Polyvagal Theory: Summary, Premises & Current Status

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SUMMARY

Autonomic state as a neural platform. Polyvagal Theory (PVT) conceptualizes autonomic state as a neural platform influencing behavioral, physiological, and psychological responses. Rather than assuming a cause-and-effect or stimulus-response model that assumes a psychophysiological parallelism (see Porges, 2022), the theory proposes that autonomic state functions as an **intervening variable** mediating the response.

Hierarchy of autonomic states: An emphasis on two vagal pathways. The model can be conceptualized as a stimulus-organism-response (S-O-R) model in which autonomic state is expressed and experienced along a continuum from fear-related immobilization involving dorsal vagal mechanisms, to fight-flight mobilization involving sympathetic mechanisms, and finally to a calm socially accessible state involving ventral vagal mechanisms. This sequence is **hierarchical**, with the latter state functionally having the capacity to co-opt the other states to enable hybrid states of mobilization without fear (play, dance) and immobilization without fear (shared moments of intimacy). From an evolutionary perspective, the sequence is a **hierarchical** representation of the evolutionary history of the vertebrate ANS (Autonomic Nervous System) as it became encoded in the ANS of humans and other social mammals.

The newest circuit, dependent on the ventral vagal complex, is the product of a **ventral migration** of cardioinhibitory neurons in the brainstem to the ventral nucleus of the vagus from the dorsal nucleus of the vagus. This ventral migration appears to have been completed in the earliest mammals as a defining characteristic of their transition from ancient extinct reptiles about 220 million years ago. The integration of cardioinhibitory neurons into the **ventral vagal complex** provided a circuit that integrated suck-swallow-vocalize-breathe processes with a newer mammalian, myelinated cardioinhibitory ventral vagal pathway that is expressed as RSA (Respiratory Sinus Arrhythmia) in the heart rate pattern. This circuit, which initially links ingestion through nursing with behavioral calming, provides the basic structures that enable co-regulation and connectedness through the lifespan.

According to the theory, survival challenges trigger a process of **dissolution** (or evolution in reverse) that disinhibits the phylogenetically older defense circuits of fight/flight or freeze/collapse. Dissolution disrupts homeostatic functions and predisposes visceral organs to disease. Within the PVT model, disruption of homeostatic functions operationally defines a physiological state of stress and psychological states of anxiety and threat. In contrast, when the ANS successfully supports homeostatic function, feelings of safety and opportunities to co-regulate and connect are spontaneously emergent. PVT is an integrative model emphasizing brainstem regulation of the ANS. PVT does not preclude the important influences of both bottom-up signals, through interoception, or top-down influences, via memories, visualizations, or associations, on these regulatory circuits.

Retrospective context. When initially formulated (Porges, 1995), PVT was conservatively organized upon several *premises* or inferences extracted from the literature. These premises were plausible explanations of important phenomena observed in psychophysiology and in perinatology for which the neurophysiological mechanisms had not been identified. By proposing these premises, the scientific community could confirm or refine these inferences through more in-depth exploration of the published literature and empirical research. The premises of PVT provided a new framing of questions that tied the neuroanatomy and neurophysiology of the ANS to clinical conditions and psychophysiological processes.

PVT focused on a plausible explanation of the 'vagal paradox' in two disparate disciplines, perinatology and psychophysiology. Functionally how could the vagus be the pathway for both RSA and bradycardia? The publication of the premises functionally framed the scientific questions for subsequent empirical research to evaluate specific clinical conditions and psychophysiological processes in which this paradox was observable. By proposing plausible relationships and identifying the specific metrics to map ventral vagal (i.e., RSA) and dorsal vagal (i.e., bradycardia) function, the research in these disciplines could incorporate a deeper neurophysiological understanding of the mechanisms underlying these observations. With this new perspective, it was optimistically hoped that vulnerabilities could be monitored to improve clinical outcomes and predict behavior.

