Critiques of Polyvagal Theory: A Comprehensive Analysis

by
Stephen W. Porges, PhD
Distinguished University Scientist,
Kinsey Institute, Indiana University

Overview

Polyvagal Theory has contributed a biologically grounded framework for understanding autonomic regulation, safety, and social engagement, informing research and clinical practice across neuroscience, psychology, medicine, and education. As the theory has gained wider visibility, it has also become the subject of critique across academic and public forums. This page provides a concise, evidence-based clarification of Polyvagal Theory's core claims, scope, and scientific grounding, with the goal of supporting accurate evaluation through reference to primary sources and contemporary neurophysiology.

The published criticisms of Polyvagal Theory, particularly those by Paul Grossman and E. W. Taylor, exhibit a recurring pattern of misinterpretation and factual inaccuracy. Despite multiple clarifications in the peer-reviewed literature (Porges, 2007b, 2022, 2023, 2025a, 2025b), these critiques continue to introduce conceptual errors into both academic discourse and clinical application. Grossman's 2023 critique reiterates themes first advanced by Taylor, drawing heavily on his anatomical and evolutionary misreadings, framed in a rhetorically authoritative style that may mislead readers unfamiliar with the theory's foundational literature.

In addition to peer-reviewed publications, public-facing sources—particularly the Wikipedia entry on Polyvagal Theory—have played a role in disseminating inaccuracies. That entry prominently cites two critiques: Neuhuber and Berthoud (2022) and Doody, Burghardt, and Dinets (2023). While both articles were peer-reviewed, their appearance in a special issue of Biological Psychology edited by Grossman suggests an editorial alignment that likely shaped both their thematic and and conceptual framing.

The critique by Neuhuber and Berthoud exemplifies an anatomical-functional misinterpretation. While questioning the functional interpretation of vagal pathways proposed by Polyvagal Theory, they do not dispute the core neuroanatomical distinctions—specifically, the separation between the dorsal motor nucleus of the vagus (DMNX) and the nucleus ambiguus (NAmb), nor the contrasting myelination of their efferents. However, they omit a critical tenet of the theory: that the Social Engagement System (SES) derives from structures of the embryological pharyngeal arches and that communication between the cardioinhibitory vagus and the SES is largely indirect, with the exception of direct pathways involving the trigeminal nerve. Wikipedia, in summarizing this critique, distorts it further—presenting it as a categorical refutation of the anatomical foundation of Polyvagal Theory, which the original article does not claim.

Similarly, the critique by Doody, Burghardt, and Dinets misrepresents the theory's phylogenetic framing. They challenge what they interpret as a rigid binary between "asocial reptiles" and "social mammals." However, Polyvagal Theory is explicitly mammalian-centric and defines social behavior through a mammalian lens. It emphasizes that sociality, in the context of Polyvagal Theory, is rooted in a mammalian-specific autonomic innovation—the ventral vagal complex—responsible for regulating the Social Engagement System. While reptiles may exhibit adaptive social behaviors, these behaviors are mediated through different signaling systems and neural architectures and serve different evolutionary functions. Thus, social behavior in reptiles, although evolutionarily adaptive, is not equivalent to mammalian sociality as defined by Polyvagal Theory. By conflating behavioral analogy with neurophysiological homology, the critique overlooks the theory's domain-specific scope. Public summaries, including Wikipedia, further amplify this misrepresentation, overstating the critique and ignoring Polyvagal Theory's focus on hierarchical autonomic evolution.

Crucially, the problem is not merely that these articles exist, but that they have been filtered through a public platform in a way that both exaggerates and misrepresents their content. This layered mischaracterization—shaped by personal bias and not by accurate scholarship—contributes to the dissemination of misleading information. It underscores the need for scholarly evaluation grounded in direct engagement with primary sources, rather than interpretations shaped by editorial alignment, selective emphasis, or secondary dissemination.

To clarify the nature of these misinterpretations, Table 1 identifies the recurrent domains of error present in critiques of Polyvagal Theory, outlining how they manifest and the consequences they carry for accurate theoretical interpretation.

