UNIVERSITY OF PADOVA

Department of General Psychology

Bachelor's Degree Course in Psychological Science

Final dissertation

Trauma and the Body: The Role of the Autonomic Nervous System

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Academic Year 2025-2026

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Introduction

1. Trauma: Clinical and Psychobiological Relevance

Psychiatric disorders related to exposure to psychological trauma represent a major public health concern due to their high prevalence and substantial societal burden. Post-traumatic stress disorder (PTSD), in particular, affects a considerable proportion of the population, with prevalence rates varying according to the nature and chronicity of exposure, as well as individual factors such as age, sex/gender, socioeconomic and socio-political context (Olff et al., 2025). For example, among military members and veterans, estimates suggest that rates may reach up to 20% (Institute of Medicine, 2014), while the estimated global lifetime prevalence is 3.9% (Koenen et al., 2017, as cited in Göçen et al., 2024). Importantly, the impact of trauma extends beyond PTSD. As highlighted by the meta-analysis by Hogg et al. (2022), psychological trauma exposure is associated with nearly a threefold increased risk of developing a mental health disorder, including Major Depressive Disorder (MDD), Bipolar Disorder (BD), anxiety disorders, dissociative symptoms, Substance Use Disorders (SUD), Obsessive-Compulsive Disorders (OCD) and sleep disorders, underscoring the broad and transdiagnostic impact of traumatic experiences (Figure 1) (Hogg et al., 2022; Olff et al., 2025).

	Psychological trauma in childhood is a transdiagnostic risk factor for mental disorder across diagnoses and spectra (OR = 2.92; 95% CI 2.60, 3.28)						
Diagnoses	Anxiety Disorders (diagnostic group comprising Generalised Anxiety Disorder, Panic Disorder and Social Anxiety Disorder)	Bipolar Disorder	Major Depressive Disorder	Obsessive-Compulsive Disorder	Borderline Personality Disorder	Psychosis (diagnostic group comprising Psychotic Disorder, Schizophrenia and Schizoaffective Disorder)	Post-traumatic Stress Disorder
Spectra	Anxiety Disorders	Bipolar Disorders	Depressive Disorders	Obsessive-Compulsive Related Disorders	Personality Disorders	Schizophrenia Spectrum Disorders	Trauma- and Stressor-Related Disorders

Figure 1. Diagnosis and Spectra meeting TRANSD criteria for psychological trauma as a transdiagnostic risk factor (Source: Hogg et al., 2022).

This highlights the need to better understand the mechanisms linking trauma exposure to such a wide range of outcomes, thus raising an important question: why is psychological trauma associated with the development of such a wide range of mental disorders? The present thesis addresses this issue by focusing on a potential transdiagnostic mechanism, namely Autonomic Nervous System (ANS) dysregulation.

2. Trauma-Related Disorders and the Nosological Approach: Clinical challenges

Despite this transdiagnostic perspective, current diagnostic systems adopt a different approach. Indeed, trauma-related conditions are traditionally distinguished on the basis of symptomatology, leading to the categorization of disorders such as PTSD, Acute Stress Disorder, Adjustment Disorder, Complex PTSD (CPTSD) and others within our current diagnostic systems (e.g., DSM-5; ICD-11).

While this symptom-based approach is useful for identification and classification purposes, and acknowledges the impact symptoms have on the person affected by the disorder, growing concerns have been raised regarding its utility in fully capturing the underlying mechanisms of trauma-related psychopathology. Specifically, some researchers suggest that this artificial categorization system is the reason why high comorbidity among disorders is so prevalent, arguing that it is not true comorbidity, but that it may instead reflect shared transdiagnostic processes underlying multiple mental disorders (Hogg et al., 2022).

Indeed, the DSM-5 identifies alterations in arousal and reactivity as core symptom clusters of PTSD (APA, 2013, as cited in Siciliano et al., 2022), and evidence from psychobiological research suggests that trauma-related disorders may be more accurately understood as different manifestations of autonomic nervous system (ANS) dysregulation – a mechanism that plays a crucial role in shaping both biological and psychological responses to trauma (Siciliano et al., 2022). From this perspective, variations in symptomatology may reflect different patterns or degrees of autonomic dysregulation, rather than qualitatively distinct disorders.

