

Hall's Neural Imprint Syndrome (HNIS)

A Synthesis of Emerging Evidence for Cellular and Bioelectrical Mechanisms

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Abstract. This paper presents a synthesis of emerging scientific evidence relevant to Hall's Neural Imprint Syndrome (HNIS), also known in external and academic contexts as Harmonic Neurodivergent Imprinting Syndrome. HNIS is an independently developed theoretical framework proposing that neurodivergent nervous systems encode traumatic and formative experience through a distinct mechanism: mirror neuron imprinting operating at a cellular and bioelectrical level. Drawing on five lines of recent scientific inquiry (predictive coding and trauma, Kotler, Mannino, Fox & Friston, 2026; bioelectrical cellular memory, Levin, 2021, and Zhang & Levin, 2025; the biological "third state", Noble & Pozhitkov, 2024; theta-burst stimulation in autistic children, *The BMJ*, 2026; and mirror neuron research in autism, Andreou, Skrimpa & Peristeri, 2025), this paper argues that HNIS occupies a distinct and currently unfilled position: it proposes a candidate cellular and bioelectrical mechanism for the rigidity that recent predictive coding models describe but leave open, grounded in a decade of independent theoretical development informed by lived neurodivergent experience. HNIS is offered as a hypothesis for testing, not as an established clinical entity.

1. Introduction: The Mechanism Gap

Two parallel problems have shaped trauma and neurodivergence research for the past decade. The first is mechanistic: despite significant advances in our understanding of how trauma affects the brain's predictive architecture, the field lacks a convincing explanation for why traumatic prediction patterns become rigid in the first place, and why that rigidity can be so resistant to intervention in neurodivergent people. The second is conceptual: the dominant popular framework for somatic trauma, advanced by Bessel van der Kolk in *The Body Keeps the Score* (2014), has provided a clinically generative but biologically imprecise metaphor that recent science is now actively challenging.

HNIS was developed independently over the preceding decade to address precisely this gap. It proposes that the missing mechanism is neural imprinting: a process by which formative and traumatic encounters are encoded not merely as psychological memory or altered pre-

diction weights, but as bioelectrical patterns at the cellular level, operating beneath and feeding the brain’s anticipatory architecture. This paper presents a formal synthesis of the scientific evidence that bears on this proposition.

2. Predictive Coding and the Open Question

In a 2026 opinion article in *Frontiers in Systems Neuroscience*, Kotler, Mannino, Fox and Friston propose a fundamental reframing of trauma: rather than being stored in somatic tissue as van der Kolk’s metaphor suggests, trauma represents a disorder of rigid threat prediction within the brain’s anticipatory architecture. Drawing on Friston’s predictive coding framework, among the most influential theoretical models in contemporary neuroscience, the authors argue that traumatic experience causes the brain to over-weight threat signals, reducing metastability: the brain’s capacity to shift fluidly between functional states.

This reframing is significant. The authors are also careful about its limits: they present the link between flow states, restored metastability and recovery as a prediction of their framework rather than an established fact, noting that direct empirical confirmation with established metastability measures has not yet been achieved. In an interview about the paper, Mannino put it plainly: “We do not yet have definitive evidence that flow heals trauma by increasing metastability, altering prediction error, or reorganising attractor dynamics” (PsyPost, 2026).

The model also leaves open what, at a biological level, makes threat predictions so rigid in the first place. HNIS proposes a candidate answer to that question. If predictive coding describes what the brain does with traumatic experience, HNIS proposes how that pattern becomes fixed: through mirror neuron imprinting operating beneath the prediction layer, in the bioelectrical substrate of the cells themselves.

3. Bioelectricity as Cellular Memory: The Levin Framework

The most significant recent scientific development for the HNIS framework comes from the work of Michael Levin and colleagues at Tufts University. Building on Levin’s (2021) work in *Cell* and extended in Zhang and Levin (2025), the Levin Lab has established bioelectricity as an ancient, intrinsic, and fundamental property of all living cells, not limited to neural or neuromuscular systems as previously assumed.

Of particular relevance to HNIS is Levin’s account of bioelectric control as a physiological layer operating above genetics, providing extra plasticity to genetically encoded hardware. Levin further describes how ion channels and gap junctions, by propagating signals conditionally on the physiological state set by their own prior activity, implement a form of historicity: a memory of physiological state carried across fields of cells.

This concept of historicity is close biological language for what HNIS has proposed phenomenologically: that the body carries a history of its encounters not only as psychological memory but as structural bioelectrical patterning. On this reading, the imprint would be a stable bioelectric state that, once established, persists and shapes subsequent cellular behaviour in ways not readily accessible to top-down cognitive or linguistic intervention. This extension to neural imprinting is the HNIS proposal, not a finding of the Levin Lab.

Levin's work also shows the regenerative potential of bioelectrical reprogramming: in animal models, brain defects induced by chemical teratogens or genetic mutation have been rescued by reinforcing appropriate bioelectrical signalling. This suggests that an imprint, while persistent, may in principle be modifiable through bioelectrical rather than purely psychological means.

