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COMMENTARY

WHY THE POLYVAGAL THEORY IS UNTENABLE

An international expert evaluation of the polyvagal theory and commentary upon Porges, S.W. (2025). Polyvagal theory: current status, clinical applications, and future directions. Clin. Neuropsychiatry, 22(3), 169-184.

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Abstract

Thirty-nine highly acknowledged experts in the areas of the physiology and the evolution of the vagus nerve and of vertebrate social behavior (many whose works have been cited in the polyvagal theory [PVT] literature as supporting the theory) were invited by the first author to participate as co-authors of this article. They were asked to evaluate the PVT and comment upon an overview of the theory written by its author (Porges, 2025a). All those invited, save one, accepted and co-authored the paper. The dissenting scholar was "unfamiliar with the PVT." This article specifically appraises--based upon the current state of knowledge of autonomic function and vertebrate evolution--several major elements of the PVT, as described in Porges (2025a) and elsewhere. These include: 1) the validity of PVT assumptions that respiratory sinus arrhythmia is a direct measure of the extent of central vagal drive to the heart; 2) PVT characterizations regarding the neuroanatomy and functions of two major brainstem vagal nuclei, the ventrally situated Nucleus Ambiguus and the Dorsal Motor Nucleus of the vagus nerve; 3) PVT assertions regarding the evolution of the vagus nerve; 4) PVT claims about the specificity of mammalian social behavior in relation to nonmammalian vertebrates, and 5) PVT interpretations of earlier seminal physiological literature. All co-authors agree that major tenets of the PVT are not supported by past or current knowledge and, in several instances, are inconsistent with the broader evidence base. Since the topics addressed constitute fundamental premises of the PVT, we conclude that the PVT is untenable, because it is not defensible based on existing neurophysiological and evolutionary evidence. The psychological elements composing the superstructure of the PVT are primarily derived from earlier psychological literature and are neither clarified nor strengthened by PVT constructs that lack evidence. This article does not intend to address alternative explanations about relations between vagal function and psychological processes, although such explanations do exist.

Key words: polyvagal, vagus nerve, parasympathetic, autonomic nervous system, respiratory sinus arrhythmia, respiratory heart-rate variability

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Introduction

The polyvagal theory (PVT), over the last decade, has become one of the most popular theoretical frameworks in psychotherapy and is often assumed to be scientifically supported. Its appeal has also expanded to several other fields, including education and different forms of healthcare. In a recent review titled "Polyvagal theory: Current status, clinical applications, and future directions," Porges (2025a) reiterates the view that the polyvagal theory rests on a phylogenetic progression of neurophysiology and the emotional capacity of vertebrates (e.g. Porges, 2007, 2011). This is amply exemplified by the first sentence of the abstract defining the scope of the review as: "*Polyvagal Theory [PVT] proposes an evolutionarily informed neurophysiological framework for understanding how the autonomic nervous system supports social engagement, emotional resilience, and adaptive physiological responses*." In this commentary, we dispute the assertion that PVT accurately portrays the evolutionary neurophysiological changes proposed and the contention that the evolution of social behavior in vertebrates reflects these proposed changes[1]. We base our arguments upon experimental studies of the vagus nerve and the autonomic nervous system in vertebrates carried out for the past six decades, and on well-established phylogenetic inferences of social evolution in vertebrates.

Psychological elements of PVT form a major part of the theory and primarily derive from pre-existing writings, theories and research. They include concepts such as psychological safety, social engagement, co-regulation, emotional freezing and psychological dissociation (e.g. Maslow, 1943; Rogers, 1954; Walters & Parke, 1965; Bowlby, 1969; Brazelton et al., 1975; Sroufe & Waters, 1977; Saurez & Gallup, 1979; Lester et al., 1985; Field et al., 1990; Roelofs et al., 2010). Physiological knowledge of the autonomic nervous system has been neither central nor essential to the development of these concepts. When the inaccurate physiological assumptions of PVT are discounted, it follows that these emotional and psychosocial processes are correspondingly divested of any PVT-hypothesized physiological framework. That is not to say that these processes have no physiological underpinnings, only that PVT does not accurately depict them. We maintain that an erroneous belief system regarding relationships between psychological states and neurophysiology may be harmful when presented as facts to healthcare providers, patients and their families. Moreover, implausible physiological explanations are neither helpful for psychotherapists nor for the credibility of psychology, psychotherapy, psychophysiology or other related fields within medicine and neuroscience.

For all these reasons, we, as a diverse group of specialists in the areas of autonomic control, vagal physiology, psychophysiology and evolution of vertebrate social behavior, find it necessary to clarify the extent to which polyvagal assertions deviate from the currently existing evidence base and consensus regarding the physiology and evolution of the vagus nerve and autonomic nervous system. We proceed first by addressing the major physiological assertions that are fundamental to PVT but are untenable. Then we point out major inaccuracies regarding citations in the paper and their interpretations.

Respiratory sinus arrhythmia is not a direct and reliable measure of central vagal outflow to the heart (mediated by the ventrally located brainstem Nucleus Ambiguus)

Most non-invasive studies of vagal control of heart rate rely on the analysis of respiratory sinus arrhythmia[2] (RSA) as an index of the extent of vagal effects upon average heart rate during a specific period of time, which is termed cardiac vagal tone, or parasympathetic

[1] Burghardt (1977) proposes that this contention is a common anthropocentric, if not mammalian, bias, which has been repeatedly challenged for many decades.

[2] Recently, Menuet et al. (2025) proposed an alternative term for respiratory sinus arrhythmia, respiratory heart-rate variability (respHRV), to clarify that the phenomenon is primarily a measure of rhythmic fluctuations of heart rate inherently tied to respiration – and not a direct measure of cardiac vagal tone (i.e. the level of vagal effects upon average heart rate during a particular period of time). Menuet et al. also address misinterpretations, particularly in the psychological world, which have arisen because of the use of RSA, as a term. Here, we choose to apply the older name RSA because of its familiarity for the target audience and to simplify discussion. However, we strongly recommend employing RespHRV in future research and theory as a more suitable and comprehensible term for respiratory-related rhythmic changes in heart rate, and have herein applied it for non-mammalian vertebrates.

control of heart rate (Katona & Jih, 1975). RSA plays a crucial and central role in PVT. Porges (e.g. 2007, 2011, 2023, 2025b) claims that RSA is a direct central nervous system (CNS) measure of vagal outflow from the brainstem Nucleus Ambiguus (NA) to the heart (i.e. efferent activity), and that this measure is not influenced by peripheral mechanisms (e.g. respiratory rate and depth, blood pressure, the baroreflex, arterial CO2 level, pulmonary stretch receptors) or other factors. This assertion is fundamental to all elements of PVT but is contrary to a voluminous body of evidence presented below.

