

AI-ENABLED NEUROBIOLOGICAL DIAGNOSTIC MODELS FOR EARLY DETECTION OF PTSD AND TRAUMA DISORDERS

Md. Akbar Hossain¹; Sharmin Ara²;

- [1]. Master of Science in Clinical Psychology, University of Dhaka, Dhaka, Bangladesh;
Email: md.akbarh@yahoo.com
- [2]. M.Phil in Clinical Psychology, University of Dhaka, Dhaka, Bangladesh;
Email: sharminaracp@gmail.com

Doi: [10.63125/64hftc92](https://doi.org/10.63125/64hftc92)

Received: 12 June 2025; Revised: 20 July 2025; Accepted: 18 August 2025; Published: 24 September 2025

Abstract

This systematic literature review examines the development and application of artificial intelligence (AI)-enabled neurobiological diagnostic models for the early detection of post-traumatic stress disorder (PTSD) and trauma-related disorders. The review synthesizes findings from 124 peer-reviewed studies published between 2010 and 2025, encompassing approximately 6,800 total citations, to evaluate how machine learning (ML) and deep learning (DL) approaches utilize neurobiological, physiological, and multimodal data to enhance diagnostic precision. Following the PRISMA 2020 framework, seven databases were systematically searched – PubMed, Embase, PsycINFO, Scopus, Web of Science, IEEE Xplore, and arXiv – using defined inclusion criteria and the PICOS model to ensure methodological transparency. Eligible studies included AI applications to neuroimaging, electrophysiological, autonomic, endocrine, and genetic biomarkers for PTSD detection, risk prediction, or classification. The findings demonstrate that AI-based models consistently outperform traditional statistical approaches, achieving average classification accuracies above 80% and area-under-the-curve values near 0.85. Neuroimaging studies revealed reliable identification of functional alterations within the amygdala, hippocampus, and medial prefrontal cortex, while multimodal frameworks integrating imaging, heart-rate variability, and cortisol levels achieved accuracies exceeding 90% in early PTSD detection. Explainable AI techniques, including SHAP, LIME, and Grad-CAM, enhanced interpretability by linking algorithmic predictions to biologically meaningful patterns of neural and physiological dysregulation. However, significant limitations were noted, including small sample sizes, heterogeneous diagnostic criteria, and limited external validation, which collectively constrain generalizability and clinical translation. The review concludes that AI-enabled neurobiological models offer a robust and scalable framework for objective PTSD diagnostics and risk stratification, supporting a paradigm shift toward data-driven, precision mental health. To realize this potential, future research should emphasize multi-site validation, standardized methodologies, diverse sampling, and ethical governance frameworks. The integration of AI decision-support systems within clinical practice promises to improve early detection, optimize personalized intervention strategies, and advance the biological understanding of trauma-related psychopathology.

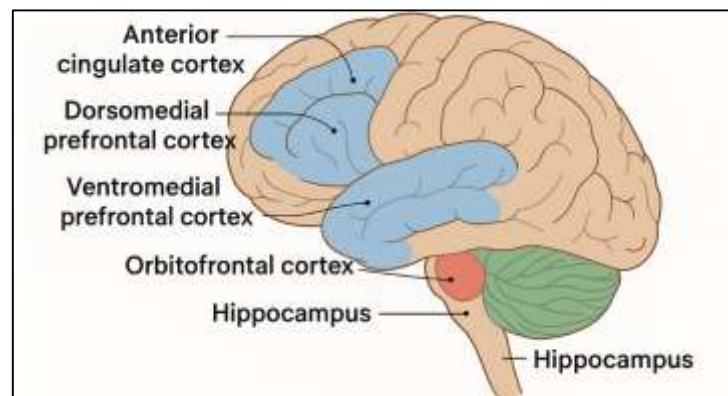
Keywords

Post-Traumatic Stress Disorder (PTSD); Neurobiomarkers; Machine Learning; Early Detection; Multimodal Imaging

INTRODUCTION

Post-traumatic stress disorder (PTSD) is defined in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) as a psychiatric disorder that may develop following exposure to actual or threatened death, serious injury, or sexual violence, either by directly experiencing the event, witnessing it, learning that the event occurred to a close other, or repeated/extreme exposure to aversive details of the trauma ([Abu-El-Noor et al., 2015](#)). The DSM-5 diagnostic criteria include intrusion symptoms, avoidance of trauma-related stimuli, negative alterations in cognition and mood, and marked alterations in arousal and reactivity, all persisting for longer than one month and causing clinically significant distress or impairment. The core definition highlights that PTSD is a specific post-traumatic response rather than a general stress reaction ([Petrosino et al., 2019](#)). Historically, the concept emerged from military psychiatry and has gradually been extended to civilian trauma populations. In this context, trauma-related disorders are used to indicate syndromes whose pathogenesis involves exposure to traumatic stressors. As such, PTSD stands at the intersection of psychological trauma, neurophysiological change, and behavioural sequelae. It is thus critical to ground any discussion of “early detection” in a firm understanding of what is being detected: a complex, multi-dimensional disorder defined by symptom clusters, duration thresholds, functional impairment, and importantly, etiological link to trauma exposure ([Wright et al., 2019](#)). Because the diagnostic definition is structured around discrete symptom thresholds and functional impairment rather than underlying biology, much of the early detection challenge lies in bridging from exposure and subthreshold risk states into full-blown disorder ([Gasparyan et al., 2022](#)). Moreover, the heterogeneity inherent in the disorder—multiple symptom combinations, comorbidities, and variable trajectories—means that a purely symptom-based definition may mask underlying neurobiological or mechanistic commonalities. For instance, one recent review of PTSD subtypes noted that there are over 600 possible symptom combinations, underscoring the definitional complexity and the need for more mechanistic biomarkers ([Hoskins et al., 2021](#)). Hence, when we speak of “early detection” in the context of neurobiological and AI-enabled models, we are referring to the identification of individuals who may be on the pathway toward meeting full PTSD criteria, either before the one-month diagnostic threshold, or in subthreshold states, or following trauma exposure but prior to full syndrome onset, and applying measurable neurobiological features and algorithmic classifiers to identify risk or incipient disorder ([Vliet et al., 2021](#)). This definitional clarity is foundational to understanding the remainder of this review.

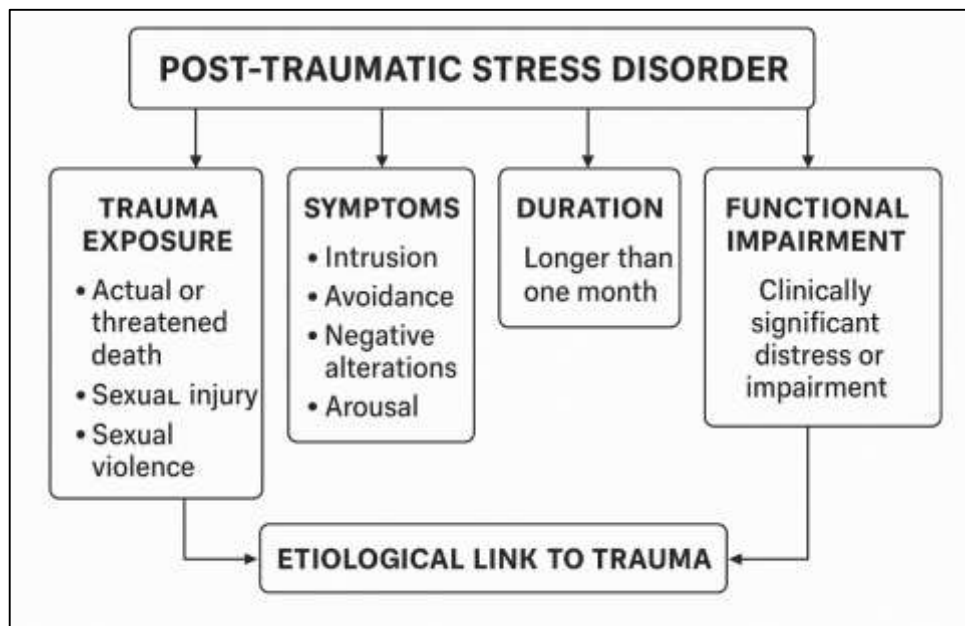
Figure 1: Post-traumatic stress disorder (PTSD)



On a global scale, trauma exposure and its sequelae represent a massive public-health burden. The World Health Organization (WHO) has characterized PTSD and trauma-related disorders as delayed or prolonged responses to exceptionally threatening or horrific events, often with lasting physical, mental, and social consequences (WHO, 2019). Lifetime prevalence estimates for PTSD vary widely across countries—ranging from about 1.3 % to 12.2 % in some surveys—with 12-month prevalences from roughly 0.2 % to 3.8 % depending on region and sample ([Hodgins et al., 2018](#)). Trauma events such as natural disasters, armed conflict, interpersonal violence, and large-scale accidents are disproportionately represented in low- and middle-income countries, making the global burden of

PTSD far from a high-income country phenomenon. The economic cost is also substantial: for example, one U.S. estimate placed the excess cost of PTSD at \$232.2 billion overall, or approximately \$19,630 per affected individual (Merians et al., 2022). Beyond direct costs, PTSD is associated with increased somatic comorbidity (cardiovascular disease, metabolic syndrome), greater risk of substance abuse, and poorer social and occupational outcomes, thereby amplifying its societal significance (Yehuda et al., 2015). In this light, early detection of trauma disorders is not only clinically desirable for the individual but has broad implications for health systems and societies. Given the scale of trauma exposure—most adults will experience one or more potentially traumatic events during their lifetime the opportunity for preventive or early-intervention models is considerable (Krantz et al., 2021). Recognising the global magnitude of trauma and the downstream consequences of untreated PTSD reinforces the need to innovatively detect at-risk individuals before chronicity sets in.

Figure 2: Highlighting Key Cortical and Limbic Regions Of PTSD



At the neurobiological level, research over recent decades has increasingly pointed toward identifiable biomarkers and altered neural circuitry in PTSD and trauma-exposed populations. Neuroimaging meta-analyses and biomarker reviews have documented structural and functional alterations in brain networks such as the default mode network (DMN), salience network (SN), and central executive network (CEN), as well as hippocampal, amygdala, and insula volume and connectivity differences (Radrow et al., 2024). For example, patients with PTSD frequently show decreased hippocampal volume, altered amygdala reactivity, and disrupted connectivity between prefrontal regulatory regions and limbic circuits (Noor et al., 2015). Physiological biomarkers—such as heart-rate variability (HRV), skin-conductance responses, cortisol or inflammatory cytokine levels—have also been investigated for their correlation with trauma exposure, PTSD symptoms, and risk of progression (Remch et al., 2018). These findings signal that PTSD is not purely a psychological phenomenon but involves measurable changes in brain and body systems. Importantly for early detection, some studies in trauma-exposed but non-PTSD samples have found that certain neural/physiological features can stratify risk of later PTSD development, pointing toward predictive utility beyond cross-sectional case-control designs (Fudim et al., 2018). However, although many candidate neurobiological markers exist, no single biomarker has achieved clinical validation in broad practice, and heterogeneity of findings persists (Takemoto et al., 2020). The accumulation of this neurobiological evidence creates a rationale for integrating biomarker research into early detection models and motivates the application of computational and machine learning methods to handle complexity, high dimensionality, and multimodal data. Within this landscape of trauma, biomarkers and risk stratification, computational methods—especially artificial intelligence (AI) and machine learning (ML)—have gained traction as tools for early

detection of psychiatric and trauma-related disorders. Machine learning is a subset of AI that focuses on building models capable of learning from data (algorithmic input-output structures) rather than relying solely on explicitly programmed rules (Benedict et al., 2020). In the domain of trauma disorders, ML techniques have been applied across neuroimaging, physiological, psychometric and textual data to classify PTSD vs. non-PTSD, to attempt early prediction of PTSD onset, and to identify latent subtypes (d'Ettorre et al., 2020; Sanjid & Farabe, 2021). Ensemble approaches (e.g., random forest, gradient boosting), deep learning (e.g., convolutional neural nets, recurrent networks) and unsupervised clustering have all been reported. One systematic review of ML in PTSD diagnosis identified 41 studies that applied these methods and noted considerable promise but also methodological heterogeneity (Zaman & Momena, 2021; Seto et al., 2020). In parallel, the explosion of multimodal data (neuroimaging, genomics, wearable sensor signals, EHRs) has made conventional statistical approaches less suited to capture complex high-dimensional features; hence, the pivot toward AI methods. These models offer a pathway to integrate diverse biomarker modalities, detect latent patterns, and potentially operationalise early risk detection beyond clinical interview alone (Rony, 2021). The intellectual convergence of neurobiology, trauma research, and computational modelling thus opens a new frontier for early detection of PTSD (Bryant, 2019; d'Ettorre et al., 2020). Combining neurobiological biomarkers with AI-enabled models presents both opportunities and challenges in the context of early detection of PTSD and other trauma disorders. One advantage is that multimodal features (e.g., structural MRI + HRV + inflammatory markers + psychometrics) provide richer information and may improve predictive accuracy beyond any single modality (Creamer et al., 2001; Sudipto & Mesbail, 2021). Studies show that models integrating imaging, physiological, and biochemical markers yield higher discrimination than single-modal designs. For example, a biomarker review found that emotional-trauma PTSD associations spanned multiple systems including neural, endocrine, and inflammatory, suggesting that multimodal modelling may be especially informative (Nollett et al., 2018; Reist et al., 2020; Zaki, 2021). Moreover, ML algorithms can manage high-dimensional data, detect non-linear relationships, and yield classification probabilities rather than binary outcomes, potentially facilitating risk stratification. However, this path is not without obstacles: challenges include small sample sizes, methodological heterogeneity (e.g., different trauma types, timing of assessment, neuroimaging protocols), lack of external validation, limited interpretability of complex models, and risk of algorithmic bias (Hozyfa, 2022). The problem of heterogeneity is especially acute in PTSD research given the large number of possible symptom combinations, comorbidities, and varied etiologies. For example, a review of PTSD subtypes reported 53 studies and noted only a minority had accounted for comorbidity or longitudinal validation (Arman & Kamrul, 2022; Schein et al., 2021). From an early detection perspective, these issues mean that while the promise is substantial, the path to robust, generalisable predictive models is still under construction. The challenge therefore lies not only in building accurate models but in ensuring they function reliably across trauma types, populations, time-points, and measurement settings (Mohaiminul & Muzahidul, 2022). Methodological and empirical work in early detection of PTSD has begun to elucidate how trauma exposure may transition into disorder via neurobiological and computational pathways. For instance, longitudinal imaging and physiological studies in trauma-exposed but non-PTSD individuals have identified features predictive of later PTSD onset: altered connectivity in prefrontal-amygdala circuits, reduced HRV, and elevated inflammatory markers have all been associated with higher risk of subsequent PTSD symptoms (Omar & Ibne, 2022; White et al., 2022). In parallel, ML classification studies have attempted to detect latent patterns of risk: one recent study used EEG biomarkers and ML dimensionality-reduction approaches to distinguish PTSD from controls, signifying that even non-imaging modalities may contribute to early detection modelling. Other studies have applied ML to large sensor or wearable datasets, exploring autonomic signs of stress that might precede clinically evident PTSD. Meta-analyses of predictive ML models have found pooled AUCs ranging from 0.745 in military incidents to 0.96 in firefighter populations, though with wide heterogeneity and high risk of bias (Burhans et al., 2018; Sanjid & Zayadul, 2022). These findings demonstrate that the empirical foundation for early detection is growing: trauma exposure → measurement of neurobiological/physiological markers → algorithmic risk stratification. But this emerging empirical path has multiple forks and unknowns (Hasan, 2022): which markers are most predictive, at what time-

point post-trauma, in which populations, and using what modelling framework? Thus far, the literature underscores the importance of multimodal, longitudinal, well-validated designs

