TEMPERAMENTAL PREDICTORS OF PTSD SYMPTOM SEVERITY IN COMBAT VETERANS

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Abstract. Background: The marked heterogeneity in post-traumatic stress disorder (PTSD) symptom severity among combat veterans with comparable trauma exposure indicates that event characteristics alone cannot explain individual vulnerability. Traditional personality models often fail to capture the functional mechanisms behind this variability. The Functional Ensemble of Temperament (FET) offers a refined perspective, decomposing broad traits into specific bio-energetic (Endurance) and dynamic (Plasticity) components that may govern the capacity to withstand chronic stress.

Objective: This study investigates the association between FET-defined temperament traits, regulatory mechanisms (coping and defenses), and PTSD symptom severity. A central aim was to test the hypothesis of "structural collapse"– specifically, whether the latent organization of temperament and symptoms differs between resilient and affected veterans (preservation versus loss of orthogonality).

Methods: In a comparative cross-sectional design, 95 male combat veterans were stratified into clinically verified PTSD (n = 44) and Non-PTSD (n = 51) groups using the SCID-5. Assessment utilized the CAPS-5 for symptom severity, STQ-77 for temperament, COPE Inventory for coping strategies, and the MIPZ for defense mechanisms. Data were analyzed using non-parametric group comparisons, Spearman

correlations, and separate Exploratory Factor Analyses (PCA) to reconstruct the latent architecture of each group.

Results: Veterans with PTSD exhibited a coherent "asthenic" profile characterized by significantly reduced intellectual and social endurance, lowered plasticity, and diminished empathy, rather than isolated emotional instability. Higher symptom severity was strongly linked to regulatory regression: a depletion of future-oriented coping (planning) and mature defenses (humor, sublimation) in favor of rigid, primitive mechanisms such as omnipotent control and denial. Crucially, factor analysis revealed divergent structural organizations. While the Non-PTSD group retained a functional separation between temperament and stress markers, the PTSD group demonstrated a "collapse of orthogonality," where reduced endurance, defensive rigidity, and clinical symptoms converged into a single, self-reinforcing dominant factor.

Conclusions: Chronic combat-related PTSD appears to function as a systemic resource failure, driven by bio-energetic exhaustion (hypo-ergonicity) and regulatory inflexibility. The observed structural fusion of constitutional traits and psychopathology suggests that clinical interventions should adopt a staged approach, prioritizing the restoration of functional resources before engaging in intensive trauma processing.

Keywords: PTSD, combat veterans, Functional Ensemble of Temperament (FET), STQ-77, coping strategies, defense mechanisms.

1. Introduction

1.1. The Epidemiological Context and the Enigma of Differential Susceptibility

Combat exposure remains a definitive risk factor for post-traumatic stress disorder (PTSD), imposing a persistent burden on service members and healthcare systems alike. However, prevalence estimates paint a complex picture, fluctuating significantly across different conflicts and cohorts. While the National Vietnam Veterans Longitudinal

Study documented lifetime prevalence rates as high as 30.9%, estimates [1] for Operations Enduring Freedom and Iraqi Freedom are generally lower, falling within the 11-20% range, with recent analyses of younger cohorts suggesting weighted rates of approximately 13.5-15.8% [2].

Beyond these aggregate statistics lies a clinically decisive observation: comparable exposure yields vastly different outcomes. The lack of a consistent linear relationship between trauma severity and symptom development shifts the focus of psychotraumatology from descriptive epidemiology to etiology [3]. The central question is no longer just how many individuals are affected, but why severe combat stress leads to chronic psychopathology in some veterans, while others – facing similar or even greater threats – maintain functional adaptation or exhibit post-traumatic growth [4].

This heterogeneity demonstrates that while trauma exposure is a necessary condition for PTSD, it is functionally insufficient to explain between-person variability. Models relying solely on trauma parameters – such as blast magnitude, proximity to death, or moral injury [5] – leave a substantial proportion of the clinical variance unexplained. To bridge this gap, focus must shift to pre-morbid individual differences. In this context, temperamental traits emerge not merely as descriptive labels, but as biologically grounded constraints on neurobehavioral functioning [6]. These stable characteristics define the limits of an individual's capacity to sustain regulation under the prolonged allostatic load of combat, thereby acting as critical moderators of vulnerability [7].

1.2 The Problem of Personality in PTSD Research

Historically, the search for premorbid markers of PTSD vulnerability has relied heavily on trait-based frameworks, particularly the Five-Factor Model [8, 9]. While empirical data consistently link elevated Neuroticism and reduced Extraversion to greater symptom severity [10], these associations are predominantly descriptive. They identify who is vulnerable but offer little insight into how that vulnerability operates at a

mechanistic level.

A primary limitation is the circularity inherent in using broad traits as causal explanations. Neuroticism, by definition, represents a disposition toward negative affect and heightened stress sensitivity. Consequently, invoking Neuroticism to explain why an individual develops stress pathology is largely tautological: it amounts to stating that a person is vulnerable to stress because they possess a trait defined by stress vulnerability [11]. Such descriptors treat the individual as a "black box," failing to specify whether the observed susceptibility stems from amygdala reactivity, deficits in prefrontal inhibition, or dysregulated neuroendocrine signaling [6]. This lack of resolution limits the utility of trait models for designing targeted interventions.

