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AUTHOR'S REPLY

WHEN A CRITIQUE BECOMES UNTENABLE: A SCHOLARLY RESPONSE TO GROSSMAN ET AL.'S EVALUATION OF POLYVAGAL THEORY

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Abstract

A recent critique advanced by Grossman et al. (2026, this issue) argues that Polyvagal Theory is scientifically untenable, asserting that its core claims regarding autonomic organization, respiratory sinus arrhythmia (RSA), and evolutionary framing are inconsistent with established neurophysiology. The present paper evaluates these assertions not by disputing individual claims in isolation, but by examining whether the critique engages Polyvagal Theory as it is articulated in the peer-reviewed literature and whether it meets the epistemic standards required for scientific refutation.

Rather than responding sequentially to individual objections, the analysis clarifies the theory's conceptual foundations, scope, and explicit conditions of falsifiability as a systems-level, pathway-specific framework of autonomic state regulation. It demonstrates that the critique repeatedly evaluates a reconstructed proxy of the theory shaped by persistent category errors, including conflation of neuroanatomy with neurophysiology, reduction of theory to measurement, and substitution of phylogenetic continuity for functional organization. These structural misrepresentations propagate across methodological, neurophysiological, evolutionary, and developmental domains, precluding meaningful empirical adjudication.

Across these domains, the paper shows that disagreements concerning RSA metrics, comparative anatomy, or evolutionary framing do not engage the theory's specified mechanisms or demonstrate conditions under which its predictions would fail. Where disagreement exists, it reflects differences in measurement preference, level of analysis, or theoretical framing rather than evidence against the theory's organizing principles. An appendix presents a historical audit showing that several central claims reiterated in the critique were identified in the literature nearly two decades earlier as mischaracterizations of Polyvagal Theory. Their continued repetition without substantive modification reflects a persistent failure of representational uptake rather than unresolved empirical controversy.

It is concluded that the charge of scientific untenability does not apply to Polyvagal Theory as formulated, but instead reflects a critique that fails to engage the theory on its own terms. Productive scientific discourse requires representational fidelity, appropriate alignment of levels of analysis, and responsiveness to theoretical and empirical clarification – criteria essential to theory evaluation but not met in the critique under review.

Key words: polyvagal theory, autonomic nervous system, vagal regulation, brainstem autonomic circuits, respiratory sinus arrhythmia, autonomic state regulation, social engagement system, developmental neurophysiology, evolutionary neurobiology, systems neuroscience, autonomic biomarkers

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Section 1. Introduction

1.1 Purpose and scope of the rebuttal

This article responds to the coordinated critique advanced by Grossman et al. (2026, this issue), which challenges the conceptual, neurophysiological, and evolutionary foundations of Polyvagal Theory. The rebuttal evaluates the critique's methodological fidelity, accuracy in construct representation, and responsiveness to clarification present in the peer-reviewed literature. While critical evaluation of theoretical frameworks is essential to scientific progress, such evaluation must accurately represent the claims under consideration. In the present case, the critique does not engage Polyvagal Theory as articulated in the primary literature, but instead relies on reconstructed claims that the theory does not make. This pattern reflects a straw-man

structure of critique in which a substituted version of the theory is evaluated in place of its actual propositions.

1.2 Historical context and recurring criticisms

Several of the central objections advanced by Grossman et al. (2026, this issue) – including claims regarding respiratory sinus arrhythmia (RSA), vagal pathway differentiation, and evolutionary framing – were raised in substantially similar form in earlier critiques and addressed directly in the peer-reviewed literature during the past two decades (Porges, 2007b, 2023, 2025a, 2025b). These objections, first articulated by Grossman and Taylor (2007) and explicitly corrected in the peer-reviewed literature (Porges, 2007b), are summarized in **table A1**. The present response therefore does not treat these issues as unresolved controversies but examines why critiques that were previously answered continue to be reiterated without engagement with those responses or with subsequent empirical and methodological developments.

1.3 Aims of the present rebuttal

Accordingly, this rebuttal serves two functions. First, it clarifies the core organizing principles and empirical commitments of Polyvagal Theory as a systems-level, pathway-specific framework of autonomic regulation, emphasizing distinctions that are routinely overlooked in critical accounts – such as the difference between anatomy and physiology; between descriptive phylogenetic sequencing and evolutionary mechanism; and between mammalian-specific neural integration and generic respiratory–cardiac coupling (Porges, 1995, 2007a, 2023, 2025a). Second, it evaluates whether the critique advanced by Grossman et al. (2026, this issue) engages claims that are foundational to, and explicitly asserted by, the theory.

Importantly, this rebuttal does not argue that Polyvagal Theory is beyond refinement, empirical testing, or revision. On the contrary, the theory was explicitly introduced as a set of testable, interdisciplinary hypotheses. Productive scientific dialogue, however, requires that critiques engage a theory on its own terms. When criticisms persistently rely on misstatements of those terms – even after formal correction – the resulting discourse risks obscuring, rather than advancing, understanding of autonomic regulation and its relevance to systems neuroscience, psychophysiology, and translational research.

To clarify the organization of this response, the sections that follow proceed in a deliberate sequence. Section 2 articulates the conceptual foundations, scope, and falsifiability of Polyvagal Theory as it is currently formulated. Sections 3 and 4 examine recurring structural and methodological misrepresentations in the critique, followed by analyses of neurophysiological organization, evolutionary framing, and developmental evidence in Sections 5 through 7. The final sections address translational relevance and the epistemic structure of the critique itself.

Section 2. Conceptual foundations, scope, and falsifiability of polyvagal theory

With evaluative standards established in Section 1, this section specifies the conceptual foundations of Polyvagal Theory as it is currently formulated. The focus is not on adjudicating critiques, but on articulating the theory's organizing principles, scope, and explicit conditions of falsifiability, thereby providing the theoretical frame within which subsequent methodological, neurophysiological, developmental, and translational analyses are evaluated (Porges, 1995, 2007a).

Polyvagal Theory emerged in 1994 as a neurophysiological framework linking autonomic regulation to behavioral organization, physiological homeostasis, and social engagement, with explicit focus on mammalian nervous system architecture (Porges, 1995). Since its initial formulation, the theory has generated a broad interdisciplinary literature and a growing set of testable hypotheses concerning autonomic state regulation, RSA, vagal efficiency, and the neural substrates of social behavior.

2.1 Polyvagal theory as a systems-level neurophysiological framework

At its core, Polyvagal Theory proposes that autonomic state functions as an intervening variable – a neurophysiological platform that shapes how organisms detect, evaluate, and respond to cues of safety, danger, and life threat (Porges, 2007b, 2023). The theory articulates organizing rules governing state regulation, explaining how brainstem-mediated autonomic control influences behavior, affect, cognition, physiological homeostasis, and social engagement. This systems-level organization is not a peripheral feature of the theory; it is foundational and operates at the level of functional integration and regulatory organization rather than isolated anatomical components or single reflex pathways.

Rather than treating autonomic activity as a linear reflexive response to environmental stimuli, Polyvagal Theory emphasizes hierarchical and state-dependent regulation. Changes in autonomic state alter the functional availability of neural circuits supporting social communication, defensive mobilization, or metabolic conservation. In this framework, autonomic regulation is not merely reactive but organizes the range of possible behavioral and physiological responses available to the organism at any given moment.

2.2 Autonomic state as an intervening neural platform

A central explanatory contribution of Polyvagal Theory is the conceptualization of autonomic state as an intervening neural platform that mediates the relationship between environmental cues and behavioral output. Autonomic state influences perceptual thresholds, emotional expression, motor behavior, and social engagement by modulating the functional integration of cortical, limbic, and brainstem circuits. In this framework, autonomic state organizes adaptive responses across a wide range of ecological and social contexts.

The theory further proposes that state transitions are governed by neural mechanisms operating largely outside conscious awareness, a process described as neuroception (Porges, 2003, 2004). Neuroception refers to the nervous system's capacity to detect cues of safety, danger, or life threat and to adjust autonomic state accordingly. This process provides a neurophysiological explanation for adaptive and maladaptive behaviors that occur independently of explicit cognitive appraisal, including – but not limited to – contexts of stress, trauma, and social engagement.

2.3 Functional organization of ventral and dorsal vagal pathways

Polyvagal Theory differentiates vagal efferent pathways according to their developmental specification, functional integration, and regulatory role (Porges, 1995, 2023, 2025a; Porges & Furman, 2011). In mammals, cardioinhibitory vagal regulation is mediated through at least two functionally distinct brainstem pathways: ventral vagal efferents originating in the nucleus ambiguus (NAmb) and dorsal vagal efferents originating in the dorsal motor nucleus of the vagus (DMNX)(Strain et al., 2024).

Ventral vagal pathways are predominantly myelinated, exhibit respiratory gating, and provide rapid, phasic modulation of cardiac output. These pathways are embedded within an integrated brainstem network coordinating cardiac regulation with respiration and related cranial motor functions (Porges, 1995; Richter & Spyer, 1990).