As a heuristic exercise we can evaluate how these premises fit with the documentation summarized above. However, first it is useful to ask: 1) How has PVT been accepted within the scientific community? 2) Is there a need to revise the initial premises published in 1995?

The theory has been well received in Science. Googles Scholar documents, as of December 2022, that the foundational articles explaining and expanding the theory have been cited in more than 14,000 peer reviewed articles. Virtually all these articles are supportive of the theory or use the theory to support their hypotheses. In addition, from 1975-2013 peer reviewed research, competitively funded by the National Institutes of Health, continuously supported the development and testing of data contributing to PVT. These facts confirm the overwhelming support of peers within several scientific disciplines.

It is important to place the stated initial premises within the context of the science being conducted during the early 1990s. During this period, the theory was driven by the prominent research questions in the two disciplines in which I was working, developmental psychophysiology and perinatology. Within psychophysiology and especially in developmental psychophysiology, there was an interest in identifying in the preverbal infant the mechanisms mediating transitory heart rate changes, including those that occurred in response to changes in stimulation. These responses were often associated with orienting and were frequently labeled 'cardiac orienting.' (e.g., Graham & Clifton, 1966; Jackson et al., 1971). From the 1960s through the 1990s the investigations of the cardiac correlates or components of classical conditioning and orienting and defense responses were prominent (e.g., Clifton, 1974; Hare, 1972; Schneiderman et al., 1966). In fact, my studies in the early 1970s with newborn infants evaluated transitory heart rate responses in newborn infants as indices of attention, orienting,

and associative learning (i.e., Porges, 1974; Porges et al., 1974; Stamps & Porges, 1974). My research also investigated the relationship between heart rate variability, as a baseline individual difference, and transitory heart rate reactions.

In the early 1970s heart rate variables were treated phenomenologically and there was little interest in underlying neural mechanisms. In fact, although the sympathetic nervous system was frequently assumed to be the mediator of autonomic reactions such as heart rate, potential vagal mechanisms were rarely acknowledged. Observations of heart rate slowing in response to and in anticipation of stimulation were inconsistent with the prevalent views that autonomic reactivity was mediated primarily by the sympathetic nervous system. Although a more parsimonious explanation would be that the heart rate slowing was a product of the parasympathetic nervous system through the transitory changes in vagal efferent tone to the heart. However, the possibility of vagal pathways as a mechanism producing transitory heart rate responses was not a common perspective within psychophysiology.

Perhaps, interest in monitoring vagal function was slowed due to a lack of valid noninvasive indices or to a historical bias in psychophysiology. As a discipline psychophysiology had historically focused on measures of the sympathetic nervous system such as electrodermal (GSR) and vasomotor responses. Even in early studies of pupillary diameter, which is innervated by both branches of the ANS, the focus was not on parasympathetic mechanisms but on sympathetic excitation in explaining pupillary dilation (Hess & Polt, 1964). These biases persist (Wang et al., 2018).

In the 1960s arousal theory was the prevailing theory linking ANS to behavior (e.g., Malmo., 1959). It was basically a sympathetic-centric theory assuming a linearity among increasing SNS activation, mobilization, and brain activity (Darrow et al., 1942). This was followed by a more generalized sympathetic-adrenal model of stress that focused on glucocorticoids (Pfaff et al. 2008; Sapolsky et al., 2002). This view still permeates our language and implies that calming is due to a down regulation of SNS and adrenal hormones, while the PNS is frequently assumed not to play a major role in dampening the impact of the sympathetic-adrenal reactions. PVT forced a reconceptualization of the dynamic interplay between SNS and PNS and the potential role of the vagus as a modulator of more systemic defense systems. An inspection of the first 20 years of the journal, *Psychophysiology*, confirms that in contrast to the sympathetic nervous system there was a conspicuous paucity of information related to the parasympathetic nervous system and specifically the vagus. Similarly, until my research introducing HRV in the 1970s, which proposed that RSA could index vagal cardioinhibitory tone (e.g., Porges, 1976), psychophysiology had a strong sympathetic bias.

