Neurally Driven Evolution: Behavior, Niche Construction, and the Acceleration of Variation Structure

A Sequel to: Perturbation, Integration, Possibility Space

A note on the level of argument. This paper argues at the intersection of philosophy of biology and evolutionary theory. It is not a biological paper in the strict sense, and it does not claim to present a fully formalized theory. Its aim is to identify a causal architecture that existing biological frameworks do not adequately describe, to embed known empirical findings within that architecture, and to derive from it precisely formulated hypotheses that are open to empirical investigation. Throughout the paper, a distinction is maintained between empirically established findings, mechanistically plausible pathways supported by partial evidence, and open hypotheses that constitute the paper's central empirical desiderata. Readers seeking formal quantitative models or exhaustive empirical review will find this paper a theoretical precursor to such work, not a substitute for it.

Abstract. The nervous system invents no new evolutionary mechanism. It accelerates and complexifies an existing one. The first paper in this series showed that autocatalytic systems generate, through their history of perturbations, a structured variation space whose topology makes certain variations more probable than others. The present paper extends this thesis to the level of neural systems. The nervous system is not a fourth inheritance system in Jablonka's sense, but a possibility-space generator operating on a dramatically shortened timescale, which acts on the perturbation structure of offspring through niche construction, without leaving the causal architecture of the basic mechanism. Three empirically grounded channels connect neural processes to heritable variation structure: activity-dependent gene expression inscribed into the somatic genome of neurons; neuroendocrine axes transmitting chronic neural integration states to the germline; and niche construction as the primary indirect channel through which behavior shapes the perturbation environment of successive generations. The Weismann barrier remains intact throughout. The category error committed by Jablonka's behavioral inheritance system is avoided by maintaining strict causal-level distinctions.

1. The Starting Point

The first paper in this series ended with an implicit question: what happens to the described mechanism when organisms develop nervous systems? Behavior appears as a new level. The timescale of perturbation integration shortens dramatically. And the question arises whether this represents an extension of the mechanism or a categorially new process.

The answer is: it is an extension, but one that must be precisely demarcated from what Jablonka described as behavioral inheritance. The difference is not gradual but categorical.

Before the nervous system, perturbation integration operates at the level of biochemical networks: metabolic autocatalysis, epigenetic restructuring, germline modification. The timescale is generational. The integration of perturbations leaves traces that slowly accumulate in variation structure across generations.

With the nervous system, a qualitatively new level emerges. Integration speed explodes. What previously required generations now occurs within a lifetime, within hours, within milliseconds. And a

decisive additional capacity appears: the system can internally model perturbations, that is, anticipate them before they arrive. This is not a departure from material substrate but a new organizational form of the same principle, implemented through synaptic weighting, network dynamics, and activity-dependent gene expression in neurons.

2. What the Nervous System Inscribes and Where

A precise answer to this question is essential for avoiding the category error that will be addressed in the next section.

Neural experience is inscribed primarily into the genome of nerve cells themselves, into the somatic cells of the brain. This is empirically well established. Neuronal activity alters chromatin structure, histone modifications, methylation patterns, and the nuclear position of genes within neurons (Lee et al., 2017). The immediate early genes Arc, c-Fos, FosB, and Egr1 mediate epigenetic and transcriptional changes that regulate synaptic plasticity and memory consolidation (Guzowski, 2002; Bhatt et al., 2020). BDNF expression is regulated by neuronal activity through histone acetylation and chromatin remodeling in a temporally and spatially specific manner (Tian et al., 2009). The transcription factor DeltaFosB accumulates in the hippocampus through learning, regulates synaptic morphology, and is required for memory formation (Bhatt et al., 2015).

All of this occurs in somatic cells. Neurons are not germ cells. What is inscribed into the neuronal genome remains there, within the individual, within the lifetime.

The Weismann barrier, the strict separation between soma and germline in vertebrates, blocks direct transmission of what neurons learn to the next generation. This is not a limitation to be circumvented but a categorical constraint that defines the structure of the problem. Any theory of neurally driven evolution must work within this constraint, not around it.

3. The Category Error in Jablonka and Why We Avoid It

Jablonka and Lamb treated behavioral inheritance as a fourth inheritance system on the same causal level as genetic and epigenetic inheritance. This is the category error: behavior is not transmitted from generation to generation through a direct causal mechanism that transfers structures independently of the interpretive capacity of the receiving system.

Genetic inheritance through DNA replication, and epigenetic inheritance through methylation patterns, histone modifications, and small non-coding RNAs, are causal mechanisms in the strict physical-chemical sense: they transfer structures directly, without requiring an interpreter. Behavioral transmission operates differently: it presupposes organisms already capable of interpretation, learning, or imitation, and it operates at the level of meaning and intentionality, not at the level of physical-chemical causality.