Table 1. Core Domains of Misrepresentation in Critiques of Polyvagal Theory

Domain	Type of Misrepresentation	Description	Impact on Interpretation of PVT
Anatomical / Neurophysiological	Misrepresentation of NAmb vs. DMNX pathways	Collapses functionally distinct vagal pathways by treating NAmb and DMNX as interchangeable; ignores the distinction between myelinated and unmyelinated efferent fibers and their pathway-specific cardioinhibitory functions.	Obscures the hierarchical, pathway-specific organization of vagal regulation that is foundational to Polyvagal Theory.
Physiological / Functional	Mischaracterization of vagal regulation	Treats vagal influences on the heart as unitary and static; fails to distinguish context-dependent shifts between ventral vagal regulation, sympathetic mobilization, and dorsal vagal shutdown.	Leads to incorrect inferences about autonomic flexibility, state-dependent regulation, and the mechanisms underlying adaptive versus defensive responses.
Methodological	Misrepresentation of RSA measurement	Critiques Polyvagal Theory using outdated or inappropriate metrics (e.g., peak-to-trough RSA) that conflate respiratory mechanics with centrally mediated cardiopulmonary regulation; ignores validated analytic approaches that more precisely isolate vagal cardioinhibitory influence.	Produces false negative conclusions about vagal regulation and undermines valid empirical tests of Polyvagal Theory.
Evolutionary	Conflation of analogy and homology	Treats basic cardiorespiratory coupling observed in fish or reptiles as evolutionarily equivalent to mammalian RSA; ignores mammalian-specific neuroanatomical reorganization, including NAmb-linked myelination and integration with social communication systems.	Misrepresents the evolutionary claims of Polyvagal Theory by substituting functional analogy for neuroanatomical homology.
Developmental / Interpretive	Misinterpretation of the Social Engagement System (SES)	Ignores the embryological origins of the SES in pharyngeal arch–derived branchial motor circuits and their integration with vagal regulation; treats SES as a metaphorical or speculative construct rather than a developmentally grounded neural system.	Distorts Polyvagal Theory's account of how autonomic regulation is functionally integrated with facial expression, vocalization, and social communication.
Conceptual / Theoretical	Strawman reconstruction of Polyvagal Theory	Attributes claims to Polyvagal Theory that it does not make (e.g., mammalian exclusivity of sociality or RSA, non-falsifiability); narrows the theory into an oversimplified "dual vagus" caricature.	Shifts critique away from the theory as formulated, leading to evaluations that are epistemically invalid and scientifically uninformative.

As shown in Table 1, these domains span anatomical, physiological, evolutionary, conceptual, and methodological dimensions. While the specific content of critiques varies, they consistently fall within this typology of error— undermining the coherence and scientific validity of Polyvagal Theory by misrepresenting core constructs or applying flawed evaluative criteria. This structured framework provides a diagnostic lens through which to assess the epistemic integrity of both academic and public-facing criticisms.

While Table 1 identifies recurrent error domains across critiques, Table 2 maps these distortions more specifically to key publications—highlighting how Taylor's foundational misrepresentations established patterns that were subsequently reproduced and extended in later critiques, particularly those by Grossman. Citations of Taylor and his colleagues (e.g., Campbell et al., 2006; Monteiro et al., 2018; Sanches et al., 2019; Taylor et al., 2022) indicate recurrent patterns across publications; specific dominant error types for individual articles are detailed in a later typological summary (see Table 4) and the annotated bibliography.

Table 2. Misrepresentations in Taylor and Grossman's Critiques of Polyvagal Theory

Category	Representative Publications	Nature of Misrepresentation	Consequences for Scientific Discourse
Anatomical	Taylor et al. (2006, 2014)	Collapses functional distinctions between DMNX and NAmb; posits a unitary vagus.	Disregards the functional significance of mammalian myelinated cardioinhibitory pathways.
Evolutionary	Monteiro et al. (2018); Taylor et al. (2022); Sanches et al. (2019)	Confuses functional analogy (fish/reptile cardiorespiratory coupling) with neuroanatomical homology (mammals).	Misrepresents the evolutionary specificity of Polyvagal Theory.
Conceptual Appropriation	Grossman & Taylor (2007)	Adopts Polyvagal Theory constructs without attribution.	Misinforms the reader while borrowing from the theory's conceptual framework.
Strawman Amplification	Grossman (2023) citing Taylor et al. (2022)	Refutes claims not made by Polyvagal Theory by attributing unsupported assumptions.	Propagates false critiques and distorts the scientific dialogue surrounding Polyvagal Theory.