In contrast, dorsal vagal pathways exert slower, tonic influences on visceral organs and are not characterized by the same respiratory-gated, rapid cardiac modulation emphasized for ventral vagal regulation in Polyvagal Theory (Porges, 1995). These distinctions are not framed as mutually exclusive control mechanisms but as functionally differentiated components of a hierarchically organized, state-dependent regulatory system.

2.4 Evolutionary and developmental framing: what the theory does – and does not – claim

polyvagal Theory does not propose anatomical exclusivity, deny phylogenetic continuity, or invoke recapitulationist logic (Porges, 1995, 2007a, 2023, 2025a; Porges & Furman, 2011). Instead, it situates mammalian autonomic regulation within an evolutionary framework emphasizing functional repurposing, developmental specification, and increasing circuit-level integration.

The theory proposes that mammals exhibit a derived reorganization of autonomic control characterized by the integration of ventral vagal cardioinhibitory pathways with brainstem circuits regulating facial expression, vocalization, ingestion, head orientation, and auditory processing (Porges, 1998).

Developmental evidence is used within Polyvagal Theory to elucidate how these integrated circuits emerge and mature, not to infer direct evolutionary ancestry. Ontogenetic sequencing is interpreted mechanistically, reflecting the progressive functional integration and experiential calibration of conserved neural elements rather than a recapitulation of phylogeny (Porges & Furman, 2011; Porges et al., 2019).

2.5 Conditions of falsifiability and empirical accountability

Polyvagal Theory is explicitly formulated as a testable neurophysiological framework. Its core propositions generate falsifiable predictions regarding the organization of brainstem autonomic circuits, the functional role of autonomic state as an intervening variable, and the conditions under which social engagement, defensive mobilization, or metabolic conservation are expressed.

Among these core predictions, the theory would be empirically challenged if ventral vagal (NAmb-originating) activity were shown to increase during conditions of life threat or defensive immobilization rather than being withdrawn as predicted by hierarchical state regulation; if robust social engagement reliably occurred in the absence of ventral vagal regulation or during dominance of dorsal vagal or sympathetic circuits; if developmental maturation of NAmb myelination failed to correspond with the emergence of RSA, vagal efficiency, and social regulatory capacities; or if central coordination of respiratory and cardioinhibitory activity were shown to be epiphenomenal rather than reflecting an integrated regulatory network.

These predictions are empirically tractable and have guided hypothesis-driven research for more than three decades (Porges, 1995, 2007a, 2023, 2025a). Their empirical evaluation reflects ongoing engagement with the theory rather than insulation from testing. Disagreements concerning measurement, interpretation, or scope do not constitute falsification unless they directly address these core predictions and specify conditions under which the theory would fail.

Section 3. Structural misrepresentations in the Grossman critique

The critique advanced by Grossman et al. (2026, this issue) raises objections across multiple domains; however, the present analysis proceeds by evaluating the structural assumptions that underlie those objections rather than responding to them sequentially. Before addressing specific methodological, neurophysiological, or evolutionary claims, it is therefore necessary to examine whether the critique engages Polyvagal Theory as articulated in the peer-reviewed literature. As demonstrated below, the critique operates on a reconstructed version of the theory, shaped by selective assumptions and category errors that precede – and ultimately invalidate – its substantive conclusions.

3.1 Reconstruction of polyvagal theory as a proxy target

A central limitation of the critique advanced by Grossman et al. (2026, this issue) is that it does not evaluate Polyvagal Theory as formulated in the scientific literature, but instead constructs a synthetic version of the theory based on selective interpretations and assumptions that the theory does not assert. This reconstructed target is then evaluated as if it were Polyvagal Theory itself.

Across multiple domains, the critique attributes to Polyvagal Theory claims that are neither foundational to nor explicitly stated by the theory, including assertions regarding anatomical exclusivity, unitary or global vagal tone, and recapitulationist evolutionary logic (Grossman et al., 2026, this issue). These attributions are not supported by citation to primary sources and conflict with longstanding clarifications in the Polyvagal Theory literature (Porges, 2007a, 2023, 2025a, 2025b). As a result, the critique does not engage the theory's organizing principles – autonomic state as an intervening variable, functional differentiation of vagal pathways, and mammalian-specific circuit integration – but instead evaluates a substituted proxy.

Scientific critique requires fidelity to the claims under examination. When a theory is reconstructed in this manner, subsequent evaluation no longer tests the original framework but rather the assumptions introduced by the critique itself. This substitution sets the stage for further category errors that propagate across anatomical, physiological, and evolutionary arguments. The persistence of these reconstructed claims – despite published corrections clarifying that Polyvagal Theory does not make these claims – is documented in Appendix A as a methodological audit of representational error.

When a theory is repeatedly reconstructed in forms that diverge from its published claims and then evaluated as if

those reconstructions were original, the resulting critique assumes the structure of a straw-man argument. As shown here, this pattern characterizes the critique under review (Grossman et al., 2026, this issue).

3.2 Conflation of neuroanatomy with neurophysiology

A primary mechanism through which this reconstruction occurs is the systematic conflation of neuroanatomical description with neurophysiological function. Throughout the critique, anatomical observations are treated as if they directly falsify functional claims made by Polyvagal Theory, resulting in conclusions that are not logically warranted (Grossman et al., 2026, this issue).

Polyvagal Theory does not assert that specific anatomical structures are unique to mammals, nor does it claim that particular nuclei or fiber types are absent in non-mammalian vertebrates. Rather, the theory is concerned with the functional organization, integration, and deployment of autonomic circuits – particularly the coordination of brainstem vagal pathways with somatomotor structures regulating ingestion, vocalization, facial expression, and listening.

Within Polyvagal Theory, RSA is defined as a pathway-specific, respiratory-gated peripheral index of ventral vagal cardioinhibitory influence originating in NAmb, rather than as a generic phenomenon of cardiorespiratory coupling or a global index of vagal tone (Porges, 1995; Porges & Furman, 2011). Observations of respiratory–cardiac interactions arising from different neural substrates, fiber types, or regulatory organizations therefore do not adjudicate the theory's claims unless they engage this specified mechanism.

Grossman et al. (2026, this issue) repeatedly argue that the identification of vagal nuclei, fiber myelination, or cardiorespiratory interactions in non-mammalian species undermines Polyvagal Theory. This argument rests on the implicit premise that Polyvagal Theory predicts strict anatomical exclusivity. That premise is incorrect and has been explicitly disavowed in the literature (Porges, 2007b, 2023, 2025a).

By substituting anatomical presence for physiological organization, the critique collapses distinctions in neural origin, temporal dynamics, and circuit integration that are central to Polyvagal Theory. In doing so, it substitutes descriptive similarity for mechanistic equivalence and evaluates the theory at a level of analysis at which it is not formulated.

3.3 Framing disagreement as consensus

Grossman et al. (2026, this issue) present their critique as a consensus document, suggesting it reflects a broadly shared disciplinary rejection of Polyvagal Theory. However, the article does not describe a formal consensus process – such as a Delphi panel, professional society endorsement, or systematic meta-analytic adjudication – by which such a designation would ordinarily be established.

Rather than reflecting an adjudicated scientific consensus, the document represents the position advanced in a manuscript authored by Grossman and endorsed by a self-selected group of co-authors, several of whom have previously published critical accounts of Polyvagal Theory. These include contributions to a special issue of *Biological Psychology* edited by Grossman, in which he was acknowledged on multiple articles and for which no formal mechanism for contemporaneous response was provided. While critique and debate are essential to scientific progress, characterizing such a position paper as consensus in the absence of an explicit consensus process, institutional endorsement, or defined adjudicative criteria risks misrepresenting the current state of scientific discourse.

The invocation of consensus in this context functions rhetorically to position Polyvagal Theory as deviant from settled knowledge, rather than engaging its specific propositions and testable predictions. This framing obscures the distinction between disciplinary preference and empirical adjudication and shifts the locus of evaluation away from falsifiable claims toward perceived authority.

3.4 Summary: consequences of structural misrepresentation

Taken together, these structural misrepresentations – reconstruction of the theory's claims, conflation of anatomy with physiology, and rhetorical appeals to consensus – preclude meaningful evaluation of Polyvagal Theory's validity. When critique operates on a substituted theoretical target and evaluates it at an inappropriate level of analysis, disagreement cannot function as empirical falsification.

These structural errors propagate into subsequent arguments concerning measurement, vagal organization, evolutionary inference, and developmental logic. As a result, later disputes are framed as refutations despite not addressing the theory's core mechanisms or falsifiability conditions as articulated in Section 2.

The sections that follow examine how these foundational misalignments shape methodological, neurophysiological, evolutionary, and translational critiques, demonstrating that the appearance of contradiction arises not from empirical failure of Polyvagal Theory, but from persistent misrepresentation of its organizing principles.