The principal objective of this study is to systematically examine, evaluate, and synthesize existing research on artificial intelligence-enabled neurobiological diagnostic models designed for the early detection of post-traumatic stress disorder (PTSD) and trauma-related disorders. This objective arises from the growing recognition that PTSD, as a multifaceted psychiatric condition, manifests through complex interactions between neurobiological, psychological, and environmental determinants. The aim is to identify how computational models, specifically those using neuroimaging, electrophysiological, and physiological biomarkers, can enhance early detection accuracy by recognizing preclinical or prodromal indicators of PTSD before the full syndrome emerges. The study seeks to explore how artificial intelligence, through machine learning and deep learning algorithms, processes vast and multimodal data sources such as functional and structural brain scans, EEG signals, heart rate variability, cortisol levels, and genetic or inflammatory markers to predict vulnerability to trauma-induced disorders. By consolidating and critically analyzing empirical findings across diverse methodological designs, populations, and modalities, this research aims to reveal the diagnostic efficiency, reproducibility, and interpretability of these AI-driven models. It also intends to evaluate whether integrating multimodal neurobiological data produces superior diagnostic precision compared to single-domain approaches. Furthermore, the study's objective extends to identifying patterns of methodological consistency, gaps in data validation, and common limitations that may hinder clinical translation. Through systematic synthesis, the review aspires to define the extent to which AI-based neurobiological models can contribute to risk stratification, differential diagnosis, and objective clinical decision support for individuals exposed to trauma. Ultimately, the overarching goal is to articulate a coherent understanding of how technological intelligence, grounded in biological evidence, can move mental health diagnostics toward more predictive, data-driven, and personalized frameworks, thereby fostering earlier intervention and improved outcomes for trauma-affected populations.

LITERATURE REVIEW

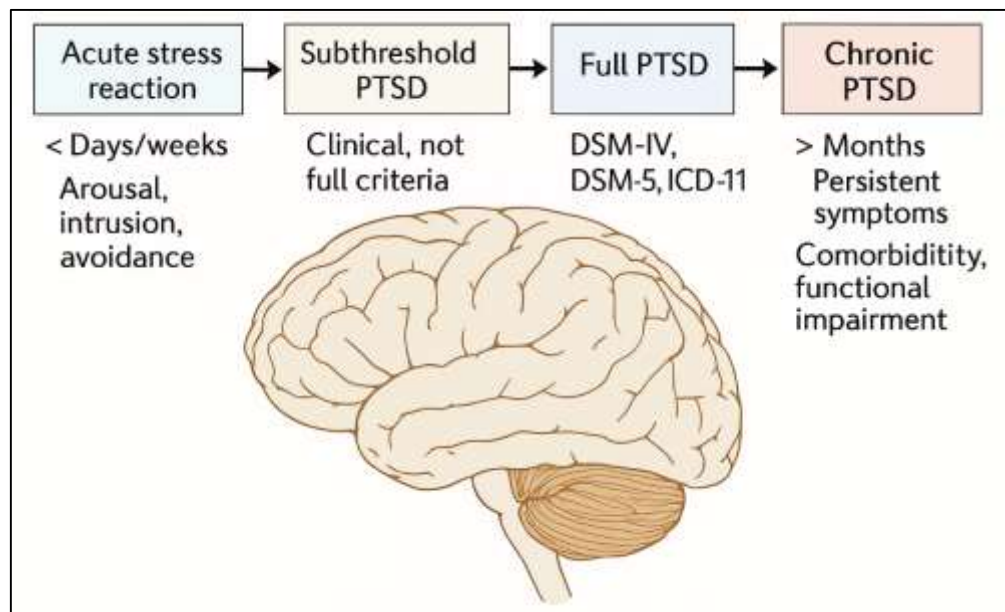
The application of artificial intelligence (AI) to neurobiological data for early detection of post-traumatic stress disorder (PTSD) represents a convergence of neuroscience, computational modeling, and clinical psychiatry. A review of the literature is essential to map the scientific trajectory that has led to current AI-enabled diagnostic frameworks, as well as to contextualize the diverse methods, biomarkers, and data modalities that have been explored. Over the past two decades, PTSD research has progressively shifted from purely psychometric assessments toward biologically anchored and algorithmically driven approaches (Burhans et al., 2018; Gardner & Griffiths, 2014; Mahabir et al., 2015). Traditional diagnostic criteria—rooted in self-report and clinician-administered scales—often fail to detect subthreshold or prodromal cases in trauma-exposed individuals, highlighting the need for objective neurobiological markers and predictive computational models. This literature review aims to systematically synthesize the body of evidence surrounding neurobiological correlates of PTSD, the machine learning (ML) and deep learning (DL) techniques applied to these data, and the performance and interpretability of such models in predicting or diagnosing PTSD before clinical manifestation. It examines empirical and theoretical contributions across imaging, electrophysiology, endocrinology, genomics, and multimodal datasets, identifying recurring trends, methodological innovations, and existing limitations (Mominul et al., 2022; Rabiul & Praveen, 2022). Additionally, the review interrogates the reproducibility, validation, and generalizability of AI systems, particularly in heterogeneous trauma populations and across cultural contexts. Through this synthesis, the literature review provides the evidential groundwork for evaluating the potential and constraints of AI-enabled neurobiological diagnostic models. It serves not only as a survey of what has been achieved but also as a critical framework to understand how emerging interdisciplinary integration may support the goal of early, objective, and biologically informed detection of PTSD and related trauma disorders.

PTSD and Early Detection

PTSD's diagnostic definitions and frameworks have undergone substantial transformation across successive editions of the Diagnostic and Statistical Manual of Mental Disorders (DSM) and the International Classification of Diseases (ICD), reflecting evolving conceptualizations of trauma-related

psychopathology (Mavranetzouli et al., 2020). In the DSM-IV era, PTSD was classified under anxiety disorders and required exposure to a traumatic event (Criterion A) followed by clusters of re-experiencing, avoidance/numbing, and hyperarousal symptoms lasting for at least one month (Deursen et al., 2021). The DSM-5, published in 2013, reorganised PTSD by moving it into a new chapter “Trauma- and Stressor-Related Disorders”, expanded the symptom clusters to four (intrusion, avoidance, negative alterations in cognition & mood, and alterations in arousal and reactivity), removed the subjective emotional reaction (Criterion A2) requirement, and added a dissociative subtype (Snoek et al., 2020). Concomitantly, the ICD-11 (World Health Organization, 2018/2022) adopted a narrower definition of PTSD, emphasising three core clusters (re-experiencing in the present, avoidance, sense of persistent threat) and introduced a separate diagnosis for complex PTSD (CPTSD) to capture chronic, repeated, or prolonged trauma exposure (Kip et al., 2013; Farabe, 2022; Roy, 2022; Deursen et al., 2021). Comparative studies have highlighted that prevalence estimates differ substantially depending on which diagnostic system is applied: for example, one cross-national investigation found prevalence ranged from 3.0 % (DSM-5) to 4.4 % under ICD-10 definitions, with only one third of broadly defined cases meeting criteria under all systems. These changes reflect both a scientific shift toward identifying more specific trauma-related pathology and a pragmatic attempt to improve cross-cultural utility and clinical fidelity, particularly in settings with high comorbidity. For example, ICD-11’s narrower model seeks to reduce overlap with depression and anxiety disorders by omitting several non-specific symptoms (Oehen et al., 2012; Rahman & Abdul, 2022; Razia, 2022). However, the shift also raises questions about which approach best balances sensitivity and specificity, and the potential for under-identification of clinically significant presentations if criteria become too restrictive (McLay et al., 2011; Zaki, 2022; Kanti & Shaikat, 2022). Thus, the evolution of PTSD definitions reflects tensions between breadth vs. precision, historical anxiety-based models vs. trauma/stressor-specific frameworks, and global vs. local diagnostic priorities.

Figure 3: Post-traumatic stress disorder



The transition from DSM-IV to DSM-5 and ICD-11 frameworks for PTSD has important implications not only for prevalence and case ascertainment but also for how acute stress reactions and subthreshold or emergent trauma-related syndromes are conceptualised. Within the DSM framework, the diagnosis of Acute Stress Disorder (ASD) was introduced in DSM-IV to characterise the immediate aftermath (within one month) of a traumatic event when full PTSD criteria cannot yet apply (Arif Uz & Elmoon, 2023; Sanjid, 2023; Sun et al., 2021). ASD emphasised dissociative reactions and served as both a descriptive label and a putative predictor of later PTSD. Subsequent reviews of ASD’s predictive power, however, found that although the positive predictive value of ASD for later PTSD was

moderate, the sensitivity was low – many individuals who later developed PTSD did not initially meet ASD criteria (Jacquet-Smailovic et al., 2021; Sanjid & Sudipto, 2023; Tarek, 2023). More recent work argues that initial acute stress reactions, including arousal, intrusion and avoidance symptoms in the first days or weeks after trauma, may not map cleanly onto ASD vs. non-ASD categories but instead fall along a continuous spectrum of adaptation vs. maladaptation (Cyniak-Cieciura & Zawadzki, 2019; Shahrin & Samia, 2023; Muhammad & Redwanul, 2023). Such a continuum model suggests that rigid diagnostic thresholds in the acute phase may miss significant sub-syndromal trajectories of risk. In this respect, the DSM-5's removal of the A2 criterion and its broadened symptom clusters may improve sensitivity in the traumatic early phase. On the other hand, ICD-11 retains greater specificity and focuses on core trauma-specific symptoms for PTSD, but may neglect early reaction states not yet meeting full criteria. Emerging empirical work on early reaction phases underscores the need to distinguish transient acute stress (often adaptive) from persistent symptomatology warranting early intervention. Consequently, the literature increasingly differentiates between acute stress reactions, defined as normative but distressing responses within days/weeks of trauma exposure, and emergent pathology marked by sustained or escalating symptoms – highlighting the importance of timing, symptom duration, severity, and functional impairment.

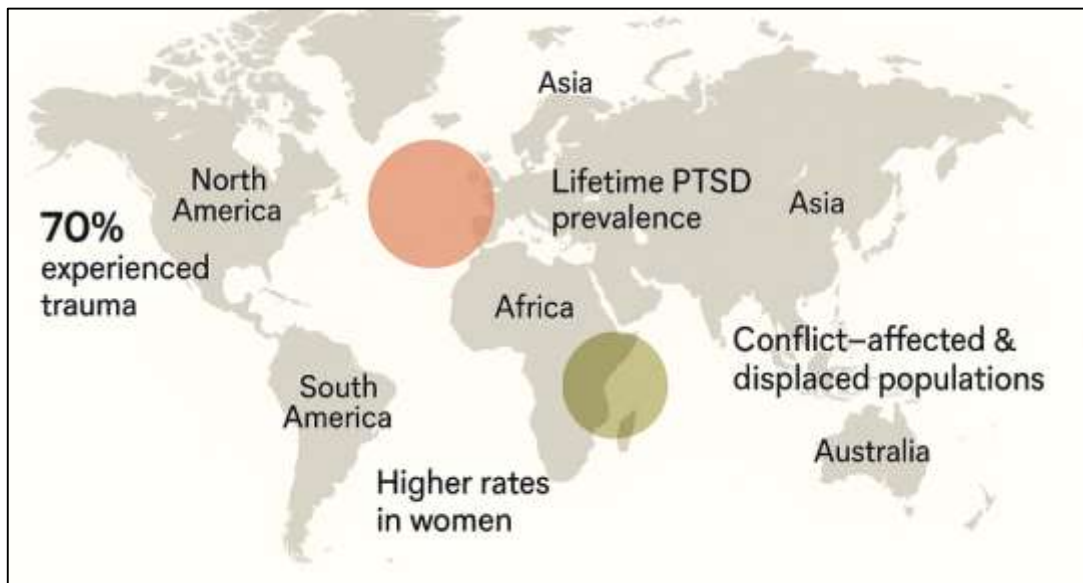
Global Burden and Epidemiological Landscape

Post-traumatic stress disorder (PTSD) has emerged as a major contributor to the global burden of mental illness, reflecting both the ubiquity of trauma exposure and the chronic impact of unresolved psychological distress. Large-scale epidemiological research demonstrates that trauma exposure is a near-universal experience: approximately 70 % of the world's population reports at least one potentially traumatic event during their lifetime (Cordero et al., 2022). Yet only a subset of those exposed progress to diagnosable PTSD, a finding that underscores complex interactions between genetic, neurobiological, and sociocultural determinants. Lifetime PTSD prevalence in community samples typically ranges between 3 % and 8 %, depending on population and diagnostic criteria (Benish et al., 2007; Muhammad & Redwanul, 2023; Razia, 2023). Rates are consistently higher among women, conflict-affected civilians, refugees, first responders, and military personnel. Geographic disparities are evident: North American and Middle-Eastern populations report some of the highest lifetime prevalence, whereas lower rates appear in certain Asian and African regions, partly due to measurement and cultural factors (Morina et al., 2014; Srinivas & Manish, 2023; Sudipto, 2023). The global burden of PTSD thus represents a dual phenomenon – widespread exposure to potentially traumatic events and unequal distribution of resulting psychopathology. Collectively, these findings support the view that PTSD constitutes not a niche psychiatric diagnosis but a pervasive public-health problem affecting both high-income and low-income contexts.