3. Aims of the Thesis and Structure

The aim of the present thesis is to explore the role of autonomic nervous system regulation, dysregulation and flexibility in trauma-related disorders. In particular, this work will adopt the polyvagal framework as a theoretical model to interpret patterns of autonomic activation and their clinical implications. Although, as abovementioned, trauma exposure is associated with a broad spectrum of psychiatric conditions, the present thesis will primarily focus on PTSD, as it represents the most extensively studied trauma-related disorder in relation to autonomic dysregulation.

It is important to note that trauma is conceptualized as a significant, yet non-deterministic, risk factor for ANS dysregulation that interacts with genetic, biological, and environmental vulnerabilities.

Chapter 1 – Classical View of the Autonomic Nervous System

1. Sympathetic and Parasympathetic Branches: Mechanisms and Functions

For a long time, the functioning of the autonomic nervous system (ANS) was formally conceptualized according to the model proposed by John Langley (1903). In this model, ANS functioning relied on the balance between two branches: the sympathetic nervous system, responsible for "*fight or flight*" responses and for preparing the body to cope with immediate threat through neurochemical and musculoskeletal changes, and the parasympathetic nervous system, which mediates "*rest and digest*" or "*feed and breed*" functions, promoting recovery and homeostasis once the threat subsided and supporting processes such as digestion and tissue repair.

In this early framework, both medical and physiological research largely focused on the antagonistic efferent effects of these two branches on target organs, whereas afferent pathways and the role of brainstem areas regulating efferent pathways tended to be ignored (Porges, 2009), treating the ANS as a mere motor system (Porges, 2003).

Although the central role of the vagus nerve (i.e., the X cranial nerve) in mediating parasympathetic activity was recognized, this early conceptualization did not distinguish between its functionally distinct efferent pathways, with supra- and sub-diaphragmatic pathways not differentiated from each other (Porges, 2009).

2. Limitations of the Classical ANS Perspective: The Vagal Paradox

Porges (2009) highlighted a major inconsistency between empirical findings and the physiological assumption of the classical dual autonomic model, referring to it as the "*Vagal Paradox*". As above mentioned, according to the traditional ANS model vagal control was conceptualized as mediating parasympathetic activity through a single central vagal pathway associated primarily with states of calm, energy conservation, and restoration.

However, data showed that vagal activation was also implicated in profound cardioinhibitory responses and states of defensive immobilization, such as fainting or shutdown, associated with threat, chronic stress and potentially lethal conditions, such as hypoxia (Porges, 2023). This apparent contradiction, whereby vagal activity could be associated both with social engagement, anti-stress functions and with defensive collapse, and potentially death, challenged the notion of a unitary parasympathetic system (Porges, 2023).

In response to this paradox, Porges (2023) proposed that the vagus nerve comprises functionally distinct efferent pathways with different evolutionary origins, behavioral functions, and originating in two different anatomic regions of the brainstem. Specifically, he distinguished between the Ventral Vagal Complex (VVC), associated with social engagement and flexible regulation, and the Dorsal Vagal Complex (DVC), linked to immobilization and shutdown responses.

Chapter 2 – Polyvagal Theory and Autonomic Flexibility

1. Three Phylogenetic Response Systems

The Polyvagal Theory (Porges, 2001, 2003, 2007, 2009, 2022) proposed a hierarchical organization of the ANS in mammals grounded in phylogenetic development. According to this framework, three functionally distinct subsystems emerged sequentially over the course of vertebrate evolution (Figure 2).

	ANS Component	Behavioral Function	Lower motor neurons
III	Myelinated vagus *(ventral vagal complex)*	Social communication, self-soothing and calming, inhibit "arousal"	Nucleus ambiguus
II	Sympathetic-adrenal system	Mobilization (active avoidance)	Spinal cord
I	Unmeyelinated vagus *(dorsal vagal complex)*	Immobilization (death feigning, passive avoidance)	Dorsal motor nucleus of the vagus

Figure 2. Phylogenetic stages of the Polyvagal Theory (Source: Porges, 2007).

The most phylogenetically ancient subsystem is the Dorsal Vagal Complex (DVC), originating in the dorsal motor nucleus of the brainstem. This system is dependent on the unmyelinated or "vegetative" vagus, which primarily regulates subdiaphragmatic organs and is shared with most vertebrates. Under conditions of overwhelming threat, it mediates immobilization responses characterized by metabolic conservation, such as behavioral shutdown and withdrawal, dissociation, bradycardia and even thanatosis and vasovagal syncope.