A more critical issue, particularly in the context of combat trauma, is the tendency of lexical models to conflate functionally distinct behavioral dimensions [12]. The construct of Extraversion, for instance, aggregates sociability (the drive for interaction), energetic capacity (endurance), and processing speed (tempo). Under normative conditions, these components often covary. However, extreme stress can dissociate them [13]. In a combat zone, a soldier may retain high sociability – actively seeking peer support as a coping resource – while simultaneously suffering from profound physical or cognitive exhaustion. Conversely, an individual may react with high behavioral speed (tempo) but lack the metabolic endurance to sustain that activity over time.

By collapsing endurance, speed, and social orientation into unitary traits, traditional inventories obscure these functional nuances. Given that chronic combat exposure imposes a relentless physiological and cognitive load, the specific capacity to sustain effort (endurance) becomes a decisive factor in adaptation [14]. Models that fail to distinguish between the style of behavior (e.g., sociability) and the energy available to fuel it (e.g., endurance) are therefore insufficient for isolating the specific pathways of breakdown. This limitation underscores the need to move beyond descriptive taxonomies toward functionally informed frameworks capable of dissecting the bio-

behavioral components of resilience [13].

1.3. The Functional Ensemble of Temperament (FET)

To address the limitations of descriptive lexical models, this study utilizes the Functional Ensemble of Temperament (FET) framework developed by Trofimova and Rusalov [6, 15]. While traditional taxonomies (e.g., the Big Five) classify individuals based on social descriptors, the FET model approaches temperament as a system of biologically grounded functional capacities [13]. It decomposes broad traits into specific energetic, dynamic, and emotional components, viewing them not as social labels but as observable behavioral regularities constrained by the properties of the nervous system [16].

Structurally, the FET organizes temperament into a matrix defined by three core functional dimensions – Endurance (Ergonicity), Speed/Plasticity (Tempo and Flexibility), and Emotionality – distributed across physical, social, and intellectual domains [6, 15]. This architecture allows for the disentanglement of functional elements often conflated in other models. For instance, it distinguishes between the drive to act, the endurance to sustain that action, and the speed of execution. In the context of trauma research, this granularity is critical: it permits the hypothesis that vulnerability to PTSD stems not from global personality "defects," but from specific deficits in functional resources – such as the inability to sustain regulatory effort (low endurance) or to disengage from threat detection (low plasticity).

1.3.1. Functional Endurance (Ergonicity) and Allostatic Load

A cornerstone of the FET model is the concept of Ergonicity, or Endurance. Unlike motivation or momentary reactivity, Endurance reflects the nervous system's capacity to sustain prolonged activity under high intensity or load [13, 16]. It represents a measure of bio-energetic supply rather than subjective intent. While the FET model theoretically links this dimension to opioid modulation and HPA axis integrity [6], for the purposes of this study, Endurance is treated as a functional marker of stress

tolerance.

Combat exposure imposes a state of chronic, high-intensity allostatic load, requiring continuous mobilization of physical, cognitive, and social resources [2, 17]. In this context, baseline Endurance becomes a decisive factor in adaptation. We hypothesize that veterans with lower Ergonicity are functionally constrained: their capacity to maintain regulatory control depletes faster under sustained stress.

This depletion manifests in clinically distinct ways. Reduced Intellectual Endurance may limit the cognitive resources required for inhibitory control, making the suppression of intrusive memories increasingly difficult [18, 19]. Similarly, diminished Social Endurance may lead to withdrawal – not necessarily due to a lack of desire for connection, but because the energetic cost of sustained social processing exceeds the individual's current capacity. Thus, symptoms often interpreted as avoidance or apathy may essentially represent protective energy-conservation strategies triggered by systemic exhaustion [20].

1.3.2. Plasticity, Tempo, and Contextual Updating

The FET framework separates the dynamic aspects of behavior into Plasticity (flexibility) and Tempo (speed) [13, 15]. Plasticity refers to the ability to shift smoothly between behavioral programs and update strategies in response to changing environmental feedback. In combat settings, survival often depends on rapid transitions between extreme vigilance/aggression and restraint.

Low Plasticity creates a vulnerability to functional rigidity. Clinically, this manifests as a failure of "contextual updating": the veteran continues to operate on combat-adapted predictive models (e.g., hypervigilance) even after returning to civilian safety [21]. This functional "stickiness" – the inability to disengage from a threat-oriented mode – aligns closely with core PTSD clusters such as re-experiencing and physiological reactivity [22].

It is crucial to distinguish this rigidity from Tempo – the sheer speed of cognitive

or motor processing. High Tempo does not imply high Endurance or Plasticity. In fact, a profile characterized by high Tempo but low Endurance is frequently associated with anxiety disorders: the system operates at a high frequency but lacks the energetic reserves to sustain it, leading to rapid "burnout" and regulatory collapse [23]. By differentiating these dimensions, the FET model allows us to isolate the specific mechanical failures – such as the inability to switch tasks versus the inability to sustain effort – that underpin the complex phenomenology of PTSD.

1.4. Regulatory Mechanisms

Clinical outcomes following traumatic exposure are shaped not only by stable temperamental traits but also by secondary regulatory systems – specifically, conscious coping strategies and unconscious defense mechanisms. While classical models typically treat these as distinct domains – contrasting the goal-directed nature of coping (e.g., transactional models) [24] with the automatic, anxiety-mitigating function of defenses (e.g., hierarchical maturity models) [25] – we propose that they are functionally interdependent and constrained by the individual's constitutional resources [26].