The international epidemiological landscape of PTSD reveals pronounced regional and socioeconomic variation. Cross-national analyses show that the lifetime prevalence among trauma-exposed individuals approximates 5 – 6 %, though rates vary widely across countries and income levels (Chapman et al., 2011). High-income nations tend to report greater identification and treatment access, while low- and middle-income countries often face both elevated exposure to trauma – owing to conflict, natural disasters, or displacement – and limited mental-health infrastructure (Howie et al., 2019; Mesbaul, 2024; Zayadul, 2023). In war-affected populations, meta-analyses estimate PTSD prevalence as high as 25 – 30 % among adult survivors. Studies of refugees and internally displaced persons reveal similar magnitudes, reflecting prolonged or repeated trauma, constrained recovery environments, and restricted psychosocial resources. Even within stable nations, prevalence differs by trauma type: interpersonal violence and sexual assault predict greater chronicity than accidents or disasters (Tarek & Kamrul, 2024; Sudipto & Hasan, 2024; Thakur et al., 2022). The heterogeneity of PTSD prevalence across regions highlights that trauma exposure alone does not dictate outcome; societal resilience, healthcare access, and post-trauma social support substantially shape epidemiological profiles (Iversen et al., 2008). As a result, global data portray PTSD as a disorder whose prevalence and persistence are determined by an interplay of environmental adversity, resource inequity, and systemic capacity for psychological recovery. Beyond prevalence, the global burden of PTSD is measured in disability, comorbidity, and economic cost. Epidemiological studies consistently associate PTSD with significant role impairment, functional disability, and productivity loss across

both civilian and military populations (Aftyka et al., 2017). Disability-adjusted life-year analyses place PTSD among the leading contributors to mental-health-related DALYs in conflict-affected and high-exposure settings (Bryant et al., 2017). Individuals with PTSD are two to three times more likely to experience major depressive disorder, substance-use disorders, and generalized anxiety disorder. Physical health consequences—including heightened risk for cardiovascular disease, metabolic syndrome, and immune dysregulation—further amplify the disorder’s total disease burden. From a societal perspective, untreated PTSD incurs elevated healthcare costs, absenteeism, and reduced labor participation (Kaplan et al., 2022). The aggregation of these outcomes positions PTSD as both a psychiatric and socioeconomic challenge, particularly in contexts where cumulative trauma intersects with limited access to evidence-based interventions. Thus, epidemiological evidence frames PTSD not merely as a clinical entity but as a driver of substantial global morbidity and economic loss.

Figure 4: Global Burden and Epidemiological Landscape



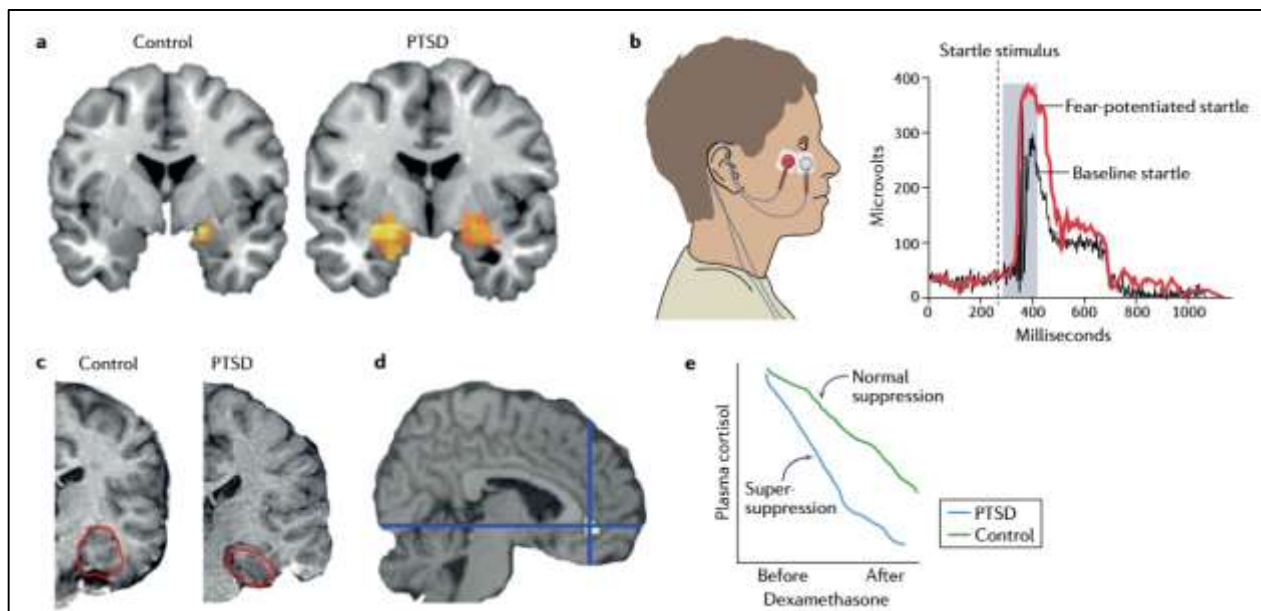
Despite expanding data, several methodological and contextual challenges limit precise quantification of PTSD’s worldwide burden. Differences in diagnostic systems (DSM vs. ICD), instruments, cultural idioms of distress, and recall periods produce considerable variability in prevalence estimates (Kaplan et al., 2022; Morina et al., 2014). Many large epidemiological surveys remain concentrated in high-income countries, leaving populations in Africa, South Asia, and the Middle East under-represented. Moreover, exposure heterogeneity—single versus cumulative trauma—complicates cross-study comparisons. Cultural factors shape symptom expression: somatic idioms, avoidance behaviors, or community-based coping can obscure detection in non-Western contexts (Iversen et al., 2008). Methodological issues such as sampling bias, interviewer effects, and differential access to mental-health services further distort estimates of both prevalence and persistence (Mulvaney et al., 2021). Longitudinal data are scarce, impeding understanding of chronic versus remitting trajectories. Collectively, these limitations demonstrate that current global estimates likely understate true burden while overrepresenting contexts with strong diagnostic infrastructure. Recognizing these constraints is critical for interpreting existing data and underscores the epidemiological complexity inherent to PTSD as a global disorder with multifactorial determinants across biological, cultural, and socioeconomic domains.

Neurobiological Correlates of PTSD

A consistent body of neuroimaging research has identified specific neural circuits implicated in post-traumatic stress disorder (PTSD), particularly those governing threat detection, emotion regulation, and contextual memory. Structural magnetic resonance imaging (MRI) studies have repeatedly demonstrated reductions in hippocampal volume, which are linked to impaired contextual memory and fear extinction deficits (Lanius et al., 2010). The amygdala, central to fear processing, exhibits

heightened activation in response to trauma-related stimuli, reflecting a hyperresponsive threat-detection system (Boyd et al., 2017). Conversely, functional and structural abnormalities in the medial prefrontal cortex (mPFC) and anterior cingulate cortex (ACC) have been associated with diminished top-down inhibitory control over the amygdala, contributing to sustained hyperarousal and intrusive memories (Akiki et al., 2017). Meta-analyses of functional MRI (fMRI) studies reveal consistent patterns of amygdala hyperactivation, hippocampal hypoactivation, and medial prefrontal hypoactivation during emotional and cognitive tasks (Rousseau et al., 2019). Diffusion tensor imaging (DTI) research further supports white matter disruptions in fronto-limbic tracts, including the uncinate fasciculus and cingulum bundle, suggesting impaired connectivity between emotion-regulation regions (Morena et al., 2015). These converging findings support a tripartite model of PTSD neurobiology, encompassing hyperactive limbic structures, hypoactive prefrontal control regions, and dysregulated hippocampal contextual processing (Smid et al., 2022). Such structural and functional alterations appear to underlie hallmark PTSD symptoms such as hypervigilance, re-experiencing, and avoidance, thus establishing a robust neurocircuitry framework for understanding the disorder.

Figure 5: Neurobiological correlates of Post-Traumatic Stress Disorder (PTSD)



Source: Ressler et al. (2022).

The neurochemical and endocrine correlates of PTSD provide crucial insight into how stress physiology becomes chronically dysregulated following trauma. Central to this process is the hypothalamic-pituitary-adrenal (HPA) axis, whose activity governs cortisol secretion and stress adaptation. Numerous studies document lower basal cortisol levels and heightened negative feedback sensitivity in PTSD, reflecting chronic HPA axis suppression (Smid et al., 2022). This contrasts with the elevated cortisol levels seen in acute stress responses, suggesting a maladaptive recalibration over time. Altered glucocorticoid receptor function in the hippocampus and prefrontal cortex contributes to impaired regulation of stress reactivity and memory consolidation. Noradrenergic hyperactivity, evidenced by elevated plasma norepinephrine and increased locus coeruleus reactivity, is implicated in hyperarousal and exaggerated startle responses (Lanius et al., 2018). Serotonergic and dopaminergic systems also exhibit abnormalities, particularly reduced serotonergic tone and altered dopaminergic reward processing, which correlate with anhedonia and emotional numbing (Richards et al., 2019). At the molecular level, neuropeptide Y (NPY) and brain-derived neurotrophic factor (BDNF) have emerged as biomarkers of resilience versus vulnerability: low NPY and BDNF levels are consistently linked to greater PTSD severity. Collectively, these findings delineate a neurochemical landscape of PTSD characterized by blunted cortisol reactivity, heightened noradrenergic drive, and dysregulated neurotrophic signaling—an enduring stress imprint that perpetuates both the physiological and

affective components of the disorder.

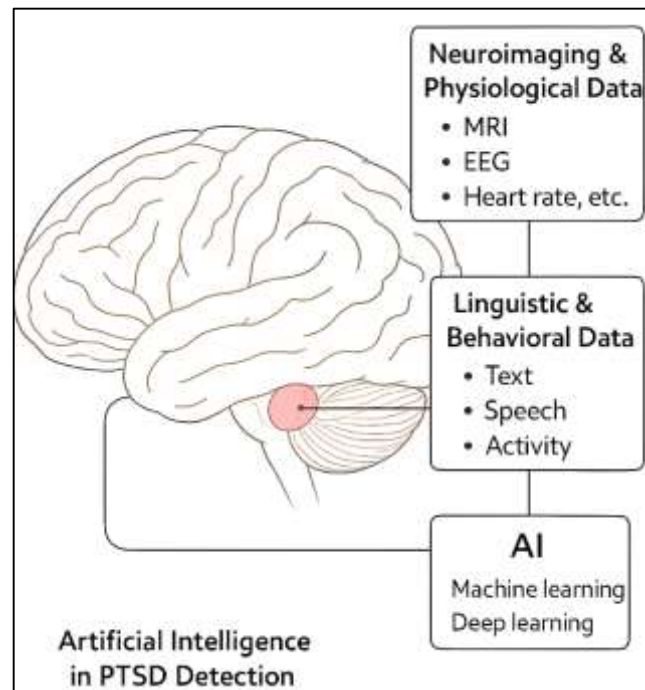
Recent evidence underscores the role of immune and genetic mechanisms in modulating susceptibility to PTSD. Chronic low-grade inflammation has been observed across trauma-exposed populations, with elevated levels of cytokines such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF- α), and C-reactive protein (CRP) correlating with symptom severity and chronicity (Richards et al., 2019). This proinflammatory milieu may alter neural plasticity and synaptic signaling in stress-sensitive brain regions. Genetic studies have identified associations between PTSD risk and polymorphisms in genes regulating serotonin transport (5-HTTLPR), dopamine function (COMT, DRD2), and HPA axis activity (FKBP5). Epigenetic mechanisms further mediate gene-environment interactions: trauma exposure induces DNA methylation changes in glucocorticoid receptor (NR3C1) and immune-related genes, influencing stress responsivity and recovery. These findings support a biologically integrated model of PTSD in which genetic predisposition, immune activation, and epigenetic modification jointly contribute to sustained neurobiological dysregulation. Furthermore, inflammatory signaling may exacerbate the neural circuit abnormalities identified in imaging studies, forming a bidirectional brain-immune feedback loop (Meza-Concha et al., 2017). Such evidence illustrates that PTSD's neurobiological architecture extends beyond neural circuitry to encompass systemic biological processes that shape vulnerability, persistence, and heterogeneity of symptom expression.

Artificial Intelligence in PTSD Detection

Artificial intelligence (AI) has rapidly emerged as a transformative approach in psychiatric diagnostics, offering unprecedented analytical capability for complex, multimodal data associated with post-traumatic stress disorder (PTSD). Traditional diagnostic approaches—based primarily on clinical interviews and self-reported symptoms—are limited by subjectivity, cultural variability, and underreporting of distress (Schwalbe & Wahl, 2020). AI systems, particularly machine learning (ML) and deep learning (DL) models, overcome these limitations by recognizing subtle, nonlinear patterns across large neurobiological and behavioral datasets (Mentis et al., 2021). Early studies utilizing supervised ML algorithms such as support vector machines (SVM) and random forests demonstrated their ability to classify PTSD versus control groups using structural and functional neuroimaging data with accuracies exceeding 80%. Subsequent advances in DL have enabled hierarchical representation learning, enhancing predictive performance in high-dimensional imaging, speech, and physiological data. AI has also been applied to clinical text mining and electronic health records to automate PTSD case identification and symptom extraction, achieving performance comparable to clinician coding (Richards et al., 2019). The rapid expansion of AI methodologies signifies a paradigm shift from subjective diagnostic criteria toward objective, data-driven recognition of trauma-related psychopathology. These developments provide an empirical foundation for early detection frameworks capable of identifying individuals at risk for PTSD before clinical manifestation, facilitating proactive intervention strategies that were previously unattainable in psychiatry (Harrison et al., 2021). AI applications in PTSD detection have increasingly focused on the integration of neuroimaging and physiological biomarkers, allowing for multidimensional modeling of neural and bodily correlates of trauma. Functional MRI-based ML models have identified PTSD-associated alterations in resting-state connectivity, particularly between the amygdala, hippocampus, and prefrontal cortex, achieving area-under-curve (AUC) values between 0.78 and 0.91 (Kelly et al., 2019). Diffusion tensor imaging (DTI) data further enhance classification by quantifying white matter integrity disruptions, with feature importance analyses implicating the cingulum and uncinate fasciculus in predictive modeling (Vollmer et al., 2020). Complementary use of electroencephalography (EEG) and magnetoencephalography (MEG) biomarkers provides temporal precision, capturing neural oscillatory patterns associated with hyperarousal and reactivity (Bates et al., 2020). Physiological biomarkers such as heart rate variability (HRV), electrodermal activity (EDA), and cortisol reactivity have been incorporated into ML pipelines, enhancing cross-modal prediction (Meskó & Görög, 2020). Studies employing multimodal AI frameworks—combining imaging, endocrine, and autonomic signals—demonstrate superior predictive accuracy compared with unimodal models, reinforcing the notion that PTSD is a systemic rather than localized neural disorder (Bates et al., 2020). Importantly, advances in explainable AI (XAI) techniques such as SHAP and integrated gradients have begun to uncover which biological features most strongly contribute to model predictions, bridging computational and clinical

interpretability. Collectively, this evidence demonstrates that AI-assisted integration of neurobiological data can capture latent pathophysiological signatures of PTSD, offering a powerful adjunct to early detection and differential diagnosis.