The Sympathetic Nervous System (SNS), which evolved later, supports active mobilization strategies, such as *fight or flight*, enabling increased metabolic output and heightened vigilance in response to danger by increasing heart rate and sweat glands activity while inhibiting gastrointestinal activity (Porges, 2001).

The most recent subsystem, unique to mammals, is the Ventral Vagal Complex (VVC) originating in the brainstem, precisely in the nucleus ambiguus (NA). The VVC tends to preserve homeostasis and mediates the control of

supradiaphragmatic visceral organs, as well as of the heart. It integrates two closely intertwined components: a visceromotor component – involved in the regulation of visceral homeostasis – and a somatomotor component – which underlies social engagement functions.

The visceromotor function refers to the myelinated supradiaphragmatic vagal pathway that controls bronchi and heart, and that is responsible for the rapid modulation of cardiac activity (the "*vagal brake*"), as well as for the inhibitory influence on sympathetic activation and stress-related neuroendocrine responses, including the hypothalamic–pituitary–adrenal (HPA) axis (Porges, 2007). It has also been associated with anti-inflammatory pathways through vagally mediated immune modulation (Porges, 2007). This function is therefore associated with growth and restoration. The somatomotor function involves visceral efferent connections with cranial nerves controlling facial expression, vocalization, listening, and eye gaze (Porges, 2007).

Through evolutionary processes, the brainstem nuclei that regulate the myelinated vagus became neuroanatomically and neurophysiologically integrated with the visceral efferent nerves that regulate the muscles of the face and head (Porges, 2007), making the two components – visceromotor and somatomotor, or bodily states and social engagement behaviors – completely and bidirectionally linked to each other, forming an integrated Social Engagement System (SES) (Figure 3). Indeed, the visceromotor component of the VVC is involved in the rapid modulation of heart and bronchi, thereby providing the metabolic resources to engage and disengage in social contexts (Porges, 2001).

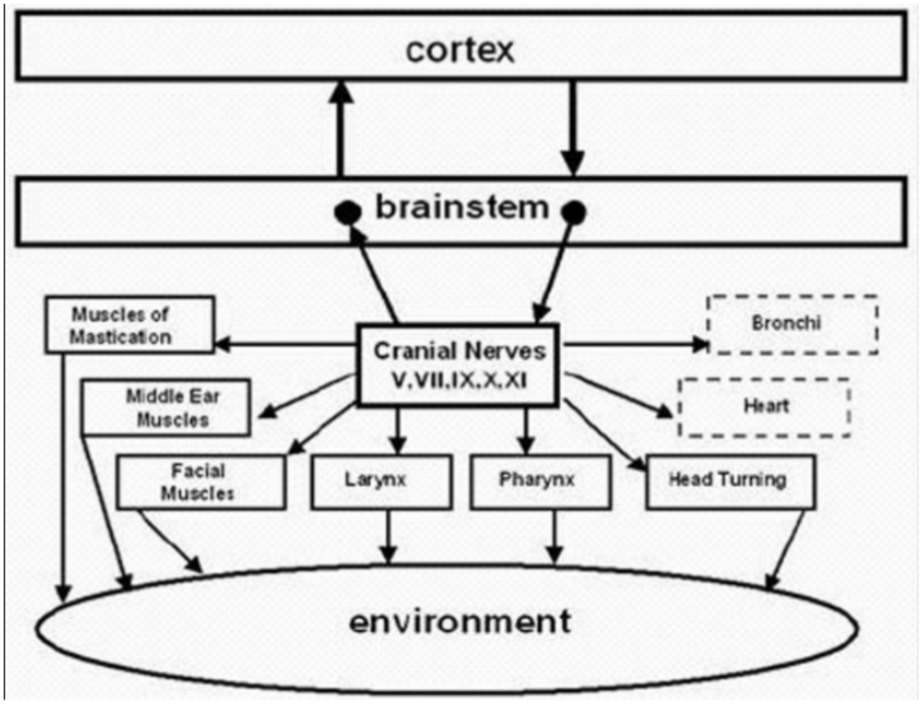

Figure 3. The Social Engagement System (Source: Porges, 2007).