From the perspective of the FET framework, psychological regulation is not metabolically neutral; it operates within an "economy" defined by the organism's available energy [13]. Temperamental endurance (Ergonicity) effectively sets the baseline budget for these processes [15, 27]. Adaptive, problem-focused coping (such as active planning) and mature defenses (such as humor, sublimation, or anticipation) are "expensive." They require sustained executive control, complex symbolic processing, and the inhibition of immediate impulses. Consequently, the capacity to maintain such high-level regulation is theoretically contingent upon sufficient levels of functional endurance.

In the context of combat exposure, which imposes extreme and prolonged allostatic load, these resource-intensive strategies may become unsustainable. We posit a mechanism of regulatory regression, driven by the depletion of bio-energetic reserves

[20]. As functional endurance is exhausted, the cognitive cost of maintaining mature defenses becomes prohibitive. The system is thus forced to switch to more "economical," primitive strategies – such as denial, omnipotent control, or regression [28]. These mechanisms offer immediate reduction of subjective distress through perceptual distortion or behavioral simplification, requiring far less metabolic and cognitive investment than the integrative work of mature adaptation.

Therefore, the dominance of rigid or primitive defenses in veterans with PTSD should not be viewed merely as a characterological flaw or a maladaptive psychological choice. Instead, it represents a functional fallback: when the temperamental "fuel" required for flexible, high-level processing runs out, the regulatory system reverts to earlier, low-energy modes of operation to preserve basic stability, even at the cost of long-term psychological integration.

1.5 Study Objectives and Hypotheses

The primary objective of this study is to ground the heterogeneity of PTSD symptoms in a functional bio-behavioral framework [29, 30]. Moving beyond descriptive trait associations, we aim to determine whether specific neurobiological dispositions – conceptualized within the Functional Ensemble of Temperament (FET) – underlie vulnerability to combat-related PTSD [6, 13]. Specifically, we examine whether deficits in Endurance (Ergonicity) and Plasticity are systematically linked to symptom severity and the organization of secondary regulatory mechanisms, such as coping strategies and psychological defenses [31].

Recognizing the cross-sectional nature of the design, the study focuses on the structural architecture of these relationships rather than strict causality. We investigate how stable constitutional traits interact with clinical status to shape the psychopathological profile [32]. Based on the FET theoretical model, we propose three core hypotheses:

We predict that veterans with PTSD will exhibit a specific functional phenotype

characterized by systemic hypo-ergonicity (reduced endurance) and diminished plasticity across motor, social, and intellectual domains. Unlike general Neuroticism [9], this "asthenic" profile reflects a fundamental deficit in the capacity to sustain activity and switch behavioral programs under load. This functional limitation is expected to distinguish the clinical PTSD group from combat-exposed but resilient peers, serving as a key marker of maladaptation [7].

We hypothesize that severe PTSD symptoms are associated with a "regression" in regulatory economy. As temperamental endurance is depleted, the cognitive resources required for mature, high-level defenses (e.g., Humor, Sublimation) and active coping become inaccessible [20]. Consequently, regulation is expected to shift toward metabolically "cheaper" but more rigid and primitive mechanisms (e.g., Omnipotent Control, Regression) [33, 34]. This shift is viewed not as a behavioral choice, but as a functional necessity imposed by resource constraints.

This hypothesis concerns the latent structure of psychopathology. In resilient veterans, we expect temperament traits and stress symptoms to remain statistically independent (orthogonal), reflecting a modular organization where stress reactions do not consume the core personality structure. Conversely, in the PTSD group, we anticipate a structural fusion of these domains. Specifically, temperament deficits and symptom severity are expected to load onto a single shared latent factor, indicating a breakdown of autonomous regulation and the integration of pathology into the core bio-behavioral system [23].

2. Materials and Methods

2.1. Participants and Recruitment

We enrolled a total of 95 male combat veterans. To ensure clinical heterogeneity while preserving diagnostic rigor, recruitment employed a dual-source strategy. The clinical subsample was recruited from the tertiary center specializing in trauma-associated disorders. To avoid sampling bias favoring only severe, treatment-seeking

cases, a community-based comparison arm was recruited through veteran networks.

Selection Criteria

To isolate the specific effects of combat trauma and minimize confounders, strict inclusion criteria were applied:

Direct participation in hostilities was confirmed via official military service records, ensuring objective adherence to DSM-5 Criterion A.

The sample was restricted to enlisted personnel (privates through sergeants). Commissioned officers were excluded to eliminate confounding variables related to command responsibility and leadership-specific stressors, allowing the study to focus on frontline combat experience.

All participants provided voluntary written informed consent.

Participants were excluded if they presented with conditions likely to distort psychometric data or independently impair regulatory capacity. These included schizophrenia spectrum or other primary psychotic disorders, bipolar disorder (current manic/hypomanic episodes), acute intoxication/withdrawal, or severe organic brain pathology (including TBI with significant cognitive impairment).

Demographic and Clinical Comparability

The PTSD (n = 44) and Non-PTSD (n = 51) groups were statistically equivalent across key demographic variables. No significant differences were found in mean age (PTSD: 34.34 ± 8.38; Non-PTSD: 35.43 ± 9.18), educational level, or time elapsed since deployment. This matching suggests that observed differences in clinical and temperamental measures are not attributable to age, education, or service-related confounders.