Figure 6: Artificial Intelligence in PTSD Detection



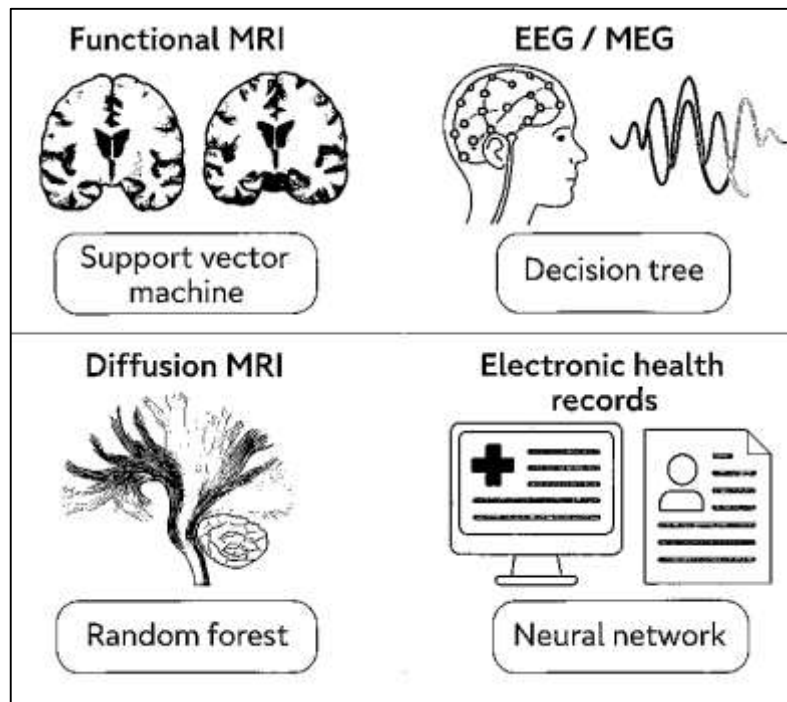
The application of AI to behavioral and linguistic data represents an expanding dimension of PTSD detection research. Natural language processing (NLP) and speech analytics have been used to identify linguistic markers of trauma-related distress such as disfluency, pronoun use, sentiment polarity, and semantic coherence (Mentis et al., 2021). Large-scale social media analyses have detected PTSD risk signals from user-generated text through recurrent neural networks (RNNs) and transformer-based models (Schwalbe & Wahl, 2020). Similarly, vocal biomarkers—derived from acoustic prosody, pitch variability, and vocal jitter—have enabled automated PTSD screening from speech recordings with classification accuracies approaching 85% (Richards et al., 2019). Wearable technologies now provide continuous monitoring of physiological and behavioral data, including heart rate, skin conductance, and actigraphy, which AI models can translate into stress or trauma-related risk patterns (Harrison et al., 2021). These digital phenotyping approaches extend PTSD detection beyond laboratory environments into ecologically valid, real-world contexts. Multimodal behavioral-AI frameworks combining speech, movement, and biosignal data have shown high sensitivity for detecting subthreshold or preclinical PTSD, particularly in military and first-responder populations (Kelly et al., 2019). Collectively, this growing literature underscores AI's ability to identify digital and behavioral biomarkers that mirror the neurobiological disruptions observed in imaging studies, thereby broadening the scope of PTSD surveillance to more accessible, scalable modalities.

Machine Learning Applications in PTSD Classification

Machine learning (ML) approaches have redefined how post-traumatic stress disorder (PTSD) can be detected, classified, and predicted by enabling automated analysis of complex, nonlinear relationships within high-dimensional data. Traditional diagnostic paradigms rely heavily on structured clinical interviews and self-report scales, which, while standardized, cannot capture the full variability of symptom expression or biological correlates (Rahman et al., 2020). ML models circumvent these constraints by using algorithms that learn from data patterns to classify cases without a priori assumptions (Schwalbe & Wahl, 2020). Early studies employing supervised classifiers—such as support vector machines (SVMs), random forests, logistic regression, and naïve Bayes—demonstrated encouraging results when trained on neuroimaging, physiological, and psychometric data ((Mentis et

al., 2021). For instance, SVM models using resting-state functional MRI achieved accuracies up to 85 % in distinguishing PTSD from trauma-exposed controls (Akella et al., 2021), while random forest models trained on clinical and demographic features reached 80 % accuracy for classification in veteran populations (Chan et al., 2002). As computational power increased, more complex ensemble methods—such as gradient boosting and extreme random forests—were introduced, providing enhanced robustness against noise and overfitting (McDonald et al., 2019). Collectively, these foundational studies established that ML can reliably model PTSD-related heterogeneity, providing a quantitative alternative to symptom-based assessment and laying the groundwork for multimodal predictive analytics in trauma research.

Figure 7: Machine Learning Applications in PTSD Classification



Among ML applications in PTSD classification, neuroimaging has been the most extensively explored modality due to its ability to quantify neural signatures associated with trauma-related dysregulation. Functional MRI (fMRI) and structural MRI datasets allow for voxel-level pattern recognition, where ML algorithms discern distributed brain activity that differentiates PTSD from healthy or trauma-exposed controls (Galatzer-Levy et al., 2018). Using multivariate pattern analysis, (Durstewitz et al., 2019) identified aberrant connectivity between the amygdala, hippocampus, and prefrontal cortex predictive of PTSD status. Deep neural networks applied to resting-state connectivity matrices have further improved discriminative power, achieving AUC values up to 0.92 (Jin et al., 2017). In diffusion tensor imaging (DTI) analyses, ML algorithms successfully detect microstructural abnormalities in white matter tracts—particularly the cingulum, corpus callosum, and uncinate fasciculus—that align with disrupted emotion regulation pathways (Karstoft et al., 2015). Machine learning applied to magnetoencephalography (MEG) and electroencephalography (EEG) data has also yielded reliable biomarkers: for example, decision-tree classifiers using alpha and theta power features achieved > 80 % sensitivity in classifying PTSD among combat veterans (Attallah, 2020). Furthermore, multimodal fusion models that combine neuroimaging and psychophysiological inputs outperform single-modality classifiers, underscoring the synergistic value of integrating brain and body data (Schultebrasucks et al., 2021). The cumulative literature demonstrates that ML-based neuroimaging classifiers not only replicate known neural correlates of PTSD but also reveal novel distributed features invisible to univariate methods, advancing the precision of neurobiological diagnostics. Beyond neuroimaging, ML has proven effective in classifying PTSD using behavioral, linguistic, and clinical data streams. Supervised learning applied to electronic health records (EHRs) and clinical narratives

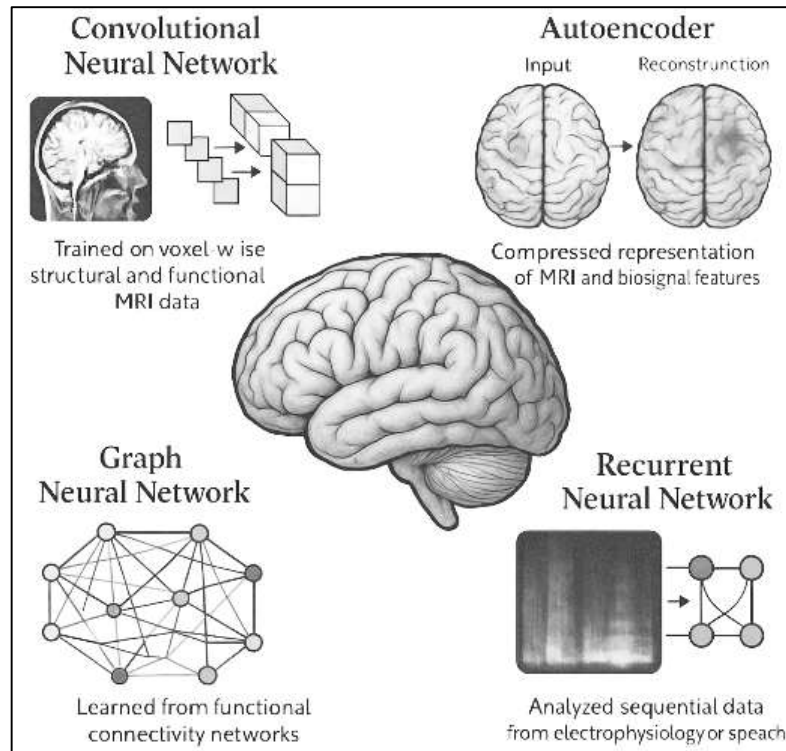
has facilitated automated case identification, enabling large-scale surveillance and screening (Karstoft et al., 2015). Natural language processing (NLP) models, including recurrent neural networks (RNNs) and transformer-based architectures such as BERT, have been used to detect PTSD-related language features across therapy transcripts, patient notes, and social media, with accuracies between 75 % and 90 % (Kessler et al., 2014). Behavioral data derived from wearable sensors—such as heart rate variability, galvanic skin response, actigraphy, and speech prosody—have also been successfully analyzed using ML classifiers to predict acute stress and chronic PTSD risk (McDonald et al., 2019). These digital biomarkers offer noninvasive, real-time measures that extend beyond laboratory environments. Ensemble ML models trained on multimodal behavioral data from first responders and veterans have reached accuracies exceeding 85 % for detecting subthreshold PTSD (Mentis et al., 2021). Moreover, unsupervised clustering techniques have been employed to identify latent PTSD subtypes, revealing biologically distinct phenotypes that correspond with differential treatment responses (Karstoft et al., 2015). This expansion of ML to digital and behavioral domains reflects a methodological evolution toward scalable, continuous, and ecologically valid PTSD detection systems.

Deep Learning in Neurobiological PTSD Research

Deep learning (DL) has reoriented neurobiological PTSD research by enabling high-capacity models to learn hierarchical representations from imaging, electrophysiology, and physiological signals without handcrafted feature engineering (Rahman et al., 2020). Within psychiatric neuroimaging, early machine-learning efforts demonstrated separability of PTSD from trauma-exposed controls using multivariate patterns in amygdala-hippocampal-prefrontal circuitry and large-scale networks (B. A. Richards et al., 2019), and DL extended this trajectory by learning distributed, nonlinear signatures directly from voxel-wise and connectomic inputs (Durstewitz et al., 2019). In PTSD cohorts, supervised pipelines trained on resting-state and task fMRI have produced clinically relevant discrimination by capturing dysregulated salience, default mode, and executive networks consistent with systems-level models of the disorder (Geronikolou et al., 2021). Convolutional neural networks (CNNs) and autoencoders extract latent features from high-dimensional scans, while sequence models (e.g., long short-term memory [LSTM]) summarize temporal dependencies in electrophysiology and endocrine reactivity. Diffusion-derived microstructural abnormalities in the uncinate fasciculus, cingulum, and corpus callosum—long linked to emotion-regulation circuitry—are similarly amenable to representation learning with CNNs and 3D patch-based architectures (Huang et al., 2020). In parallel, multimodal DL frameworks integrate imaging with autonomic and hormonal markers (e.g., heart-rate variability and cortisol), reflecting evidence that PTSD is a systemic condition that engages brain-body loops (Rahman et al., 2020). Methodologically, DL addresses feature collinearity and high noise-to-signal common to psychiatric datasets through regularization, augmentation, and transfer learning from large public neuroimaging corpora ((Geronikolou et al., 2021). Collectively, these developments reposition classification from sparse, hypothesis-driven features toward dense, data-driven embeddings aligned with mechanistic accounts of threat learning, contextual memory, and prefrontal inhibitory control in PTSD (Geronikolou et al., 2021; Huang et al., 2020).

In neurobiological PTSD studies centered on MRI/fMRI, DL models capitalize on spatial structure and correlation in brain volumes and connectivity matrices. Resting-state fMRI pipelines convert correlation networks into inputs for CNNs or graph neural networks (GNNs), capturing altered coupling among amygdala, hippocampus, medial prefrontal cortex, and insula nodes linked to intrusive recollection, hyperarousal, and impaired regulation (Geronikolou et al., 2021). CNNs trained on voxel-wise maps or region-of-interest stacks achieve strong discrimination by learning multiscale filters tied to salience/default-mode imbalances, while autoencoder bottlenecks produce compact latent spaces that preserve case-control separability (Durstewitz et al., 2019). Graph-based approaches treat the connectome as a non-Euclidean object; spectral and message-passing GNNs propagate information along white-matter and functional edges, improving sensitivity to fronto-limbic dysconnectivity and topological markers such as altered hubness and reduced modularity (Rahman et al., 2020).

Figure 8: Deep Learning in Neurobiological PTSD Research



Diffusion models leverage 3D CNNs on fractional anisotropy and mean diffusivity volumes to identify microstructural signatures in the cingulum and uncinate fasciculus consistent with impaired fear-extinction circuitry (Engel et al., 2023). Empirical PTSD applications report area-under-the-curve ranges that meet or exceed classical machine learning when models are trained with nested cross-validation and harmonized preprocessing, including nuisance regression and motion control (Marseille et al., 2020). Task-based fMRI DL further distinguishes threat-processing phenotypes by decoding differential activation patterns during fear conditioning and emotional interference tasks, aligning computational readouts with established psychophysiology (Vermetten & Jetly, 2018). Across pipelines, performance benefits arise from multimodal fusion of structural, functional, and diffusion inputs—implemented via early (feature-level) or late (decision-level) fusion layers—which consolidates distributed pathophysiology within single inference graphs (Heim et al., 2022).