Section 4. Measurement, metrics, and methodological misrepresentation

Having established that the critique advanced by Grossman et al. (2026) operates on a reconstructed version of Polyvagal Theory and evaluates it at an inappropriate level of analysis, this section examines how those structural misrepresentations extend into methodological claims. The focus is on whether objections concerning measurement – particularly with respect to RSA – accurately reflect the theory–metric relationship specified by Polyvagal Theory and the neurophysiological processes the metrics are designed to index.

4.1 Respiratory sinus arrhythmia as a pathway-specific index

A central pillar of the critique advanced by Grossman et al. (2026, this issue) is the claim that Polyvagal Theory rests on an erroneous understanding of RSA. Specifically, the critique asserts that evidence of respiratory–cardiac coupling in non-mammalian vertebrates contradicts core propositions of the theory. This objection, however, rests on a misrepresentation of how RSA is defined and employed within Polyvagal Theory.

Within Polyvagal Theory, RSA is not conceptualized as a generic phenomenon of respiratory–cardiac interaction, nor is it treated as a global or trait-like measure of vagal tone. Rather, RSA is specified as a pathway-specific, context-sensitive index of

ventral vagal cardioinhibitory influence, mediated by myelinated efferent fibers originating in NAmb (Porges, 1995, 2007a, 2023, 2025a). Critically, these efferents exhibit respiratory gating, such that their inhibitory influence on the cardiac pacemaker is rhythmically modulated across the respiratory cycle. This gating reflects central coordination between medullary respiratory rhythm–generating circuits and NAmb-originating vagal output, rather than peripheral mechanical coupling.

Importantly, Polyvagal Theory does not claim that respiratory–cardiac coupling is unique to mammals. Instead, it proposes that mammals exhibit a derived functional organization in which respiratory–cardiac coupling is generated through a centrally coordinated cardiopulmonary oscillator integrating respiratory rhythmogenesis with ventral vagal cardioinhibitory control (Porges, 1995, 2007a). This organization enables rapid, phasic modulation of cardiac output in support of behavioral flexibility and social engagement and is identified as a mammalian synapomorphy at the level of functional organization, reflecting derived circuit integration rather than the emergence of novel anatomical components (Porges, 2023, 2025a).

This definition constrains the interpretation of RSA to a specific neural mechanism embedded within an integrated brainstem cardiopulmonary network. Observations of respiratory-linked heart rate variability arising from different neural substrates, fiber types, or regulatory organizations do not meet this definition and therefore do not adjudicate the theory's claims.

4.2 Respiratory gating and central cardiopulmonary integration

Within Polyvagal Theory, respiratory gating is not treated as a confound or methodological artifact but as a defining feature of ventral vagal regulation. The rhythmic modulation of cardiac output across the respiratory cycle reflects central coordination between medullary respiratory oscillators and NAmb–originating cardioinhibitory vagal neurons, rather than peripheral mechanical coupling or reflexive respiratory effects.

This organization is consistent with classic demonstrations of coupled respiratory–cardiac control within the brainstem, in which respiratory rhythm–generating circuits directly modulate cardiac vagal motoneuron activity (Richter & Spyer, 1990). Extensive human and animal research further demonstrates that respiratory activity phasically gates the *timing* – rather than the magnitude – of vagal–cardiac motoneurone firing, while leaving tonic levels of vagal outflow largely unchanged (Eckberg, 2003).

In this framework, respiration and RSA are parallel outputs of a common brainstem regulatory network rather than sequential cause–effect processes. Experimental manipulations that abolish respiratory motor drive – such as apnea, mechanical ventilation, or high-frequency jet ventilation – markedly attenuate or eliminate RSA despite preserved cardiovascular reflexes, indicating a central rather than peripheral origin of respiratory-frequency cardiac modulation (Eckberg, 2003).

By contrast, analytic approaches that treat respiratory modulation as noise to be removed invert the neurophysiological logic underlying the measure. When respiratory gating is stripped from heart period variability, what is eliminated is not contamination but the very signal that reflects coordinated brainstem regulation.

By collapsing all respiratory–cardiac interactions into a single undifferentiated category, the critique erases distinctions in neural origin, temporal organization, and functional integration that are central to Polyvagal Theory. Under such conditions, RSA no longer maps onto the hypothesized mechanism – not because the theory fails, but because the construct being measured has been redefined at an incompatible level of analysis. This shift transforms a pathway-specific, centrally gated index into a generic correlate of breathing, rendering any subsequent test incapable of adjudicating the theory's actual claims.

4.3 Limitations of global "vagal tone" constructs

A recurring assumption in the critique advanced by Grossman et al. is that cardiac vagal regulation can be adequately characterized by a unitary construct of "vagal tone," an assumption that has been articulated explicitly in earlier methodological treatments of RSA (e.g., Grossman & Taylor, 2007). Polyvagal Theory explicitly rejects this premise, arguing instead that vagal regulation of the heart reflects the engagement of multiple efferent pathways with distinct developmental origins, temporal dynamics, and functional roles (Porges, 2007a, 2023).

As articulated in earlier formulations of the theory, tonic measures of cardiac vagal tone, while useful for characterizing general visceral states and broad autonomic balance, provide limited insight into response strategies, stimulus sensitivity, or the specificity of neural mechanisms engaged by environmental and visceral challenges. As a global construct, cardiac vagal tone obscures distinctions between NAmb–mediated regulation indexed by RSA and influences arising from DMNX, thereby limiting its utility for understanding how heart rate patterns change in response to psychological stimuli, reflect health risk, or respond to interventions (Porges, 2007a, p.139).

Because these pathways differ in myelination, central control, and response characteristics, global indices of vagal tone lack the neurophysiological specificity required to adjudicate pathway-specific hypotheses. Measures that aggregate vagal influences into a single scalar value collapse circuit-level organization into an undifferentiated output metric, obscuring the distinction between tonic background activity and phasic, state-dependent modulation.

Within Polyvagal Theory, RSA is therefore not treated as a proxy for total vagal activity, but as a selective, pathway-specific index of ventral vagal cardioinhibitory influence under defined physiological and contextual conditions. Variability in RSA is expected and informative, reflecting dynamic shifts in autonomic organization rather than instability or inconsistency of regulation.

Independent physiological evidence supports this distinction. Respiratory gating primarily organizes the *timing* of vagal–cardiac motoneurone firing as part of centrally coordinated cardiorespiratory control, rather than directly determining the magnitude of vagal outflow. Within this framework, RSA amplitude reflects the consequences of changes in central autonomic regulation rather than a direct effect of peripheral respiratory mechanics (Eckberg, 2003). Accordingly, changes in RSA amplitude cannot be interpreted as linear readouts of tonic vagal tone, nor can they be reduced to voluntary alterations in respiratory pattern.

Critiques that evaluate Polyvagal Theory using global vagal tone constructs therefore conflate pathway-specific regulation with aggregate output and impose a level of analysis incompatible with the theory's claims. Such approaches do not test the theory as formulated, but instead substitute an alternative construct that obscures the functional, temporal, and organizational distinctions central to Polyvagal Theory.

4.4 Analytic methods and the interpretation of RSA

Much of the empirical critique of Polyvagal Theory relies on legacy analytic approaches – particularly peak–valley methods that assume stationarity, fixed respiratory frequencies, and linear coupling between respiration and heart rate (e.g., Grossman & Taylor, 2007). These assumptions are frequently violated in biological systems, especially during behavioral challenge, developmental transition, or shifts in autonomic organization.

Peak–valley estimation strategies have long been shown to be data-dependent and vulnerable to systematic bias arising from respiratory frequency, waveform, and signal characteristics (Byrne & Porges, 1993). Comparative analyses further demonstrate that commonly used RSA metrics – including peak–valley, spectral, and time-domain approaches – are not equivalent and yield divergent inferences regarding autonomic regulation, particularly under nonstationary conditions (Lewis et al., 2012). Treating such estimates as interchangeable obscures the physiological processes they are intended to index and conflates analytic convenience with neurophysiological validity.

Polyvagal Theory conceptualizes autonomic regulation as inherently nonstationary, with state-dependent changes in both respiratory patterning and vagal output reflecting central coordination rather than peripheral reactivity (Porges, 1995, 2007a). Analytic methods that impose stationarity, constrain respiration, or collapse variability into a single summary metric are therefore poorly suited to testing the theory's core predictions and may yield distorted estimates of RSA precisely under the conditions most relevant to its claims.

In response to these limitations, refined analytic approaches were developed to quantify RSA under conditions of variable respiration and shifting baselines. The Porges–Bohrer method – originally outlined in Porges (1986) and formalized in subsequent publications – was designed to extract rhythmic heart period variability from aperiodic physiological signals using frequency-domain techniques aligned with biological signal structure (Porges & Bohrer, 1990). Importantly, this analytic framework does not treat respiratory modulation as noise, but as an essential feature of the physiological signal reflecting coordinated brainstem control. Lewis et al. (2012) further demonstrate that failure to account for these distinctions leads to non-equivalence among RSA estimates and obscures mechanistic interpretation.