2.2. Procedure and Design

The study utilized a comparative cross-sectional design incorporating a structured, two-stage assessment protocol.

Stage 1: Clinical Verification. Participants underwent a comprehensive

psychiatric evaluation by board-certified psychiatrists experienced in psychotraumatology. The diagnostic process included a review of medical and service history, followed by the Structured Clinical Interview for DSM-5 (SCID-5), PTSD module. Diagnoses were established according to DSM-5 criteria and cross-referenced with ICD-10 (F43.1) to ensure nosological consistency.

Based on this assessment, participants were stratified into two cohorts:

1. PTSD Group (n = 44): Veterans meeting full diagnostic criteria for current Post-Traumatic Stress Disorder.

2. Non-PTSD Group (n = 51): Combat-exposed veterans with no current stress-related diagnosis. This group served as a trauma-exposed control, representing resilient adaptation rather than a lack of exposure.

Stage 2: Psychometric Assessment. Following diagnostic allocation, participants completed a battery of self-report instruments assessing temperament, coping, and defense mechanisms. Data collection took place in a controlled, distraction-free environment. To prevent criterion contamination, psychometric testing was conducted independently of the clinical interview.

2.3. Ethical Considerations

The study adhered to the ethical standards of the Declaration of Helsinki. Strict data protection protocols were implemented, with all personal information anonymized and coded prior to analysis.

2.4. Instrumentation

The study employed a multi-level assessment battery designed to map the structural interplay between clinical symptoms, constitutional temperament, and regulatory mechanisms. We conceptualized these variables across three complementary domains: the clinical phenotype (PTSD severity), the constitutional baseline (temperament traits), and dynamic regulation (coping and defenses).

2.4.1. Clinical Assessment

Post-traumatic symptom severity was evaluated using the CAPS-5, widely recognized as the diagnostic gold standard. This structured interview differentiates trauma-specific pathology from general distress by assessing the frequency and intensity of symptoms directly linked to the index trauma. The instrument covers the four DSM-5 symptom clusters: Intrusion (B), Avoidance (C), Negative Alterations in Cognition/Mood (D), and Hyperarousal (E). The primary dependent variable for analysis was the CAPS-5 total severity score.

2.4.2. Temperament Assessment

Constitutional traits were profiled using the STQ-77, grounded in the Functional Ensemble of Temperament (FET) framework. Unlike lexical models (e.g., Big Five), the FET focuses on functional components of behavior – energy, speed, and plasticity – rather than social descriptors. The STQ-77 assesses 12 scales across four domains:

Ergonicity (Endurance): The capacity to sustain activity under load across Motor (ERM), Social (ERS), and Intellectual (ERI) domains.

Plasticity: The flexibility to switch between behavioral programs (Motor, Social, Intellectual).

Tempo: The speed of processing and execution (Motor, Social, Intellectual).

Emotionality: Sensitivity and reactivity, assessed via Neuroticism, Impulsivity, and Self-Confidence.

For this study, these scales were operationalized as stable bio-behavioral dispositions hypothesized to constrain regulatory capacity under stress.

2.4.3. Assessment of Regulatory Mechanisms

Conscious regulatory efforts were measured using the Russian adaptation of the COPE Inventory. This 60-item instrument categorizes habitual stress responses into three functional styles: Problem-focused (e.g., active coping, planning); Emotion-focused (e.g., seeking social support, reinterpretation); Maladaptive/Avoidant (e.g., denial, behavioral disengagement).

Analysis focused on the availability of cognitively demanding strategies versus avoidant patterns in the clinical group.

Unconscious regulation was assessed via the Methodology for Archiving Psychological Defense (MIPZ). This tool organizes defenses hierarchically, allowing for the quantification of "defensive regression." Mechanisms are stratified into three maturity levels: Primitive/Psychotic (e.g., denial, distortion); Immature/Neurotic (e.g., displacement, repression); Mature/Adaptive (e.g., sublimation, humor).

This hierarchy was used to test whether PTSD severity correlates with a shift toward ontogenetically earlier forms of defense.

2.5. Statistical Analysis

Data analysis was performed using IBM SPSS Statistics 26. Due to the non-normal distribution typical of clinical samples, non-parametric methods were prioritized. Statistical significance was set at $p < 0.05$ (two-tailed), with effect sizes (r) calculated to quantify group differences.

The Mann–Whitney U test was used to compare the PTSD and Non-PTSD groups across temperament traits and regulatory variables. To examine the relationship between constitutional factors and clinical status, we computed Spearman's rank correlations (ρ) between STQ-77 scales, defense mechanisms, and CAPS-5 severity scores.

To test the hypothesis of a "collapse of orthogonality," we conducted Exploratory Factor Analysis (Principal Component Analysis with Varimax rotation) separately for the PTSD (n=44) and Non-PTSD (n=51) cohorts. This split-file approach allowed us to compare the latent structure of psychopathology in each group directly.

Factor retention was guided by eigenvalues (>1.0) and scree plot inspection. Variables with loadings $\geq |0.40|$ were considered salient. The analysis specifically sought to determine whether clinical symptoms formed an independent factor (orthogonality) or converged with temperament and regulatory traits into a single dominant dimension (structural collapse).