As shown in Table 2, Taylor's publications repeatedly collapse anatomical distinctions and confuse phylogenetic constructs central to Polyvagal Theory. Grossman's later writings amplify these misrepresentations, embedding them in critiques that are rhetorically authoritative but empirically misaligned. This inheritance of error contributes to a layered mischaracterization of Polyvagal Theory across both academic and public domains.

While Tables 1 and 2 categorize general and foundational misrepresentations, Table 3 engages directly with major criticisms advanced by Grossman—evaluating them against established empirical and neurophysiological evidence.

Table 3. Empirical Evaluation of Grossman's Major Criticisms of Polyvagal Theory

Domain	Claim	Argument Summary	Evaluation & Counter-Evidence
Anatomy	Dual-vagus model is non-anatomical	Claims ventral and dorsal vagal pathways lack meaningful anatomical distinction.	Refuted by comparative neuroanatomy (Strain et al., 2024; Porges, 1995, 2007b, 2025b).
Physiology	RSA is a respiratory artifact	RSA is a mechanical consequence of breathing.	Misrepresents the common cardiopulmonary oscillator (Porges, 2007a, 2023; Richter & Spyer, 1990), which links RSA primarily to NAmb–mediated cardioinhibitory pathways rather than DMNX-mediated regulation.
Physiology	Bradycardia disproves vagal brake	Uses threat-induced bradycardia to dismiss ventral vagal influence.	Confuses dorsal shutdown with ventral regulation (Reed et al., 1999; Porges, 2025b; Strain et al., 2024), as DMNX activation elicits bradycardia independent of locomotor suppression.
Evolution	Reptile cardiorespiratory coupling disproves mammalian specificity	Cardiorespiratory coupling is not mammal-specific.	Confuses analogy and homology (Porges, 1998, 2023, 2025a).
Interpretation	PVT overextends physiology to behavior	Claims vagal metrics do not justify behavioral predictions.	Polyvagal Theory distinguishes physiological state from emotion (Dale et al., 2022; Lewis et al., 2012).

Table 3 consolidates the principal claims advanced by Grossman (2007, 2023) and evaluates them against contemporary empirical and neurophysiological findings. Across anatomical, physiological, evolutionary, and interpretive domains, these critiques consistently reflect misunderstanding rather than empirically grounded disagreement—reinforcing the need for evaluation rooted in primary literature.

Table 3 functions not merely as a summary of neuroscientific evidence that refutes Grossman's criticisms, but also as a demonstration of the internal coherence of Polyvagal Theory. By aligning with contemporary findings across neuroanatomy, physiology, developmental psychobiology, and evolutionary biology, the theory maintains conceptual integrity and cross-disciplinary consistency—highlighting its explanatory robustness rather than vulnerability to reductionist critique.

Taken together, the critiques addressed here—whether anatomical, physiological, evolutionary, or conceptual—reveal more about the interpretive lens of their authors than about the validity of Polyvagal Theory itself. The persistence of mischaracterization, particularly in public-facing summaries, underscores the necessity of returning to primary sources when evaluating the theory's claims. As this analysis demonstrates, Polyvagal Theory remains a coherent and empirically anchored framework, whose interdisciplinary reach continues to inform both basic neuroscience and translational applications.

Methodological Misrepresentation and the RSA Artifact Claim

Grossman's repeated assertion that RSA is merely a respiratory artifact stems from his reliance on the peak-to-trough method of RSA quantification—a technique long recognized as methodologically flawed. First critiqued by Byrne & Porges (1993) and later confirmed by Lewis et al. (2012), this method violates key statistical assumptions, is overly sensitive to respiration and signal trend, and fails tests of normality and stationarity. In contrast, the Porges–Bohrer method is more responsive to vagal blockade, less influenced by respiratory variables, and more suitable for valid parametric analysis.

Despite longstanding critiques, Grossman has continued to apply the peak-to-trough approach, producing systematic bias and a misleading interpretation of RSA's relationship to ventral vagal tone. His refusal to adjust analytic methods or acknowledge empirical refutations has allowed this error to persist across more than two decades of publications.

As a result, many of the published criticisms of Polyvagal Theory are not grounded in competing evidence, but in a singular, uncorrected methodological flaw. Addressing such errors is critical to restoring scientific accuracy and analytic accountability in discourse surrounding Polyvagal Theory.

