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Commentary

The Polyvagal Theory in Contemporary Psychology: Why Popularity Should Not Be Confused With Scientific Validity

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In recent years, the Polyvagal Theory (PVT) has gained remarkable popularity within clinical psychology, psychotherapy, trauma studies, and applied neuroscience. Despite its widespread adoption in training programs and clinical narratives, its core assumptions lack robust neuroanatomical, neurophysiological, and methodological validation. This paper critically examines the scientific foundations of the Polyvagal Theory, highlighting conceptual ambiguities, methodological weaknesses, and epistemological risks associated with its uncritical use in psychology. Drawing on contemporary autonomic neuroscience and psychoneuroendocrinoimmunology (PNEI), the paper argues that the success of the theory reflects narrative appeal rather than empirical robustness. This raises significant epistemological and methodological concerns for clinical psychology. The paper concludes by calling for a return to biologically plausible, falsifiable, and realistic integrative models of psychological regulation.

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Introduction

The Polyvagal Theory^{[1][2]} has become one of the most cited and taught frameworks in contemporary applied psychology. It is frequently invoked to explain emotional regulation, trauma responses, social behavior, and therapeutic change.

However, the increasing diffusion of a theory does not necessarily correspond to its scientific solidity^[3].

As emphasized by Grossman^[4] and Bottaccioli,^[5] scientific psychology must remain anchored to **biological plausibility, empirical verification, and epistemological coherence**, especially when dealing with models that claim neurophysiological grounding.

This paper argues, with other authors,^{[5][4][6]} that the Polyvagal Theory fails to meet these criteria.

Core Claims of the Polyvagal Theory

In its essential formulation, the Polyvagal Theory proposes a hierarchical organization of the vagus nerve into functionally distinct subsystems, including a so-called ventral vagal complex associated with safety and social engagement, and a dorsal vagal complex associated with immobilization and shutdown^[1].

Neuroanatomical and Physiological Plausibility

While conceptually appealing, these claims require direct anatomical and physiological evidence, which remains insufficient in the current scientific literature^[7].

One of the central criticisms of the Polyvagal Theory concerns the lack of empirical validation for the proposed vagal subsystems. Contemporary autonomic neuroscience shows that regulation emerges from distributed and integrated networks rather than from linear hierarchical switches^[7].

Evolutionary Inconsistencies in the Comparative Anatomy Evidence

For over two decades, the available comparative animal science literature has already raised substantial and unresolved challenges to the evolutionary narrative proposed by Porges, according to which the two ventral and dorsal vagal subsystems are the result of an evolutionary process, whereby the ventral vagal system (characterized by myelinated neural fibers) is phylogenetically more recent and characteristic of mammals (and particularly humans).

There is evidence of the presence of myelinated vagal fibers in cartilaginous fish, lungfish, amphibians, reptiles, and birds,^{[8][9][10][11][12][13][14]} which undermines the foundations of Porges's hypotheses.

Conceptual Problems in the Hierarchical "Stratification" Narrative

The Polyvagal Theory is therefore based on assumptions that are discredited from a comparative anatomical perspective, but also from a strictly conceptual one. Indeed, Porges refers to two twentieth-century scientists, Jackson and MacLean, respectively for their concepts of evolution as a hierarchical and conservative process, according to which more evolutionarily recent systems control older ones (Jackson), and of cerebral anatomical "stratification," according to which it is possible to identify the increase in complexity in the upper layers of the brain compared to the more phylogenetically ancestral lower layers (MacLean).

Both concepts have long been dismissed as simplistic metaphors at best, representing unfounded narratives that are both fascinating from a communicative perspective and flawed from an evolutionary biological perspective^{[15][16]}.

Moreover, vagal pathways interact continuously with sympathetic, endocrine, immune, and cortical systems, undermining a simplistic dichotomy between safe and defensive vagal states^[4].