2.6. AI-Assisted Language Refinement

Generative artificial intelligence tools, specifically the ChatGPT 5.2 architecture (OpenAI), were utilized exclusively for the translation and stylistic refinement of the manuscript to ensure adherence to international academic English standards. The authors performed a comprehensive review of the final text to verify terminological accuracy and retain full responsibility for the scientific content and data integrity.

3. Results

3.1. Group Differences: The Asthenic Temperament Profile

Comparison of STQ-77 profiles revealed a distinct functional configuration separating combat veterans with PTSD from the non-clinical group. While PTSD is often framed primarily as a disorder of emotional dysregulation, the current data indicate a systemic depletion of the energetic and dynamic foundations of temperament. The clinical group exhibited a consistent "asthenic" profile, characterized by compromised endurance and flexibility across social and intellectual domains (Table 1).

Table 1. Comparative Analysis of Temperament Traits (STQ-77)

Scale	Functional Aspect	Non-PTSD (μ)	PTSD (μ)	Δ	p-value	Interpretative Note
ERS	Social Endurance	13.68	12.67	−1.01	0.03	Reduced capacity to sustain social interaction
TMS	Social Tempo	14.00	12.84	−1.16	0.01	Slowed communicative processing
ERI	Intellectual Endurance	14.02	13.12	−0.90	0.01	Increased cognitive fatigability
PL	Plasticity	13.80	13.12	−0.68	0.04	Reduced behavioral flexibility
EMP	Empathy	14.00	12.80	−1.20	<0.001	Attenuation of affective responsiveness
PRO	Probabilistic Processing	14.11	13.14	−0.97	0.02	Impaired anticipatory evaluation
NEU	Neuroticism	lower	higher	—	<0.01	Elevated threat sensitivity

Note. Group differences assessed using the Mann–Whitney U test.

The most pervasive deficit appeared in the ergonic (energetic) dimension.

Veterans with PTSD demonstrated significantly lower Social Endurance (ERS) and Intellectual Endurance (ERI), accompanied by slowed Social Tempo (TMS). This pattern suggests a fundamental constraint on the nervous system's capacity to sustain prolonged cognitive focus and interpersonal engagement. Critically, these low endurance scores should be distinguished from depressive withdrawal or lack of motivation; within the FET framework, they reflect a biological limitation on energy mobilization – a functional inability to maintain activity under load.

In parallel, the clinical group showed reduced Plasticity (PL) and Probabilistic Processing (PRO). This decline points to a heightened behavioral rigidity, manifesting as difficulty in shifting between tasks or updating anticipatory models when situational contexts change. Such temperamental rigidity aligns with the clinical inability to disengage from threat-oriented processing modes even when objective danger has subsided.

The most profound group difference was observed in Empathy (EMP) ($p < 0.001$). In this context, the sharp reduction in empathy scores likely represents a temperamental correlate of "affective blunting" – a protective downregulation of emotional resonance often developed under conditions of chronic allostatic strain.

Collectively, these findings describe a phenotype where psychopathology is anchored in a generalized reduction of functional resources. The PTSD group is differentiated not merely by higher neuroticism, but by a specific inability to energize and flexibly adjust behavior, supporting the hypothesis of an asthenic reorganization of temperament.

3.2 Regulatory Mechanisms

The assessment of regulatory mechanisms – coping strategies and psychological defenses – revealed a systemic reorganization of stress management in the PTSD group, consistent with the Regulatory Regression hypothesis. Unlike the resilient combat-exposed group, veterans with PTSD exhibited a marked withdrawal from cognitively

demanding, flexible strategies in favor of more rigid, primitive patterns. This shift appears to be a global functional adaptation to chronic stress rather than a collection of isolated symptoms.

3.2.1. Coping Strategies (COPE)

In the domain of conscious coping, the most significant deficits appeared in executive, future-oriented functions. The PTSD cohort showed substantially reduced utilization of: Planning ($p < 0.001$) and Active Coping ($p < 0.001$).

These strategies inherently require sustained cognitive effort and the ability to project oneself into the future to organize goal-directed behavior. Their attenuation suggests a functional constriction of temporal perspective: behavior becomes increasingly reactive and anchored in the immediate present rather than guided by long-term objectives. Crucially, this pattern likely reflects not a lack of motivation or intentional avoidance, but a reduced accessibility of resource-intensive regulatory operations under heavy symptom load.

3.2.2. Defense Mechanisms (MIPZ) A parallel regression was observed in the hierarchy of unconscious defense mechanisms. The data indicate a "defensive economy" trade-off: a statistically significant reduction in mature mechanisms accompanied by a rise in primitive and immature defenses (Table 2).

Table 2. Shifts in Defense Mechanisms (MIPZ)

Defense Mechanism	Level	Direction (PTSD)	p-value	Clinical Interpretation
Humor	Mature	↓ Decrease	< 0.001	Reduced capacity for cognitive-affective reappraisal
Sublimation	Mature	↓ Decrease	0.04	Impaired transformation of aggressive impulses
Altruism	Mature	↓ Decrease	< 0.001	Withdrawal from pro-social regulatory roles
Omnipotent Control	Immature	↑ Increase	< 0.001	Rigid hyper-responsibility and vigilance
Regression	Primitive	↑ Increase	0.01	Reversion to somatic or developmentally earlier modes
Compulsive Behavior	Immature	↑ Increase	0.01	Ritualized binding of anxiety

This reconfiguration suggests that under conditions of chronic pathology, the psyche abandons metabolically expensive adaptive defenses (which require integration and flexibility) in favor of distortion-based strategies that offer immediate, albeit rigid, anxiety reduction.