PVT introduced the possibility that the two vagal circuits could contribute to the distinctly different biobehaviorial roles. Although PVT provides a basis for hypotheses to be tested regarding the two vagal circuits, the ability to monitor the separate functions of the two vagal motor circuits continues to be difficult to study. Initially, I approached this question from a functional level as it related to infant survival and psychophysiological reactivity.

Subsequently, I looked to neurophysiology and neuroanatomy for tools to further explore and monitor these discrete systems. Unfortunately, physiologists and anatomists were not familiar with the vagal paradox and asked different questions and used different methodologies. When I ventured into the realm of neurophysiology with these questions, I was confronted with discipline-based limitations of methods and perspectives. The ability to asked questions about neural function were dependent on pharmacological blockade and surgery. Since the motor fibers of both the dorsal and ventral vagi are dependent on acetylcholine to communicate, selective blockade was not an option. Similarly, surgical manipulations tended to sever the entire nerve including both ventral and dorsal pathways as well as the abundant sensory pathways that populate the vagus.

I embraced these limited technologies and paradigms to explore vagal function from the perspective of physiology. In the 1980s I used selective pharmacological manipulations to effectively block total vagal efferent activity in rats, cats, and rabbits ((McCabe et al., 1984, 1985; Yongue et al., 1982) to study the neural contribution to heart rate variability focusing on RSA. In a quest to explore vagal regulation, I even collected beat-to-beat heart rate data during experiments in which brainstem nuclei related to vagal function were stimulated (see Porges, 1995, Figures 3 & 4, pp.307-308).

Invasive stimulation of brainstem nuclei has an additional confound, anesthesia. Since surgery is necessary to expose the vagus nerve or the source nuclei in the brainstem, the animal subject needs to be anesthetized. This led me to conduct research on the impact of inhalant anesthesia on the ANS. My research confirmed that a commonly used inhalant anesthesia in humans depressed RSA, virtually independent of heart rate, in studies conducted during a clinical procedure (Donchin et al., 1985). Initially, as PVT emphasized the link between consciousness and ventral vagal function, the ventral vagus was labeled the 'smart' vagus. This was later dropped, since it implied an 'executive' function, which was not the intention. The term was meant to highlight the autonomic state that might optimize higher brain functions, while the dorsal vagus initially labeled the 'vegetative' vagus, since it was involved in background homeostatic processes as well as being recruited in survival reactions.

In a search to learn about the vagus I also explored the anatomy literature. I discovered that anatomists had different biases and limitations, since their tools were dissection and histology, and focused on structure (rather than functional physiology). Although structure is important and provides insights into the connections between areas of the brain and visceral organs, it is agnostic about the actual functional dynamic recruitment of neural pathways in the regulation of organs. For example, it is possible that there are identifiable pathways from vagal source nuclei that are not recruited in the dynamic regulation of the heart or are only recruited during life threat situations. These are not the questions that can be answered by traditional anatomical methods.

Although I searched for tools and methods to confirm that function of two vagal nuclei on the heart, investigators in physiology and anatomy were limited by their disciplines' methods and research questions, which were not sufficient to conceptualize the selective functions of the

two vagal pathways. Thus, neurophysiology and neuroanatomy, both limited by their techniques, were of little help in refining a conceptualization of the function of the two vagal pathways.

By the mid 1980s (see Dellinger et al., 1987; Porges 1986) we confirmed that RSA was more sensitive to vagal blockade than the previously assumed 'gold standard' of heart rate change used by physiologists. More recently, we confirmed that our method was selectively more sensitive compared to several other methods (see Lewis et al 2012), which due to lack of sensitivity to ventral vagal function; methodological limitations have led to faulty inferences (see Grossman & Taylor, 2007). Thus, although the neural mechanisms contributing to heart rate variability were known in 1994, the neural mechanisms regulating the temporal features of transitory heart rate responses were at best speculative.