Methodological Flaws in HRV and RSA-Based Inference

A recurring methodological issue in the Polyvagal literature is the misuse of heart rate variability (HRV) as a supposed validation of the theory. HRV is a non-specific index of autonomic modulation influenced by multiple psychophysiological systems, including respiration, metabolic state, and circadian rhythms^{[4][17]}. Interpreting HRV changes as evidence of distinct vagal subsystems therefore represents a category error rather than a valid scientific inference.

The Polyvagal Theory is based on the fundamental assumption that the ventral and dorsal vagal regions of the mammalian brainstem each have specific and unique effects on heart rate control as well as socioemotional behavior, but it is significant that only one measurable phenomenon (respiratory sinus arrhythmia - RSA), as an index of vagal processes, serves as the linchpin for virtually every premise^[18].

A review indicated that RSA is highly susceptible to multiple confounding factors, such as respiratory parameters and individual differences in heart rate variability^[19]. This suggests that interpretations based on RSA alone may mask underlying physiological complexities, undermining the robustness of conclusions drawn from these measurements.

For instance, although studies have shown associations between polyvagal constructs and emotional regulation, the predictions made by the theory concerning physiological reactivity under stress have yet to be consistently validated across varied contexts. Notably, socioeconomic adversity and parental relationships also play critical roles in physiological reactivity and emotional regulation, suggesting that a more integrative approach may be necessary to fully address these intricate dynamics^[20].

The polyvagal hypothesis assumes that RSA is a mammalian phenomenon since Porges^[1] states, “RSA has not been observed in reptiles,” but Grossman^[18] counters that using RSA as equivalent to a general vagal tone index or even a cardiac vagal tone index is conceptually a category mistake.

Brainstem Circuitry: Freezing, DVMN, and the Bradycardia Claim

Vagal responses to emotional freezing in mammals appear to be mediated primarily by the ventrally located dorsal motor nucleus of the vagus (DVMN)^[21], not by the dorsal motor nucleus of the vagus as claimed by the polyvagal theory that “the immobilization defense system recruits unmyelinated vagal motor pathways to the heart to produce an immediate and massive slowing of heart rate”^[2].

There is no credible evidence that the DVMN plays any role in massive bradycardia as claimed by the polyvagal theory^{[18][21]}.

Limits of Clinical Extension: Psychopathology and Intervention Claims

Another area of criticism concerns the application of Polyvagal Theory to explain psychopathologies. While the theory posits that dysregulated vagal responses significantly contribute to emotional and behavioral disorders, the exclusive focus on autonomic pathways can minimize the complexity of psychological conditions influenced by a multitude of factors, including environmental and cognitive aspects^[22].

Applications of Polyvagal Theory in clinical practice and interventions also face critiques regarding their practical efficacy. While there is a clear intent to utilize the theory in therapeutic frameworks, real-world applications encounter challenges related to inconsistencies in individual responses to interventions based on theoretical constructs. For example, while the theory emphasizes enhancing social connections to regulate the autonomic nervous system, research indicates that the effectiveness of such interventions is influenced significantly by contextual factors and individual differences^[23].

Epistemological Concerns: Falsifiability and Narrative Drift

The Polyvagal Theory increasingly functions less as a testable scientific model and more as a clinical narrative. This shift entails several epistemological risks, including reductionism disguised as integration, loss of falsifiability, and didactic distortion^[3]. A theory that cannot be clearly operationalized or potentially disproven cannot serve as a foundational scientific framework.

The degree of neural activation of the vagus nerve represents the convergence of multiple central and peripheral factors of the person, understood as a biopsychosocial entity. It is not, as proposed by Porges's reductionist polyvagal theory, the sole cause of complex biopsychosocial phenomena.

The Microbiota Gap: Why a Host-Centered Vagal Narrative Is Incomplete

Still in the context of the modern model of human health, which considers each person as a biopsychosocial holobiont that therefore also includes the enormous interaction between human and non-human cells of the microbiota, it is significant to note that, despite the fact that for over twenty years we have been witnessing the so-called "microbiota revolution," so named for the enormous impact that microbiota science is having on many established biomedical paradigms, even in the most recent publications of the polyvagal theory, there is no mention of this particularly important factor^{[24][25]}.