4.5 Derived metrics and functional interpretation

In addition to refining the measurement of RSA itself, Polyvagal Theory has motivated the development of derived relational metrics designed to capture the functional organization of autonomic regulation across changing physiological contexts. These approaches do not treat autonomic activity as a static quantity, but instead quantify how centrally mediated regulatory signals are expressed at peripheral effectors as autonomic state and contextual demands vary.

One such metric, vagal efficiency, indexes the functional coupling between centrally mediated vagal regulation and cardiac response across dynamic conditions. Rather than estimating the magnitude of vagal activity per se, vagal efficiency assesses how effectively changes in central vagal regulation are translated into changes in heart period, thereby operationalizing a systems-level measure of regulatory capacity (Porges, 2025c). This formulation aligns with the theory's emphasis on functional organization and state-dependent regulation rather than tonic output.

Empirical studies demonstrate that vagal efficiency is sensitive to neuromodulatory intervention and physiological context – such as sleep state, developmental regulation, and exposure to early adversity – while remaining insensitive to peripheral cholinergic blockade. These findings support its interpretation as a marker of central autonomic coordination rather than peripheral vagal transmission alone (Porges et al., 1999; Porges et al., 2019; Dale et al., 2022; Kolacz et al., 2025). By indexing relational change across conditions, vagal efficiency remains interpretable under nonstationary circumstances in which respiratory frequency, autonomic state, and behavioral context fluctuate.

Related time–frequency approaches have likewise been developed to quantify coordination among physiological rhythms without collapsing them into single summary measures. Weighted coherence, for example, was introduced as a method for detecting rhythmic co-occurrence between physiological signals (Porges et al., 1980; Porges, et al., 1981). When applied to respiratory–cardiac data, weighted coherence quantifies the degree to which respiratory and cardiac rhythms share temporally aligned oscillatory structure, providing a formal index of coordinated timing rather than amplitude or output magnitude.

Crucially, high coherence between respiratory and cardiac signals reflects shared central organization of timing rather than mechanical causation. Consistent with evidence for a common brainstem cardiopulmonary oscillator, respiratory rhythm–generating circuits directly modulate vagal–cardiac output, producing coordinated oscillations across respiratory and cardiac domains (Richter & Spyer, 1990). The convergence of oscillatory structure indexed by coherence therefore indicates that respiration and vagal–cardiac modulation are parallel outputs of shared brainstem regulatory circuits, rather than that breathing mechanics directly generate cardiac variability (Porges et al. 1981; Porges & Coles, 1982).

Together, these derived metrics extend the measurement framework of Polyvagal Theory by capturing functional coordination and regulatory efficiency under conditions of dynamic state change. Rather than replacing RSA, they complement it by indexing how central autonomic organization is expressed across physiological contexts – particularly those in which assumptions of stationarity or fixed respiratory patterning do not hold.

4.6 Summary: measurement disputes do not constitute falsification

Disputes concerning the measurement or interpretation of RSA do not constitute a falsification

of Polyvagal Theory. Such disputes pertain to how specific hypotheses are operationalized and tested, not to whether the theory's organizing principles or core predictions are valid.

Polyvagal Theory specifies RSA as a pathway-specific, respiratory-gated peripheral expression of centrally mediated ventral vagal regulation. When defined and measured at this level of analysis, variability in RSA is expected and informative, reflecting dynamic shifts in autonomic organization rather than instability or inconsistency of regulation. Independent physiological evidence confirms that respiratory gating provides a temporal portal through which central vagal regulation becomes observable at the heart, rather than a peripheral mechanism that directly generates cardiac variability.

Measurement approaches that collapse pathway-specific regulation into global constructs, impose assumptions of stationarity, or treat respiratory modulation as noise redefine the construct under investigation. Under such conditions, RSA no longer maps onto the hypothesized mechanism – not because the theory fails, but because the measurement no longer corresponds to the theory's specified level of organization.

Accordingly, critiques that rely on mismatched constructs or inappropriate analytic assumptions evaluate a reconstructed version of Polyvagal Theory rather than the framework articulated here. Measurement choices may constrain empirical tests and shape inference, but they do not adjudicate falsifiability unless they directly engage the theory's stated mechanisms, boundary conditions, and predictions. The implications of this pathway-specific, centrally organized framework become clearer when examined in relation to the functional segregation, developmental origins, and evolutionary organization of vagal pathways.

Section 5. Functional segregation of vagal pathways

With methodological distinctions clarified, this section examines whether converging neurophysiological, developmental, and behavioral evidence supports the functional segregation of vagal efferent pathways proposed by Polyvagal Theory. Specifically, it evaluates whether distinctions between pathways originating in NAmb and DMNX reflect meaningful organization at the level of circuit integration, temporal dynamics, and regulatory role, rather than theoretical overextension or semantic differentiation.

5.1 Capacity for influence versus functional organization

A common analytical error in discussions of autonomic regulation is the assumption that shared influence over a peripheral target implies functional equivalence among the pathways involved. From this perspective, the capacity of multiple vagal pathways to modulate cardiac activity is taken to indicate that the vagal system operates as a functionally undifferentiated unit. This inference, however, conflates the ability of a pathway to affect an end organ with its role within a hierarchically organized regulatory system. At the center of this analysis is the proposition that mammalian vagal regulation is organized not merely to influence visceral organs, but to integrate cardiac control with cranial motor systems supporting social communication and survival-related behaviors – a principle that constitutes the core organizing claim of Polyvagal Theory (Porges, 1998).

Polyvagal Theory does not propose exclusive cardiac control by any single vagal pathway, nor does it deny that both NAmb and DMNX efferents can influence heart rate under particular conditions. Rather, the theory differentiates vagal pathways according to their developmental specification, myelination, central integration, temporal dynamics, and context-dependent recruitment. Shared end-organ influence does not imply equivalence at the level of neural organization or regulatory function.

5.2 Ventral vagal regulation originating in the nucleus ambiguus

In mammals, vagal efferents originating in NAmb are predominantly myelinated, exhibit respiratory gating, and provide rapid, phasic modulation of cardiac output. These pathways are embedded within an integrated brainstem network that coordinates cardiac regulation with respiration, vocalization, facial expression, listening, and ingestion. This organization enables rapid state transitions and supports physiological flexibility and social engagement – functional properties characteristic of mammalian autonomic regulation.

Developmentally, NAmb-originating cardioinhibitory neurons are specified as branchial motor (special visceral efferent) neurons and differentiate in coordination with pharyngeal arch–derived cranial motor systems. This embryological lineage accounts for their integration with the musculature of facial expression, vocalization, swallowing, and middle-ear regulation, providing a mechanistic basis for coupling cardiac regulation with social communication and ingestive behaviors (Machado & Brody, 1988; Gourine et al., 2016; Porges, 1998).

Together, these developmental and anatomical findings demonstrate that NAmb-originating cardioinhibitory pathways are not nominal subdivisions of a unitary vagal system, but are specified through distinct embryological processes and embedded within integrated branchial motor circuits.

Critically, this integration reflects a functional organization rather than an incidental anatomical arrangement. The ventral vagal system therefore cannot be reduced to a generic cardioinhibitory influence; it constitutes a pathway-specific regulatory circuit whose properties are defined by its developmental specification, central connectivity, and state-dependent engagement (Porges, 1998; Machado & Brody, 1988; Gourine et al., 2016).

5.3 Dorsal motor nucleus of the vagus pathways and metabolic regulation

In contrast to ventral vagal pathways originating in NAmb, vagal efferents arising from DMNX are predominantly unmyelinated, lack respiratory rhythmicity, and exert slower, more tonic influences on visceral organs, particularly subdiaphragmatic targets. These pathways are not integrated with branchial motor circuits and therefore do not support the rapid, phasic, context-sensitive modulation of cardiac output characteristic of ventral vagal regulation.

Within Polyvagal Theory, DMNX-mediated regulation is associated with metabolically conservative response strategies that are phylogenetically older

and hierarchically recruited within the mammalian autonomic nervous system under conditions in which rapid mobilization or social engagement is not possible, such as severe threat, hypoxia, or energy depletion. This association does not imply dysfunction or pathology; rather, it reflects the persistence of evolutionarily conserved regulatory mechanisms that support survival by reducing metabolic demand and maintaining visceral homeostasis under constrained conditions (Porges, 1998; Porges, 2007a, 2023).