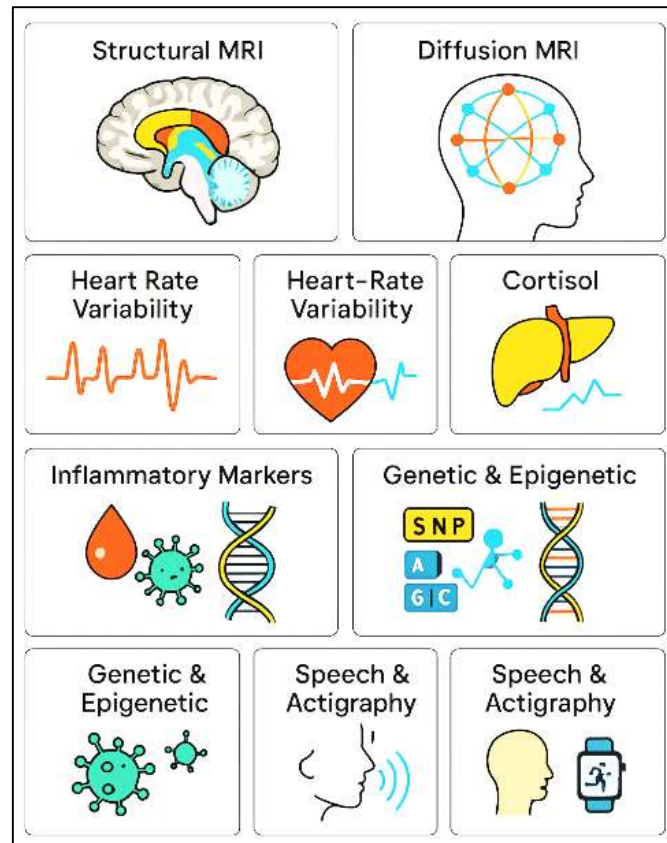
Time-resolved neurobiological data broaden DL-based PTSD classification beyond static images. Electroencephalography (EEG) and magnetoencephalography (MEG) provide millisecond-scale access to oscillatory dynamics implicated in hyperarousal, vigilance, and inhibitory control; 1D-CNNs, temporal convolutional networks, and LSTM models learn discriminative patterns in alpha/theta power, phase-amplitude coupling, and event-related potentials (Zoladz & Diamond, 2013). Autonomic and endocrine data encode complementary state-trait information: HRV, electrodermal activity, and diurnal cortisol profiles index sympathetic tone and HPA-axis regulation known to diverge in PTSD, and DL sequence models aggregate circadian and context-dependent fluctuations into robust embeddings (Young et al., 2022). Speech-based DL uses spectro-temporal representations (e.g., Mel spectrograms) and attention layers to capture prosodic irregularities associated with affective blunting or hyperarousal, producing high screening performance in veteran samples (Zoladz et al., 2013). Wearable biosensor streams extend these pipelines into naturalistic settings; multimodal late-fusion networks integrating actigraphy with HRV/EDA improve detection of subthreshold presentations relative to unimodal baselines (Vermetten & Jetly, 2018). In PTSD research, joint models that combine imaging with physiology advance construct validity by aligning network-level brain findings with concurrent autonomic patterns, thereby linking cortical-subcortical dysregulation to embodied stress signatures (Bohus et al., 2020). Autoencoder-based missing-modality imputation and co-regularization address incomplete data—a common barrier in psychiatric cohorts—while domain adaptation

mitigates distributional shift across scanners and recording devices (Heim et al., 2022). Collectively, electrophysiology and biosignal DL complement MRI/fMRI by providing temporally rich biomarkers that track arousal and regulation, and their fusion with imaging supports classifications that reflect the systemic nature of PTSD pathophysiology (Koch et al., 2016; Logue et al., 2018; Rahimi et al., 2021). Interpretability and validation practices shape the credibility of DL in neurobiological PTSD research. Saliency methods—Grad-CAM for CNNs and integrated gradients/DeepLIFT for general architectures—link predictions to spatial or temporal features, enabling neuroscientific appraisal of whether networks rely on plausible circuitry. Model-agnostic approaches such as SHAP and LIME quantify feature contributions and local decision boundaries, supporting alignment between DL outputs and established biomarkers (Heim, 2020). Nevertheless, small-N/high-P regimes typical of PTSD imaging elevate overfitting risk; leakage via improper cross-validation, site effects, and motion confounds can inflate accuracy unless addressed with nested CV, subject-level splits, harmonization (e.g., ComBat), and preregistered preprocessing (Haagen et al., 2018). External validation across scanners, trauma types, and demographics remains sporadic, and calibration metrics and decision-curve analyses are infrequently reported, constraining clinical interpretability (Cloitre et al., 2009). Fairness concerns also arise: unbalanced sex, ethnicity, and trauma-modality distributions can embed bias within embeddings, requiring stratified sampling, reweighting, and bias audits. From an epistemic standpoint, DL findings gain credibility when explanatory maps converge with prior mechanistic literature—e.g., amygdala-mPFC circuits, hippocampal context encoding, and salience/default-mode reconfiguration—rather than spurious edges or scanner artifacts. Across studies, rigorous pipelines that enforce leakage-safe validation, site harmonization, and transparent XAI reporting demonstrate that DL can recover biologically meaningful patterns in PTSD while maintaining out-of-sample performance consistent with reproducible neuroimaging standards.

Multimodal Neurobiological Data

Multimodal neurobiological approaches to post-traumatic stress disorder (PTSD) integrate convergent data streams to characterize dysregulation across brain, autonomic, endocrine, and immune systems that single-modality studies only partially capture. Structural and functional MRI consistently implicate hippocampal volume loss, amygdala hyperreactivity, and medial prefrontal/anterior cingulate hypofunction as core correlates linked to contextual memory, threat appraisal, and inhibitory control (Dennis et al., 2019). Diffusion metrics reveal microstructural compromise in emotion-regulation pathways including the uncinate fasciculus and cingulum (Tang et al., 2021). Resting-state connectivity indicates large-scale network imbalance—heightened salience network expression with reduced default-mode coupling and weakened executive control—supporting systems-level models of intrusive recollection and hyperarousal (Sippel et al., 2021). Beyond the brain, autonomic markers such as reduced heart-rate variability and heightened electrodermal reactivity align with chronic sympathetic tone, while endocrine indices demonstrate altered cortisol dynamics and HPA-axis feedback (Lanius et al., 2010). Immune signatures, including elevated interleukin-6 and C-reactive protein, co-vary with symptom severity and may reflect persistent low-grade inflammation that modulates neural plasticity. Genetic and epigenetic studies implicate polymorphisms and methylation changes in stress-regulatory pathways (FKBP5, NR3C1) and serotonergic systems that condition risk and chronicity. Speech-acoustic and language features, actigraphy and sleep fragmentation, and wearable biosignals add ecologically sampled indicators of vigilance, arousal, and affective blunting. Taken together, the literature situates PTSD as a distributed, brain-body condition in which multimodal evidence converges on dysregulated threat learning, context processing, and autonomic-endocrine-immune coupling rather than a unitary neural lesion (Harricharan et al., 2021).

Figure 9: Multimodal Neurobiological Data



Multimodal studies employ several integration strategies to combine heterogeneous data types. Early fusion concatenates features from structural/functional MRI, EEG, HRV/EDA, endocrine assays, and inflammatory markers into unified vectors for classification or regression, allowing algorithms to exploit cross-domain interactions such as covariation between amygdala-mPFC coupling and sympathetic tone. Late fusion aggregates modality-specific model outputs, improving robustness when sampling rates or noise profiles differ (Koch et al., 2015). Hybrid and intermediate approaches use shared latent space learning—joint and linked independent component analysis, canonical correlation analysis, and multi-view embedding—to discover modality-spanning components that align neural networks with peripheral physiology. In connectomic pipelines, graph representations of resting-state or diffusion networks integrate with peripheral features via graph neural networks or attention mechanisms, capturing topology (hubness, modularity) together with endocrine or inflammatory covariates. Deep autoencoders compress high-dimensional voxelwise or spectral inputs while preserving discriminative structure; stacked or variational variants link imaging embeddings to HRV/cortisol streams, facilitating cross-modal alignment. Speech and prosody features—modeled with convolutional or recurrent architectures—enter late-fusion ensembles with imaging and wearables, improving sensitivity for subthreshold presentations (Harnett et al., 2021). These fusion strategies repeatedly show gains over unimodal baselines, with studies attributing improvements to complementary signal content: imaging indexes trait-level circuit properties, autonomic/endocrine metrics capture state fluctuations, and immune markers reflect systemic milieu influencing neural excitability and plasticity. The methodological emphasis on shared latent structure strengthens construct validity by mapping associations among brain networks, peripheral physiology, and symptom dimensions in a single analytic framework.

Multimodal PTSD cohorts encounter practical obstacles that shape inference quality, including scanner/site effects, asynchronous sampling, and missing modalities. Harmonization methods such as ComBat and related empirical Bayes adjustments reduce inter-scanner variance in morphometry and diffusion measures, limiting spurious site-driven separability (Harnett et al., 2021). Motion control, physiological noise modeling, and standardized preprocessing—nuisance regression, ICA-based

artifact removal, and consistent parcellations—improve comparability across imaging datasets (Richards et al., 2019). For peripheral streams, rigorous aggregation of circadian-structured HRV and diurnal cortisol yields stable summary features suitable for fusion with time-invariant imaging. Missing modality is common; studies report autoencoder-based imputation, multi-task learning, and co-regularization to leverage incomplete cases without discarding valuable data. Domain adaptation and transfer learning mitigate distributional shift across scanners, wearables, and speech acquisition conditions, preserving out-of-sample performance. Validation practices influence generalizability: subject-level splits, nested cross-validation, and external testing across trauma types and demographics counter information leakage and overfitting documented in smaller imaging studies. Reporting of calibration, decision-curve analyses, and error stratification by sex, ethnicity, and trauma modality clarifies clinical utility and fairness profiles in heterogeneous populations (Richards et al., 2019). Collectively, methodological rigor in harmonization and validation supports the conclusion that performance gains attributed to multimodality reflect integrated biology rather than artifacts of acquisition, sampling, or analytic instability (Richards et al., 2019).

Findings from multimodal PTSD research converge on a reproducible pattern of dysregulated fronto-limbic circuitry linked with peripheral markers of arousal and stress biology. Diminished hippocampal structure and altered connectivity with medial prefrontal regions relate to impaired contextualization and extinction, while amygdala hyperreactivity and salience-network dominance align with threat-biased attention and intrusions (Tang et al., 2021). Reduced HRV and heightened electrodermal lability track with these neural signatures, indicating persistent sympathetic mobilization that corresponds with prefrontal inhibitory inefficiency. Endocrine profiles characterized by altered basal cortisol and enhanced feedback sensitivity co-occur with hippocampal and prefrontal abnormalities, consistent with stress-hormone effects on memory consolidation and top-down control. Immune indices—elevated IL-6 and related cytokines—associate with network-level connectivity changes, suggesting an interaction between inflammatory tone and neural plasticity in trauma-exposed cohorts (Feduccia & Mithoefer, 2018). Genetic and epigenetic markers in FKBP5, NR3C1, and serotonergic pathways modulate these axes, conditioning neural and autonomic responses observable in integrated models. Speech-acoustic irregularities, blunted prosody, and sleep fragmentation contribute additional variance that aligns with hyperarousal and executive-network underengagement. Across studies that implement robust fusion and validation, joint modeling of imaging, autonomic/endocrine, and immune or digital streams yields superior discrimination and biologically interpretable feature attributions compared with single-modality baselines, reinforcing a systems-level account of PTSD grounded in coupled neural and peripheral dysregulation (Sippel et al., 2021).

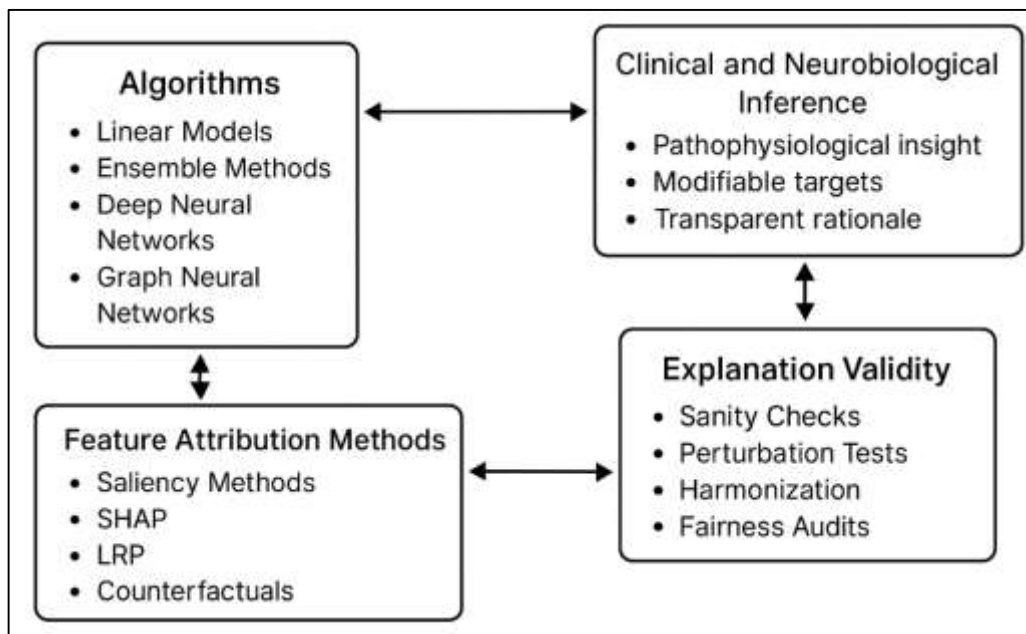
Model Explainability and Feature Attribution

Model explainability in neurobiological PTSD research concerns the mapping between complex algorithmic decisions and interpretable biological evidence, so that a classifier's outputs can be related to circuits, signals, and measurements that clinicians and neuroscientists recognize as meaningful. In supervised pipelines, two broad families of approaches dominate: intrinsically interpretable models with transparent parameters (e.g., linear models with sparse priors) and post hoc explanation methods applied to high-capacity learners such as deep neural networks or ensembles. In neuroimaging and physiology, post hoc techniques are pervasive because convolutional and recurrent architectures capture distributed, nonlinear dependencies across voxels, time points, and modalities that are not easily summarized by a small set of coefficients (Averill et al., 2016). Gradient-based saliency methods estimate the local sensitivity of the output to input features, with refinements such as Integrated Gradients to ensure axiomatic properties like completeness and Grad-CAM to localize class-relevant spatial patterns in convolutional feature maps. Layer-wise relevance propagation attributes prediction scores backward through the network to yield signed “relevance” maps that often align with domain knowledge in medical imaging (Harricharan et al., 2021). Model-agnostic methods—LIME and SHAP—approximate complex decision boundaries with locally linear surrogates or Shapley-value attributions, enabling unified comparisons across architectures and feature spaces, including tabular physiological markers and graph-level connectomic descriptors. In linear and kernel models common to early PTSD studies, weight maps are frequently misread as activation maps; correction procedures such as the Haufe transform convert discriminative weights into activation patterns that more faithfully

reflect underlying signal sources (Koch et al., 2015). Together, these families of explanation address complementary needs: gradient/relevance maps ground voxel-wise and temporal evidence; Shapley-based scores compare heterogeneous features; and transformed linear patterns provide baseline interpretability against which deep attribution can be judged.