4. The Third State: Cellular Reorganisation Beyond Binary Categories

A complementary line of evidence emerges from research into what Noble and Pozhitkov (2024) have described as a “third state”, a condition beyond the traditional binary of life and death in which cells from deceased organisms, placed in new environments, spontaneously reorganise into novel multicellular structures with unexpected functions.

In the most studied examples, skin cells from frog embryos have self-assembled into xenobots, capable of movement and self-repair, while human lung cells have formed anthrobots capable of moving through their environment and promoting repair of damaged neural tissue nearby. The proposed mechanism involves ion channels and pumps in cell membranes functioning as electrical circuits, generating bioelectrical signals that coordinate cellular behaviour.

For HNIS, the significance of this research is twofold. First, it supports the proposition that cells retain organisational capacity and bioelectrical signalling beyond their original context. Second, it supports the Orchestrated Dualism framework (Hall, 2026) by showing that binary categorical distinctions (life and death, predetermined and emergent, self and environment) often fail to capture what is actually occurring at the biological threshold. If cellular systems retain adaptive, reorganising capacity in such extreme conditions, the proposition that neurodivergent nervous systems encode experience at a cellular and bioelectrical level below conscious cognition becomes more plausible, though it remains to be tested directly.

5. Mirror Neurons in Autism: Different Responsiveness, Not Absence

HNIS is grounded in mirror neuron science. Mirror neurons, first identified by Rizzolatti and colleagues in macaques and subsequently studied as a distributed system in humans, fire both during action execution and action observation, providing a neural substrate for imitation, empathy, social learning, and the transmission of experience between individuals.

In neurodivergent nervous systems, HNIS proposes that the mirror neuron system operates with greater sensitivity and fidelity: encoding relational and traumatic experience with more intensity, across a broader bandwidth, and with less automatic filtering. The critical question for the framework is whether empirical evidence supports a “different responsiveness” model over the more common deficit framing.

A review published in *Applied Sciences* in June 2025 (Andreou, Skrimpa & Peristeri) examines this evidence, including studies of mu rhythm suppression, the primary EEG proxy for mirror neuron system activation, in autism. Its findings are mixed: some studies report reduced mu suppression in autistic participants, while others find no group difference, and the authors conclude there is no consensus supporting a simple “broken mirror” account. That absence of a clear deficit is compatible with the HNIS position. The framing also has precedent: in 2007, Jaime Pineda of the University of California, San Diego, summarising his group’s EEG work, said that mirror neurons in autism appear to be abnormally responsive and abnormally integrated into wider social-cognitive circuits rather than absent (*ScienceDaily*, 2007).

This framing is foundational for HNIS’s therapeutic positioning. If mirror neurons in neurodivergent systems are differently and in some respects more responsive rather than less functional, then these systems may be more susceptible to imprinting: encoding experience more deeply, more broadly, and at lower thresholds than neurotypical systems. If so, intervention should address the bioelectrical substrate of that encoding rather than only the prediction layer above it.

6. Bioelectrical Intervention: Theta-Burst Stimulation Evidence

If HNIS is correct that formative and traumatic imprinting occurs at a bioelectrical level beneath the prediction layer, then bioelectrical rather than purely cognitive intervention should be able to produce change. Recent clinical evidence is consistent with this.

A randomised, sham-controlled trial published in *The BMJ* in 2026 enrolled 200 autistic children aged four to ten across three hospitals in China; about half also had an intellectual disability. Children received accelerated continuous theta-burst stimulation, a non-invasive technique using patterned magnetic pulses, targeting the left primary motor cortex, in ten sessions a day for five consecutive days. Compared with sham stimulation, the active group showed greater improvement in social communication and in language, with small to mod-

erate effect sizes, and the improvement was still present at the one-month follow-up, which was the only follow-up. Side effects such as restlessness and scalp discomfort were more common with active stimulation, but were mild to moderate and resolved without treatment.

The relevance of this finding to HNIS is mechanistic rather than direct. The trial was not designed to test HNIS. But theta-burst stimulation is not a cognitive intervention: it involves no language, narrative, reframing, or conscious processing. It introduces new bioelectrical signals directly into neural tissue, below the level of prediction and language, where HNIS proposes the imprint resides. That a brief bioelectrical intervention produced measurable change in social communication, including in children with intellectual disability, is consistent with what the HNIS framework would predict. Longer follow-up is needed before the durability of such effects can be judged.

In Levin's terms, the stimulation may be introducing new bioelectrical patterning into a system whose historicity has been shaped by prior experience: not erasing an imprint but creating new pathways alongside it, altering the system's dynamics from the substrate up.

7. HNIS in the Scientific Landscape: A Triangulation

The five lines of evidence reviewed above bear on HNIS from distinct but complementary directions. It is worth making the triangulation explicit.

Predictive coding (Kotler et al., 2026) frames trauma as a disorder of rigid prediction, while leaving open the biological basis of that rigidity. HNIS supplies a candidate mechanism: mirror neuron imprinting at the bioelectrical level that fixes prediction patterns from below.

Bioelectrical cellular memory (Levin, 2021; Zhang & Levin, 2025) establishes that living cells carry bioelectrical historicity, a memory of physiological state implemented through ion channels and gap junctions. HNIS proposes that this historicity, in neurodivergent nervous systems, is the substrate in which formative and traumatic imprinting persists.