Firstly, although a major source of RSA may be of central origin, RSA is inherently a peripheral measure, and its quantification is entirely based upon fluctuations in heart rate, a process that is importantly influenced by, but peripheral to, direct CNS control. RSA is a physiological phenomenon that primarily serves to coordinate respiratory and cardiovascular functions (Grossman & Wientjes, 1986; Elstad et al., 2018; Fisher et al., 2022; Grossman, 2024; Menuet et al., 2025), inherently peripheral in nature.

Secondly, RSA is a sub-component of vagal cardiac regulation, specifically contributing to coordination of heart rate and breathing, but does not represent the large diversity of potential cardiac effects induced by vagal regulation (Menuet et al., 2025). This specific subcomponent of cardiac regulation, RSA, reflects only the phasic, dynamic adjustments in vagal efferent activity that manifest as heart-rate fluctuations during the breathing cycle – not the tonic, sustained influence of vagal activity on mean heart rate (i.e. cardiac vagal tone). Critically, equivalent levels of cardiac vagal tone may be associated with divergent magnitudes of RSA; likewise, equivalent levels of RSA may be associated with divergent levels of cardiac vagal tone (e.g. Grossman et al. 1991; Grossman & Kollai, 1993; Grossman and Taylor, 2007; Menuet et al., 2025).

Thirdly, in a number of investigations, RSA has proven to be neither a sensitive nor accurate index of individual differences in cardiac vagal activity (i.e. trait differences; see review of studies in Grossman, 2024). Variations in RSA magnitude between individuals do not necessarily reflect individual differences in cardiac vagal tone.

Fourthly, numerous studies show that various factors peripheral to central vagal outflow can substantially confound the relation between within-individual (i.e. state) variations in RSA and cardiac vagal tone (see **figure 1**). Most prominent, perhaps, are the *often-profound* effects of respiratory frequency and tidal volume (e.g. Hirsch & Bishop, 1981; Saul et al., 1989; Grossman et al., 1991; Grossman & Kollai, 1993; Ritz, 2024), importantly due to respiratory-rate-related patterning of vagal influences upon heart rate (Grossman & Taylor, 2007). A part of these effects is also mediated through changes in venous return to the heart and the consequent direct stretch of the sinoatrial node (Perlini et al., 1995; Skytioti & Elstad, 2022), another part from arterial baroreceptors responding to fluctuations in blood pressure caused mechanically by the act of breathing.

Several other factors, both peripheral and central, additionally affect relations between central NA vagal outflow and RSA magnitude within-individuals. In fact, we know from animal studies that much of the respiratory modulation of heart rate (which defines RSA) is governed by two respiratory-control centers in the brainstem regulating inspiration and expiration (Farmer et al., 2016; Menuet et al., 2020; Buron et al., 2025). Recent studies, in which the first recordings have been made from the human vagus nerve, provide direct evidence of likely similar respiratory modulation of heart rate via rapidly conducting fibers in the vagus nerve (Ottaviani et al., 2020; Patros et al., 2022; Farmer et al., 2025). In animal experiments, disturbance of each respiratory-control center can lead to altered relationships between RSA and cardiac vagal tone (or parasympathetic control of heart rate)[3]. So to repeat, the central generation of RSA via cardiac vagal activity, thus, only reflects respiratory modulation of cardiac NA preganglionic neurons and cardiac post-ganglionic neurons (McAllen et al., 2011; see **figure 2** for definitions of ***preganglionic*** and ***postganglionic***), and does not represent all other phasic (i.e. non-respiratory) and tonic influences regulating cardiac vagal activity of the NA, the dorsal motor nucleus of the vagus (DMV) or intrinsically at the heart, itself (Rajendran et al., 2019).

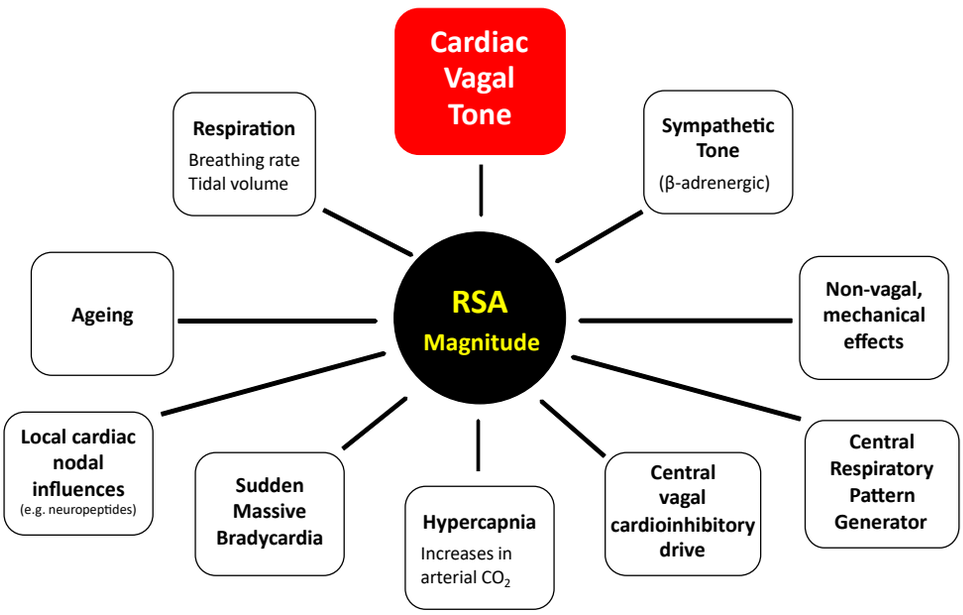
Sympathetic activity also influences RSA magnitude (e.g. Grossman & Kollai, 1993; Hedman et al., 1995; Taylor et al., 1998, 2001; Vaile et al., 1999; Billman, 2013; Fisher et al., 2022): These effects can be ***substantial***, and demonstrate that RSA is not a specific measure of cardiac vagal activity (ironically, a major cause – and hope – of initial enthusiasm in the estimation and application of RSA). Ageing is yet another confounding factor (Porta et al., 2014; Lakatta, 2025), which may be caused by brainstem neuro-degenerative mechanisms (e.g. gradual uncoupling of respiratory activity and cardiac vagal discharge) and structural changes (e.g. changes in vascular stiffness). The local intrinsic nervous system of the heart, itself, (composed of intracardiac neurons) is potentially still another potently influencing factor that can confound the relationship between RSA and cardiac vagal tone (Rajendran et al., 2019; Gee et al., 2023). There are others, too (see **figure 1**).