5.4 Developmental specification and circuit integration

Although ventral vagal cardioinhibitory neurons originate from dorsal brainstem regions early in development, they undergo ventral migration, are specified as branchial motor neurons, and subsequently differentiate into NAmb. Developmental neuroanatomical studies in mammals have identified cardioinhibitory neurons distributed across three brainstem regions – DMNX, NAmb, and intermediate zones – interpreted as reflecting no migration, complete migration, and abortive migration during embryogenesis (Nosaka et al., 1979). These findings demonstrate that NAmb specialization reflects an active embryological process rather than a nominal anatomical distinction.

This embryological trajectory is functionally decisive and uniquely mammalian. During mammalian development, a subset of vagal cardioinhibitory neurons originating dorsally undergo ventral migration into NAmb, where they become specified as special visceral efferent neurons rather than remaining general visceral efferents of DMNX. This migration enables cardiac inhibitory control to become integrated with the mammalian branchial motor system that regulates the striated muscles of the face, larynx, pharynx, and middle ear. As first articulated within Polyvagal Theory, this reorganization links moment-to-moment regulation of cardiac output with the coordinated control of sucking, swallowing, breathing, and vocalization, as well as facial expression and listening – capacities that support nursing, vocal communication, and social engagement in mammals (Porges, 1998).

Human developmental and comparative neuroanatomical evidence demonstrates that NAmb pathways differentiate later than dorsal vagal pathways and become functionally embedded within mammalian pharyngeal arch–derived motor systems, supporting rapid, state-dependent coordination across autonomic and somatomotor domains rather than isolated visceral control (Machado & Brody, 1988; Gourine et al., 2016). This organizational principle constitutes the core of Polyvagal Theory. By situating cardiac vagal regulation within a mammal-specific branchial motor network, the theory provides a plausible mechanistic account linking observable behaviors – such as facial expression, vocal prosody, listening, and the coordinated suck–swallow–breath–vocalize sequences of early life – to underlying vagal function. In this framework, ventral vagal regulation cannot be reduced to generic cardioinhibition; its defining feature is the integration of cardiac control within a unified circuit that coordinates physiological state with social communication and survival-related behaviors.

5.5 State-dependent recruitment and hierarchical regulation

Polyvagal Theory conceptualizes vagal organization within a framework of state-dependent recruitment rather than a fixed anatomical hierarchy. Under conditions of safety, ventral vagal pathways originating in NAmb are preferentially engaged, supporting social engagement, physiological flexibility, and efficient regulation of cardiac output. As environmental or internal demands increase, ventral vagal influence is withdrawn, permitting shifts toward mobilization mediated by sympathetic pathways or, under extreme conditions, toward metabolically conservative strategies associated with DMNX-mediated regulation.

This hierarchical organization reflects principles of neural regulation observed throughout the nervous system, in which higher-order, integrative circuits are inhibited or disinhibited as functional demands change. Importantly, hierarchical recruitment does not require absolute suppression or exclusive activation of any single pathway. Multiple autonomic influences may be concurrently active, with regulatory dominance shifting as a function of context, threat level, and physiological state.

Within this framework, the term *dorsal vagal shutdown* refers to a shift in regulatory dominance toward metabolically conservative vagal strategies, not to an anatomical cessation of ventral vagal activity or an inherently pathological process. Such shifts are adaptive under conditions of extreme threat, injury, or energy depletion and reflect conserved survival strategies rather than dysfunction. Interpreting these state transitions as categorical failures or structural deficits mischaracterizes adaptive, evolutionarily conserved regulatory strategies that are recruited under conditions of extreme threat, injury, or metabolic constraint.

5.6 Summary: functional differentiation as explanatory necessity

When evaluated at the level of circuit integration, temporal dynamics, and state-dependent recruitment, converging evidence from neuroanatomy, embryology, physiology, and behavior supports the functional segregation of NAmb– and DMNX–mediated vagal pathways (Machado & Brody, 1988; Gourine et al., 2016; Porges, 1998, 2023). Treating the vagal system as a functionally undifferentiated parasympathetic unit collapses distinctions required to explain rapid state transitions, coordinated social engagement, and adaptive metabolic regulation in mammals.

The differentiation proposed by Polyvagal Theory does not represent theoretical embellishment or unnecessary complexity. Rather, it reflects the organizational requirements of a mammalian nervous system in which cardiac regulation is embedded within branchial motor circuits that coordinate facial expression, vocalization, listening, and suck–swallow–breath sequences with autonomic state. This organizational principle – first articulated within Polyvagal Theory – provides a plausible mechanistic framework linking observable social and survival-related behaviors to underlying vagal regulation (Porges, 1998, 2023, 2025a).

Functional segregation is therefore an explanatory necessity, not a discretionary hypothesis. By specifying how distinct vagal pathways are developmentally organized, differentially integrated, and selectively recruited across physiological states, Polyvagal Theory accounts for patterns of behavior and regulation that cannot be explained by models treating vagal influence as a unitary or purely tonic parasympathetic process (Porges, 2023).

With the neurophysiological organization of vagal pathways established, the next section examines how this integrated regulatory architecture emerged over evolutionary time and the selective pressures that shaped its development.

Section 6. Comparative neurobiology and evolutionary repurposing

With the functional segregation and state-dependent organization of vagal pathways established, this section examines how such integration emerged over evolutionary time. Polyvagal Theory situates mammalian autonomic regulation within an evolutionary framework that emphasizes conservation of neural elements alongside functional repurposing, altered central connectivity, and increasing circuit-level integration. Accordingly, this section evaluates whether comparative neuroanatomical and physiological evidence is consistent with this organizational model (Butler & Hodos, 2005).

6.1 Evolutionary continuity and functional reorganization

Polyvagal Theory does not propose that mammals possess autonomic structures absent in other vertebrates, nor does it treat extant non-mammalian species as ancestral intermediates. Instead, it emphasizes that evolutionary processes conserve neural elements while reorganizing their functional integration, temporal coordination, and regulatory deployment.

Across vertebrates, vagal pathways contribute to visceral regulation and respiratory–cardiac coordination, reflecting deep evolutionary continuity. However, continuity at the level of anatomical presence does not entail equivalence at the level of circuit organization. Functional reorganization – through changes in central connectivity, embryological specification, myelination, and coordination with somatomotor systems – can yield distinct regulatory strategies without introducing novel structures (Porges, 1998; Striedter, 2005; Butler & Hodos, 2005).

From this perspective, the presence of vagal efferents or respiratory–cardiac interactions in non-mammalian species is expected and theoretically unproblematic. What differentiates mammals is not the appearance of isolated traits, but the emergence of integrated brainstem networks capable of rapid, flexible state regulation in service of sustained social interaction.

6.2 Mammalian reorganization of brainstem autonomic integration

In mammals, ventral vagal cardioinhibitory pathways originating in NAmb are integrated with cranial motor circuits governing facial expression, vocalization, ingestion, head orientation, and auditory processing. This coordination supports precise coupling of respiration, cardiac output, and social signaling – capacities essential for behaviors such as suckling, nursing, vocal communication, and reciprocal caregiving.

These functional demands impose selective pressures favoring rapid state transitions, physiological flexibility, and co-regulation between individuals. Integration of autonomic and social communication circuits enables mammals to downregulate defensive responses in contexts of safety, facilitating proximity, nurturance, and cooperative interaction. Importantly, this reorganization reflects altered central connectivity and circuit coordination rather than the emergence of novel autonomic components.

Polyvagal Theory identifies this integrated configuration as a mammalian synapomorphy at the level of functional organization, reflecting a derived coordination of conserved neural elements into a regulatory architecture that supports social engagement. This framing does not imply exclusivity of individual components, but emphasizes the system-level integration that differentiates mammalian autonomic regulation (Porges, 1998; Machado & Brody, 1988; Gourine et al., 2016; Richter & Spyer, 1990).

6.3 Homology, analogy, and the limits of trait-based comparison

A recurrent source of confusion in comparative critiques of Polyvagal Theory is the conflation of homology with functional equivalence. Structures that share common ancestry may participate in markedly different regulatory networks across species, just as superficially similar behaviors may arise from distinct underlying neural organizations.

Affiliative or social behaviors observed in birds, reptiles, or fish may be analogous to mammalian social engagement at the behavioral level while relying on different autonomic substrates and patterns of circuit integration. In the absence of evidence for comparable coordination between ventral vagal cardioinhibitory pathways and cranial motor systems supporting facial expression, vocalization, and auditory processing, such behaviors cannot be assumed to reflect homologous autonomic organization.

Comparative neurobiology therefore requires evaluation at the level of circuit integration, embryological specification, and functional coupling, rather than enumeration of individual traits such as vagal myelination or respiratory–cardiac interaction. Trait-based comparison, without analysis of system organization, is insufficient to adjudicate evolutionary claims formulated at the systems level (Butler & Hodos, 2005; Striedter, 2005; Tinbergen, 1963).

6.4 Developmental evidence and evolutionary inference without recapitulation

Polyvagal Theory does not invoke recapitulationist logic, nor does it treat developmental sequencing as a replay of evolutionary history. Instead, developmental evidence is used to elucidate how conserved neural elements are progressively specified, integrated, and coordinated into functional regulatory systems.