Importantly, this hierarchical organization implies that autonomic responses to challenge are recruited sequentially (Porges, 2007): when social engagement strategies fail to ensure safety, sympathetic mobilization emerges; when mobilization proves ineffective or the threat is perceived as inescapable, dorsal vagal immobilization may dominate. Therefore, in response to challenge, our response strategy starts with the newest structure (VVC) and subsequently, if it fails to provide safety, it reverts to the most primitive systems (SNS, DVC). From an evolutionary perspective, this organization ensured adaptive flexibility, as the human nervous system evolved not only to survive in safe environments, but also in dangerous and life-threatening ones (Porges, 2007).

According to Polyvagal Theory, the selection of these autonomic responses is not solely driven by *top-down* conscious appraisal involving cortical areas (i.e., temporal cortex), but also by an automatic and implicit process, involving subcortical limbic structures (i.e., amygdala and PAG), referred to as *neuroception* (Porges, 2007). Neuroception was proposed as a plausible mechanism to describe the nervous system capacity to continuously evaluate environmental and internal (i.e., visceral) cues for safety, danger, or life threat, without conscious awareness. It also mediates both the expression and

disruption of not only positive social behavior, but also visceral homeostasis and emotional regulation (Porges, 2009).

Through this process, the autonomic nervous system rapidly shifts between states of social engagement, mobilization, or immobilization, depending on how the environment is perceived. If the environment is perceived as safe, the ANS inhibits the more primitive fight, flight or freeze behaviors, which are metabolically costly for the ANS and compromise homeostasis (Porges, 2022), whereas it activates the evolutionarily more advanced VVC. Instead, if the environment is perceived as threatening, disruption of homeostatic functions in the form of SNS or DVC activity occurs (Porges, 2022).

Notably, while most individuals can reliably evaluate whether there is actual risk in the environment, neuroception can become biased in individuals following traumatic experiences, leading to the detection of threat even in objectively safe contexts. (Porges, 2007). This mismatch results in physiological states mediated by the SNS and DVC even in safe situations.

Within this conceptualization, the individual's autonomic state shapes behavioral, cognitive, and physiological reactions to contextual cues, such that the same stimulus may elicit different responses depending on the prevailing autonomic state of the person (Porges, 2009).

Indeed, evidence suggests that following adversity and traumatic experiences the autonomic nervous system may undergo a process of retuning, resulting in increased sensitivity to stimuli directly or even indirectly related to the events (Corrigan et al., 2011). Consequently, traumatized individuals may become more easily triggered and locked into states of defense (i.e., sympathetic mobilization/hyperarousal state or dorsal immobilization/hypoarousal state; Porges, 2022) which, according to polyvagal theory, block the access to the calming pathways through the ventral vagus (Williamson et al., 2013, 2015; Kolacz et al., 2020, as cited in Porges, 2022) and are therefore incompatible with social engagement behaviors (Porges, 2009). This autonomic dysregulation results in a reduction in autonomic flexibility and in a variety of symptoms depending on which defensive state the person is.

2. The Role of Autonomic Flexibility and Regulation

Before delving into the specificity of trauma-related disorders, it is important to highlight the role of autonomic flexibility as a key indicator of general psychophysiological health, as well as its underlying psychophysiological indices. A growing body of literature emphasizes the importance of autonomic regulation and flexibility in both maintaining and assessing psychophysiological health. These processes are increasingly recognized as crucial not only for the affective, cognitive and physical neuroscientific field, but also for clinical applications. Moreover, autonomic imbalance has been associated with increased morbidity and mortality across a range of medical conditions, both physical and psychiatric (Agorastos et al., 2023).

Autonomic flexibility refers to the capacity of the autonomic nervous system to maintain or restore a state of balance in response to environmental demands, by dynamically shifting between different autonomic states in accordance with internal and external cues. In contrast, reduced flexibility is characterized by a persistent bias toward states of hyperarousal or hypoarousal. Within a polyvagal framework, such dysregulated patterns may correspond to prolonged sympathetic activation or dorsal vagal dominance, and are especially present in people suffering from mental disorders (Agorastos et al., 2023).

Thereby, measures of ANS reactivity can be considered a transdiagnostic psychophysiological marker of not only self-regulation, but also cognitive and emotional control, and overall psychophysiological health (Agorastos et al., 2023).

3. Psychophysiological Markers of Autonomic Flexibility

These theoretical considerations highlight the importance of identifying objective physiological markers of autonomic regulation and flexibility. Currently, several psychophysiological indices are used in research to assess ANS functioning. Among these, Heart Rate Variability (HRV) represents the most widely used and well-established measure.