RSA, therefore, does not provide a direct or consistently reliable index of cardiac vagal tone, either as a state or trait measure. No specific procedure for estimation of RSA obviates this conclusion (see Grossman et al., 1990; Grossman, 1992; Quigley et al., 2024; Grossman & Sinichi, 2025). Although RSA is sometimes a more sensitive within-individual index (i.e. state, rather than trait), various constraints can seriously alter the correlation between RSA and cardiac vagal tone under many conditions (see Grossman, 2024; Menuet et al., 2025: **figure 1**). Additionally relevant to this discussion, a recent expert-evaluation/consensus-statement, among specialists in the field, points out common misconceptions and measurement problems regarding RSA (Menuet et al., 2025). These have broad impact, not only upon PVT, but also upon the entire area of research into relations between the vagus nerve and psychological functioning.

Collectively, then, there is hardly basis for polyvagal assertions, which tie RSA amplitude to a special physiological repurposing of the NA to (p. 172) *"endow mammals with the capacity for rapid cardiac modulation--a prerequisite for adaptive, co-regulated social behavior and behavioral flexibility."* Additional inferences about psychological functioning, derived from the mistaken notion that RSA directly measures cardiac vagal tone or

[3] Farmer et al. (2016) showed that cardiac vagal tone persists in the absence of RSA upon abolishment of a brainstem post-inspiratory center, i.e. they provide evidence for the existence of centrally derived, NA-mediated tonic cardiac vagal drive that is non-oscillatory. RSA 'approximates the amplitude of oscillatory (respiratory-phase related) variations in cardiac vagal activity about heart rate's mean level, rather than the total magnitude of cardiac vagal activity' (Menuet et al., 2025).

Figure 1. *Diverse constraints and influences upon respiratory sinus arrhythmia (RSA) when employing RSA magnitude as an index of cardiac vagal tone*



This list of constraints and influences is not meant to imply conceptual relationships among them, merely that each has been individually demonstrated to relate to RSA magnitude, independently of cardiac vagal tone. Cardiac vagal tone is the extent of average parasympathetic impact specifically upon heart rate during a specified period of measurement and is also called parasympathetic control of heart rate (Katona & Jih, 1975). Central cardioinhibitory vagal drive refers to the extent of vagal output from the brainstem (the Nucleus Ambiguus) specifically targeted to the heart to affect heart rate; cardioinhibitory refers to those parasympathetic brainstem neurons whose activation typically contribute to the slowing of heart rate. Local cardiac nodal influences refer to potential local cardiac events that occur at the sinoatrial node and/or other areas of the heart that are responsive to cardiac vagal efferent discharge and may interact with neuropeptides or other substances in the immediate periphery, which may inhibit or allow transmission of vagal traffic (see Gee et al., 2023). There is a broad system of intrinsic cardiac ganglia at the heart that are responsive to vagal stimulation (acetylcholine and other released substances) but interact with neuropeptides (often released by concurrent sympathetic stimulation) to determine final effects upon heart rate and other cardiac functions: neuropeptides are protein-rich compounds produced and released by neurons that can act as neurotransmitters or hormones to influence diverse bodily functions (https://tinyurl.com/ytxnu6v5). The other terms should be self-evident in terms of their definitions (for details, see Grossman 2024; adapted from Grossman, 2024)

even "general vagal tone" are without evidence. Importantly, neither "general" vagal tone related to states or traits, nor mass sympathetic discharge during particular conditions, are credible concepts given what is known about the specific regional control of organs by the autonomic nervous system necessary to maintain homeostasis (Jänig & Häbler, 2000; Jänig, 2022).

Additional putative indices of vagal functioning derived from RSA measurement, "vagal efficiency," and weighted coherence, are mentioned in the article we comment upon (p. 179). These indices are, as well, without scientific justification since they are based on false assumptions: The claim (p. 173) is made that weighted coherence--the normalized coherence between the respiratory and heart period signals at the time-varying respiratory frequency--"*specifically quantifies the efficiency of the central cardiopulmonary oscillator, integrating the pre-Bötzinger complex, nucleus tractus solitarius (NTS), and NA (Smith et al., 1991; Richter & Spyer, 1990, 2001; Mendelowitz, 1999)*." However, there is no supporting neurophysiological evidence nor mention of this measure in the latter references nor any basis with which we are familiar in the remaining scientific literature. Indeed, results from Porges (1986) contradict the assertion (p. 172) that "*weighted coherence remains stable*" across varying levels of cholinergic blockade: Weighted coherence, in fact, increased with blockade in that study.

So-called "vagal efficiency" is simply a measure derived from RSA and heart-rate measurement, based on the mistaken premise that RSA is an accurate measure of either central vagal outflow to the heart or cardiac vagal tone. It is thus also without scientific justification. The idea is self-refuting that vagal efficiency reflects central brainstem coordination immune to peripheral influences and can also show that "*vagal signals… are not producing expected downstream effects – indicating impaired central-peripheral integration*" (p. 179): Like weighted coherence, "vagal efficiency" is merely a calculation derived from two peripheral measures.