The sharp increase in Omnipotent Control is particularly revealing. Clinically, this defense likely functions as a compensatory attempt to manage the unpredictability of trauma through absolute vigilance and an exaggerated sense of personal responsibility. However, this configuration appears to be inherently self-limiting. Maintaining a state of Omnipotent Control requires continuous attentional engagement and autonomic arousal. In the context of the reduced temperamental Endurance (Ergonicity) identified in this sample, reliance on such a "high-cost" defense risks accelerating resource depletion, potentially creating a maladaptive feedback loop between defensive rigidity and exhaustion.

3.3. Temperamental and Regulatory Correlates of Symptom Severity

Spearman correlation analysis within the PTSD cohort identified a distinct structural link between temperamental resources, regulatory mechanisms, and clinical severity (CAPS-5). The observed patterns indicate that symptom intensity is not random, but systematically covaries with specific deficits in functional capacity and regulation.

A significant inverse relationship emerged between Intellectual Ergonicity and overall PTSD severity ($\rho \approx -0.48$, $p < 0.001$). Veterans with lower capacity for sustained cognitive effort exhibited more profound clinical impairment. Functionally, this suggests that the processing of traumatic content requires mental stamina; when this "intellectual battery" is depleted, the cognitive integration of memories is compromised, leaving the individual vulnerable to intrusive symptoms.

Conversely, Neuroticism acted as a consistent predictor of distress, showing a strong positive correlation with CAPS-5 scores ($\rho \approx +0.52$, $p < 0.001$). This confirms its role as a dispositional amplifier: higher baseline emotional instability lowers the

threshold for stress tolerance, resulting in a heavier, more generalized symptom burden across all clusters.

The most striking associations appeared in the domain of psychological defenses, revealing a clear divergence based on maturity level. The use of Sublimation – a complex, adaptive defense – decreased sharply as symptom severity rose ($\rho = -0.63$). In contrast, reliance on Denial – a primitive, reality-distorting mechanism – showed a robust positive link with severity ($\rho = +0.66$). This points to a "regulatory economy" principle: severe PTSD appears incompatible with high-cost, cognitively demanding defenses, forcing a shift toward simpler, more rigid strategies that distort reality rather than process it.

Collectively, these associations outline a specific phenotype of severe PTSD. It is characterized not merely by the presence of trauma, but by a functional triad: depleted cognitive endurance, heightened emotional lability, and a regressive collapse of mature defenses. This suggests that the severity of the disorder is intrinsically tied to the availability of bio-behavioral resources needed to manage psychopathology.

3.4. The Collapse of Orthogonality

To determine whether PTSD involves a fundamental reorganization of psychological functioning rather than a simple accumulation of symptoms, we conducted exploratory factor analysis (PCA) separately for each cohort. This approach allowed us to compare the latent architecture of temperament, regulation, and clinical symptoms in resilient versus PTSD-affected veterans.

In the combat-exposed but resilient group, the analysis revealed a clear structural compartmentalization. Clinical symptom indicators (CAPS-5 and SCID-5 status) loaded onto a distinct, isolated factor (Factor 4), remaining statistically independent from core temperamental traits (Endurance, Plasticity) and regulatory mechanisms. This configuration suggests that in resilient veterans, post-traumatic stress manifestations operate as a relatively autonomous functional domain. While stress symptoms are

present, they do not structurally integrate with the veteran's core personality or regulatory systems. Essentially, the psychopathology remains "modular" – an overlay that does not disrupt the organizational integrity of the underlying temperament.

In sharp contrast, the PTSD group demonstrated a breakdown of this modular architecture. Instead of distinct factors, the structure was dominated by a massive first component – provisionally termed the Syndrome–Regulatory Factor – which accounted for a substantial proportion of the total variance. Within this dominant dimension, high PTSD symptom severity converged tightly with: Elevated Neuroticism; Significant deficits in Social and Intellectual Endurance (negative loadings); Reduced Plasticity; A reliance on primitive defense mechanisms (specifically Denial and Dissociation).

Comparing these two structures highlights a phenomenon we define as a collapse of orthogonality. In the absence of the disorder, constitutional traits and stress symptoms function as orthogonal (independent) dimensions. In chronic PTSD, this independence is lost. We observe a structural fusion where diminished endurance, rigid defenses, and clinical symptoms become mutually constitutive. This suggests that severe PTSD is not merely a high-intensity reaction superimposed on a healthy personality, but a systemic condition where regulatory deficits and pathology become entrenched within a single, self-reinforcing functional cycle.

4. Discussion

Our study proposes a shift in the conceptualization of combat-related PTSD, moving beyond a descriptive analysis of symptom clusters to an examination of the functional constraints that underlie vulnerability. By grounding the analysis in the Functional Ensemble of Temperament (FET) [6, 13], we demonstrate that severe post-traumatic pathology is closely tied to a depletion of neurobiological and regulatory resources. Specifically, diminished Ergonicity (Endurance) appears to be a critical factor; when the capacity for sustained activity is compromised, higher-order regulation fails, leading to a more severe clinical presentation.