Third-state cellular reorganisation (Noble & Pozhitkov, 2024) shows that cells retain organisational capacity and adaptive signalling beyond conventional biological categories. HNIS extends this to propose that neural imprinting similarly persists as cellular architecture rather than only as psychological record.

Mirror neuron research in autism (Andreou et al., 2025; Pineda, 2007) does not support a simple deficit model, and has long been read by some researchers as pointing to atypical rather than absent responsiveness. This is compatible with HNIS's proposition that these systems encode experience differently, and in some respects at lower thresholds.

Theta-burst stimulation (*The BMJ*, 2026) shows that brief, direct bioelectrical intervention can produce measurable short-term change in autistic children at a level below cognitive processing, consistent with the HNIS prediction that the imprint resides in the bioelectrical substrate rather than in the prediction layer alone.

None of these lines of evidence, taken alone, demonstrates HNIS. Taken together, they form a convergent body of support for a framework that was developed independently, from a lived and theoretical starting point, before much of this literature had emerged. That convergence is the paper's primary finding.

8. Implications for Intervention: U-Space and KITHen

The synthesis presented here has implications for two programs under development within Project Humanity: U-Space, a mutual rescue model pairing displaced neurodivergent people with displaced companion animals as co-participants in recovery, and KITHen, a virtual connection program pairing neurodivergent individuals with shelter animals through live-streamed footage and digital engagement.

If HNIS is correct that imprinting occurs through the mirror neuron system at a bioelectrical level, then the therapeutic value of human-animal connection may operate through this mechanism. Companion animals do not offer language, narrative, or cognitive reframing. They offer sustained, non-threatening relational presence: an ongoing source of gentle mirror neuron activation that may introduce new patterning into an imprinted system over time.

The virtual engagement model of KITHen preserves this therapeutic essence while avoiding the practical constraints of physical animal interaction. Observing animals' movement, behaviour, and presence engages the mirror neuron system through observation alone, consistent with established findings on mu rhythm changes during action observation. In neurodivergent systems whose mirror neurons are differently responsive rather than absent, this activation may be particularly significant.

This is not a claim that animal observation cures trauma. It is the more modest and testable proposition that sustained, voluntary, gentle mirror neuron activation through human-animal connection may introduce new resonance patterns alongside existing imprints, gradually altering the bioelectrical historicity of the system from below.

9. Limitations and Future Directions

This paper makes no claim that HNIS is a finalised clinical doctrine. It is a theoretical framework in active development, and the evidence synthesised here is convergent rather than directly confirmatory. Several limitations must be acknowledged.

First, the cellular and bioelectrical mechanisms proposed by HNIS have not yet been tested directly in the context of trauma or neurodivergence. The Levin Lab's work on bioelectrical historicity has been conducted primarily in developmental and regenerative contexts; the extension to neural imprinting in human neurodivergent systems remains theoretical, albeit grounded in the same cellular mechanisms.

Second, mirror neuron science remains contested. Mu rhythm suppression as a proxy for mirror neuron activity has methodological limitations, findings in autism are inconsistent, and the causal relationship between mirror neuron responsiveness and social-communication differences in autism is not established.

Third, the theta-burst evidence is short-term and was not collected to test HNIS; its relevance here is as consistency with the framework, not confirmation of it.

Fourth, the therapeutic implications for U-Space and KITHen are prospective rather than evidenced. The human-animal connection hypothesis, while consistent with the HNIS framework, requires empirical testing in the program contexts proposed.

Future research directions include: direct measurement of bioelectrical patterning in neurodivergent people with trauma histories; comparative studies of bioelectrical and cognitive intervention approaches; longitudinal assessment of mirror neuron activation during human-animal interaction in neurodivergent participants; and empirical testing of U-Space and KITHen outcomes against HNIS predictions.

10. Conclusion

The central argument of this paper is simple. Recent science has converged, from several independent directions, on a picture of biological memory and encoding that is consistent with what HNIS proposed on theoretical and phenomenological grounds. Cells carry a history of their encounters in bioelectrical patterns. Mirror neuron function in autism appears to be different rather than simply deficient. The biological basis of traumatic prediction rigidity remains an open question in current models. And brief, direct bioelectrical intervention has produced measurable change in autistic children.

HNIS sits at the intersection of these findings. It is not the last word on any of them. It is a framework, independently developed, prior-art documented, and now finding convergent scientific support, that proposes a cellular and bioelectrical mechanism the field is beginning to approach but has not yet named.

The body does not keep the score. The cells carry the tuning. And in neurodivergent systems, that tuning may run very deep.

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Author note. HNIS is cited as Hall’s Neural Imprint Syndrome in formal and citation contexts, and as Harmonic Neurodivergent Imprinting Syndrome in external and academic contexts. Both refer to the same framework, originated by Ash Hall (Ashlar Hall; earlier work published as Maggie Hall). This synthesis was first written in May 2026. Version 1 of this preprint, October 2026, corrects citation details in the original working draft against the published sources.

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