The "vagal paradox", often mentioned by Porges (e.g. 2023) to indicate that RSA sometimes correlates with changes in cardiac vagal tone and other times fails to, is not paradoxical at all when RSA is no longer equated with cardiac vagal tone, but when RSA is realistically considered as a vulnerable and approximate index of cardiac vagal tone, constrained, in its accuracy, by many factors. As Farmer et al. (2016) astutely stated: "Although RSA has been found to correlate with [cardiac] vagal tone, it is worth noting that the two measures are not identical and, as reported in the present study, may have different origins."

Important, finally, to mention, the arguments above do not imply that RSA magnitude bears no relationship to psychological functioning, since there is a large body of literature that documents associations, although

rarely strong in rigorous meta-analytic investigations (e.g. Graziano & Derefinko, 2013; Holzman & Bridgett 2017; Balzarotti et al., 2017; Beauchaine et al., 2019; Alen et al., 2022; Behnke et al., 2022; Wu et al., 2023; Z. Wang et al., 2025). However, such relationships, when they do exist, are likely to be mediated by many factors other than either cardiac vagal tone or the action of respiratory centers on cardiac vagal motor neurons. They require additional explanatory frameworks (see Grossman, 2024; Menuet et al., 2025).

Specious functional distinctions between vagal dorsal (Dorsal Motor Nucleus) and ventral (Nucleus Ambiguus) regions of the brainstem

Primary to PVT is the idea that human behavioral functioning to aversive and appetitive psychosocial stimuli depends upon a hierarchy of autonomic responses: First, calm and prosocial behavior is said to be parasympathetically mediated by the brainstem "ventral vagal complex" (most directly, the Nucleus Ambiguus [NA]). Second, PVT asserts that individuals experiencing severe stress mount sympathetic nervous system reactions of fight or flight. Third, in very dire or life-threatening circumstance, a defensive pattern of emotional freezing or psychological dissociation may occur, which is parasympathetically mediated by the Dorsal Motor Nucleus of the vagus nerve (DMV). Thus, this hypothesis posits a sequential ladder of autonomic responses. Certainly, this sequence of autonomic reactions is not grounded in evidence: the functional distinctions made in polyvagal proposals – between dorsal and ventral groups of neurons – are unsupported by the scientific evidence and are often contradicted, as explained below.

PVT (e.g. Porges, 2007, 2011, 2023, 2024) relies upon an implausible claim about the functioning of the DMV: Via unmyelinated (slowly conducting) efferent fibers, the DMV is primarily responsible for mediating massive heart-rate decelerations (bradycardia) under various conditions, often related to emotionally traumatic events, and termed "dorsal vagal shutdown" (p. 179; and Porges, 2007, 2025b). The actual evidence and scientific consensus are, however, that the ventral vagal nucleus NA is predominantly involved in mediating both small and large heart-rate reactions (McAllen & Spyer, 1976; Geis & Wurster, 1980; Jones et al., 1995; Y. Wang et al., 2000; Jones, 2001; Cheng et al., 2002, 2004; Machhada et al., 2015, 2016, 2020; Farmer et al., 2016; Gourine et al., 2016; Ottaviani & Macefield, 2022: Veerakumar et al., 2022). The DMV has limited influence upon heart-rate control in mammals such as rats, cats, dogs and sheep, with numerous studies showing only modest effects. For example, a recent study with rats showed that strong stimulation of the DMV neurons using an optogenetic approach had no significant effect on heart rate (Kellet et al., 2024). Another recent mouse investigation demonstrated small DMV-mediated heart rate decelerations of 50 bpm (equal to or less than a 10% reduction in heart rate), related to activation of oxytocin receptor-positive DMV neurons (X. Wang et al., 2025).

Noteworthy, however, is an additional well-controlled experiment with mice (Strain et al., 2024) that is at odds with these general findings and observed significant bradycardia to DMV stimulation, possibly due to genuine species differences between these mice and other animals. Also, as an important aside, DMV stimulation resulted in a decrease, rather than increase, in anxiety-like behavior--contrary to PVT assumptions about the dorsal vagus DMV effects upon socio-emotional behavior.

There are also other indications that, among rabbits, stimulation of the DMV elicits a potent bradycardia (Ellenberger et al. 1983). Such studies once more indicate the possibility of species differences regarding NA vs. DMV influences upon heart rate. Indeed, rabbits and possums are known to exhibit profound bradycardia during emotional freezing (Gabrielsen & Smith, 1985; Giannico et al., 2014). However, studies both of rabbits (Schwaber & Schneiderman, 1975) and of a large mammal, sheep, (Booth et al., 2021) show myelinated fibers originating in the DMV, contrary to PVT claims (p. 171). Furthermore, in dogs, stimulation of unmyelinated fibers, assumed by PVT to originate in the DMV, has only small effects upon heart rate, whereas stimulation of myelinated fibers produces bradycardia (Donald et al., 1967). We know of no experimental physiological evidence, which even vaguely suggests that the dorsal vagus or unmyelinated cardioinhibitory fibers in humans or other primates may be responsible for bradycardia during the stress-related immobilization of emotional freezing or dissociation.

Recent research, furthermore, indicates that profound bradycardia is neither common nor frequent among humans during emotional freezing or psychological dissociation (Beutler et al., 2022; Roelofs & Dayan, 2022; Danböck et al., 2024; Beutler-Traktovenko et al., 2025). Experimental and review findings reveal no characteristic massive heart-rate decreases during emotional freezing or dissociation: either no changes in heart rate are found or very small decreases that may be related to metabolically related reduction of activity during immobility.

Additionally, Neuhuber and Berthoud (2022) summarized evidence on involvement of the ventral NA in emotional freezing and other defensive responses and concluded that vagal responses to emotional freezing in mammals appear to be primarily mediated by the ventrally located NA, not the DMV. These vagal neuroanatomists conclude that the empirical literature *"does not support a role of the "dorsal vagal complex"..... Moreover, the term "dorsal vagal complex" denotes the unit formed by the medial NTS, DMV and area postrema controlling the gastrointestinal tract (Travagli & Anselmi, 2016) and should not be linked to passive defensive behavior in freezing as proposed by the PVT."* It is unclear why the current PVT paper and other recent ones neglect this and other lines of evidence.

In a nutshell, there clearly is no basis for the notions that the ventral vagal NA has evolved to support prosocial behavior in mammals, nor that the dorsal vagal area mediates defensive responses. The concept of "dorsal vagal shutdown" (pp. 174 & 176) in humans is, thus, completely without evidence. Additionally, experimental evidence indicates that vertebrate species have myelinated cardiac vagal neurons originating in each of the brainstem vagal nuclei, the NA and the DMV.