This is not surprising, as the heart and brain are deeply interconnected at a pathophysiological level. Indeed, a high comorbidity has been observed between

stress-related disorders, or emotionally challenging events, and cardiovascular disease or failure (Agorastos et al., 2023), highlighting the central role of neuroautonomic regulation in both mental and physical health.

HRV reflects the beat-to-beat variation in heart rate (R-R intervals), resulting from the dynamic interplay between sympathetic and parasympathetic influences on cardiac activity (Tun et al., 2018). As such, HRV represents a reliable biomarker of autonomic functioning and control (Park, 2014) and, consequently, of emotional and physiological regulation and health (Agorastos et al., 2023). It is also associated with the capacity to adapt to continuously changing internal and external demands (Kenemore et al., 2024). Moreover, HRV is widely used in research due to its non-invasive nature and relative ease of measurement through Electrocardiogram (ECG), and can be visually presented through tachograms, which depict the variation in R–R intervals over time (Figure 4).

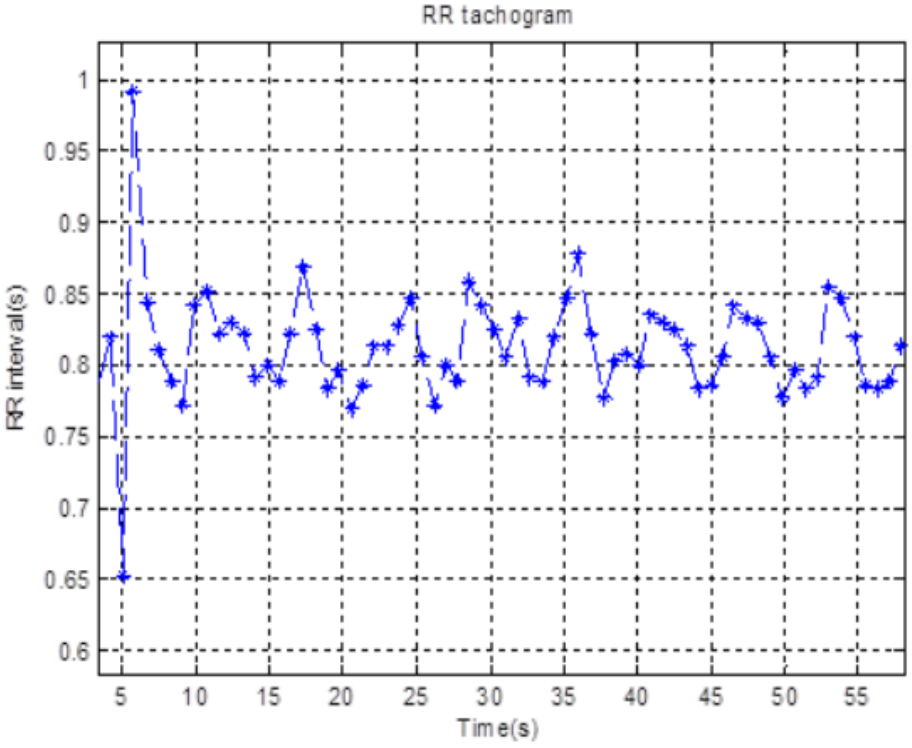

Figure 4. RR Tachogram for HRV Analysis (Source: Tun et al., 2018).

Reduced HRV has been consistently associated with a wide range of psychiatric disorders (e.g., PTSD, anxiety disorders, depression, schizophrenia, bipolar disorder, autism), early cognitive decline, suicide and psychosis risk, emotional dysregulation as well as higher mortality, especially due to cardiovascular causes

(Agorastos et al., 2023; Park, 2014; Williamson et al., 2015), and appears significantly associated with the overactivation of the SNS and/or decreased PNS (vagal) activity (Schneider, 2020).

In contrast, higher resting HRV is reliably associated with good adaptability of the cardiovascular system (Schneider, 2020) and both *top-down* and *bottom-up* cognitive processing, with consequent higher emotion regulation (Park, 2014), resilience (Ge, 2020) and overall psychophysiological health, and mainly depends on parasympathetic dominance (Agorastos et al., 2023).

A more specific component of HRV is Respiratory Sinus Arrhythmia (RSA), which reflects heart rate fluctuations associated with the respiratory cycle, and it is considered a more direct index of vagally mediated parasympathetic activity. As such, RSA represents a useful biomarker of autonomic regulation, particularly of VVC functioning (Porges, 2026).