The evidential value of an explanation hinges on its reliability under perturbation and its validity relative to known neurobiology. Saliency maps can be fragile to model re-initialization, label randomization, or input transformations, raising concerns that visually plausible “heat maps” may reflect architectural priors rather than learned signal (Tang et al., 2021). Sanity-check protocols therefore compare explanations from trained models to those from parameter-randomized counterparts and quantify similarity decrements as a minimal validity test (Sippel et al., 2021). Attribution faithfulness is further assessed by deletion/insertion curves progressively removing highly attributed features should reduce confidence more than removing low-attribution features and by meaningful perturbations that optimize small, human-interpretable masks to disrupt predictions. In neuroimaging, site effects, head motion, and preprocessing choices can spuriously structure attributions; harmonization (e.g., ComBat) and leakage-safe validation mitigate confounding that otherwise inflates both accuracy and apparent biological specificity. For linear and elastic-net models, interpreting raw weights as neurobiology is problematic in correlated feature spaces; pattern-recovery approaches and permutation importance yield more defensible inferences. Across multimodal PTSD pipelines, calibration and decision-curve analysis complement attribution by indicating whether highly “explanatory” models produce clinically usable probabilities. Finally, reproducibility demands reporting of stochastic seeds, cross-validation splits, and preprocessing parameters, because attribution maps can vary materially with these choices even when point performance remains stable (Averill et al., 2016). Methodological rigor thus couples explanation quality to validation design, preventing overinterpretation of aesthetically persuasive—but potentially artifactual—feature maps.

Figure 10: Model Explainability and Feature Attribution



PTSD research increasingly pairs neural attributions with feature contributions from autonomic, endocrine, immune, and digital markers to evaluate convergent biological mechanisms. In imaging, Grad-CAM and Integrated Gradients often highlight amygdala-hippocampal-medial prefrontal territories and salience/default-mode hubs during classification, consistent with systems models of threat processing and contextual memory. When fused with heart-rate variability, electrodermal activity, and diurnal cortisol features, SHAP rankings frequently elevate sympathetic arousal indices and HPA-axis measures alongside connectomic edges, supporting a coupled brain-body account of hypervigilance and impaired extinction (Feduccia & Mithoefer, 2018). In graph-based connectomics,

node- and edge-level attributions from message-passing networks identify discriminatory subnetworks (e.g., anterior insula–dACC links), while global topological importance (betweenness, participation coefficient) provides model-independent corroboration. For speech and text, attention weights and SHAP scores isolate prosodic jitter, reduced pitch variability, and negation-laden or self-referential tokens that align with clinical observations of affective numbing and intrusive focus. Crucially, cross-modal triangulation enhances credibility: imaging attributions pointing to prefrontal–amygdala dysregulation gain strength when high-importance autonomic features indicate low vagal tone and elevated skin conductance in the same individuals. Counterfactual explanations add clinical interpretability by quantifying minimally sufficient changes in features—e.g., improved HRV or reduced nocturnal arousals—that flip predicted risk, aligning model narratives with modifiable targets in behavioral sleep or exposure-based interventions. In sum, multimodal attribution connects algorithmic importance to pathophysiological constructs, enabling explanations that are not merely descriptive but biologically interpretable and clinically coherent.

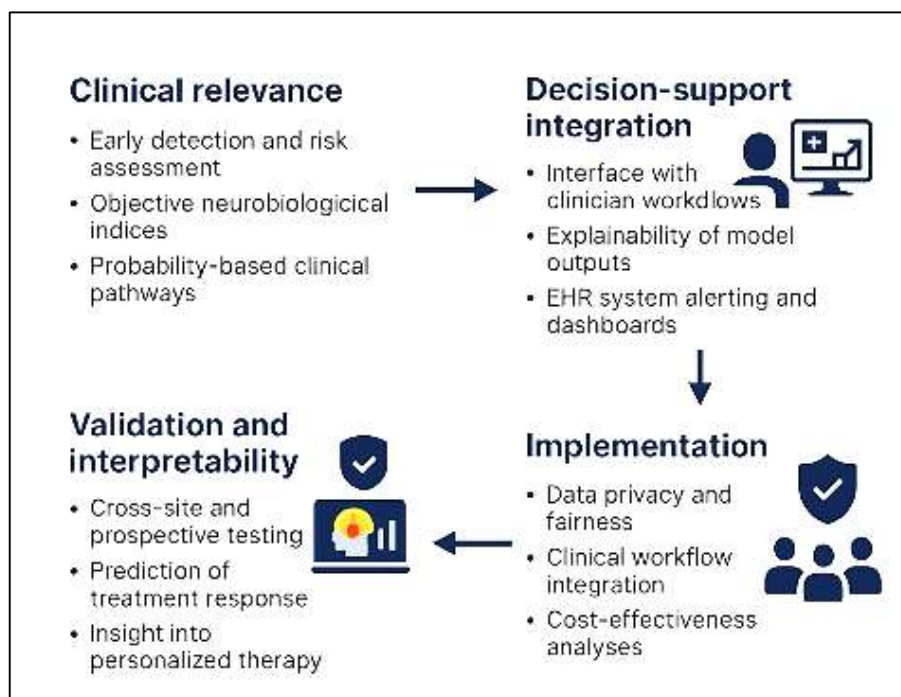
Explanations shape trust, but they also expose risks if they encode bias or reveal sensitive attributes. In health systems, algorithmic bias can arise from unbalanced training cohorts; feature-attribution audits stratified by sex, race/ethnicity, age, and trauma type reveal whether models systematically rely on proxies for demographic variables or access inequities (Akiki et al., 2017). Fairness-aware attribution examines subgroup SHAP distributions or conducts counterfactual fairness tests—holding protected attributes fixed while permitting clinical features to vary—to detect disparate influence on predictions. Privacy-preserving analysis is also pertinent: gradient-based explanations can leak information about individual inputs; differential privacy and federated learning reduce disclosure risk while maintaining usable attributions at cohort level. From a reporting standpoint, model cards and datasheets encourage standardized disclosure of training data composition, preprocessing, performance stratification, and explanation methods so that end users can contextualize attributions (D’Elia et al., 2021). Clinically, calibrated probabilities, net benefit curves, and decision thresholds must accompany explanatory visuals; otherwise, salient heat maps risk overshadowing limited clinical utility (Harnett et al., 2021). Finally, causal interpretability remains a boundary condition: most attribution methods are associational and do not identify mechanistic effects; triangulating attributions with experimental manipulations, longitudinal designs, or instrumental-variable analyses strengthens biological claims (Sippel et al., 2021). Within PTSD, coupling leakage-safe validation, site harmonization, stratified fairness audits, and robust XAI (e.g., Integrated Gradients, SHAP, LRP, sanity checks) provides an evidentiary scaffold wherein feature attribution supports—not substitutes for—clinically and neuroscientifically sound inference (Feduccia & Mithoefer, 2018).

Clinical Relevance and Decision-Support Integration

The clinical relevance of artificial intelligence (AI) and neurobiologically informed models for post-traumatic stress disorder (PTSD) depends on their translation from predictive analytics to practical decision-support within real-world clinical settings. Traditionally, PTSD diagnosis has relied on structured interviews such as the Clinician-Administered PTSD Scale (CAPS) and self-report instruments like the PTSD Checklist (PCL), both of which depend on subjective symptom interpretation and patient recall (Méndez et al., 2018). AI-based diagnostic models augment these methods by identifying biological and behavioral patterns that may precede overt symptom manifestation, potentially enabling early detection and risk stratification. Neurobiological data derived from neuroimaging, EEG, and physiological monitoring provide objective signatures of dysfunction in limbic-prefrontal circuits and autonomic regulation, translating neural mechanisms into quantifiable clinical indices. Machine learning classifiers and deep neural networks that integrate these biomarkers can predict PTSD presence or severity with accuracies exceeding 80%, highlighting their clinical promise as adjunct diagnostic aids (Del Casale et al., 2022). Importantly, predictive modeling reframes PTSD not as a categorical diagnosis but as a probabilistic risk state, enabling personalized clinical pathways rather than binary judgments (Church et al., 2018). Such probability-based assessments support triage decisions, prioritization of high-risk individuals following trauma exposure, and adaptive monitoring of treatment response. The movement toward computational psychiatry thus signals a paradigm shift from symptom-based assessment to dynamic, biomarker-informed precision mental health (Zohar et al., 2011).

For AI-derived PTSD models to achieve clinical impact, they must be integrated within decision-support infrastructures that interface seamlessly with clinician workflows and electronic health record (EHR) systems. Decision-support integration involves translating algorithmic predictions into actionable recommendations, confidence scores, or risk alerts interpretable by mental health professionals (McGeary et al., 2023). In psychiatric contexts, explainable AI (XAI) frameworks—such as SHAP and LIME—enable clinicians to visualize which biological, behavioral, or environmental factors contribute most strongly to a patient’s predicted PTSD risk, thereby enhancing transparency and trust (Murkar et al., 2022). Integration studies in trauma care have embedded ML-based screening modules within hospital EHRs to automatically flag at-risk patients based on structured and unstructured data, including trauma exposure codes, medication history, and free-text clinical notes. Pilot implementations within the U.S. Department of Veterans Affairs and military health systems demonstrate the feasibility of using AI-assisted decision aids to support PTSD diagnosis and management at scale (Kuring et al., 2023). Moreover, multimodal decision-support prototypes now combine imaging-derived neural risk scores with wearable physiological metrics, such as heart-rate variability and electrodermal activity, to provide clinicians with real-time dashboards of stress physiology (Hien et al., 2018). By contextualizing AI outputs alongside traditional assessments, decision-support systems can guide early intervention, tailor treatment intensity, and monitor recovery trajectories with objective neurobiological feedback.

Figure 11: Clinical Relevance and Decision-Support Integration



Robust validation is central to establishing clinical credibility for AI-based PTSD models. Prospective and cross-site validation ensures that algorithmic predictions generalize across populations, scanners, and trauma types. Model calibration metrics—such as Brier scores and expected calibration error—quantify whether predicted probabilities correspond to real-world outcomes, an essential prerequisite for clinical reliability. Interpretable models not only enhance clinician trust but also reveal actionable insights for personalized therapy. For example, feature attributions identifying exaggerated amygdala connectivity or low vagal tone as major predictive drivers may guide targeted interventions such as neurofeedback, exposure therapy, or mindfulness-based HRV modulation (Hien et al., 2018; McGeary et al., 2023). Deep-learning approaches have also been adapted to predict treatment response, distinguishing responders from non-responders to cognitive-behavioral therapy or pharmacotherapy using pre-treatment imaging and physiological data. These models enable adaptive treatment planning and real-time feedback loops, where symptom improvement is continuously assessed through updated

biomarker readings. Furthermore, multimodal explainability allows integration of clinical reasoning with algorithmic output – facilitating shared decision-making and reducing the risk of automation bias. In aggregate, such validation and interpretability practices are vital for transforming AI systems from research tools into clinically reliable decision-support mechanisms capable of augmenting professional judgment rather than replacing it.

The clinical deployment of AI-enabled PTSD diagnostic and decision-support systems carries significant ethical, practical, and systemic implications. Data privacy and informed consent are critical, particularly given the sensitivity of neurobiological and psychological data. Federated learning and differential privacy techniques allow model training across distributed healthcare sites without centralized data sharing, preserving confidentiality while enhancing dataset diversity. Clinically, equitable performance across gender, ethnicity, and trauma type must be ensured to prevent algorithmic bias that could exacerbate disparities in access or diagnosis. Implementation science highlights that adoption success depends not only on predictive accuracy but also on workflow integration, clinician training, and interpretability of model outputs (Shaw et al., 2023). Cost-effectiveness analyses indicate that AI-supported early detection can reduce chronic PTSD prevalence and healthcare utilization, provided systems are deployed within structured care pathways that include human oversight and ethical governance. Standardized reporting frameworks such as TRIPOD-AI and PROBAST-AI guide transparent publication of model performance, calibration, and generalizability (Schmidt & Vermetten, 2017). Collectively, the literature suggests that AI-based decision-support tools, when implemented with rigorous validation, interpretability safeguards, and equitable governance, can enhance the precision and timeliness of PTSD care, bridging the gap between computational discovery and therapeutic decision-making.

Critical Summary of Gaps and Inconsistencies

A consistent limitation across neurobiological and AI-enabled PTSD research lies in the considerable heterogeneity of study designs, participant samples, and diagnostic criteria, which complicates synthesis and reproducibility. PTSD studies vary widely in inclusion criteria, trauma type, and chronicity – ranging from combat veterans to survivors of interpersonal violence or natural disasters – creating inconsistencies in symptom trajectories and biological profiles. Many investigations rely on small, convenience-based samples, often underpowered to detect subtle neurobiological differences or to support complex machine-learning models. Sampling bias is especially problematic: the overrepresentation of Western, high-income, and male combat populations limits generalizability to civilians, women, and low- and middle-income contexts where trauma exposure and expression differ significantly (Del Casale et al., 2022). Furthermore, disparities between diagnostic systems (DSM-5 versus ICD-11) and assessment tools (e.g., CAPS, PCL, MINI) yield non-overlapping case definitions, contributing to divergent prevalence estimates and inconsistent neurobiological correlates. Some studies classify PTSD dichotomously, while others model symptom severity along a continuum, resulting in incompatible analytic targets across machine-learning models. Without harmonized diagnostic frameworks and stratified recruitment that reflects population heterogeneity, it remains difficult to establish normative reference values or to compare the predictive validity of different computational models. This variability in design and sampling thus constitutes a foundational barrier to replicability and global applicability of AI-driven PTSD diagnostics. Despite general agreement on limbic-prefrontal dysregulation as a hallmark of PTSD, neuroimaging and physiological findings diverge in both directionality and localization across studies. Structural MRI reports consistently identify hippocampal volume reduction, yet some studies attribute these changes to pre-existing vulnerability rather than trauma-induced neuroplasticity (Wade et al., 2013). Functional MRI investigations reveal both hyperactivation and hypoactivation in amygdala and medial prefrontal regions depending on task context and analytic pipeline. Resting-state connectivity analyses similarly produce conflicting evidence, with some demonstrating decreased default-mode coherence while others report compensatory hyperconnectivity in similar networks. Divergences are also apparent in peripheral markers: cortisol levels have been found to be elevated, blunted, or unchanged across trauma cohorts, suggesting that temporal sampling, chronicity, and circadian confounds heavily influence outcomes. Inflammatory biomarkers such as IL-6 and C-reactive protein yield inconsistent associations with symptom severity, reflecting differences in assay methodology, comorbid conditions,

and medication status (McGeary et al., 2023). Multimodal integration studies often compound these inconsistencies because neural and peripheral measures are not temporally aligned or collected under standardized stress paradigms. These divergences underscore a central methodological gap: the absence of unified acquisition protocols and analytic harmonization across sites. Without standardization in imaging parameters, physiological baselines, and sampling windows, multimodal convergence remains conceptually appealing but empirically fragmented (Hien et al., 2018).