Evolutionary speculations regarding cardiac vagal control

PVT places much emphasis upon the evolution of the brainstem vagal center (dorsal and ventral). Therefore, a rebuttal of several PVT assertions regarding evolutionary trends of the vagus nerve is important, given the opposing, empirical state of knowledge.

To begin, PVT often asserts, as fact, that myelinated cardioinhibitory vagal fibers (efferent fibers that con-

tribute to heart-rate slowing) are found only in mammals and provide for rapid responses to environmental and social stimuli (since myelin speeds up the propagation of nerve impulses from brain to heart). Porges (p. 170, this journal) states: *Reptiles and birds "lack the myelinated cardioinhibitory vagal efferents that define mammalian autonomic flexibility and social engagement."* Also, Porges (p. 173): *"In mammals, myelinated cardioinhibitory fibers within the ventral vagal complex − rhythmically gated by medullary respiratory circuits − enable rapid, context-sensitive modulation of cardiac output (Porges, 1995, 2007, 2023; Mendelowitz, 1999; Neff et al., 2003). This innovation distinguishes mammals from reptiles, where vagal cardioinhibitory control is mediated primarily by unmyelinated fibers from the DMVX [dorsal motor nucleus], and where a coordinated cardiopulmonary oscillator is absent (Richter & Spyer, 1990; Porges, 2023). Consequently, respiratory–heart rate interactions, as a marker of central cardioinhibitory gating, are minimal or absent in reptiles. In these species, autonomic regulation is coupled primarily to meet the metabolic demands of movement and remains largely decoupled from social interaction (Porges, 2007, 2021; Liu et al., 2024)."*

It is incorrect that reptiles and birds lack myelinated, cardiac vagal efferents. Numerous studies have shown that the cardiac vagus nerve in fishes, amphibians, reptiles and birds includes myelinated efferent axons, able to induce near-instantaneous changes in heart rate (e.g. fish [Short et al., 1977; Taylor & Butler, 1982; Barrett & Taylor, 1985a,b,c]; lungfish [Monteiro et al., 2018]; amphibians [T. Wang et al., 1999]; reptiles [Sanchez, et al., 2019; Duran, et al., 2020]; birds [Schwaber & Cohen, 1978a & b; Abdalla & King, 1979; Lang & Levy, 1989]; overall [Taylor et al. 2014 and 2022]).

In addition, non-mammalian vertebrates have cardiac vagal preganglionic neurons located both in the DMV and ventro-laterally outside the DMV, which in mammals comprise the NA. Both groups provide vagal efferent fibres to the heart, which exert evolving levels of influence on heart rate, being able to generate respiration-related cardiac responses (RSA or other types of vertebrate respiration-related heart-rate variability). See Taylor et al., 2022 and **figure 2**. Already almost 50 years ago (Short et al., 1977; Taylor & Butler, 1982; Barrett & Taylor, 1985a, b & c), a series of studies commenced on the dogfish *Scyliorhinus canicula*, recently renamed the catshark, (representing a primitive group of fishes, lacking sympathetic innervation of the heart). These investigations have shown that the catshark heart is supplied with CNS-generated and respiration-related, vagal efferent activity from neurons located in the DMV, having myelinated axons with rapid conduction rates. This activity modulates heart rate, generating cardiorespiratory synchrony, and is possibly the most direct, closely coupled cardiac modulation recorded throughout the vertebrate groups, including the mammals. In contrast, for air-breathing vertebrates, from lungfish to mammals, inspiration-related increases in heart rate likely follow withdrawal of vagal inhibition, due to neuronal interactions in the CNS, possibly arising from neurons located in the NA (as described for mammalian RSA; see **figure 2**).

Taylor and colleagues have continued to expand upon these findings over the last half century (see references just above), and Taylor et al. (2022) summarized existing data that fully counter PVT claims (see **figure 2**). Porges (e.g. 2007 and 2011) referred to the work of Taylor and colleagues; however, over the last two decades, the PVT literature has continued to disregard consistent empirical evidence: Namely, cardiac vagal preganglionic neurons supplying myelinated efferent axons to the heart are found both in the DMV and in ventral vagal areas, outside the DMV, within the brainstem of all major groups of vertebrates from sharks to mammals**.**