Indeed, this phenomenon is primarily mediated by cardiac-linked vagal efferent connections from the Nucleus Ambiguus (Tonhajzerova et al., 2016), which, according to Polyvagal Theory, constitutes the anatomical basis of the VVC (Porges, 2007). Nevertheless, it is important to note that sympathetic activity may also contribute to RSA variability (Tonhajzerova et al., 2016).

Physiologically, RSA is characterized by an increase in heart rate during inhalation and a decrease during exhalation, reflecting dynamic vagal modulation of cardiac activity in synchrony with respiration. Psychologically, growing research has associated greater resting RSA amplitude with better cognitive control of emotions, and reduced resting RSA (i.e., reduced vagal modulation) with maladaptive cognitive-affective regulation (Tonhajzerova et al., 2016).

Taken together, HRV and RSA provide complementary indices of autonomic flexibility, offering valuable insight into the regulation of physiological and emotional responses.

Chapter 3 – PTSD in the Polyvagal Framework

1. PTSD: Clinical Features and Psychophysiological Interpretation

At the beginning of my thesis, the limits of the classical nosological approach in classifying disorders were mentioned. However, for the purpose of clarity and consistency with the existing literature, the present work focuses on trauma related disorders, particularly PTSD. Indeed, PTSD represents a particularly relevant model for examining autonomic dysregulation, as it encompasses both hyperarousal and hypoarousal symptom clusters.

According to the DSM-5 (American Psychiatric Association, 2013), PTSD symptomatology includes intrusion symptoms (e.g., flashbacks, trauma-related nightmares, dissociative experiences, extreme sensitivity to internal and external trauma-related cues), persistent avoidance of stimuli associated with the traumatic event, marked cognitive and affective alterations (e.g., amnesia, persistent negative cognitions and mood, anhedonia, avolition, feelings of detachment or estrangement from others) and marked alteration in arousal and reactivity (e.g., irritability, anger outbursts, self-destructive behavior, exaggerated startle response, problems with attention and sleep). Additionally, a dissociative subtype has also been identified, with derealization and depersonalization being the core symptoms.

From a psychophysiological and polyvagal perspective, these manifestations do not represent mere psychopathology, but rather a survival adaptation to traumatic experiences. Specifically, symptoms represent state-dependent autonomic responses, reflecting chronic autonomic imbalance and rigidity in the form of restricted access to the VVC and dominance of defensive states – i.e. sympathetic mobilization and dorsal vagal shutdown – due to altered neuroception biased toward danger (Porges, 2025).

2. Hyperarousal and Sympathetic Mobilization

Research confirms that individuals with PTSD show increased sympathetic activity, reflected in higher Heart Rate (HR) and Skin Conductance (SC) levels

(Campbell et al., 2019), alongside reduced HRV (Schneider, 2020; Thome et al., 2016), which has been associated with enhanced sympathetic activation (Agorastos et al., 2023) and decreased vagal functioning and consequent behavioral inflexibility (Thome et al., 2016). Moreover, hightened RSA withdrawal (i.e., decreased RSA) is observable in individuals with PTSD (Campbell, 2021).
Nevertheless, it is not clear yet whether lower HRV represents a vulnerability marker for PTSD or if it develops during its course. Therefore, further research is needed (Schneider, 2020). Additionally, it has been observed that epinephrine and norepinephrine levels, typically associated with sympathetic activity, are higher in individuals with PTSD and affect the severity of symptoms (Lemieux and Coe, 1995, as cited in Williamson et al., 2015).
This hyperarousal state appears to be responsible for symptoms such as high-risk behaviors, suicidal impulses, self-harm (Corrigan et al., 2011), increased startle reflex (Williamson, 2015), irritability and hostility (Peters et al., 2018) – all symptoms that may represent an attempt to regulate this altered autonomic state (Corrigan et al., 2011).
Additionally, it is important to note that growing literature associates low resting HRV with undifferentiated threat responses and difficulties in differentiating between safety and threat (Thome et al., 2016), which, within a polyvagal framework, may reflect altered neuroception.