This perspective reframes PTSD not merely as a consequence of maladaptive fear conditioning or cognitive errors, but as a condition defined by the insufficiency of bio-energetic support necessary for psychological stability. While the cross-sectional nature of the data precludes definitive causal claims, the structural coherence of these associations suggests that clinical outcomes are strictly limited by the patient's available energetic reserves [20], rather than by trauma exposure alone.

4.1. Ergonicity as a Functional Resource

Our findings reveal a consistent pattern of "hypo-ergonicity" across the PTSD cohort, characterized by a generalized reduction in endurance across motor, social, and intellectual domains. Within the FET model, this dimension serves as a psychometric proxy for the organism's capacity to sustain intensity and duration of activity. While the model theoretically grounds these traits in opioid and HPA-axis regulation [6], the clinical picture observed here points to a functional outcome: a systemic depletion of the resources required for sustained effort.

Combat exposure imposes an extreme allostatic load, demanding continuous mobilization of physical, cognitive, and emotional reserves. The profile observed in veterans with PTSD suggests a state of "allostatic bankruptcy," where the chronic demands of adaptation have outstripped available regulatory capacity [35]. Regardless of whether this reflects a pre-existing constitutional vulnerability or trauma-induced exhaustion, the functional result is identical: a compromised ability to maintain prolonged engagement with the environment.

This bio-energetic perspective offers a pragmatic reinterpretation of core PTSD symptoms. Clinical features traditionally categorized as avoidance, apathy, or depressive withdrawal can be understood as adaptive, energy-conserving strategies. High-order functions such as complex planning (Intellectual Ergonicity) and sustained empathy (Social Ergonicity) are metabolically expensive. Faced with systemic exhaustion, the nervous system appears to downregulate these resource-intensive processes to preserve

vital autonomic stability. Thus, the low Plasticity and Empathy scores observed in this study likely reflect secondary functional constraints imposed by an energy deficit, rather than primary defects in affect or character. This view aligns with emerging translational research linking PTSD to mitochondrial inefficiency [36], suggesting that the "asthenic" profile is a behavioral signature of physiological survival.

4.2. Regulatory Collapse

The observed shift in the PTSD group – away from mature regulation toward primitive strategies – provides strong empirical support for the hypothesis of "Regulatory Collapse." This phenomenon is best understood through the lens of bio-energetic economy: psychological regulation is not cost-free. Distinct strategies impose vastly different demands on an individual's limited cognitive and energetic resources.

Mature defenses, such as humor, sublimation, or anticipation, can be considered "expensive" [37]. They require high-level cognitive integration, the capacity to tolerate complexity, and sustained inhibitory control. These processes presuppose a surplus of Endurance and Plasticity. In contrast, primitive defenses like denial or omnipotent control are "cheap": they operate automatically to reduce immediate distress without requiring complex processing or deep integration [34].

This resource constraint effectively explains the marked deficit in Planning and Active Coping found in the clinical group. Planning requires the projection of the self into the future – a cognitively taxing operation that becomes inaccessible when functional reserves are critically low [24]. Consequently, the behavioral rigidity associated with low Plasticity should not be interpreted as motivational resistance or a character flaw. Instead, it represents a functional necessity: the nervous system "downgrades" to less demanding regulatory modes because it lacks the energetic capacity to sustain more adaptive, but metabolically costly, strategies.

From this perspective, resilience is not merely the presence of specific "good" defenses, but rather the maintenance of regulatory flexibility – the functional budget

required to deploy complex strategies as situational demands change. The present data suggest that in chronic PTSD, this budget is structurally compromised.

4.3. The Paradox of Omnipotent Control

The prominence of Omnipotent Control among veterans with PTSD serves as a critical bridge between psychodynamic constructs and the bio-energetic constraints of the FET model. Psychologically, this defense operates as a radical compensatory reaction to the helplessness inherent in combat: the individual unconsciously internalizes the belief that survival depends solely on their absolute vigilance and total personal responsibility [28].

However, from a functional perspective, this strategy proves paradoxically maladaptive. Maintaining a state of "omnipotent" readiness requires continuous autonomic activation and unrelenting attentional focus – processes that are metabolically expensive. In the context of the reduced Endurance (Ergonicity) observed in our sample, such high-cost regulation rapidly depletes the organism's remaining functional reserves. Consequently, a self-reinforcing cycle emerges: the strategy deployed to bind anxiety ultimately accelerates physiological exhaustion. This deepening fatigue destabilizes emotional regulation, thereby fueling further anxiety and compelling the individual to rely even more heavily on rigid control. This bio-behavioral trap likely contributes to the treatment resistance often seen in chronic PTSD, as the very mechanism intended for survival actively prevents the restoration of regulatory resources.

4.4. Structural Implications

The exploratory factor analysis highlighted a striking divergence in how psychological variables are organized within each group. In combat veterans without PTSD, trauma symptoms formed a discrete factor, statistically detached from temperament and regulatory traits. This structural independence suggests that even when stress symptoms are present, they remain compartmentalized; the stress response does not dismantle the individual’s core functional architecture.

In the PTSD cohort, however, this boundary dissolved. We observed a convergence where high symptom severity, diminished endurance, and rigid defenses loaded onto a single, dominant factor. We term this pattern the “Collapse of Orthogonality.” It represents a fundamental loss of separation between stable constitutional traits and state-dependent psychopathology, where the disorder is no longer an isolated reaction but has become intertwined with the personality structure itself.