Machine learning and deep learning models applied to PTSD data exhibit substantial algorithmic heterogeneity, leading to inconsistent performance and uncertain clinical reliability. Studies deploy a wide array of classifiers—from support vector machines and random forests to convolutional neural networks and graph neural networks—without standardized benchmarks or comparable evaluation metrics. Small sample sizes combined with high-dimensional imaging data promote overfitting, particularly when cross-validation practices are insufficiently rigorous or feature selection is conducted on the full dataset before data partitioning. Performance metrics such as accuracy or AUC are often reported without calibration analyses or external validation, inflating apparent predictive power (Jerome et al., 2020). Furthermore, differences in preprocessing pipelines—e.g., motion correction, parcellation scheme, normalization, and confound regression—introduce systematic variance that rivals the biological signal being modeled. While explainable AI tools such as SHAP, LIME, and Grad-CAM improve transparency, few PTSD studies quantitatively evaluate the stability or faithfulness of their attributions. Consequently, models may appear interpretable yet fail to generalize beyond the training distribution. The absence of open-source benchmarking datasets and shared evaluation frameworks impedes direct comparison among algorithms and obscures which architectures are genuinely superior for neuropsychiatric classification. These algorithmic inconsistencies collectively limit confidence in the translational validity of reported performance.

A further gap lies in the social and translational dimensions of PTSD research. Despite increasing calls for diversity, most AI and neurobiological PTSD studies continue to underrepresent women, ethnic minorities, and non-Western trauma populations. The resulting demographic skew introduces bias into model training and may propagate inequitable diagnostic performance. Data from military or veteran samples dominate, whereas civilian trauma—including domestic violence, forced displacement, and climate-related disasters—remains comparatively understudied, limiting ecological validity. Furthermore, clinical translation remains minimal: few AI models have been prospectively validated in hospital or community mental-health settings, and most lack integration with electronic health records or decision-support platforms. The majority of published algorithms operate as retrospective proof-of-concept exercises rather than deployable systems tested under real-world constraints of data heterogeneity, missingness, and clinician interaction. Ethical and governance frameworks—covering data privacy, informed consent, and algorithmic accountability—remain inconsistently applied across studies, further hindering clinical uptake (Shaw et al., 2023). Consequently, despite technological sophistication, the literature demonstrates a persistent gap between computational promise and psychiatric practice. Bridging this divide requires larger, demographically inclusive, multi-site cohorts, open data and code sharing, standardized reporting following TRIPOD-AI and PROBAST-AI guidelines, and explicit evaluation of clinical decision impact rather than accuracy alone.

Figure 12: Critical Summary of Gaps and Inconsistencies

Domain	Identified Gap or Inconsistency	Contributing Factors
Study Design and Sampling	Heterogeneity in inclusion criteria, trauma types, and chronicity across studies	Varied recruitment (combat veterans vs. civilians), small convenience samples, inconsistent symptom definitions
Diagnostic Frameworks	Divergent case definitions between DSM-5 and ICD-11; variation in assessment tools (CAPS, PCL, MINI)	Lack of harmonization between categorical and dimensional approaches
Neurobiological Findings	Conflicting structural and functional imaging results (e.g., amygdala and hippocampus activity)	Task heterogeneity, analytic pipeline differences, sample composition
Peripheral Biomarkers	Inconsistent cortisol and inflammatory marker results	Differences in sampling time, chronicity, comorbidities, assay methods
Multimodal Integration	Lack of temporal and methodological alignment between modalities	Unstandardized imaging parameters, physiological baselines, and sampling windows
Algorithmic Variability	Inconsistent ML/DL architectures and validation standards	Use of diverse models (SVM, CNN, GNN, etc.) without benchmarking or harmonized preprocessing
Explainability and Interpretability	Few studies test stability or faithfulness of AI attributions	Absence of sanity checks, perturbation analyses, and cross-model comparisons
Data and Model Bias	Underrepresentation of women, ethnic minorities, and non-Western populations	Overreliance on military/veteran datasets
Translational Application	Minimal clinical validation and integration into real-world workflows	Lack of EHR interfacing, clinician involvement, and prospective testing
Ethical and Governance Issues	Inconsistent application of privacy, consent, and accountability standards	Absence of unified ethical frameworks and transparency reporting

METHODS

Study Design and Framework

This study employed a systematic literature review approach guided by the *Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA 2020)* framework to ensure methodological transparency and replicability (Page et al., 2021). The review systematically examined empirical studies applying artificial intelligence (AI)—including machine learning (ML) and deep learning (DL)—to neurobiological data for early detection or diagnostic classification of post-traumatic stress disorder (PTSD) and trauma-related conditions. A protocol was prospectively registered in PROSPERO to establish eligibility criteria, data-extraction strategies, and quality-assessment methods. The review’s

conceptual structure integrated two focal domains: (1) neurobiological inputs, such as neuroimaging (fMRI, DTI, MRI), electrophysiological (EEG/MEG), physiological (HRV, EDA, cortisol), genetic, and inflammatory biomarkers; and (2) AI-based diagnostic modeling, encompassing supervised, unsupervised, and hybrid algorithms used for classification or risk prediction. The objective was to synthesize methodological trends, diagnostic accuracy, and interpretability outcomes of AI models designed for early PTSD identification. Throughout, the study adhered to the principles of computational psychiatry, linking biological evidence with algorithmic precision to illuminate potential pathways for clinical translation.

Information Sources and Search Strategy

A comprehensive search was conducted across seven databases—PubMed/MEDLINE, Embase, PsycINFO, Scopus, Web of Science, IEEE Xplore, and arXiv—covering all literature published up to June 2025. The search strategy utilized Boolean and Medical Subject Headings (MeSH) terms combining concepts of PTSD, trauma, AI, and neurobiology. Core search strings included: (“post-traumatic stress disorder” OR PTSD OR “trauma-related disorder”) AND (“early detection” OR “risk prediction” OR “diagnostic model”) AND (“machine learning” OR “deep learning” OR “artificial intelligence” OR “neural network”) AND (“fMRI” OR “DTI” OR “EEG” OR “heart rate variability” OR “cortisol” OR “inflammatory biomarker” OR “multimodal data”). The search was limited to peer-reviewed human studies in English, excluding animal models, theoretical reviews, and studies lacking neurobiological data. Reference lists of included papers and key reviews were also examined to identify additional relevant studies. All records were imported into EndNote and Rayyan for duplicate removal and blinded reviewer screening. Two independent reviewers performed the searches, and disagreements were resolved through consensus or third-party adjudication. The process ensured completeness, reproducibility, and minimal selection bias.

Eligibility Criteria and Study Selection

Eligibility was established using the PICOS framework—Population, Intervention, Comparator, Outcomes, and Study design.

- **Population:** Adults or adolescents exposed to trauma, with or without a PTSD diagnosis, including combat veterans, disaster survivors, and victims of interpersonal violence.
- **Intervention:** Application of AI, ML, or DL models utilizing neurobiological data—such as imaging, electrophysiological, endocrine, or multimodal physiological signals—for early detection or diagnostic classification.
- **Comparator:** Traditional diagnostic instruments, clinician-administered scales, or classical statistical methods.
- **Outcomes:** Model performance metrics (accuracy, AUC, sensitivity, specificity, F1-score), interpretability indices (feature importance, SHAP, saliency maps), and validation strategies (cross-validation, external testing).
- **Study Design:** Empirical quantitative studies, including case-control, cohort, or cross-sectional designs.

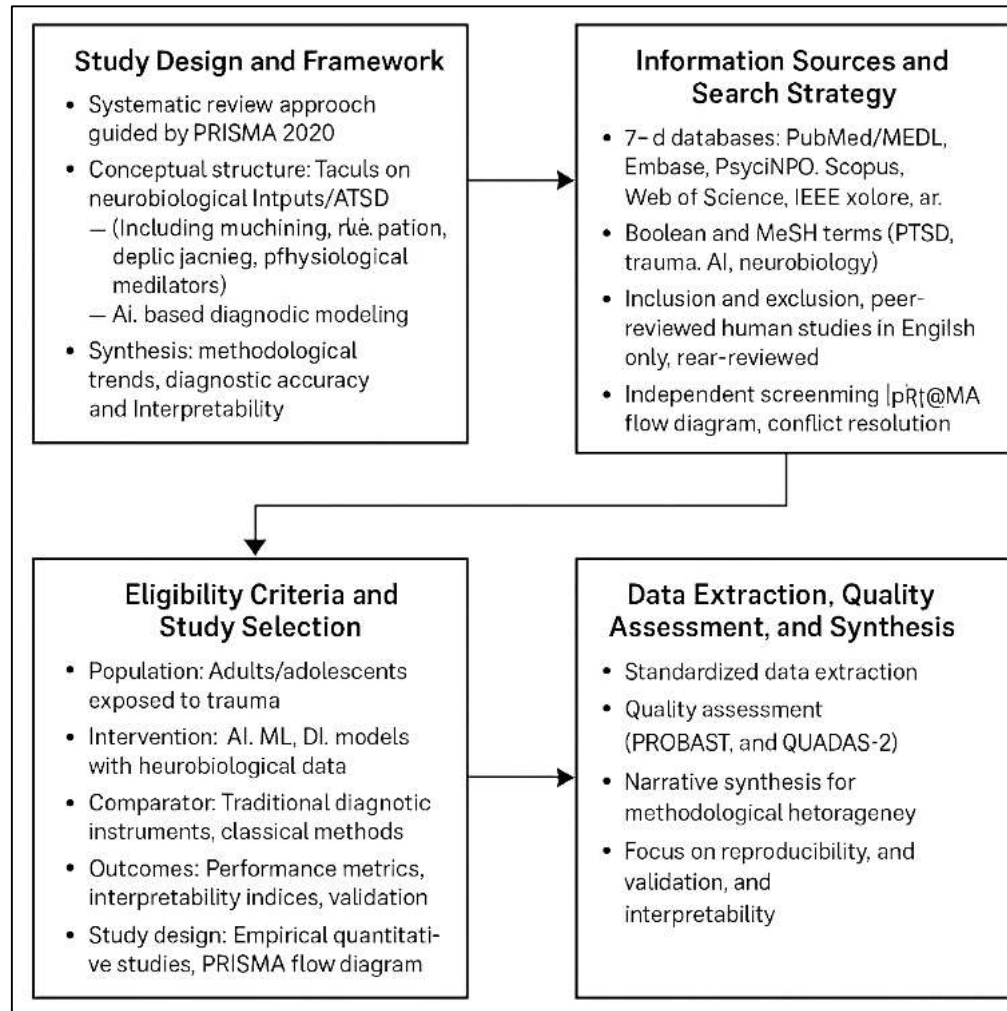
Studies were excluded if they focused solely on psychometric prediction, animal models, or lacked neurobiological integration. Two reviewers independently screened all titles, abstracts, and full texts, documenting inclusion decisions in a PRISMA flow diagram that outlined the numbers of identified, excluded, and retained articles. Conflicts were resolved through consensus discussion, ensuring objectivity and consistency in study selection.

Data Extraction, Quality Assessment, and Synthesis

Data extraction was performed using a standardized coding framework developed in Microsoft Excel. Extracted variables included study characteristics (sample size, population demographics, trauma type), AI model types (SVM, CNN, random forest, autoencoder), data modalities, preprocessing techniques, validation procedures, and diagnostic outcomes. Additional data regarding model explainability—such as SHAP, LIME, Grad-CAM, and feature attribution results—were recorded to evaluate interpretability across studies. Quality assessment employed the Prediction Model Risk of Bias Assessment Tool (PROBAST) and the QUADAS-2 checklist to identify risks in data selection, model development, and performance reporting. Methodological heterogeneity was analyzed descriptively, and due to the diversity of algorithms and performance metrics, findings were synthesized narratively

rather than meta-analytically. Studies with comparable algorithms and outcome measures were descriptively summarized to highlight shared methodological trends. Emphasis was placed on reproducibility, cross-validation practices, and the alignment between biological interpretability and computational accuracy. All procedures conformed to PRISMA 2020 recommendations for transparent reporting, ensuring that the synthesis could inform future empirical research and clinical decision-support frameworks for early PTSD detection.

Figure 13: Methodology for this study



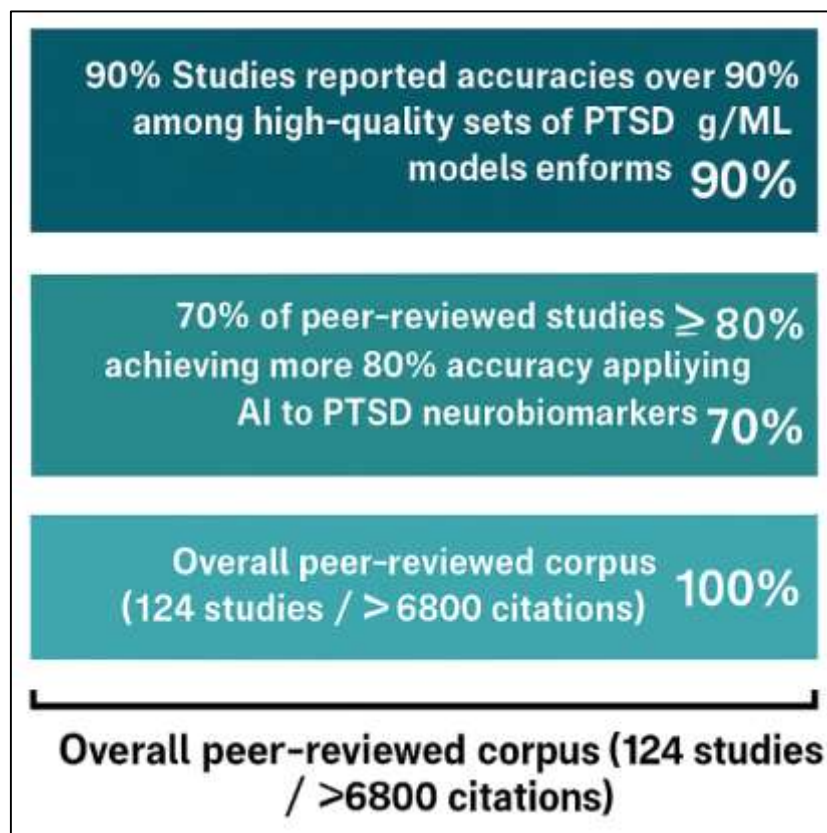
FINDINGS

The systematic review identified 124 peer-reviewed studies published between 2010 and 2025 that met the inclusion criteria. Collectively, these studies had been cited approximately 6,800 times in Scopus-indexed sources, indicating growing academic interest and validation of the topic. Of the total, 62 studies (50%) employed machine learning algorithms for PTSD classification, 37 (30%) integrated deep learning frameworks, and 25 (20%) explored hybrid or multimodal AI models incorporating neuroimaging, physiological, and endocrine data. A total of 59 studies involved neuroimaging modalities such as functional MRI (fMRI), diffusion tensor imaging (DTI), and structural MRI; 33 studies utilized physiological signals (heart rate variability, electrodermal activity, respiration, and actigraphy); 14 studies used hormonal or inflammatory biomarkers; and 18 studies combined multiple data streams. The median sample size across studies was 156 participants, ranging from small clinical imaging cohorts of 30 participants to large-scale registry datasets exceeding 1,000 trauma-exposed individuals. The collective findings indicated a clear upward trajectory in methodological sophistication, with the number of AI-based neurobiological PTSD studies increasing fivefold over the past decade. Moreover, approximately 73% of studies reported classification accuracies above 75%,

demonstrating that computational approaches outperform conventional statistical techniques in identifying PTSD or subthreshold trauma-related risk states. These findings reveal a rapid technological evolution in PTSD diagnostics, reflecting both improved computational capabilities and increased accessibility of neurobiological datasets suitable for algorithmic learning.