3. Hypoarousal, Dissociation and Dorsal Vagal Shutdown

In contrast to hyperarousal sympathetic states, PTSD may also manifest through hypoarousal (McTeague et al., 2010; D'Andrea et al., 2013) and dissociative symptoms, particularly in individuals exhibiting the dissociative subtype of the disorder (APA, 2013).
From a psychophysiological perspective, this state is characterized by reduced arousal and behavioral disengagement, often accompanied by symptoms such as hopelessness, loss of energy, emotional numbing, and feelings of helplessness (Corrigan et al., 2011), with high levels of anxiety and/or depression (McTeague et al., 2010). In more extreme cases, this state may also involve passive coping responses in the form of suicidal ideation, collapse, compliance,

and resignation in the face of clear imbalance of power and inescapable threat (Corrigan et al., 2011; Porges, 2007).

Within a polyvagal framework, these responses may be understood as reflecting the activation of the Dorsal Vagal Complex (DVC), which mediates immobilization strategies when active defense is no longer possible (Porges, 2007). This state promotes survival through energy conservation, enabling the individual to sustain overwhelming conditions for a long period of time, if needed. Indeed, this type of PTSD manifestation is often observable in individuals who experienced severe or repeated, chronic trauma exposure (McTeague et al., 2010; D'Andrea et al., 2013).

Although such responses may be adaptive in the short-term, particularly in situations of extreme and inescapable danger, their chronic activation can lead to persistent low mood, hopelessness, and worthlessness that can mimic or coexist with MDD (Corrigan et al., 2011).

These hypoarousal states are particularly relevant in the dissociative subtype of PTSD, where symptoms such as depersonalization and derealization reflect a profound disengagement from both internal (e.g., bodily or emotional) and external (environmental) experience (APA, 2013).

Unfortunately, the psychobiological mechanisms and biomarkers underlying these symptoms have not been clarified yet (Beutler et al., 2022). Nevertheless, some evidence suggests that dissociative responses may be associated with distinct autonomic patterns: for example, Bichescu-Burian and colleagues (2016) reported decreased HR during trauma-related recall in individuals with borderline personality disorder and a history of peritraumatic dissociation, a finding that has been interpreted as indicative of a dissociative response pattern, while Seligowski and collaborators (2019) found that women with PTSD and dissociation have decreased startle response and SCR and higher RSA. Within a broader psychophysiological framework, such blunted physiological symptoms have often been associated with parasympathetic dominance or vagally mediated responses (D'Andrea et al., 2013).

4. Autonomic Dysregulation and Reduced Flexibility in PTSD

To summarize, in healthy and resilient individuals, the autonomic nervous system flexibly transitions between states of social engagement, mobilization, and immobilization. In contrast, individuals with PTSD often exhibit a restricted autonomic range, characterized by prolonged sympathetic activation or dorsal vagal dominance, and reduced access to ventral vagal regulation. According to polyvagal theory, at the core of this autonomic dysregulation there may be an impaired functioning of the Ventral Vagal Complex (VVC) potentially due to trauma, adversity, or cumulative stress. Indeed, the VVC plays a central role in downregulating sympathetic activity and supporting flexible transitions between restorative and aroused states (Porges, 2025).

More broadly, some authors (e.g., Williamson et al., 2015; Schneider, 2020) are proposing autonomic dysfunction and reduced flexibility, defined as a diminished capacity to adaptably shift between different autonomic states in response to social and environmental cues, as a core feature of PTSD. Such dysregulation has widespread consequences and contributes to the broader health burden associated with PTSD, as autonomic functioning influences not only social and psychological functioning, but also metabolic, immunologic and cardiovascular systems (Williamson et al., 2015).

Beyond PTSD, recent literature increasingly recognizes the transdiagnostic role of autonomic dysfunction across a wide range of psychiatric disorders, including panic disorder, depression, bipolar disorder, schizophrenia, sleep disorders, attention-deficit/hyperactivity disorder, generalized anxiety disorder, substance abuse, and autism (Agorastos et al., 2023; Göçen et al., 2024). Importantly, this dysregulation may not only accompany these conditions, but also contribute to symptom severity and maintenance, suggesting a potential transdiagnostic role (Göçen et al., 2024).

From a clinical perspective, this framework suggests that interventions targeting autonomic regulation may play a key role in improving outcomes in PTSD, and potentially other psychological disorders (Corrigan et al., 2011). In particular, approaches aimed at enhancing vagal functioning (Agorastos et al., 2023) and

restoring autonomic flexibility may support more adaptive emotional and behavioral responses as well as coherent neuroception.