Is Grossman Critiquing Polyvagal Theory or a Misrepresentation of It?

Close analysis of Grossman's critiques (2007, 2023) indicates that his arguments frequently engage a version of Polyvagal Theory that diverges from its formulation in the primary literature. This divergence arises through selective quotation, reliance on outdated models of vagal physiology, and misinterpretation of comparative neuroanatomical evidence. As a result, premises are attributed to Polyvagal Theory that it does not advance (e.g., that RSA is unrelated to respiration or that NAmb exists only in mammals), and these attributed positions are subsequently challenged.

This pattern reflects a categorical misunderstanding rather than a disagreement grounded in competing empirical data. In particular, Grossman repeatedly conflates functional analogy with neuroanatomical homology, overlooking the phylogenetic rationale articulated within Polyvagal Theory. Similarly, the failure to distinguish between DMNX and NAmb obscures the anatomical basis of the theory's hierarchical model of autonomic regulation.

Although the foundational papers of Polyvagal Theory are frequently cited in these critiques, the points under criticism are not linked to an accurate representation of the theory's original neurophysiological claims. Instead, Grossman's commentaries rely on a highly siloed treatment of specific neuroscience subdisciplines—such as comparative anatomy or neurophysiology—without engaging integrative theoretical models that relate structure, function, development, and behavior. Consequently, the critiques focus disproportionately on secondary interpretations and downstream clinical applications rather than on the core constructs articulated in the foundational literature. Framed as methodological critique, this body of work perpetuates misrepresentations first introduced by Taylor and colleagues, substituting metaphor for mechanism and evaluating a version of Polyvagal Theory that was never advanced in its primary sources.

Ethical Implications

Misrepresentations of Polyvagal Theory have generated epistemic harm—confusing clinicians, misguiding educators, and undermining translational clarity. These consequences are not merely academic; they obstruct the accurate application of a theory grounded in validated neuroanatomy, robust physiological metrics, and a coherent neuroethological model of autonomic regulation.

Recent findings strengthen the empirical foundation of Polyvagal Theory. For example, Strain et al. (2024) demonstrated via optogenetic activation that neurons in the DMNX—a key component of the dorsal vagal complex—produce bradycardia and modulate anxiety-like behavior without triggering locomotor suppression. This distinction aligns with Polyvagal Theory's differentiation between dorsal-mediated threat responses and immobilization without fear, addressing longstanding misconceptions, including those perpetuated in the Biological Psychology special issue edited by Grossman.

This context raises further ethical concerns. The guest-edited issue featured multiple critiques of Polyvagal Theory—such as Neuhuber and Berthoud (2022) and Doody et al. (2023)—without offering rebuttals or scholarly responses. Given Grossman's established opposition to Polyvagal Theory, the absence of countervailing views introduces potential editorial bias. Such asymmetry risks misrepresenting scholarly consensus and propagating error in both academic and public domains.

As Porges (2025b) notes, scientific critique should engage with theories as they are written—not as caricatures. Ethical scientific discourse demands fidelity to evidence, currency in interpretation, and transparency in method. These standards are essential not only for accurate evaluation of Polyvagal Theory, but for preserving the integrity of scientific exchange more broadly.

Misrepresentation in Public Discourse

Several criticisms frequently attributed to Polyvagal Theory—such as oversimplification or over-commercialization—are, in fact, critiques of how the theory has been represented or applied by third parties, not flaws inherent to the theory itself.

- **Oversimplification:** Claims that Polyvagal Theory oversimplifies autonomic-emotional relationships often stem from secondary interpretations that disregard its neurophysiological depth. Constructs such as the "vagal brake" and "neuroception," while empirically supported, are frequently reduced to metaphor in popular discourse. These simplifications reflect a lack of engagement with the primary literature, not intrinsic features of Polyvagal Theory.

- **Over-commercialization:** Polyvagal Theory has been adapted in coaching, wellness, and branding contexts that may lack scientific rigor. While this trend raises valid concerns about translational ethics, the theory itself embeds ethical considerations and should not be held accountable for distortions introduced by external actors.