Our claim is categorially precise: behavioral patterns do not constitute a fourth inheritance channel. They constitute a mechanism of niche construction through which organisms modify the perturbation environment of their offspring. This perturbation environment then acts on the offspring's autocatalytic systems through the mechanisms described in the first paper, epigenetic, metabolic, and genomic, which are genuine causal inheritance mechanisms.

The causal chain is complete and materially grounded at every step. But it runs through the environment, not directly from parent to child. That distinction is categorical, not merely descriptive.

4. Niche Construction as the Primary Channel

Niche construction is the strongest and most precisely describable channel through which neural evolution acts on heritable variation structure. Organisms actively modify aspects of their own environments and, in doing so, alter the selection pressures and perturbation structures to which their offspring are subject (Odling-Smee, Laland & Feldman, 2003). This is empirically established as a general phenomenon in evolutionary biology.

Crucially, this modified selection and modified perturbation structure persists as long as the constructed niche persists, often far longer than the lifetime of the organisms that produced it (Laland, Matthews & Feldman, 2016). A beaver dam alters the perturbation environment of beaver offspring for generations after the dam-building individuals have died. Bird song learned and transmitted culturally alters the acoustic perturbation environment of offspring, shaping their neural developmental conditions. Tool use in primates alters the nutritional and mechanical perturbation environment, changing the metabolic selection pressure on subsequent generations.

In all these cases, the causal pathway is the same: neural behavior generates environmental structures that function as perturbations on the autocatalytic systems of offspring, shaping their variation space topology through the mechanisms described in the first paper. The neural system does not bypass the basic mechanism. It feeds into it through the environment.

A recent experimental study provides direct evidence for the evolutionary significance of this pathway: experimental removal of niche construction substantially altered the pace and mechanisms of resistance evolution in a host-parasite system, demonstrating that niche construction effects on selection are real, measurable, and distinct from direct genetic effects (Huttener et al., 2025, biorXiv).

The evolutionary significance of niche construction is that it allows neural complexity to influence variation structure across generations without violating the Weismann barrier and without requiring a new inheritance mechanism. The more complex the neural system, the more elaborate and stable the niche it constructs, and the more systematically it shapes the perturbation environment of offspring.

5. Indirect Material Channels: Neuroendocrine Axes and Circulating RNAs

Beyond niche construction, two indirect material channels connect neural processes to the germline. Both maintain categorical clarity: they involve material carriers and do not breach the Weismann barrier. They differ substantially in their current empirical status, which is explicitly marked below.

5.1 The Neuroendocrine Channel

The brain regulates the entire body, including the gonads, through the hypothalamic-pituitary-adrenal (HPA) axis and related neuroendocrine systems. The following represents empirically established findings. Chronic neural perturbation integration, specifically sustained stress, demonstrably alters germline epigenetic structure through this axis.

Rodgers et al. (2015) showed in a rigorous mouse model that chronic paternal stress alters the microRNA content of sperm and reprograms offspring HPA stress axis regulation. They demonstrated this causally by injecting nine specific sperm microRNAs into naive zygotes, which recapitulated the offspring phenotype with remarkable fidelity. This is direct causal evidence of the neuroendocrine channel: a neural integration state (chronic stress) produces a molecular germline signature (altered

sperm miRNAs) that reshapes offspring neurodevelopment. Gapp et al. (2021) showed that long-term paternal psychological stress induces inter- and transgenerational inheritance of reproductive, behavioral, and metabolic alterations, mediated through differential DNA methylation regions in sperm that partially evade embryonic reprogramming. Franklin et al. (2010) showed that maternal separation stress alters methylation at specific gene promoters in F1 sperm, with changes transmissible to F2 offspring cortex.

The following represents a mechanistically plausible pathway supported by the above findings but not yet fully mapped in mechanistic detail. The connecting pathway runs through glucocorticoid signaling: chronic neural stress activates sustained glucocorticoid release, which reaches gonadal tissue and alters the epigenetic programming of developing germ cells. The precise intermediate steps between glucocorticoid receptor activation in gonadal tissue and the specific small RNA changes observed in sperm remain an active area of investigation.

5.2 Circulating Small RNAs

The following represents a mechanistically plausible but empirically less mature pathway. Neuronal activity influences the production of exosomes and small non-coding RNAs released into the bloodstream. These can in principle reach germ cells. What is established is that sperm contain a rich repertoire of small RNAs that are sensitive to paternal environmental conditions and function post-fertilization to alter offspring development (Rodgers et al., 2015). The question of how much of this repertoire is shaped by neural activity specifically, as opposed to other physiological processes, remains open as an empirical question and constitutes one of the desiderata of the present framework.