Interestingly, Porges makes no mention of the microbiota, despite coining, among other things, the term *neuroception* to indicate the automatic and unconscious neurobiological process through which the nervous system continuously assesses the internal and external environment to determine whether a situation is safe, dangerous, or life-threatening.

Neuroception: Operational Ambiguity and Category Errors

The term *neuroception*, introduced within the Polyvagal Theory to describe an automatic and unconscious neural evaluation of safety and threat^{[1][2]}, presents significant epistemological weaknesses.

First, it lacks a clear and independent operational definition, making it difficult to distinguish *neuroception* from established constructs such as threat appraisal, salience detection, or interoceptive processing.

Second, *neuroception* does not specify identifiable neural mechanisms or falsifiable predictions, functioning instead as a post hoc explanatory label applied to observed autonomic or behavioral outcomes.

This circularity undermines its scientific status, as the construct explains phenomena by redescribing them rather than by generating testable hypotheses^[3].

Moreover, *neuroception* conflates multiple levels of analysis (neural, psychological, and evolutionary) without adequate theoretical mediation, resulting in a category error^[18].

Consequently, *neuroception* operates more as a narrative heuristic than as a scientifically grounded construct, placing it closer to a prescientific explanatory framework than to a robust theoretical concept.

In the modern science of the Microbiota-Gut-Brain-Mind axis^[26], the hypotheses formulated by the polyvagal theory struggle to find a logical and coherent place with the most recent scientific findings, which emphasize the enormously integrated, complex, and bidirectional interaction that characterizes this axis.

The absence of the microbiota in the Polyvagal Theory is epistemologically relevant, not merely a clinical limitation.

According to holobiont theory, the human organism must be understood as an integrated biological system composed of host and microbial genomes, whose interactions fundamentally shape regulation and adaptation.

The microbiome exerts a powerful epigenetic influence through metabolites, immune signaling, and neuroactive compounds that modulate gene expression, autonomic functioning, and stress responsivity across the lifespan.

Any theoretical model claiming to explain autonomic regulation or unconscious environmental assessment therefore entails an implicit commitment to this multilayered informational ecology.

By excluding the microbiota, the Polyvagal Theory violates the principle of causal completeness and reduces regulation to a host-centered neural narrative.

This omission also contradicts the theory's emphasis on continuous environmental monitoring, as microbiota-mediated epigenetic signaling represents a primary interface between organism and environment.

Consequently, neglecting the microbiome places the Polyvagal Theory outside contemporary systems biology, epigenetics, and psychoneuroendocrinology frameworks.

The uncritical adoption of the Polyvagal Theory in clinical psychology may lead to oversimplified interpretations of trauma and emotional dysregulation, psychologization of biological conditions, and the use of interventions presented as vagal regulation without sufficient empirical support^[5].

As repeatedly argued within PNEI, scientific rigor constitutes an ethical obligation in healthcare professions.

Toward Network-Based Models of Regulation Compatible with PNEI

Rejecting the Polyvagal Theory as a central explanatory model does not imply rejecting embodiment or bottom-up processes. Rather, it calls for network-based models of regulation, integration with psychoneuroendocrinology, and recognition of regulation as a dynamic, complex, and multilevel adaptive process^{[7][5]}.

Conclusion

The popularity of the Polyvagal Theory should not shield it from scientific scrutiny.

Psychology, as a science and a profession, must resist the temptation of elegant but weakly supported models.

The clinical community deserves theories that are not only intuitive but true enough to be useful—and humble enough to be questioned.

The future of psychology depends on its ability to integrate complexity without abandoning rigor, favoring theories that are not only intuitive but sufficiently supported by data to be clinically and scientifically responsible.

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Data Availability

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Author Contributions

M.A. was the sole author and is responsible for all aspects of the manuscript.

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