- **Critique of the anatomical distinction between NAmb and DMNX:** Neuhuber and Berthoud (2022) argue that the anatomical separation proposed by Polyvagal Theory lacks empirical support. However, their critique overlooks well-established evidence differentiating NAmb and DMNX—including their

embryological origins, efferent myelination, and functional specificity. Transcriptomic data (Coverdell et al., 2019; Jalil et al., 2023) reveal molecular markers unique to NAmb neurons related to autonomic function and myelination. Strain et al. (2024) further demonstrated that selective optogenetic activation of DMNX neurons induces bradycardia and modulates anxiety-like behavior, confirming the functional and behavioral relevance of the dorsal vagal complex as described in Polyvagal Theory.

- **Phylogenetic critiques based on non-mammalian vagal structures:** Some critiques cite vagal features in lungfish or reptiles to challenge Polyvagal Theory's evolutionary claims. These arguments conflate functional analogy with phylogenetic homology. Polyvagal Theory does not suggest that vagal regulation is exclusive to mammals; rather, it identifies the NAmb-mediated, myelinated vagus integrated with cranial social engagement as a mammalian adaptation. Equating general vagal function across species with this specialized system reflects a misunderstanding of Polyvagal Theory's evolutionary specificity.

It is essential to differentiate between a theory's empirical foundation and its public portrayal. The Wikipedia entry on Polyvagal Theory exemplifies this conflation, often treating misuse or misrepresentation by third parties as if they reveal flaws in the theory itself. This constitutes a category error in scholarly evaluation.

These criticisms, when examined closely, reveal not theoretical vulnerabilities but challenges in accurate dissemination. Upholding the scientific integrity of Polyvagal Theory requires precise communication and ethical stewardship by those who apply it in clinical, educational, and public domains.

Typology of Errors in Critiques of Polyvagal Theory

To synthesize the recurring issues identified across critiques of Polyvagal Theory, Table 4 presents a typology of common error categories. These range from rhetorical misrepresentations to empirical and methodological missteps. Each error type is defined, exemplified by its appearance in published critiques, and paired with its resulting scientific distortion.

This diagnostic classification underscores a central concern of the current document: that the most persistent and visible criticisms of Polyvagal Theory often fail to engage with the theory on its empirical and conceptual terms. Rather than offering grounded scientific challenges, these critiques frequently reflect misunderstandings of evolutionary biology, neuroanatomy, and methodological validity—or arise from editorial and translational dynamics external to the theory itself.

Table 4. Typology of Recurrent Errors in Critiques of Polyvagal Theory

Error Type	Description	Representative Citations
Mischaracterization of Theoretical Claims (incl. Strawman Arguments)	Distorts or exaggerates Polyvagal Theory's original claims—often by attributing positions the theory does not hold, narrowing the theory into a simplified dual-vagus caricature, asserting non-falsifiability while ignoring operationalized metrics, or omitting core hierarchical principles. Includes explicit strawman arguments and scope misalignment.	Doody et al. (2023); Grossman (2023); Grossman & Taylor (2007); Taylor et al. (2014)
Conflation of Analogy and Homology	Conflates functional similarity (e.g., cardiorespiratory coupling) with evolutionary continuity. Treats basic coordination (e.g., in fish or reptiles) as equivalent to mammalian RSA, without acknowledging the phylogenetic and neuroanatomical innovations emphasized in Polyvagal Theory—particularly the NAmb-mediated myelinated vagal system and its integration with the Social Engagement System.	Campbell et al. (2005, 2006); Taylor et al. (2022); Sanches et al. (2019)
Anatomical Oversight	Ignores or collapses distinctions between key vagal structures (DMNX vs. NAmb), overlooks myelination, and fails to represent the hierarchical organization foundational to Polyvagal Theory. In addition, it omits afferent pathways and the integrated role of the Social Engagement System in Polyvagal Theory's neurophysiological framework.	Neuhuber & Berthoud (2022); Taylor et al. (2014)
Methodological Misrepresentation	Critiques Polyvagal Theory based on flawed or outdated physiological measurements, ignoring more current statistical models and metrics used within Polyvagal Theory. Often advocates techniques documented to distort the assessment of cardioinhibitory vagal influence (e.g., peak-to-trough RSA).	Grossman (2023); Grossman & Taylor (2007)
Editorial or Interpretive Bias	Centers critiques within publication venues lacking balanced representation or peer rebuttal, or attributes flaws in public or commercial interpretations of Polyvagal Theory to the theory itself. This includes editorial gatekeeping and conflating secondary misuse (e.g., oversimplified therapies or commercial branding) with theoretical invalidity.	Grossman (2023); Doody et al. (2023)

Conclusion

A comprehensive evaluation of the published criticisms of Polyvagal Theory reveals a recurring structure of error: the most influential objections—across anatomical, physiological, evolutionary, and conceptual domains—are repeatedly traceable to the scholarly contributions and editorial influence of Paul Grossman. Whether via direct authorship, collaboration with E. W. Taylor, or editorial curation of the 2023 Biological Psychology special issue, Grossman has played a central role in shaping the public and academic discourse around Polyvagal Theory.