Within perinatology, prevailing clinical questions were and continue to be related to interpreting fetal and neonatal heart rate patterns to detect risk, predict outcomes, and to guide interventions to enhance survival and minimize brain damage due to hypoxia. Perinatologists are familiar with heart rate monitoring and are trained to interpret 'beat-to-beat' heart rate variability as an index of viability and the features (slope and magnitude) of bradycardia as risk indices of hypoxia. Interestingly, within perinatology, similar to psychophysiology, although these features were relevant to clinical practice, there was little discussion about the neural mechanisms underlying these phenomena. Thus, PVT was not and does not contradict existing theory in either discipline; both disciplines tended to be agnostic to the neural mechanisms mediating the heart rate response patterns that have been used to index clinical or psychological phenomena. Rather, PVT had added neurophysiological mechanisms as an additional layer to explain these relevant phenomena.

PREMISES OF PVT, DERIVED FROM THE SCIENTIFIC LITERATURE

As I developed the theory, I extracted principles from the literature that I summarized as premises (see below) upon which a theory could be established to generate testable hypotheses. From my perspective the premises were not controversial, but logically derived from the literature. The conceptualization of PVT required a 'transdisciplinary' approach, because the assumed foundational disciplines of clinical medicine and psychophysiology did not have the tools to conceptualize the questions generated by PVT. Since the premises were dependent on my interpretations of the literature, I welcomed alternative interpretations. Below are the five premises as presented in the first publication of the theory (Porges, 1995).

> **Premise 1:** Neurogenic bradycardia and RSA are mediated by different branches of the vagus and need not respond in concert.
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> **Premise 2**: Neurogenic bradycardia associated with orienting is a phylogenetic vestigial relic of the reptilian brain and is mediated by DMNX [dorsal vagal nucleus].

Premise 3: Withdrawal of cardiac vagal tone through NA [nucleus of the ventral vagus, nucleus ambiguus] mechanisms is a mammalian adaptation to select novelty in the environment while coping with the need to maintain metabolic output and continuous social communication.

Premise 4: The ability of NA to regulate special and general visceral efferents may be monitored by the amplitude of RSA.

Premise 5: Emotion, defined by shifts in the regulation of facial expressions and vocalizations, will produce changes in RSA and branchiomotor tone mediated by NA.

CURRENT STATUS OF PREMISES

Premise 1 and Premise 2 relate to the proposed mediation of neurogenic bradycardia and RSA being dependent on different vagal circuits with the ventral vagal nucleus regulating RSA and the dorsal vagal nucleus regulating neurogenic transitory bradycardia. The literature reviewed in previous papers outline the theory (e.g., Porges, 1995, 2007) provided conclusive evidence that in mammals, the two branches of the vagus are profound regulators of autonomic function relevant to adaptive biobehavioral reactions. A more recent review confirms these conclusions (see attached, Porges under review). Moreover, empirically the two indices are identifiable in the beat-to-beat heart rate pattern.

When first presented, the primary theoretical issues of PVT focused on the neural mechanisms underlying transitory bradycardia in neonatology and psychophysiology. The documentation of that mammalian species differ in their accessibility to dorsal vagal neurogenic bradycardia is now clearly mapped. Once an evolutionary hierarchy is incorporated into the model, then observing a depression in the ventral vagal cardiac influence (via RSA), contributes to an accurate prediction of the vulnerability of fetuses and high-risk infants to neurogenic bradycardia. Empirically, this was tested in risk infants (see above description of Reed et al., 1999). The study confirmed two important attributes of PVT: 1) It supported the foundational premise that the mechanisms underlying RSA and neurogenic bradycardia are mediated by different source nuclei of the vagus; 2) Consistent with the Jacksonian principle of dissolution, PVT supported that hypothesis that the neural regulation of the heart follows a response hierarchy in response to the life threat challenge of hypoxia. Once the 'protective' autonomic state characterized by ventral vagal regulation is depressed, the nervous system has efficient access to defense mechanisms; these are sequentially mediated by the sympathetic nervous system, that support mobilization, while the dorsal vagus can support neurogenic bradycardia to conserve metabolic resources via immobilization.