Conclusion

1. Summary of Main Findings

Overall, the present work highlights the central role of the ANS in understanding both normal and pathological functioning. In particular, Polyvagal Theory provides a useful framework to conceptualize how different autonomic states shape behavioral, cognitive, and physiological responses to internal and environmental cues. A key result emerging from the literature is that psychophysiological health is defined by the capacity of the autonomic nervous system to flexibly shift between states of social engagement, mobilization, and immobilization in response to contextual demands. In this sense, autonomic flexibility emerges as a core marker of adaptive functioning.

Conversely, psychopathology – and especially trauma-related disorders such as PTSD – is characterized by autonomic dysregulation and reduced flexibility, reflected in a restricted range of autonomic responses and a tendency to remain stuck in defensive states of hyperarousal or hypoarousal. Empirical evidence supports this view, showing that individuals with PTSD exhibit altered autonomic patterns, including increased sympathetic activation, reduced RSA and HRV or blunted physiological responses, although evidence is mixed. These alterations are associated with impaired emotional regulation, biased threat detection (i.e. altered neuroception), and difficulties in adapting to environmental demands.

Notably, such autonomic dysfunction may be shaped by traumatic experiences, with different psychophysiological outcomes depending on individual factors such as genetic vulnerability, environmental context, and developmental stage, as well as the characteristics of the traumatic event itself.

Finally, autonomic dysfunction is not specific to PTSD, but is increasingly recognized across a wide range of psychiatric conditions, supporting the idea of autonomic dysfunction as a transdiagnostic construct. Taken together, these findings suggest that the autonomic nervous system plays a fundamental role not only in the development and maintenance of psychopathology, but also as a potential target for assessment and intervention.

2. Limitations and Future Research

Polyvagal theory provides a biologically grounded and holistic framework for understanding human behavior, health and pathology, and highlights the key role of ANS functioning and flexibility (Porges, 2025). This theory has stimulated a wide range of research across multiple disciplines, extending beyond psychology (Williamson et al., 2015), and has contributed to the development of testable psychophysiological markers, such as RSA, HRV, and vagal efficiency.

Furthermore, Polyvagal Theory has been increasingly applied in clinical practice, offering a useful framework for understanding and treating patients' heterogeneous symptomatology in terms of state-dependent functioning (Porges, 2025), whether characterized by sympathetic dominance, dorsal vagal dominance, or their alternation.

However, despite its theoretical and clinical influence, several limitations have been highlighted in the literature. In particular, Grossman et al. (2026) critically discussed the reliance on RSA as a primary index of vagal functioning, arguing that it represents only an indirect and approximate measure of cardiac vagal tone, as it reflects heart rate modulation only, and it is influenced by multiple physiological factors. In contrast, Porges (2026) emphasized that RSA should be interpreted as a specific index of ventral vagal activity, rather than a global measure of vagal tone.

Additional criticisms concern the evolutionary interpretation of the theory, as evidence suggests that complex social behaviors are not exclusive to mammals, challenging the assumption that such functions are uniquely linked to the ventral vagal system (Grossman et al., 2026). Moreover, the clear functional distinction between different vagal pathways and their direct association with complex psychological states has been questioned (Grossman et al., 2026).

Nevertheless, Polyvagal Theory does not posit strict anatomical exclusivity, but rather emphasizes functional, physiological systemic differences at the ANS level (Porges, 2026).

These ongoing debates highlight the need for further empirical research aimed at clarifying the relationship between autonomic markers and psychophysiological outcomes, as well as the importance of integrating different methodological and

theoretical approaches, to clarify the underlying psychophysiological mechanisms of psychopathology, avoiding oversimplification or excessive reliance on any single theoretical or methodological framework.

Despite these limitations, and even if Polyvagal theory remains controversial and subject to ongoing debate, it represents an impactful perspective that recognizes psychopathology as an embodied phenomenon, shaped by bidirectional interactions between psychological and physiological processes. More broadly, this perspective reflects a paradigm shift from predominantly brain-centered, *top-down* models toward a more integrative and holistic approach, in which the entire nervous system is considered central to both the development of psychopathology, and the processes of recovery and healing.

Additionally, this framework may represent a way to conceptualize mental illness along a continuum, ranging from autonomic rigidity to autonomic flexibility, without oversimplifying complex phenomena. This approach moves beyond the categorical diagnostic distinctions between disorders in favor of a more transdiagnostic approach. Of course, further research is needed to better clarify and empirically support this perspective.

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