This concentration of dissent, particularly when amplified through publication venues that exclude rebuttal, raises legitimate concerns about scholarly objectivity and the simulation of consensus. Scientific critique must rest on accurate representation of a theory's foundational claims, not caricature or rhetorical deflection. As this manuscript has shown, many critiques of Polyvagal Theory misrepresent its core constructs, often via a small number of categorical errors that recur across publications and platforms.

Such repetition should not be mistaken for convergence or consensus. Rather, it underscores the need for greater epistemic accountability in both scholarly and public domains. Readers and evaluators are urged to consult the primary literature directly, where the empirical foundations and conceptual architecture of Polyvagal Theory are most clearly articulated.

References

Byrne, E. A., & Porges, S. W. (1993). Data-dependent filter characteristics of peak-valley respiratory sinus arrhythmia estimation: A cautionary note *Psychophysiology*, 30(6), 672–678. https://doi.org/10.1111/j.1469-8986.1993.tb02061.x

Campbell, H. A., Taylor, E. W., & Egginton, S. (2005). Does respiratory sinus arrhythmia occur in fishes? *Biology Letters*, 1(4), 484–487. https://doi.org/10.1098/rsbl.2005.0365

> **Note: Conflation of Analogy and Homology —** Treats fish cardiorespiratory coupling as homologous to mammalian RSA, mistaking functional similarity for phylogenetic continuity and overlooking the NAmb-based, myelinated mammalian specialization central to Polyvagal Theory.

Campbell, H. A., Leite, C. A. C., Wang, T., et al. (2006). Evidence for a respiratory component… in the rattlesnake. *Journal of Experimental Biology*, 209(14), 2628–2636. https://doi.org/10.1242/jeb.02278

> **Note: Conflation of Analogy and Homology —** Uses reptilian RSA-like coupling to imply evolutionary equivalence with mammalian RSA, bypassing Polyvagal Theory's emphasis on mammalian NAmb innovations and SES integration.

Coverdell, T., Ivison, R., Tao, J., and Campbell, J. (2019). 582-P: Disambiguating the nucleus ambiguus with single-cell transcriptomics. *Diabetes 68* (Suppl. 1):582. https://doi.org/10.2337/db19-582-P

Dale, L. P., Kolacz, J., Mazmanyan, J., Leon, K. G., Johonnot, K., Bossemeyer Biernacki, N., & Porges, S. W. (2022). Childhood maltreatment influences autonomic regulation and mental health in college students. *Frontiers in Psychiatry*, 13, 841749. https://doi.org/10.3389/fpsyt.2022.841749

Doody, J. S., Burghardt, G. M., & Dinets, V. (2023). The evolution of sociality and the polyvagal theory. *Biological Psychology*, 180, Article 108569. https://doi.org/10.1016/j.biopsycho.2023.108569

> **Note: Mischaracterization of Theoretical Claims (incl. Strawman Arguments) —** Recasts Polyvagal Theory as claiming reptiles are "asocial" and treats the theory's mammalian-specific autonomic innovation argument as a denial of reptilian sociality, conflating behavioral analogy with Polyvagal Theory's neuroanatomical scope.

Grossman, P. (2023). Fundamental challenges and likely refutations of the five basic premises of the polyvagal theory. *Biological Psychology*, 180, 108589. https://doi.org/10.1016/j.biopsycho.2023.108589

> **Note: Mischaracterization of Theoretical Claims (Strawman Argument) —** Attributes claims to Polyvagal Theory that it does not make (e.g., mammalian exclusivity of RSA). Misrepresents the theory's anatomical specificity and physiological nuance. Fails to acknowledge the distinct cardioinhibitory myelinated pathways of the nucleus ambiguus (NAmb) and their integration with cranial motor systems that constitute the Social Engagement System, as well as the coordinating role of the common cardiopulmonary oscillator in mammalian autonomic regulation.