These points were stated in the first two premises. The documentation in the sections above firmly supports the premise that two different vagal mechanisms are responsible for transitory bradycardia and RSA. The third premise incorporates the value of awareness of dissolution in documenting the complex role of the ANS in reaction to challenge, which follows a predictable phylogenetically-order response hierarchy.

Premise 3 is supported by hundreds of publications across several laboratories documenting the reduction of cardiac vagal tone via a depression of RSA during metabolic demands and attentive challenges.

Premise 4 proposes that the role of the nucleus ambiguus in the ventral vagal complex enables RSA to index the status of the system, which was later labeled as the "social engagement system" (Porges, 1998).

Premise 5 further proposes that the ventral vagal complex is a nexus for the expression of features of emotion (e.g., vocal intonation, facial expressions), which in turn are also mirrored in RSA (see Porges, 1998; Porges et al., 1994).

The five premises are dependent on the following facts extracted from the scientific literature:

1. Transitory bradycardia and RSA, although functional outputs of the vagus, are conveyed through different vagal pathways. In general, transitory bradycardia is a survival reaction in response to threat cues. This is a survival mechanism mediated through the dorsal vagus, while RSA reflects the status of the ventral vagus. RSA reflects the functional support of homeostatic functions through ventral vagal pathways. During challenges and threats the two pathways may work synergistically, resulting in depressed RSA during episodes of bradycardia. This does not preclude the potential influence of both vagal pathways on tonic heart rate levels or transitory heart rate responses in safe contexts during states of optimal ventral vagal tone.
2. The three primary neural systems involved in autonomic regulation form a phylogenetically ordered response hierarchy, which is mirrored in development and responds in a predictable order when under survival challenge.
3. The ventral vagal nucleus, nucleus ambiguus, is part of an integrated ventral vagal complex controlling the striated muscles of the face and head via special visceral efferent pathways that are involved in sucking, swallowing, breathing, vocalizing, and listening. These structures are involved in nursing and develop into a social engagement system to detect and signal safety and threat to conspecifics. This system is involved in the adaptive expression of emotion.
4. The current widespread interest in noninvasive vagal nerve stimulation is revealing that stimulating the afferents of vagus, as well as afferent pathways of the facial and trigeminal nerves, will stimulate ventral vagal tone.

CURRENT STATUS OF PVT

The foundation of Polyvagal Theory (PVT) is based on the extraction of well accepted principles from the scientific literature. These included the well documented phylogenetic and ontogenetic sequence of brainstem structures regulating cardioinhibitory processes that form the basis of a response hierarchy consistent with the Jacksonian principle of dissolution. Given its strong scientific foundation, there has been little criticism of the theory in the scientific

literature. There have, however, been misrepresentations of the theory that have been used to argue that the theory is not scientifically supported. In general, the misrepresentations can be traced to two sources: publications by Edwin W. Taylor and colleagues, and social media posts by Taylor's colleague, Paul Grossman. Taylor's research has had a decades long commitment to understanding vagal function and especially respiratory-heart rate interactions from a comparative approach that has provided atheoretical descriptions of vagal circuits and functions in several vertebrate species. Unfortunately, in several papers he and his colleagues have blatantly misrepresented Polyvagal Theory. While Taylor has used peer reviewed publications as a vehicle to disseminate his misinterpretations, Grossman has become a social media influencer promoting and at times creatively elaborating and extending Taylors misrepresentations.

Grossman and Taylor have systematically structured a *straw man* argument based on misrepresentations of PVT to create the appearance of scientifically valid arguments. In Taylor's case he has used false attributions of PVT to highlight the importance of his findings. In contrast to Taylor, Grossman has not conducted research to generate data to falsify PVT. Instead, Grossman has elaborated on Taylor's misrepresentation to position himself in social media as the 'debunker' of PVT. Since the points of contention are not supported by facts, the work by Grossman and Taylor fails to challenge any of the tenets embedded in PVT. Rather than identifying points of disagreement, their *straw man* argument is dependent on faulty attributions of the theory and NOT on PVT. The net result of their efforts has been to sow confusion around the theory, especially among those not familiar with the foundational papers.