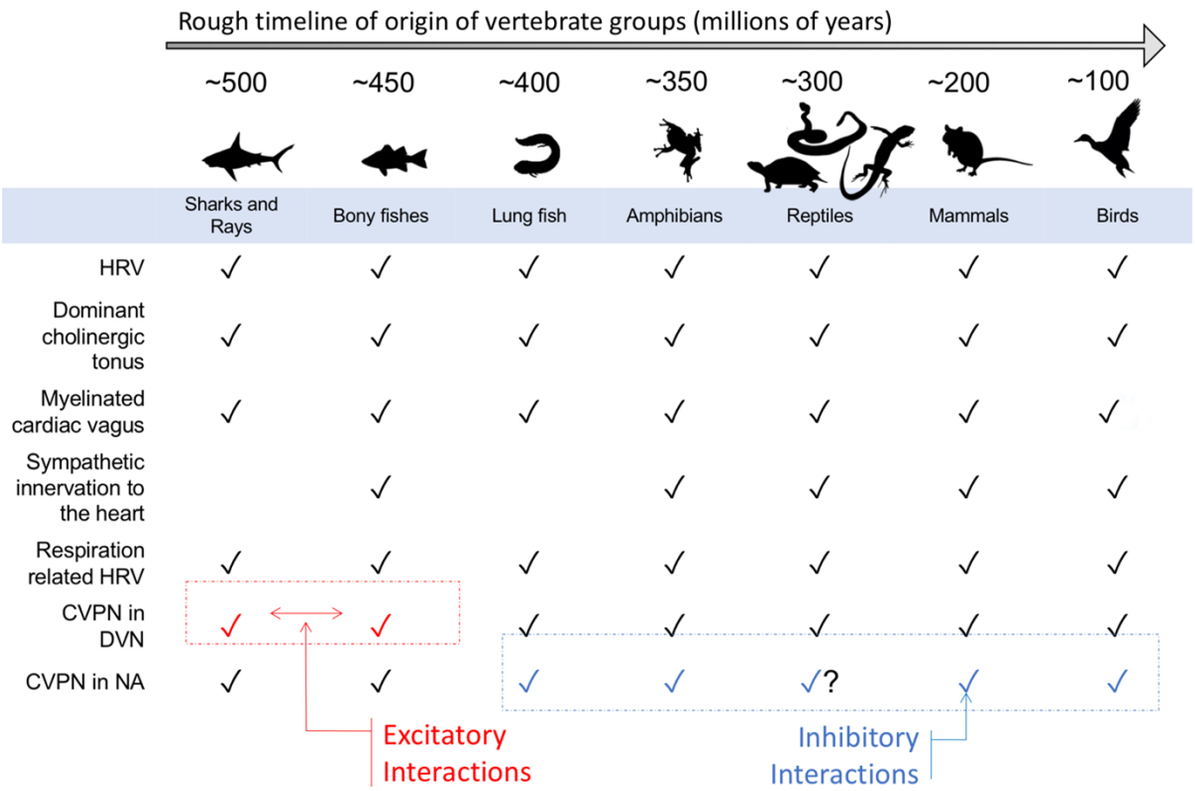
Contrary to Porges (quote above), respiratory heart-rate interactions are very much present among all groups of vertebrates. All known vertebrate species studied to date, from fish to mammals and birds, show forms of respiration-related heart-rate variability, which become, with the evolution of lung breathing, progressively closer to resembling, mammalian RSA (see **figure 2**; Taylor et al., 1999; Grossman & Taylor, 2007; Carravieri et al., 2016; Taylor et al., 2022). In consequence, we conclude that mammals inherited RSA from their vertebrate ancestors, having physiological attributes essential to the coordination of their various respiratory and cardiovascular systems, which likely serve to optimize respiratory gas exchange and cardiovascular functioning (e.g. Elstad et al., 2018; Grossman, 2024; Shanks et al., 2025). The most striking changes in type of respiration-related heart-rate variability between vertebrate groups are probably related to the necessary cardio-ventilatory adaptations of species evolving from aquatic to terrestrial habitats, not to putative "emergent" mammalian social behavior.

Furthermore, the reference to Richter & Spyer (1990), in the paper here under comment, does not exist in the literature, and the 2001 paper by the same title (Richter & Spyer, 2001) does not mention any aspect bearing on such evolutionary considerations; they focus solely upon the respiratory pattern generator of mammals. Thus, these authors nowhere deny the presence of a coordinated cardiorespiratory rhythm generator in nonmammalian vertebrates. To the contrary, another early paper by Richter, Spyer and colleagues (Richter et al., 1991), in fact, attempts "*to indicate how during phylogeny, biological organisms have developed an optimized common cardiorespiratory network that controls a single cardiorespiratory center.*" They state: "*In all animal species, respiratory motor movements are controlled by nervous substrates in the supraspinal brain (Babak 1913; Heymans and Heymans 1927; Ishihara 1907)."* This shows that Richter and Spyer, in 1991, actually endorsed a model of centralized brainstem networks, which exists across vertebrate classes, not only mammals, for integrating respiratory and cardiovascular control**.** Three reviews of the literature (Grossman & Taylor, 2007; Taylor et al., 2014, 2022) have concluded, based on experimental evidence, that the PVT proposition that centrally controlled, cardiorespiratory coupling is restricted to mammals can now be refuted.

Elsewhere in his paper, Porges endorses a literal application of the biogenetic hypothesis of Ernst Haeckel (1866), which proposed that ontogeny recapitulates phylogeny. Porges writes (p. 170): *"evolutionary transition is recapitulated during embryological development, where cardioinhibitory neurons migrate ventrally from the DMVX to the NAmb. This ontogenetic trajectory mirrors the phylogenetic transformation that gave rise to the mammalian VVC, reinforcing the idea that developmental processes echo evolutionary adaptations."*

Although Porges treats this notion as fact, the biogenetic hypothesis has long been soundly dismissed on a number of empirical grounds, importantly including falsification by means of detailed comparative embryological research and mismatching embryonic and adult stages of development (e.g. Garstang, 1922; de Beer, 1951; Gould, 1977; Richards, 2019; Martynov et al., 2022). The assumption that ontology literally recapitu-

Figure 2. *Neuroanatomical and physiological factors involved in determining types of respiration-related heart-rate variability (HRV) in vertebrates*



Respiration-related HRV are likely generated by the activation of cardiac vagal preganglionic neurons (CVPN) in the DMV in water-breathing vertebrates (all fish and amphibian tadpoles); preganglionic neurons originate in the DMV (and NA) and are attached to preganglionic axons descending toward the body; postganglionic fibers from the synapses of preganglionic fibers are distributed at different areas of the heart. Lung-breathing vertebrates (lungfish, adult amphibians, reptiles, birds and mammals) show RSA-like HRV, possibly generated by neurons located in the NA, as clearly demonstrated in mammals. All vertebrate groups have species shown to possess: 1) a tonic cardiac inhibition via the parasympathetic vagus, 2) CVPN in 2 or more locations in the brainstem, plus 3) myelinated axons in the cardiac efferent supply, providing rapid conduction rates. Dotted lines, putative location of central nervous interaction for respiratory-related HRV. Red dotted lines and checks (……√√), groups in which experimental evidence of excitatory neuronal interactions between respiratory neurons and CVPN in the DMV; blue dotted lines and checks (……√√), groups in which there is support for the existence of inhibitory neuronal interactions between respiratory neurons and CVPN in the NA, as shown in mammals. Respiratory sinus arrhythmia (RSA) is a type of respiration-related HRV present in all lung breathing vertebrates; ? indicates points under current investigation (adapted from Taylor et al., 2022).

lates phylogeny is central to the polyvagal edifice, since Porges also writes (p. 170): *"Crucially, this neuroanatomical integration underlies two defining mammalian features: the coordination of suck–swallow–breathe patterns that enable nursing, and the production of vocal prosody……This shared circuitry reflects a functional continuity between early-life feeding behaviors and later-emerging social communication."*

The assertion that species ontogenetic development literally recapitulates the evolutionary course of mammals is an erroneous application of Haeckel's claims (Burghardt, 2005) and cannot support the notion of the repurposing of the brainstem ventral vagus to support mammalian social behavior.