Grossman, P., & Taylor, E. W. (2007). Toward understanding respiratory sinus arrhythmia: Relations to cardiac vagal tone, evolution and biobehavioral functions. *Biological Psychology*, 74(2), 263–285. https://doi.org/10.1016/j.biopsycho.2005.11.014

> **Note: Mischaracterization of Theoretical Claims (incl. Strawman Arguments) —** Sets up a strawman of Polyvagal Theory's evolutionary and vagal claims, borrows Polyvagal Theory constructs without proper attribution, and collapses NAmb/DMNX distinctions while relying on invalid RSA framing.

Jalil, M., Coverdell, T. C., Gutierrez, V. A., Crook, M. E., Shi, J., Stornetta, D. S., Schwalbe, D. C., Abbott, S. B. G., & Campbell, J. N. (2023). Molecular disambiguation of heart rate control by the nucleus ambiguus [Preprint]. *bioRxiv*. https://doi.org/10.1101/2023.12.16.571991

Kolacz, J., Roath, O. K., Lewis, G. F., Karrento, K., et al. (2025). Cardiac vagal efficiency is enhanced by percutaneous auricular neurostimulation in adolescents with nausea: Moderation by antidepressant drug exposure. *Neurogastroenterology & Motility*, 37(5), e15007. https://doi.org/10.1111/nmo.15007

Lewis, G. F., Furman, S. A., McCool, M. F., & Porges, S. W. (2012). Statistical strategies to quantify respiratory sinus arrhythmia: A comparative evaluation. *Biological Psychology*, 89(2), 349–364. https://doi.org/10.1016/j.biopsycho.2011.11.009

Monteiro, D. A., Taylor, E. W., Sartori, M. R., Cruz, A. L., Rantin, F. T., & Leite, C. A. (2018). Cardiorespiratory interactions previously identified as mammalian are present in the primitive lungfish. *Science Advances*, 4(2), eaaq0800. https://doi.org/10.1126/sciadv.aaq0800

> **Note: Conflation of Analogy and Homology —** Infers evolutionary homology from functional similarity in lungfish cardiorespiratory interactions, overlooking Polyvagal Theory's emphasis on mammalian-specific neuroanatomical reorganization, particularly NAmb-linked myelination and integration with the Social Engagement System as evolutionary innovations.

Neuhuber, W. L., & Berthoud, H.-R. (2022). Functional anatomy of the vagus system: How does the polyvagal theory comply? *Biological Psychology*, 174, 108425. https://doi.org/10.1016/j.biopsycho.2022.108425

Note: Anatomical Oversight — Reduces Polyvagal Theory to a simplistic “dual vagus” narrative while bypassing developmental, myelination, and hierarchical distinctions between NAmb and DMNX. Ignores afferent pathways and omits the Social Engagement System framework, thereby overlooking Polyvagal Theory's emphasis on integrated functional circuits and evolutionary organization.

Porges, S. W. (1995). Orienting in a defensive world: Mammalian modifications of our evolutionary heritage: A Polyvagal Theory. *Psychophysiology*, 32(4), 301–318. https://doi.org/10.1111/j.1469-8986.1995.tb01213.x

Porges, S. W. (1998). Love: An emergent property of the mammalian autonomic nervous system. *Psychoneuroendocrinology*, 23(8), 837–861. https://doi.org/10.1016/S0306-4530(98)00057-2

Porges, S. W. (2003). Social engagement and attachment: A phylogenetic perspective. *Annals of the New York Academy of Sciences*, 1008(1), 31–47. https://doi.org/10.1196/annals.1301.004

Porges, S. W. (2004). Neuroception: A subconscious system for detecting threats and safety. *Zero to Three*, 24(5), 19–24.