The above explanation of the theory and its supporting literature document the features of PVT that have been obfuscated by Grossman and Taylor. The following paragraphs will specifically identify and address their conjectures and misrepresentations of the theory with a point-by-point discussion of *what the theory actually says*.

1. **Misrepresentation of the scientific bases of PVT.** Grossman has used social media to promote his claim that there is no scientific evidence for Premises 1 and 2. Although Taylor's research is not relevant to PVT, Grossman frequently cites Taylor's publications to support his claims (Campbell et al., 2005, 2006; Monteiro et al., 2018; Sanches et al., 2019; Taylor et al., 2006, 2022). Interestingly, other than stating that the Premises are false, Grossman has provided no alternative interpretation of the literature. In addition, other than depending on Taylor's misrepresentations, he has not reported any documented contradictions between the literature and PVT. Unfortunately, others, who have assumed that his statements were scientifically sound, have quoted him and set off a cascade of misinformation in social media.

2. **Misrepresentation of the uniqueness of mammalian RSA in PVT**. Taylor and his group blur the well-documented distinctions between mammalian RSA and heart rate-respiratory interactions in other vertebrates. As emphasized in the theory and throughout this paper, mammalian RSA is dependent on the ventral vagus and the

functional output of 'myelinated' cardioinhibitory vagal fibers originating in the ventral vagus. In contrast, in other vertebrate species heart rate-respiratory interactions involve the dorsal vagal nucleus and communicate to the heart, in general, via unmyelinated fibers. As described in the sections above, there is scientific consensus that neuroanatomical structures and neurophysiological pathways involved in producing mammalian RSA are distinguishable from respiratory-heart rate interactions in other vertebrates. However, in building his argument, Taylor obfuscates this distinction and redefines RSA as being inclusive of all manifestations of respiratory-heart rate interactions observed in all vertebrates. With his definition of RSA, he argues that if species other than mammals express RSA (i.e., his definition of RSA as any form of heart rate-respiratory interaction) then PVT is false.

Taylor and his colleagues repeatedly press their inaccurate argument, since they have incorrectly assumed that heart rate-respiratory coupling being solely mammalian is a foundational principle of PVT. Following their logic, observations of heart rate-respiratory coupling in other vertebrate species would be inconsistent with the theory. Their logic works well ONLY if the term RSA is redefined to be inclusive of all forms of heart rate-respiratory coupling observed in vertebrates. Then, since PVT uses the construct of RSA, they could assume that any statement regarding the uniqueness of RSA as being mammalian would be false. However, their strategy misses two important points about RSA that relate to PVT: 1) the specific vagal pathways mediating RSA in mammals, unlike their ancestral vertebrates, originate in the ventral vagus, and 2) RSA is a portal to the function of the ventral vagus enabling the testing of polyvagal-informed hypotheses and is NOT a foundational construct of the theory.

3. **Misrepresentation of the role of myelinated vagal fibers in PVT**. Consistent with the scientific literature, PVT proposes that only mammals have a myelinated cardioinhibitory vagal pathway originating from the ventral vagus. The foundational PVT papers have consistently stated that these two features, *in combination* and not independently, reliably distinguish mammalian RSA from respiratory interactions observed in other vertebrate species. Even with these strong statements qualifying the neuroanatomical structures involved in mammalian RSA, Taylor and his colleagues have misrepresented PVT by stating incorrectly that PVT assumes that only mammals have a myelinated vagal pathway (Monteiro et al., 2018). Consistent with their 'straw man' argument, with this 'new' PVT attribution, Taylor and his group highlight their finding of a myelinated cardioinhibitory pathway originating in the dorsal vagal nucleus in the lungfish as falsifying PVT. They make their argument despite the fact that the cardioinhibitory vagal pathway in lungfish originates in the dorsal vagal nucleus. Taylor's statements misrepresent the theory's foundational papers that limit the neurophysiological origin of mammalian RSA to myelinated cardioinhibitory vagal pathways originating ONLY in the ventral vagus. He and his colleagues have repeatedly misrepresented the theory as stating that only mammals have myelinated cardioinhibitory pathways without qualifying their anatomical origin in the ventral vagus. In this manifestation of misrepresenting the theory, they have repurposed the