6. The Causal Collapse as Evolutionary Threshold

The concept of causal collapse, developed in detail elsewhere (Stegemann, 2025), refers to the state arising when a system's causal relationships become non-factorizable: the point at which the internal causal structure of the system can no longer be decomposed into independent contributions from its parts, and the system as a whole becomes the irreducible locus of causation. In the context of neural evolution, this threshold marks the transition from stimulus-driven behavior to behavior generated by an integrated internal history. The nervous system ceases to be a relay and becomes a causal core in its own right.

With increasing neural complexity, this threshold appears at a point in the continuous process of increasing integration complexity at which the nervous system begins to actively shape niche construction rather than passively undergo it. Below the threshold, niche construction is simple and largely stimulus-driven. Above it, niche construction becomes anticipatory, cumulative, and culturally transmissible.

In birds, this threshold is empirically visible. Migratory behavior, song learning, and tool use in corvids are behaviors that cannot be explained by immediate stimuli. They presuppose an internal causal structure sufficiently complex to generate behavior that is largely independent of short-term perturbations. The internal attractor structure of the nervous system, built through a lifetime of perturbation integration, becomes the proximate cause of behavior.

The evolutionary consequence is that once neural complexity crosses this threshold, the speed at which variation structure can be influenced across generations increases, because the niche constructions produced by high-complexity neural systems are more stable, more elaborate, and more systematically organized than those of simpler systems.

7. Neural Evolution Without New Inheritance: What the Theory Claims

The present theory makes a precise claim that distinguishes it from the Extended Evolutionary Synthesis on one side and from naive neuro-Lamarckism on the other.

It does not claim that the nervous system constitutes a new inheritance system. It claims that the nervous system is a possibility-space generator operating on a new timescale, which feeds into the basic mechanism through three materially grounded channels: activity-dependent somatic genome modification in neurons (retained within the individual), neuroendocrine germline transmission (reaching the next generation through the HPA axis and related systems), and niche construction (shaping offspring perturbation environments without direct genome contact).

The Weismann barrier is not violated at any point. The category error of treating behavior as an inheritance system is explicitly avoided. And the basic mechanism described in the first paper, perturbation integration reshaping variation space topology, is not replaced but extended: the nervous system provides a new primary site of perturbation integration, operating faster, handling more complexity, and connecting to germline variation structure through indirect but causally traceable channels.

The result is that neural evolution and genomic evolution are not two separate processes but one process operating at multiple timescales and through multiple integration sites. What is new with the nervous system is not the logic but the speed and the organizational level at which integration occurs.

8. Empirical Questions and Theoretically Grounded Answers

Three questions were formulated at the close of the initial version of this paper. On the basis of the theoretical framework developed here, each admits of a theoretically grounded answer that specifies the expected empirical signature and the experimental design most likely to detect it. These answers are theoretical predictions, not established findings.

First question: Which specific neural activity patterns leave which epigenetic traces in germline cells through neuroendocrine channels, and in which genomic regions?

The theoretical answer follows from the logic of the HPA axis mechanism. It is tonic, sustained shifts in neural integration states, specifically chronic amygdala hyperactivation, persistently altered cortical oscillations under deprivation, and chronically elevated glucocorticoid signaling, rather than singular phasic activation events, that reach the germline. The mechanism is hormonal: chronic neural states produce sustained systemic release of glucocorticoids and CRH, which dock at receptors in gonadal tissue and alter the expression of enzymes responsible for RNA biogenesis and DNA methylation in developing germ cells.

The genomic regions affected will be found primarily in regulatory promoter and enhancer regions of genes governing nervous system development, metabolic homeostasis, and endocrine regulation in offspring, precisely the loci that control the plasticity and sensitivity of the successor system. The system does not transmit concrete experiential content to offspring. It calibrates the sensitivity of the offspring's postnatal possibility space for similar perturbations. This is a structural, not an informational, transmission: the next generation inherits a tuned responsiveness, not a record.

Second question: Can it be shown that populations with more complex neural niche construction histories display different variance structure in the flexible genomic region than populations without this history?

The signature is theoretically predictable. A population that has maintained a stable, complex neural niche across generations, through elaborate tool use, transmitted migration routes, or stable social structures, progressively stabilizes and canalizes the perturbation environment it inhabits. This dampens unpredictable environmental variation and thereby reduces phenotypic and genomic variance in the regions originally responsible for adapting to the now-buffered challenges. Canalization in the sense of Waddington operates on the previously open regions.