Porges, S. W. (2007a). The polyvagal perspective. *Biological Psychology*, 74(2), 116–143. https://doi.org/10.1016/j.biopsycho.2006.06.009

Porges, S. W. (2007b). A phylogenetic journey through the vague and ambiguous Xth cranial nerve: A commentary on contemporary heart rate variability research. *Biological Psychology*, 74(2), 301-307. https://doi.org/10.1016/j.biopsycho.2006.08.007

Porges, S. W. (2022). Polyvagal theory: A science of safety. *Frontiers in Integrative Neuroscience,* 16, 871227. https://doi.org/10.3389/fnint.2022.871227

Porges, S. W. (2023). The vagal paradox: A polyvagal solution. *Comprehensive Psychoneuroendocrinology*, 16, 100200. https://doi.org/10.1016/j.cpnec.2023.100200

Porges, S. W. (2025a). Polyvagal Theory: Current status, clinical applications, and future directions. *Clinical Neuropsychiatry*, 22(1), 7–26. https://doi.org/10.36131/cnfioritieditore20250301

Porges, S. W. (2025b). Polyvagal theory: a journey from physiological observation to neural innervation and clinical insight. *Frontiers in Behavioral Neuroscience*, 19, 1659083. https://doi.org/10.3389/fnbeh.2025.1659083

Porges, S. W., & Furman, S. A. (2011). The early development of the autonomic nervous system provides a neural platform for social behavior: A Polyvagal Perspective. *Infant and Child Development*, 20(1), 106–118. https://doi.org/10.1002/icd.688

Reed, S. F., Ohel, G., David, R., & Porges, S. W. (1999). A neural explanation of fetal heart rate patterns: A test of the Polyvagal Theory. *Developmental Psychobiology*, 35(2), 108–118. https://doi.org/10.1002/(SICI)1098-2302(199909)35:2%3C108::AID-DEV4%3E3.0.CO;2-N

Richter, D. W., & Spyer, K. M. (1990). Cardiorespiratory control. In A. D. Loewy & K. M. Spyer (Eds.), Central regulation of autonomic functions (pp. 189–207). *Oxford University Press*. https://doi.org/10.1093/oso/9780195051063.003.0011

Sanches, P. V., Taylor, E. W., Durán, L. M., Cruz, A. L., Dias, D. P., & Leite, C. A. C. (2019). Respiratory sinus arrhythmia is a major component of heart rate variability in undisturbed, remotely monitored rattlesnakes (Crotalus durissus). *Journal of Experimental Biology*, 222, jeb197954. https://doi.org/10.1242/jeb.197954

> **Note: Conflation of Analogy and Homology —** Relabels reptilian coupling as RSA homologous to mammalian NAmbmediated RSA, disregarding PVT's mammalianspecific neuroanatomical and behavioral integration.

Strain, M. M., Conley, N. J., Kauffman, L. S., Espinoza, L., Fedorchak, S., Castro Martinez, P., … Boychuk, C. R. (2024). Dorsal motor vagal neurons can elicit bradycardia and reduce anxiety-like behavior. *iScience*, 27(3), 109137. https://doi.org/10.1016/j.isci.2024.109137

Taylor, E. W., Campbell, H. A., Levings, J. J., Young, M. J., Butler, P. J., & Egginton, S. (2006). Coupling of the respiratory rhythm in fish with activity in hypobranchial nerves and with heartbeat. *Physiological and Biochemical Zoology*, 79(6), 1000–1009. https://doi.org/10.1086/507663

> **Note: Conflation of Analogy and Homology —** Interprets respiratory–cardiac coupling in fish as evidence of evolutionary continuity with mammalian respiratory sinus arrhythmia, without addressing the mammalian-specific NAmb-linked, myelinated vagal pathways and their integration with the Social Engagement System emphasized in Polyvagal Theory.

Taylor, E. W., Wang, T., & Leite, C. A. C. (2022). An overview of the phylogeny of cardiorespiratory control in vertebrates with some reflections on the "polyvagal theory." *Biological Psychology*, 172, 108382. https://doi.org/10.1016/j.biopsycho.2022.108382

> **Note: Mischaracterization of Theoretical Claims (incl. Strawman Arguments) —** Reframes Polyvagal Theory as asserting mammalian exclusivity of cardiorespiratory coupling, and critiques this reformulated claim rather than engaging the theory's stated emphasis on mammalian neuroanatomical specialization, particularly ventral vagal complex integration supporting social engagement.

Find the most recent papers from Dr. Stephen Porges here:

February 2026: When a Critique Becomes Untenable: A Scholarly Response to Grossman et al.'s Evaluation of Polyvagal Theory

September 2025: Polyvagal theory: a journey from physiological observation to neural innervation and clinical insight

For additional information on Polyvagal Theory visit us at: polyvagal.org

PVI
polyvagal institute