Prosocial behavior in vertebrates

The PVT proposes that evolution of the vagus nerve is closely tied to evolution of social behavior in mammals in a particular manner. Porges states (p. 170): *"While reptiles and birds retain a brainstem region identified as the NA, they lack the myelinated cardioinhibitory vagal efferents that define mammalian autonomic flexibility and social engagement."* In his table 2 (p. 171) and earlier publications, Porges asserts that reptiles lack any integrated prosocial behavioral system, are primarily defensive, and are absent of any behavioral flexibility in contextual autonomic regulation, social engagement, or discernment of safety *vs*. danger. As the bottom line, PVT claims that reptiles and other non-mammalian vertebrates lack sociality, primarily due to the absence of rapidly responding, myelinated vagal fibers that permit flexible dynamic interactions with their social and physical environment. In contrast, according to PVT assertions, mammals do show substantial prosocial behavior facilitated by the myelinated group of ventral neurons, which mediate prosocial behavior and rapid autonomic responses to varying situational contexts, socio-emotional or otherwise.

We have already presented substantial counter-evidence that myelinated vagal efferent fibers are common across the range of vertebrate classes, are often cardioinhibitory and frequently originate in ventral regions of the brainstem. So those findings would be sufficient, in themselves, for contesting the PVT assertions. How-

ever, it seems pertinent also to clarify the mischaracterizations of non-mammalian and mammalian sociality as propounded by the PVT.

Firstly, although reptiles and other non-mammalian vertebrate species, of course, neither communicate with language nor significantly express emotions via facial expressions, as humans do, nonmammalian vertebrates do use postures, gestures, sounds, chemosensory stimuli and other behaviors to express emotions and to communicate (Burghardt, 1977; Doody et al., 2021). Furthermore, many nonmammalian vertebrates show significant levels of social behavior that overlap with many mammals, including (depending upon species) long-term pair-bonding, monogamy, mate guarding, extended and/or communal parental care sometimes including nursing the offspring, sexual selection, complex courtship, social proximity seeking, shoaling and schooling and other social affiliative behaviors, communal nesting, prosocial choices, food sharing, social learning, developing theory of mind, reciprocal helping, eavesdropping, cooperative hunting, territoriality, dominance hierarchies, group vigilance, social-stress buffering, signaling and posturing (e.g. Doody et al., 2013, 2021, 2023; Keefner, 2016; Halliwell et al., 2017, Delmé et al., 2023). Secondly, quick-reacting, myelinated vagal efferents among these vertebrates, as previously documented, also allow for differentiated and dynamic autonomic responses to various stimuli, socio-emotional or otherwise (see previous discussion). Additionally, it might be mentioned that many insects completely lack vagal nervous system structures but, nevertheless, exhibit highly developed forms of social behavior, despite very small brain volumes (Muscedere et al., 2014).

All in all, then, after 30 years of PVT assertions, there remains no evidence of an emergent repurposing of brainstem ventral vagal regions to facilitate social affiliative responses. The brainstem ventral vagal region appears to contribute to affiliative, as well as to defensive and other socio-emotional heart-rate responses.

Citation and reference issues

The article under comment includes 47 references, 37 authored by S.W. Porges or colleagues mostly with whom he has previously published (all in the psychological or psychophysiological domain). The remaining 10 articles are in the field of physiology, none of which directly support any of the major premises of PVT enumerated in this or other publications (e.g. Porges, 2023 or Porges 2025b; also see below). Half of the physiological articles are inaccurately cited, both with respect to authorship and journal source. Perhaps more importantly, citations of some of those and other articles inaccurately suggest support of that previous research in favor of PVT assertions. In addition to the citation problems previously discussed regarding papers by Richter and Spyer, we provide further examples below:

Porges (p. 173) writes***: "****Weighted coherence specifically quantifies the efficiency of the central cardiopulmonary oscillator, integrating the pre-Bötzinger complex, nucleus tractus solitarius (NTS), and NAmb (Smith et al., 1991; Richter & Spyer, 1990, 2001; Mendelowitz, 1999).*" However, nowhere in any of these references do any of these articles provide any support that the method of weighted coherence can be used to quantify "*the efficiency of the central cardiopulmonary oscillator.*"

Also contrary to the statement, "*while RSA amplitude declines under vagal blockade, weighted coherence remains stable (Porges, 1986)*", the actual weighted-coherence results of that 1986 paper indicated a clear linear increase as vagal tone withdrew during blockade. Obviously, this does not strengthen the current argument (Porges, 2025a, p. 172) that weighted coherence represents *"a central rather than a peripheral origin for respiratory–heart rate coupling.*"

Several other citations and references are also incorrect, either citing the wrong journal attribution (Neff et al. 2003); all the wrong authors (Cao et al., 2019: should be Coverdell et al., 2019), or all the wrong authors and incomplete title (Liu et al., 2024: should be Hornung et al., 2024). Additionally, regarding the latter two studies, they were transcriptomic investigations focused upon the expression of DNA in neurons of DMV and the NA solely of mice and rats, not any reptiles. Contrary to the implications of Porges's (p.170) statement, the studies were not at all intended to *"delineate the unique features of the mammalian autonomic and social engagement systems when compared to those of reptiles."*

Porges (p .173) also states: *"Methodological advances such as weighted coherence challenge approaches advocated by Grossman and Taylor (2007), who argue for statistical correction of RSA for respiratory variables under the erroneous assumption that RSA is a mechanical artifact of breathing."* Nowhere do Grossman & Taylor (2007) suggest that *"RSA is a mechanical artifact of breathing"* but continually emphasize the central coordination of ventilation and cardiac activity. Their argument regarding statistical or experimental control of respiratory variable pertains merely to the employment of RSA as index of within-individual changes in cardiac vagal tone: Numerous studies have documented this issue (e.g. Saul et al., 1989; Grossman & Kollai, 1993; Grossman & Taylor, 2007; Quigley et al., 2024; Menuet et al., 2025), which does not necessarily have direct impact upon the issue of central vs. peripheral influences on RSA.