In mammals, the postnatal emergence of RSA and increasing autonomic flexibility parallel the maturation, myelination, and circuit integration of ventral vagal pathways with cranial motor systems supporting social communication. These developmental trajectories provide mechanistic insight into how functional organization is constructed – through the progressive coordination of cardiac regulation with suck–swallow–breath–vocalize sequences and facial–vocal signaling – rather than serving as evidence of phylogenetic ancestry (Porges & Furman, 2011).

From an evolutionary developmental perspective, novel regulatory strategies need not arise from new anatomical structures, but can emerge through changes in developmental timing, patterns of connectivity, and functional integration of conserved neural elements. Contemporary evolutionary developmental biology

emphasizes that heterochrony, circuit reorganization, and functional repurposing are sufficient to generate lineage-specific regulatory architectures without introducing novel components (Gould, 1977; West-Eberhard, 2003).

Accordingly, developmental observations are interpreted as revealing principles of system assembly and calibration that inform evolutionary inference at the level of organization and function. They do not imply that ontogeny recapitulates phylogeny, but rather that evolution operates through modifications in the timing, integration, and deployment of conserved neural circuits.

6.5 Summary: evolutionary repurposing rather than structural novelty

Comparative neurobiological evidence does not contradict the evolutionary framing of Polyvagal Theory. Instead, it supports a model in which conserved autonomic elements are reorganized through changes in central connectivity, embryological specification, and circuit integration to yield lineage-specific regulatory strategies that generate testable predictions at the level of functional organization (Porges, 1998; Porges, 2023; Butler & Hodos, 2005).

By distinguishing anatomical continuity from functional organization, Polyvagal Theory avoids category errors that arise when the presence of individual traits is treated as evidence against systems-level specialization. Mammalian autonomic regulation is characterized not by exclusivity of components, but by the derived coordination of vagal, respiratory, and social communication circuits that support rapid state regulation, co-regulation between individuals, and sustained social engagement.

With this evolutionary architecture established, the next section examines how these integrated circuits are calibrated, modulated, and reshaped across the lifespan through development, experience, and context-dependent plasticity, giving rise to individual differences in autonomic regulation and vulnerability or resilience under conditions of stress and safety.

Section 7. Development, embryology, and the emergence of social regulation

Building on the evolutionary architecture established in Section 6, this section examines how developmental and embryological processes modulate, calibrate, and refine mammalian autonomic regulation across the lifespan. Polyvagal Theory draws on developmental neuroscience not to infer evolutionary ancestry, but to clarify how conserved neural elements are progressively specified, integrated, and functionally deployed through maturation and experience to support state regulation, social engagement, and physiological flexibility (Porges & Furman, 2011).

7.1 Developmental sequencing without recapitulation

Polyvagal Theory does not invoke the discredited notion that ontogeny recapitulates phylogeny. Instead, it adopts a contemporary developmental systems perspective in which the timing, sequencing, and integration of neural circuits reflect functional construction and calibration rather than evolutionary replay.

During mammalian development, autonomic regulation undergoes progressive reorganization. Early-developing vagal influences support basic visceral homeostasis and metabolic regulation, whereas later-maturing pathways – particularly ventral vagal cardioinhibitory efferents originating in NAmb – become increasingly integrated with respiratory and social communication circuits. This sequencing reflects differences in embryological specification, myelination, and central connectivity, not ancestral layering (Porges & Furman, 2011; Machado & Brody, 1988; Gourine et al., 2016).

Developmental data are therefore used mechanistically: to explain how functional integration emerges, how regulatory flexibility increases, and how vulnerability to dysregulation may arise when maturation is disrupted. These patterns do not imply that developmental stages correspond to evolutionary stages, nor do they assume linear progression across species.

7.2 Embryological specification of ventral vagal pathways

Embryological evidence provides a mechanistic basis for the functional distinctions emphasized in Polyvagal Theory. Ventral vagal cardioinhibitory neurons are specified as branchial motor (special visceral efferent) neurons and subsequently populate NAmb. Their embryological lineage aligns them with pharyngeal arch–derived cranial motor systems rather than with general visceral efferent neurons of DMNX.

This shared embryological origin accounts for the integration of cardiac regulation with muscles controlling facial expression, vocalization, swallowing, and middle-ear function. It also explains why ventral vagal regulation is coordinated with behaviors essential for feeding, caregiving, and reciprocal social interaction in mammals.

Although these neurons originate from dorsal brainstem regions early in development and undergo ventral migration, their embryological specification and final circuit integration distinguish them functionally from dorsal vagal pathways. Developmental migration therefore does not undermine functional differentiation; it clarifies how distinct regulatory circuits are assembled within the mammalian brainstem (Nosaka et al., 1979; Machado & Brody, 1988; Gourine et al., 2016; Porges, 1998).

7.3 Maturation of autonomic flexibility and social engagement

The maturation of ventral vagal regulation parallels the emergence of autonomic flexibility, affective modulation, and social engagement capacities during infancy and early childhood. Measures such as RSA and vagal efficiency increase with gestational age and postnatal development, reflecting the progressive myelination, central integration, and functional engagement of NAmb–originating cardioinhibitory pathways (Porges, et al., 2019).

These maturational changes correspond with improvements in state regulation, feeding coordination, vocalization, and reciprocal social interaction. As ventral vagal circuits become more effectively integrated with respiratory and cranial motor systems, infants acquire greater capacity to regulate physiological state in response to social and environmental cues.

When maturation is disrupted – such as in

preterm birth, early adversity, or chronic stress – autonomic regulation may remain biased toward defensive mobilization or metabolically conservative strategies, limiting regulatory flexibility and increasing vulnerability to dysregulation. From this perspective, development reveals periods of heightened sensitivity during which experience can shape the calibration and deployment of autonomic circuits (Feldman, 2007; Porges et al., 2019; Doussard-Roosevelt et al., 1997).

7.4 Clinical and developmental implications

Developmental evidence has direct translational relevance insofar as it clarifies the mechanisms through which experience shapes autonomic regulation. Interventions that support co-regulation, sensory safety, and caregiver–infant interaction have been shown to influence the maturation and functional deployment of ventral vagal pathways, enhancing autonomic stability and regulatory flexibility.

These effects are consistent with a model in which experience modulates the calibration and integration of autonomic circuits rather than directly altering isolated peripheral outputs. From this perspective, clinical and developmental interventions operate by shaping the conditions under which autonomic state regulation becomes flexible, context-sensitive, and resilient, rather than by imposing specific physiological responses – a model consistent with developmental accounts of co-regulation (Feldman, 2012).

7.5 Summary: development as mechanism, not metaphor

Developmental and embryological evidence supports Polyvagal Theory's emphasis on functional integration, hierarchical regulation, and state-dependent modulation without invoking recapitulationist logic. Ontogenetic sequencing reflects the progressive specification and calibration of conserved neural elements into a mammalian autonomic architecture capable of supporting social engagement, co-regulation, and adaptive flexibility.

By treating development as a source of mechanistic insight rather than evolutionary metaphor, Polyvagal Theory aligns with contemporary developmental neuroscience and systems biology (Porges & Furman, 2011). This perspective clarifies how autonomic regulation emerges, why it is sensitive to timing and experience, and how disruptions in development can shape long-term patterns of physiological regulation, vulnerability, and resilience.

With the developmental foundations established, the next section turns to the translational and clinical implications of this framework, evaluating how its application reflects scientific rigor, empirical accountability, and ongoing refinement rather than conceptual appeal alone.

Section 8. Translational relevance and clinical implications

The preceding sections establish Polyvagal Theory as a systems-level framework specifying how autonomic state regulation organizes behavior, physiology, and social engagement across development and evolution. This section examines the translational implications of this framework, focusing on whether the theory provides mechanistic insight that is clinically useful, empirically generative, and relevant across domains of health, mental health, and human functioning.

Importantly, translational application within Polyvagal Theory does not function as a substitute for empirical validation. Rather, it reflects the extension of mechanistically grounded hypotheses into ecologically valid contexts in which those hypotheses can be further evaluated, refined, and challenged. From this perspective, translation expands the empirical domain of theory testing rather than insulating theoretical claims from falsification.

8.1 Autonomic state regulation as a clinical target

Polyvagal Theory reframes clinical phenomena not as isolated symptoms or trait-like deficits, but as expressions of autonomic state organization. From this perspective, dysregulated behavior, affective instability, and physiological vulnerability reflect constraints on state flexibility rather than fixed pathology. Clinical change is therefore conceptualized as a shift in the underlying autonomic platform that supports more adaptive patterns of engagement and regulation.