Simultaneously, variance structure increases in precisely those genomic grammar regions that govern neural plasticity, learning, and the anatomical prerequisites for niche exploitation. The G-matrix signature is therefore not a general increase in variance, but a directional redistribution: canalization in previously open regions, and elevated flexibility in the regions that support niche construction capacity itself.

Third question: Is there a measurable correlation between the complexity of neural perturbation integration and the speed of genomic grammar change in the flexible genomic region?

The correlation is predicted to be positive and non-linear. A highly developed nervous system generates a cascade of rapidly changing behavioral niches through anticipatory and cumulative niche construction. Because these niches function as the primary generators of intergenerational perturbation background, the flexible genomic region is placed under permanent, structured innovation pressure. The molecular clock for genomic grammar change, including rates of gene duplication, reorganization of regulatory networks, and transposon activation in neural progenitor cells, runs significantly faster in lineages with high neural complexity, such as corvids, cetaceans, and primates, than in neurally simpler lineages inhabiting static or physically determined niches.

Each of these three predictions generates a concrete experimental design: multi-generational epigenomic profiling of germline cells in populations under controlled chronic stress regimes; G-matrix measurement across populations with different niche construction histories; and comparative molecular clock analysis of flexible genomic regions across lineages stratified by neural complexity. All are technically feasible with current methods. None has been systematically executed with the framework presented here as the guiding hypothesis.

9. Relation to the First Paper: Continuity and Extension

The relationship between this paper and its predecessor is explicit and structural. The first paper established that autocatalytic systems generate structured variation spaces through perturbation history, that this structuring operates through epigenetic mutational bias, transposon mobilization, and attractor dynamics, and that the resulting variation space topology is partially heritable through germline epigenetic mechanisms. It identified the G-matrix as the population-level signature and fluctuation-response theorems as the formal framework for causal continuity across levels.

The present paper takes this framework and asks: what changes when perturbation integration acquires a dedicated high-speed system, the nervous system, as its primary site? The answer is: the timescale shortens, the complexity of integrable perturbation patterns increases dramatically, and two new channels connect the neural integration site to heritable variation structure. But the logic remains the same. The topology of the variation space is shaped by perturbation history. The nervous system accelerates and elaborates the production of perturbation history, both within the individual

through lifetime learning, and across generations through niche construction and neuroendocrine transmission.

The theory is therefore not a collection of separate claims about separate mechanisms. It is a single claim about a single mechanism, instantiated at multiple levels and timescales.

References

Bhatt, D.L. et al. (2015). Experience-dependent induction of hippocampal DeltaFosB controls learning. Journal of Neuroscience 35(40).

Dias, B.G., Ressler, K.J. (2014). Parental olfactory experience influences behavior and neural structure in subsequent generations. Nature Neuroscience 17.

Franklin, T.B. et al. (2010). Epigenetic transmission of the impact of early stress across generations. Biological Psychiatry 68.

Gapp, K. et al. (2021). Sperm epigenetic alterations contribute to inter- and transgenerational effects of paternal exposure to long-term psychological stress via evading offspring embryonic reprogramming. Cell Discovery.

Guzowski, J.F. (2002). Insights into immediate-early gene function in hippocampal memory consolidation. Hippocampus 12.

Huttener, R. et al. (2025). Experimental removal of niche construction alters the pace and mechanisms of resistance evolution. biorXiv.

Laland, K.N., Matthews, B., Feldman, M.W. (2016). An introduction to niche construction theory. Evolutionary Ecology 30.

Lee, P.R. et al. (2017). Activity-dependent gene expression in neurons. The Neuroscientist.

Odling-Smee, F.J., Laland, K.N., Feldman, M.W. (2003). Niche Construction: The Neglected Process in Evolution. Princeton University Press.

Rodgers, A.B. et al. (2015). Transgenerational epigenetic programming via sperm microRNA recapitulates effects of paternal stress. PNAS 112(44).

Stegemann, W. (2025). The mechanism of phenomenal experience. Zenodo. https://doi.org/10.5281/zenodo.17698505

Stegemann, W. (2026). Perturbation, Integration, Possibility Space: Why Highly Adaptive Phenomena Occur and Why Neo-Darwinism Cannot Explain Them. Zenodo. https://doi.org/10.5281/zenodo.19536994

Tian, F. et al. (2009). Role of BDNF epigenetics in activity-dependent neuronal plasticity. Neuropharmacology.

Weismann, A. (1893). The Germ-Plasm: A Theory of Heredity. Charles Scribner's Sons.

Whitney, O. et al. (2014). Core and region-enriched networks of behaviorally regulated genes and the singing genome. Science 346.