The repeated claims, furthermore, that Grossman & Taylor (2007) and other Grossman publications do not recognize the predominant CNS role in cardiopulmonary coordination are contradicted by decades of their own research. Grossman & Taylor (2007) states: "*covariation between RSA magnitude and respiratory parameters during alert states inherently reflects the interaction of cardiovascular control mechanisms and higher central nervous system behavioral control of breathing."* Most recently Grossman (2024) writes: *"Brainstem areas, central to generation of the breathing pattern, have been shown to modulate heart rate and drive RSA…..*" Of course, peripheral influences have also long been documented to have influences upon RSA (see Grossman, 2024). The various studies (e.g. those of Neff et al., 2003; Richter & Spyer, 2001; Mendelowitz, 1999) mentioned in the Porges article, indeed, focus upon central respiratory pattern-modulated changes in heart rate (i.e. RSA) but definitely do not assume that magnitude, or other parameters, of RSA cannot be influenced by peripheral reflexes, nor imply, as Porges (p. 102) claims, that RSA is *"a central, rather than peripheral, biomarker of autonomic regulation."*

Porges (p. 173) states: "*Despite calls by Grossman for statistical correction of RSA for respiration (e.g., Grossman & Taylor, 2007) their arguments fail to acknowledge the evolutionary distinctiveness and the central, brainstem-mediated mechanism of RSA (Richter & Spyer, 1990, 2001; Porges, 2023, 2024)."* It is unclear to us how statistical control for respiration (when employing RSA as an index of cardiac vagal tone) bears upon evolutionary considerations or the concept of cen-

tral cardiorespiratory pattern generator, nor does the article here under discussion elucidate this argument. Furthermore, Richter & Spyer, neither in 2001 nor 1991, address this issue at all.

Conclusions

Our commentary and the following conclusions pertain not only to the current review paper but also to two other recent similar articles (Porges, 2023 and Porges, 2025b), as well as to the entire PVT literature.

1. Functional distinctions between brainstem dorsal and ventral groups of vagal preganglionic neurons in mammals postulated by PVT are clearly wrong. The brainstem Dorsal Motor Nucleus has not been shown to mediate vagal cardioinhibitory responses to emotional freezing or dissociation in humans. The ventral vagal Nucleus Ambiguus, however, is involved in defensive reactions during emotional freezing, at least in other mammals. Therefore, the so-called "ladder of autonomic responses" is contradicted by existing evidence.
2. Much evidence indicates that the ventral vagal Nucleus Ambiguus of mammals is primarily responsible for mediating vagal heart-rate changes, whether it be the normal beat-to-beat control of heart rate, or abrupt and large heart-rate slowing (i.e. bradycardia). The Dorsal Motor Nucleus typically contributes modestly to vagally mediated heart-rate change, or in some mammal species, perhaps, not at all, and has rarely been shown to induce abrupt bradycardia. Also, myelinated fibers originating in the DMV may be responsible for bradycardia when it occurs.
3. Based on existing scientific literature, neither emotional freezing nor psychological dissociation among humans typically induce "massive" or "lethal" slowing of heart rate. Taken together with the first two points, the notion of "dorsal vagal shutdown" is wholly untenable.
4. RSA, the only quasi-vagal phenomenon upon which PVT relies, is neither a direct nor very accurate index of vagal outflow from brain to heart, nor of cardiac vagal tone, nor of "general vagal activity" (the latter, a currently discredited notion). The paradoxical nature of the so-called "vagal paradox" (Porges, 2023), as well as claims about additional RSA-derived measures (e.g. so-called "vagal efficiency"), are fully negated by the fact that RSA is not an accurate, direct measure of CNS-generated cardiac vagal activity.
5. RSA only sometimes correlates with magnitude of parasympathetic, or vagal, effects upon mean heart rate. Correlations between individual differences in cardiac vagal tone and RSA magnitude are often particularly weak.
6. Contrary to PVT, non-mammalian vertebrates, such as reptiles, amphibians and birds, also exhibit complex social behaviors, including both positive and negative emotional regulation, and are not merely defensively oriented.
7. Contrary to PVT, non-mammalian vertebrates also possess myelinated vagal fibers that have high conduction velocities and enable rapid and dynamic autonomic responses, via vagal efferent cardioinhibitory neurons, which serve various purposes – behavioral, socio-emotional or otherwise.
8. PVT assertions about central vagal control of the heart are neuroanatomically misinformed and often fictitious.
9. The PVT assumptions about the evolution of vagal control of heart rate are overwhelmingly in contradiction to past and current comparative physiological and neuroanatomical evidence.
10. What remains of PVT, as superstructure, are psychological concepts, almost all, if not all, predating PVT by many decades (e.g. psychological safety, social engagement, co-regulation, emotion regulation, emotional freezing, dissociation) and principally derived from early attachment theory, non-directive psychotherapy, trauma research and, most recently, ancient Eastern practices (mindfulness and yoga). Psychological and body-mind therapeutic methods have very long been in existence and often may confer benefits on their own. However, they relate to physiology differently than proposed by PVT. These psychological concepts and associated practices are not enhanced by erroneous PVT assertions.
11. In sum, PVT proposes a line of argumentation that ignores the overwhelming scientific consensus. The proposal to use an inaccurate framework and unproven measures to guide treatment of diverse vulnerable groups in society is neither scientifically valid nor ethically acceptable. The PVT framework promotes mistaken ideas about how the human mind and nervous system function together and introduces new *mental* fictions and fantasies about the mind-body relationship, thus contributing to greater, rather than less, distance between lived experience of psychological states and perceptions of bodily functions. Furthermore, broad influence of this scientifically inaccurate theory is likely to hinder the development, integration and true understanding of valid neuroscience and physiology research in relation to psychology and the practice of psychotherapy.
12. Beyond the scope of this article but of great relevance: psychologists, psychiatrists, psychotherapists and allied professionals currently influenced by PVT would do well to reorient and consider other already existing, as well as novel, psychophysiological explanations that are in line with modern conceptions and evidence regarding autonomic regulation of bodily functions. Abundant research and theoretical formulations do exist to address current knowledge of autonomic efferent and afferent regulation in relation to psychological processes.

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