This framing has direct implications for assessment and intervention. Measures indexing ventral vagal regulation, autonomic flexibility, and state transitions – such as RSA, vagal efficiency, and dynamic cardiorespiratory coupling – serve as peripheral windows into central regulatory organization. These metrics provide mechanistic targets for evaluating intervention effects and for identifying individuals at heightened risk for dysregulation across clinical populations.

8.2 Trauma, threat, and defensive state organization

In contexts of trauma and chronic stress, Polyvagal Theory predicts that autonomic state organization becomes biased toward defensive strategies characterized by sympathetic mobilization or dorsal vagal dominance. These states constrain social engagement, dampen physiological flexibility, and alter perceptual and affective processing.

Importantly, Polyvagal Theory does not conceptualize trauma responses as failures of cognition or volition, but as adaptive neural strategies shaped by neuroception of threat. Defensive states persist when cues of safety are insufficient to support ventral vagal engagement. From this perspective, effective intervention requires altering the autonomic context – through co-regulation, environmental safety, and targeted neuromodulation – rather than relying solely on cognitive reinterpretation or top-down control.

Empirical research supports this model, demonstrating associations between trauma exposure, reduced RSA, altered vagal efficiency, and increased vulnerability to affective and somatic symptoms (Dale et al., 2022; Porges, 2023). Interventions that enhance cues of safety and support autonomic flexibility have been shown to improve regulation across trauma-exposed populations.

8.3 Developmentally informed and relational interventions

Because autonomic regulation is calibrated through experience – particularly in early life – Polyvagal

Theory places emphasis on relational and developmental interventions that support co-regulation. Caregiver–infant interactions, therapeutic alliances, and social environments function as regulators of autonomic state, shaping the maturation and deployment of ventral vagal circuits.

Interventions such as family-based care in neonatal settings, trauma-informed psychotherapy, and relationally focused treatments derive mechanistic coherence from Polyvagal Theory insofar as they prioritize safety, predictability, and reciprocal engagement. Empirical studies demonstrate that interventions emphasizing co-regulation can enhance autonomic stability and improve outcomes in populations ranging from preterm infants to adults with trauma histories (Dale et al., 2022; Porges et al., 2019).

8.4 Beyond mental health: broader translational domains

The translational relevance of Polyvagal Theory extends beyond mental health into domains including developmental pediatrics, education, occupational health, and social policy. Because autonomic state regulation influences attention, learning, immune function, and metabolic processes, environments that support safety and co-regulation can have cascading effects across health and performance outcomes.

Within this broader context, Polyvagal Theory offers a biologically grounded rationale for interventions that prioritize relational safety, environmental predictability, and physiological regulation. These principles are increasingly reflected in trauma-informed educational practices, workplace wellness initiatives, and community-based health programs (Porges, 2025b).

8.5 Summary: translation through mechanism

Polyvagal Theory's translational value derives from its specification of mechanism rather than metaphor. By identifying autonomic state regulation as an organizing platform for behavior and physiology, the theory generates testable hypotheses, informs measurement strategies, and guides intervention design across clinical and applied settings.

Critiques that focus narrowly on measurement disputes or anatomical enumeration overlook this translational coherence. Whether one adopts Polyvagal Theory in whole or in part, its contribution lies in linking brainstem autonomic organization to observable patterns of regulation, vulnerability, and resilience in ways that are empirically tractable and clinically meaningful.

Section 9. Epistemic structure of the critique and standards of scientific evaluation

The preceding sections have examined the conceptual foundations, methodological commitments, neurophysiological organization, evolutionary framing, developmental evidence, and translational relevance of Polyvagal Theory as articulated in the peer-reviewed literature. Against this integrated account, the present section evaluates the critique advanced by Grossman et al. (2026, this issue) not in terms of disciplinary preference or rhetorical force, but in relation to the epistemic standards required for scientific refutation.

Scientific critique functions not merely by expressing disagreement, but by accurately representing the claims under evaluation, engaging those claims at the appropriate level of analysis, and specifying conditions under which they could be falsified. When these conditions are not met, critique ceases to operate as empirical adjudication and instead reflects interpretive persistence – repetition of objections without responsiveness to theoretical clarification or empirical development.

9.1 Levels of analysis and category error

A defining feature of the critique advanced by Grossman et al. (2026, this issue) is the persistence of objections that have remained substantively unchanged despite extensive theoretical clarification, methodological refinement, and empirically documented revision of Polyvagal Theory over more than two decades. Core claims concerning RSA, functional differentiation of vagal pathways, and evolutionary framing reappear in largely the same form as earlier critiques, notwithstanding direct responses and revisions articulated in the peer-reviewed literature (Porges, 2007b; Lewis et al., 2012; Porges, 2023). This persistence stands in contrast to Polyvagal Theory's documented responsiveness to critique, including explicit theoretical clarification, refinement of boundary conditions, and development of measurement strategies designed to engage those revisions.

At the same time, a recurring limitation of the critique is its failure to respect levels of analysis. Polyvagal Theory is articulated as a systems-level framework specifying principles of organization, integration, and state-dependent regulation. Many objections raised by Grossman et al. (2026, this issue) operate at mismatched levels – treating anatomical presence, isolated physiological phenomena, or analytic conventions as if they directly falsified claims formulated at the level of functional organization.

When objections persist without engaging published clarification or revised formulations, disagreement ceases to be hypothesis driven. Persistence alone does not constitute falsification, particularly when the theoretical target has been explicitly specified and empirically elaborated. Moreover, when observations at one level of analysis are used to refute claims at another without explicit bridging hypotheses, category error results. For example, documenting respiratory–cardiac coupling in non-mammalian species does not adjudicate claims concerning mammalian-specific circuit integration; nor does demonstrating variability among RSA metrics address predictions concerning pathway-specific regulation. In the absence of explicit mapping between levels of analysis, such objections cannot function as falsification.

9.2 Reduction of theory to measurement

A central epistemic limitation of the critique advanced by Grossman et al. (2026, this issue) is its reliance on a reconstructed version of Polyvagal Theory rather than the theory as articulated in the peer-reviewed literature. As demonstrated in Sections 2 and 3, this reconstruction occurs through selective attribution of claims, redefinition of core constructs, and omission of explicitly stated boundary conditions and qualifications. Once a substituted theoretical target is established, empirical objections directed at that target cannot adjudicate the validity of the original theory itself.

Scientific critique requires representational fidelity

to the claims under evaluation. When a theory's scope, mechanisms, or conditions of application are altered prior to evaluation, disagreement becomes decoupled from the theoretical object under scrutiny. In such cases, critique tests assumptions introduced by the critic rather than the theory as formulated. No accumulation of empirical data can resolve disagreement rooted in misrepresentation, because evidentiary standards are applied to a displaced theoretical object rather than the theory itself (Porges, 1998, 2007a, 2007b, 2023; Grossman & Taylor, 2007).

A related epistemic error arises when Polyvagal Theory is reduced to the validity of particular measurement techniques. While empirical testing necessarily depends on measurement, no theory is reducible to any single operationalization. Polyvagal Theory specifies mechanisms, boundary conditions, and falsifiable predictions; measurement strategies are tools for engaging those predictions, not substitutes for the theory itself.

Disputes concerning RSA metrics, analytic assumptions, or statistical approaches therefore do not constitute refutation unless they demonstrate that no valid measurement strategy could, in principle, index the proposed mechanism. The critique advanced by Grossman et al. (2026, this issue) does not meet this standard. Instead, it evaluates the theory through the lens of preferred metrics while disregarding alternative analytic approaches explicitly developed to align with the theory's specified level of organization. Under such conditions, disagreement reflects methodological preference rather than empirical falsification of the theory's claims.

9.3 Substitution of consensus for adjudication

The critique further relies on rhetorical appeals to consensus rather than explicit falsification. Scientific consensus, when it exists, emerges from convergent empirical evidence and transparent adjudication of competing hypotheses. Consensus, where it exists, is an outcome of empirical adjudication rather than a substitute for it. In the absence of defined consensus procedures or engagement with falsifiability conditions, claims of consensus function rhetorically rather than epistemically.

By positioning Polyvagal Theory as deviant from settled knowledge without specifying the empirical criteria by which it would be adjudicated false, the critique shifts evaluation from evidence-based testing to sociological positioning. Such framing does not advance scientific understanding and risks conflating disciplinary preference with empirical judgment.

9.4 Criteria for scientific refutation

For a critique to function as a scientific refutation, it must (a) accurately represent the theory under evaluation; (b) engage claims formulated at the same level of analysis; (c) specify empirical conditions under which those claims would fail; and (d) demonstrate that such conditions obtain. As shown throughout this rebuttal, the critique advanced by Grossman et al. (2026, this issue) does not satisfy these criteria.

Instead, the critique evaluates reconstructed claims, employs mismatched levels of analysis, substitutes methodological preference for mechanism, and invokes consensus without adjudication. Under these conditions, disagreement cannot be interpreted as empirical falsification.

