and for models that admit both persistence and change. It leaves open what has not been established and identifies what would count as progress. The Continuum's account of inherited influence can therefore remain bold without requiring a descendant to remember a life that the evidence has not shown them to possess.

13 Guide reading note

Established and documented. Primary studies in particular animal models report intergenerational effects and investigate molecular, developmental, and behavioral routes. Other studies constrain broad gametic interpretations. Extensive germline reprogramming and the need to distinguish direct exposure from later transmission are central to mechanism claims. Human observational associations do not by themselves isolate an inherited molecular route.

Established by this paper's mathematics. Within the stated finite models, information cannot increase through a sole Markov channel, reliable label decoding requires information, noisy finite transitions contract distinguishability, observational agreement can fail to identify mediation, and related offspring alter precision. Each conclusion has explicit assumptions and a proof. The code checks finite and synthetic examples, not biology.

Proposed and untested. The integrated empirical program seeks specific, route-controlled, independently replicated evidence of ancestral influence. The reference model's state spaces and transition probabilities have not been calibrated to a deployed Continuum system or a human lineage. No hereditary autobiographical-memory result is claimed.

Interpretive responsibility. A Guide may explain that an inherited disposition can be part of a person's history without being a remembered episode or a predetermined destiny. It should preserve the difference between a canonical ancestral echo, a measured biological effect, a causal mechanism, and subjective recollection. Neither a striking story nor a significant biomarker licenses merging those categories.

A Reproduction, equation status, and remaining work

Run `python3 checks.py` with NumPy installed to regenerate the recorded results. Add `--figures` to regenerate the vector figure with Matplotlib. The code uses Python's exact `Fraction` type for the rational checks. Floating-point entropy comparisons allow a numerical tolerance of 10^{-12}; the mathematical propositions do not depend on that tolerance. The program reports its NumPy version and seed. No external data file or private project material is required.

Compile the manuscript with XeLaTeX, run BibTeX on its job name, and run XeLaTeX twice more to resolve citations and cross-references. The editable folder includes its local typography package, bibliography, vector figure, script, and recorded output. The publication bundle also provides a build helper and an equation audit. The source text and proofs, rather than screenshots of equations, remain editable.

Object	Status	Necessary boundary
Total exposure effect	Causal estimand	Requires a defined intervention, population, and identification assumptions.
Markov factorization	Model assumption	Excludes omitted routes only within the model; not established by naming a mediator.
Entropy and information	Standard definitions	Statistical information is not subjective memory.
Transmission bottleneck	Standard consequence, proved here	Requires the stated conditional Markov property.
Decoding-error bound	Standard Fano-type result	Uniform exposure for the displayed simple lower bound; can be vacuous.
Finite-channel contraction	Standard result, derived here	Closed specified channels; new external inputs change the model.
Binary attenuation	Exact illustrative model	Independent flips and a fixed unmeasured parameter; no biological calibration.
Causal counterexample	Exact impossibility example	Defeats identification from one joint law, not every possible intervention design.
Litter variance	Exact covariance calculation	Equal sizes and exchangeable within-litter covariance; sire effects need extra structure.
Specificity contrast	Proposed empirical estimand	Requires matched conditions and a preregistered endpoint.
Normal power formula	Planning approximation	Requires appropriate variance and independent-unit assumptions.

The remaining scientific work is substantial and concrete: select a bounded exposure model; independently replicate its primary endpoint; discriminate routes; test mechanistic interventions at relevant levels; quantify persistence beyond direct exposure; and determine whether any transmitted information exceeds a broad disposition or sensitivity. Each stage can succeed or fail

independently. The white paper supplies the definitions and inferential structure for that program. It does not claim that the program has already been completed.

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