word myelinated from being associated ONLY in mammals with cardioinhibitory pathways originating in the ventral vagal nucleus to a general feature of cardiorespiratory interaction independent of nucleus of origin (i.e., either ventral or dorsal vagal nucleus).

4. **Taylor and colleagues have questioned the assumption that the dorsal vagal nucleus is an evolutionarily older structure than the ventral vagus.** The literature including Taylor's work (Taylor, 1999) has reliably documented in modern vertebrates representing groups of vertebrates, which evolved prior to mammals, that the prominent cardioinhibitory vagal neurons originated in the dorsal nucleus of the vagus. Thus, it is indisputable that estimating an evolutionary timeline through phylogeny, cardioinhibitory neurons originated first in the dorsal nucleus of the vagus and then consistent with Taylor's own work (2022) migrated ventrally. In the earliest (now extinct) mammals this ventral migration was sufficiently complete to embed cardioinhibitory functions with activities of branchiomotor neurons (i.e., special visceral efferent pathways) that regulate the striated muscles of the face and head promoting ingestion (e.g., nursing) and social communication via facial expression and vocalizations.

5. **RSA has historically been used to describe a mammalian heart rate rhythm.** It has a history of use that has been agnostic of the heart rate-respiratory interactions of other vertebrates. In fact, Taylor in his earlier papers (i.e., prior to 2000) uses the term RSA only when discussing mammals. Perhaps, Taylor's atheoretical agenda, to document that respiratory-heart rate interactions are a highly conserved phenomena across several vertebrate species, has contributed to his repeated, inaccurate statements regarding their underlying neural mechanisms. Although the phenomenon is highly conserved during evolution and even evidenced in mammals, the underlying mechanisms have been modified through evolution (e.g., Richter & Spyer, 1990). These points are emphasized in PVT. The foundation of PVT focuses on the structural and functional consequences of mammalian modifications of this highly conserved system. This point was unambiguously stated in the title of the paper introducing PVT (Porges, 1995) - *Orienting in a defensive world: Mammalian modifications of our evolutionary heritage. A polyvagal theory.*

   Taylor's generalization of common mechanisms underlying heart rate-respiration interactions across vertebrate species has its limitations. Evolution continues to repurpose and modify how the mammalian autonomic nervous system is both structured and functions. If we do not acknowledge the evolutionary repurposing of structures, we would be vulnerable to being criticized as accepting 'recapitulation' theory, i.e, a disproven theory that assumes evolution not only preserves structure, but also function.

CONCLUSION

The scientific method seeks to distinguish valid points from conjectures. Theories flourish only if they are useful in describing phenomena that can inform future investigations. Of course, theories must be modified and informed by empirical research and, when necessary, replaced by alternative theories that are more effective in explaining naturally occurring phenomena. If we use this as an acceptable standard, then PVT provides a testable model describing how the autonomic nervous system reacts to threat and safety. The theory specifically provides an understanding of the core features of the mammalian nervous system needed to co-regulate and trust others. It also provides insights into the consequences of autonomic state for mental and physical health. Perhaps, most importantly, the theory gives voice to the personal experiences of individuals who have experienced chronic threat (i.e., trauma and abuse) and structures an optimistic journey towards more optimal mental and physical health. It is this core, described by PVT, that links our biological imperative to connect with others to neural pathways that calm our autonomic nervous system. These systems, in the context of mammalian physiology, are foundational processes through which behavioral experiences can lead to sociality and optimal health, growth, and restoration.