Consequently, relying solely on symptom intensity offers an incomplete clinical picture. Two veterans may present with identical CAPS-5 scores [38] yet possess fundamentally different structural organizations. We propose that the preservation of orthogonality serves as a latent marker for resilience and treatment readiness, whereas its collapse signals a systemic, self-reinforcing condition that may require a distinct therapeutic strategy.

4.5. Clinical Implications

The observed coupling of temperamental exhaustion with symptom severity suggests that the timing of treatment may be as critical as the modality itself. While evidence-based trauma interventions – such as Prolonged Exposure, CPT, or EMDR – are highly effective [39, 40], they are not metabolically neutral. These therapies demand sustained cognitive effort, inhibitory control, and emotional tolerance. For veterans presenting with the specific "asthenic" profile identified in this study, where functional endurance is compromised and regulatory orthogonality is collapsed, the demand-to-resource ratio may be unfavorable. In such cases, premature confrontation with traumatic material risks precipitating resistance, dropout, or further destabilization.

Consequently, the FET framework supports a functional logic for sequencing therapy, moving from physiological restoration to cognitive integration [21]:

1. Stabilization and Bio-energetic Restoration. Before engaging top-down processing, the clinical priority is to rebuild the bottom-up energetic baseline. This phase

focuses on normalizing sleep architecture, autonomic regulation, and sensorimotor grounding to interrupt the cycle of physiological depletion.

2. Enhancing Regulatory Flexibility. Once basic endurance improves, therapy can target the rigidity of the "control-based" defenses identified in our results. The goal here is not yet trauma processing, but rather helping the patient recognize the high metabolic cost of hypervigilance and developing less energy-intensive strategies for managing anxiety.

3. Trauma Processing. Direct exposure and narrative integration are introduced only after sufficient functional reserves are re-established and the patient demonstrates the capacity to separate the self-structure from symptom reactivity.

This staged progression is proposed here as a theoretical heuristic derived from the structural analysis, offering a hypothesis for future clinical trials to test whether resource-first sequencing improves outcomes in severe, chronic cases.

4.6 Limitations and Future Directions

The interpretation of these findings is constrained by the cross-sectional nature of the study, which restricts our ability to definitively distinguish between pre-existing temperamental vulnerabilities and functional alterations resulting from trauma exposure. Consequently, it remains unclear whether the observed low endurance acts as a constitutional diathesis for PTSD or represents a “scar” of chronic allostatic load. Resolving this directionality requires prospective research designs that track service members from pre-deployment baselines through the reintegration phase [41].

Furthermore, while the FET framework is rooted in neurobiological theory, the present study relied exclusively on psychometric assessment. To move beyond descriptive associations, future investigations should aim to validate self-reported endurance against objective physiological data. Integrating STQ-77 profiles [15] with biomarkers such as HPA axis dynamics, inflammatory markers, or mitochondrial efficiency would allow for a more precise characterization of the biological substrates

underlying regulatory collapse. Finally, as the current sample was restricted to male combat veterans, testing the generalizability of the proposed structural model in female service members and diverse civilian trauma populations remains a critical priority for future work.

5. Conclusion

This study advances a bio-behavioral perspective on combat-related PTSD, challenging the notion that the disorder is solely a psychological reaction to trauma exposure. Our findings indicate that symptom severity in veterans is intrinsically linked to specific functional constraints within the Functional Ensemble of Temperament (FET) – namely, a deficit in Endurance (hypo-ergonicity) and reduced Plasticity. Rather than reflecting mere personality traits, these parameters appear to signify a systemic limitation in the nervous system's capacity to sustain cognitive and social effort under chronic allostatic load.

A key insight concerns the "economy" of regulation. The observed shift toward primitive defense mechanisms, particularly the dominance of Omnipotent Control, can be understood as a forced adaptation to these energetic constraints. When bio-behavioral resources are depleted, the regulatory system seemingly reverts to "cheaper," more rigid strategies to maintain stability, sacrificing flexibility for immediate anxiety reduction.

The most significant structural finding is the phenomenon we identify as a "collapse of orthogonality." While resilient veterans maintained a statistical separation between their core temperamental traits and stress symptoms – suggesting a modular containment of trauma – this structural barrier appears to dissolve in chronic PTSD. In the clinical group, temperament, regulation, and symptom severity converged into a single fused dimension. This implies that in severe PTSD, pathology is no longer superimposed upon the personality but becomes structurally intertwined with it, creating a self-reinforcing cycle of depletion and distress.

These structural and functional insights hold direct clinical implications. If

resilience is constrained by bio-energetic capacity, then standard trauma-focused interventions (which are cognitively demanding) may be premature for patients with a "collapsed" profile. The data support a staged therapeutic logic: treatment should prioritize the restoration of basic regulatory resources – sleep, autonomic stability, and endurance – before engaging in intensive trauma processing. By aligning therapeutic demands with the patient's functional reality, clinicians may reduce the risk of destabilization and improve long-term outcomes.

In summary, this research moves beyond descriptive epidemiology to propose a functional mechanism of PTSD vulnerability. By integrating the FET framework with the analysis of defensive organization, we highlight how the erosion of constitutional resources underpins the persistence of combat trauma, pointing toward a need for treatments that address not just the memory of the event, but the physiological capacity to process it.

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