9.5 Summary: disagreement is not disproof

Scientific progress depends on rigorous critique grounded in accurate representation and shared evaluative standards. Polyvagal Theory invites such critique by articulating explicit mechanisms and falsifiability conditions. However, disagreement that does not engage those mechanisms on their own terms cannot function as refutation.

The analysis presented here demonstrates that the critique advanced by Grossman et al. (2026, this issue) does not undermine Polyvagal Theory's core propositions. Rather, it reveals persistent misalignment between the theory as formulated and the theory as evaluated. Clarifying this distinction is essential not only for adjudicating the present debate, but for maintaining the epistemic integrity of scientific discourse in systems neuroscience and psychophysiology.

Conclusion

This rebuttal has evaluated the critique advanced by Grossman et al. (2026, this issue) not by disputing individual claims in isolation, but by examining whether the critique engages Polyvagal Theory as it is formulated in the peer-reviewed literature and whether it meets the epistemic standards required for scientific refutation. Across conceptual, methodological, neurophysiological, evolutionary, developmental, and translational domains, the analysis demonstrates that the critique repeatedly evaluates a reconstructed proxy of the theory rather than its stated mechanisms, scope, and falsifiability conditions, as documented across Sections 3–9.

Polyvagal Theory is articulated as a systems-level framework specifying how autonomic state regulation organizes behavior, physiology, and social engagement through functionally differentiated and developmentally integrated neural circuits. The theory does not rest on claims of anatomical exclusivity, global vagal tone, or recapitulationist evolution. Instead, it advances testable hypotheses concerning pathway-specific regulation, hierarchical state transitions, and mammalian-specific integration of autonomic and social communication circuits. These hypotheses have guided empirical research for more than three decades and continue to generate refinements in measurement, theory, and application. These hypotheses are biologically grounded, generate falsifiable predictions tied to identifiable neurophysiological substrates, and have continued to guide methodological, developmental, and translational research rather than remaining insulated from empirical test.

The critique reviewed here does not falsify these core propositions. Disagreements concerning RSA metrics, comparative anatomy, or evolutionary framing do not constitute refutation unless they directly engage the theory's specified mechanisms and demonstrate conditions under which its predictions fail. As shown throughout this paper, such engagement is largely absent. Where empirical disagreement exists, it reflects differences in measurement preference, level of analysis, or theoretical framing rather than evidence against the theory's organizing principles.

Importantly, clarifying these distinctions is not an exercise in theoretical defense for its own sake. Accurate representation is a prerequisite for productive scientific dialogue. When critiques persistently misstate a theory's claims – despite explicit correction – the resulting discourse risks substituting rhetorical disagreement for empirical adjudication and obscuring mechanisms that remain clinically and scientifically generative.

Polyvagal Theory remains open to refinement, empirical challenge, and revision. Its continued value lies not in immunity from critique, but in its capacity to integrate neurophysiology, development, evolution, and clinical science into a coherent framework of autonomic regulation. Future progress will depend on critiques and tests that engage the theory at its intended level of organization, specify falsifiability conditions with precision, and evaluate evidence accordingly. Only under those conditions can disagreement advance understanding rather than entrench misunderstanding.

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Appendix A. Documentation of recurrent misrepresentations

This appendix documents a set of claims reiterated in the critique advanced by Grossman et al. (2026, this issue) that had been explicitly addressed and documented as misrepresentations in peer-reviewed literature nearly two decades earlier (Porges, 2007b). The purpose of this appendix is not rhetorical rebuttal, but methodological clarification: to demonstrate that several objections presented as unresolved scientific controversies reflect a failure of representational uptake rather than the absence of empirical response, and to support transparent evaluation rather than empirical adjudication of the theory itself.

A.1 Scope and rationale

Polyvagal Theory has been the subject of critical discussion since its initial formulation. Scientific critique is essential to theory refinement; however, such critique presupposes engagement with the theory as articulated and responsiveness to published clarification. When objections are reiterated without modification following explicit correction, the resulting discourse no longer reflects empirical disagreement but rather a breakdown in scholarly communication.

Appendix A therefore serves as an audit of representational fidelity. It identifies claims attributed to Polyvagal Theory in the Grossman et al. (2026, this issue) critique, summarizes the corrective responses previously published in the peer-reviewed literature, and notes whether those corrections were acknowledged or incorporated in the current critique.

A.2 Recurrent claims and published corrections

Table A1 summarizes several recurrent claims advanced by Grossman and colleagues across publications spanning nearly two decades, alongside citations to peer-reviewed sources in which those claims were directly addressed. These include, but are not limited to:

- The assertion that Polyvagal Theory proposes strict anatomical exclusivity of vagal pathways to mammals
- The characterization of RSA as a global or unitary index of "vagal tone"
- The claim that Polyvagal Theory relies on recapitulationist evolutionary logic
- The interpretation of respiratory–cardiac coupling in non-mammalian species as a direct falsification of the theory

In each case, the Polyvagal Theory literature explicitly clarifies that the theory does not advance these claims, and specifies the level of analysis at which its propositions are formulated (Porges, 2007a, 2007b, 2023).

A.3 Implications for Scholarly Evaluation

The persistence of these reconstructed claims despite formal correction has methodological implications. When critiques fail to incorporate published responses, subsequent debate risks becoming circular, with disagreement framed as unresolved controversy rather than as repetition of previously addressed points.

This pattern undermines the cumulative nature of scientific discourse. Productive critique requires not only the articulation of objections, but also engagement with the evolving literature and responsiveness to clarification. Absent such engagement, disagreement cannot function as empirical adjudication.

A.4 Purpose of Inclusion

The inclusion of this appendix is not intended to foreclose future critique or to assert theoretical finality. Rather, it is intended to clarify the historical record, delineate points of actual disagreement from those of representational error, and support transparent evaluation of Polyvagal Theory on its own terms.

By documenting these patterns explicitly, Appendix A provides readers and reviewers with the context necessary to assess whether the critique advanced by Grossman et al. (2026, this issue) advances scientific understanding or reiterates previously corrected mischaracterizations.

Table A1. *Documentation of recurrent misrepresentations*

Recurrent claim attributed to Polyvagal Theory	Where the claim appears in Grossman et al. (2026, this issue) and earlier critiques	Clarification and correction in the Polyvagal Theory literature	Representative citations
Polyvagal Theory proposes that vagal pathways regulating the heart are anatomically exclusive to mammals	Grossman & Taylor (2007); reiterated in Grossman et al. (**2026, this issue**)	Polyvagal Theory explicitly distinguishes anatomical presence from functional organization. It does not claim exclusivity of vagal structures, but specifies mammalian-specific integration, myelination, and circuit coordination of NAmb-originating pathways	Porges (1995, 2007a, 2007b, 2023)
RSA is treated as a global or unitary index of "vagal tone"	Grossman & Taylor (2007); Grossman et al. (**2026, this issue**)	RSA is defined as a pathway-specific, respiratory-gated index of ventral vagal (NAmb-originating) cardioinhibitory influence, not as a measure of total vagal output or tonic parasympathetic activity	Porges (1995, 2007a, 2023); Porges & Furman (2011); Lewis et al. (2012)
Evidence of respiratory–cardiac coupling in non-mammalian species falsifies Polyvagal Theory	Grossman & Taylor (2007); Grossman et al. (**2026, this issue**)	Polyvagal Theory does not deny respiratory–cardiac coupling outside mammals; it proposes that mammals exhibit a derived, centrally coordinated cardiopulmonary oscillator integrating respiration with ventral vagal cardiac control	Porges (1995, 2007a, 2023); Richter & Spyer (1990)
Polyvagal Theory relies on recapitulationist evolutionary logic (ontogeny recapitulates phylogeny)	Grossman & Taylor (2007); Grossman et al. (**2026, this issue**)	The theory explicitly rejects recapitulation. Developmental data are used mechanistically to explain circuit maturation and integration, not to infer evolutionary ancestry	Porges (2007b, 2023); Porges & Furman (2011)
Dorsal vagal regulation is framed as inherently pathological or equivalent to shutdown	Grossman et al. (**2026, this issue**)	Polyvagal Theory characterizes DMNX-mediated regulation as an evolutionarily conserved, metabolically conservative strategy that is adaptive under specific conditions, not intrinsically pathological	Porges (1998, 2007, 2023)
Disagreement with preferred RSA metrics constitutes falsification of Polyvagal Theory	Grossman et al. (**2026, this issue**)	Measurement disputes do not constitute falsification unless they demonstrate failure of the specified mechanism. Polyvagal Theory anticipates nonstationarity and motivated development of alternative analytic methods	Byrne & Porges (1993); Porges & Bohrer (1990); Lewis et al. (2012)

Note. This table documents claims repeatedly attributed to Polyvagal Theory in the Grossman et al. critique and identifies peer-reviewed sources in which those claims were explicitly clarified as mischaracterizations. The persistence of these attributions reflects a failure of representational uptake rather than unresolved empirical controversy.