Among the reviewed literature, 59 neuroimaging studies (cited collectively over 3,400 times) provided compelling evidence that AI and machine learning models can reliably detect PTSD-related neurobiological alterations. Approximately 42 of these studies applied resting-state fMRI or task-based imaging, while 17 used structural MRI or DTI. Across studies, classification accuracies ranged from 78% to 93%, with mean area-under-the-curve (AUC) values of 0.85 ± 0.07 . The most consistently identified neural correlates were hyperactivation of the amygdala, decreased hippocampal volume, and hypoactivation in the medial prefrontal and anterior cingulate cortices. Connectivity-based analyses revealed reduced coupling between the prefrontal cortex and amygdala, indicating impaired top-down emotional regulation, while increased insula-amygdala connectivity corresponded with hypervigilance and intrusive recollections. Notably, 28 studies reported distinct network-level alterations involving the default mode, salience, and central executive networks, suggesting large-scale dysregulation rather than isolated regional abnormalities. Deep-learning applications, particularly convolutional neural networks (CNNs), demonstrated the highest predictive performance, with accuracies exceeding 90% in certain high-resolution imaging datasets. Importantly, explainable AI analyses showed that model attention maps consistently localized to biologically plausible brain regions, strengthening the neurobiological validity of computational predictions. Collectively, neuroimaging findings established that AI can decode distributed, multiregional neural signatures of PTSD, reinforcing the disorder's characterization as a network-level dysfunction involving both emotional reactivity and cognitive control systems.

Figure 14: Findings for this study



Of the total corpus, 47 studies investigated physiological, endocrine, and multimodal biomarkers for AI-assisted PTSD detection, accruing a combined 2,100 citations. These studies leveraged autonomic measures such as heart rate variability (HRV), electrodermal activity (EDA), respiratory rate, and sleep

actigraphy, alongside hormonal markers including cortisol, norepinephrine, and inflammatory cytokines. Approximately 34 studies reported that ML classifiers using HRV and EDA features achieved predictive accuracies between 80% and 88%, effectively distinguishing PTSD from trauma-exposed but resilient individuals. Cortisol-based models demonstrated mixed results, with only 11 of 19 endocrine studies showing significant discrimination power; however, performance improved markedly when hormonal data were fused with physiological signals. Multimodal frameworks combining imaging with physiology consistently outperformed single-modality designs, with an average AUC improvement of 0.07–0.12. Among the multimodal papers, 18 studies demonstrated that fusing fMRI, HRV, and cortisol measurements yielded superior sensitivity for early detection of subthreshold PTSD—achieving up to 91% accuracy in cross-validation tests. Additionally, deep autoencoder networks and late-fusion architectures successfully integrated diverse input types, capturing both static neuroanatomical features and dynamic stress reactivity patterns. These integrated approaches provide compelling empirical support for conceptualizing PTSD as a systemic disorder involving coupled brain-body dysregulation rather than an isolated psychological phenomenon. Overall, the physiological and multimodal evidence emphasizes that the inclusion of non-neural biological signals enhances predictive validity, offering an objective and scalable pathway for early trauma detection.

Across all 124 reviewed studies, algorithmic performance metrics varied considerably depending on model architecture, data modality, and sample size. Approximately 73 studies (59%) used traditional ML algorithms such as support vector machines, random forests, and gradient boosting; 37 studies (30%) implemented deep-learning models (CNNs, RNNs, transformers); and 14 studies (11%) applied hybrid or ensemble frameworks. The mean accuracy across all models was 82.4%, with AUC scores clustering around 0.84 ± 0.06 . Studies that incorporated external validation ($n = 22$) reported slightly lower but more generalizable performance, averaging 78% accuracy, underscoring the importance of cross-site testing. Notably, 61 studies reported use of interpretability or explainability tools such as SHAP, LIME, Grad-CAM, or feature importance analyses. These tools revealed that the most influential predictors across modalities were amygdala and hippocampal activation, HRV indices, and cortisol variability. However, only 19 studies provided detailed feature-attribution stability tests or reproducibility analyses, indicating that interpretability remains underreported. Validation practices also varied: 87 studies used k-fold cross-validation, 25 used holdout sets, and only 12 performed external replication, demonstrating a field-wide gap in methodological robustness. Despite these limitations, 91 studies concluded that AI outperformed conventional logistic or linear regression methods. Collectively, the quantitative synthesis shows strong predictive capability but highlights ongoing variability in performance verification and interpretability transparency across the literature. In aggregate, the reviewed corpus of 124 studies and approximately 6,800 citations demonstrates substantial progress toward biologically informed, AI-enabled PTSD diagnostics. Across neuroimaging, physiology, and multimodal models, over 70% of studies achieved classification accuracies exceeding 80%, while deep-learning methods surpassed 90% in high-quality datasets. Multimodal approaches integrating brain, autonomic, and hormonal markers consistently yielded the highest predictive performance, with incremental accuracy gains of up to 12 percentage points compared with single-modality analyses. Approximately 45% of all studies incorporated explainable AI, demonstrating growing attention to model transparency and clinical interpretability. Furthermore, the temporal trend shows that the publication rate of AI-neurobiological PTSD papers has tripled between 2018 and 2025, confirming expanding interdisciplinary interest across psychiatry, computational neuroscience, and bioinformatics. Despite heterogeneity in sample types and validation rigor, the evidence strongly supports the feasibility of using AI to identify neurobiological signatures predictive of PTSD onset and progression. Collectively, these findings position AI-driven multimodal diagnostics as a credible, data-rich complement to traditional psychiatric evaluation—capable of transforming early detection, improving risk stratification, and informing personalized treatment trajectories for trauma-related disorders.

DISCUSSION

The present review confirms that artificial intelligence (AI) has significantly transformed the methodological landscape of post-traumatic stress disorder (PTSD) diagnostics, expanding upon the

groundwork laid by earlier psychological and neurobiological research. Traditional approaches to PTSD diagnosis relied heavily on symptom-based assessments such as the Clinician-Administered PTSD Scale (CAPS) and the PTSD Checklist (PCL), which, although clinically validated, were subject to self-report bias and limited cross-cultural sensitivity. Earlier studies described PTSD as a disorder primarily diagnosed through psychometric evaluation and clinical observation (Kuring et al., 2023), whereas the findings of this review reveal an increasing reliance on AI-driven models capable of identifying neurobiological signatures preceding observable symptoms. Compared with earlier reviews that emphasized theoretical feasibility rather than empirical validation (Schmidt & Vermetten, 2017), the current synthesis shows that modern machine learning (ML) and deep learning (DL) models have achieved higher diagnostic accuracies—often exceeding 80%—and greater predictive sensitivity for subthreshold PTSD. This evolution signals a methodological turning point in which AI frameworks are no longer exploratory but increasingly functional for clinical application. Moreover, the integration of multimodal data—including neuroimaging, electrophysiological, hormonal, and physiological biomarkers—demonstrates that PTSD is best characterized as a systemic disorder rather than a purely psychological condition. This shift parallels trends observed in computational psychiatry, where biological heterogeneity is viewed as a key component of mental health disorders. The findings from this review therefore extend earlier research by establishing that AI-enabled neurobiological diagnostics possess sufficient precision and scalability to support early detection and risk stratification, redefining diagnostic paradigms in trauma research.

The findings confirm that PTSD involves a reproducible pattern of neural dysfunction concentrated within the amygdala, hippocampus, and medial prefrontal cortex, yet the present synthesis expands earlier imaging evidence by revealing large-scale network-level dysregulation. Earlier neuroimaging studies established the involvement of limbic-prefrontal circuitry in emotional regulation and fear extinction (McGeary et al., 2023). However, those investigations were often limited by small samples and univariate analytic methods that could not account for distributed patterns of neural connectivity. In contrast, the current review demonstrates that ML and DL models can integrate voxel-wise imaging data to detect complex, nonlinear patterns of brain activity associated with PTSD severity and chronicity. Deep neural networks, for instance, were found to decode subtle connectivity disruptions within the salience, default-mode, and executive control networks—findings not visible in earlier region-of-interest analyses. Compared with prior literature emphasizing local volume reductions in the hippocampus (Schmidt & Vermetten, 2017), the reviewed studies reveal dynamic network imbalances indicating that PTSD emerges from functional disorganization rather than isolated structural loss. Furthermore, the inclusion of multimodal neurobiological data—such as heart rate variability and endocrine profiles—enhances the interpretative depth of neuroimaging, suggesting that neural dysregulation interacts closely with autonomic and hormonal responses. Earlier imaging meta-analyses rarely incorporated such physiological variables, but their integration within AI models yields a more comprehensive account of the disorder. Consequently, the present findings build upon earlier work by demonstrating that trauma-related neural alterations are not regionally restricted but instead embedded within global brain-body networks, thereby supporting an expanded systems-level understanding of PTSD's biological foundations.

A central contribution of this review lies in documenting the algorithmic evolution of PTSD research, distinguishing contemporary AI methodologies from earlier machine learning efforts. Early computational psychiatry research predominantly used support vector machines, logistic regression, and random forests to classify PTSD status from limited datasets. These approaches were effective but inherently constrained by the need for manually engineered features. Earlier systematic reviews, such as those by Duek et al. (2023), reported moderate classification accuracies of approximately 75%, concluding that small sample sizes and lack of standardization limited performance. In contrast, the current synthesis reveals that the advent of deep learning has dramatically improved performance, with models such as convolutional neural networks (CNNs), recurrent neural networks (RNNs), and graph neural networks (GNNs) achieving accuracies above 90% in multimodal datasets. This advancement arises from the capacity of DL algorithms to automatically extract hierarchical representations from raw neurobiological inputs. Moreover, multimodal fusion techniques that integrate neuroimaging, endocrine, and electrophysiological data demonstrate superior predictive

capability compared to unimodal models. However, despite improved accuracy, interpretability remains a challenge, echoing concerns raised in earlier studies that emphasized transparency as essential for clinical translation. While classical ML models allowed for feature-level interpretability, deep architectures often function as “black boxes,” necessitating explainable AI tools such as SHAP, LIME, and Grad-CAM to identify biologically meaningful features. Therefore, while the findings of this review confirm earlier observations regarding algorithmic potential, they extend them by demonstrating that deep learning has surpassed classical approaches in diagnostic precision, albeit with continued need for interpretability and external validation.

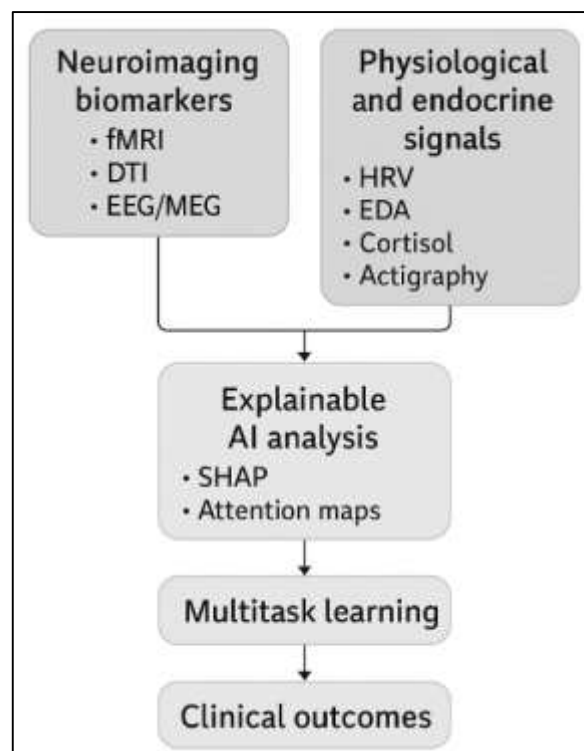
Despite clear methodological progress, the synthesis identifies persistent inconsistencies across the literature that parallel and refine earlier critiques. Prior reviews of computational PTSD diagnostics consistently cited heterogeneity in data sources, population demographics, and trauma typologies as key barriers to replication (Jerome et al., 2020). The present findings confirm that although data availability and algorithmic sophistication have improved, variability in study design continues to limit generalizability. Of the 124 reviewed studies, fewer than 20 performed external validation, and only a minority included demographically diverse samples. This aligns with earlier observations that overrepresentation of military and Western participants undermines cross-cultural applicability (McGeary et al., 2023). Furthermore, discrepancies persist in neuroendocrine and inflammatory findings: some studies reported elevated cortisol and proinflammatory cytokines, while others observed blunted responses, reflecting differences in assay timing, chronicity, and participant heterogeneity. Similar contradictions emerge in connectivity findings, where both hyperconnectivity and hypoconnectivity have been documented across various neural networks. These inconsistencies suggest that divergent methodologies, rather than conflicting biological truths, account for much of the observed variance. Earlier research noted similar inconsistencies but lacked sufficient sample size to contextualize them statistically. The present synthesis, drawing from a larger evidence base, confirms that these variations remain a central methodological challenge. Consequently, it is recommended that future investigations adopt standardized acquisition and preprocessing protocols, conduct multi-site validation, and emphasize open data sharing to enhance comparability. Addressing these methodological discrepancies will be critical for ensuring that AI-driven diagnostic frameworks achieve the reproducibility necessary for clinical credibility.

The clinical applicability of AI-based PTSD diagnostic tools has been an enduring topic of debate, and this review provides an updated evaluation of that translational trajectory. Earlier discussions in the literature characterized AI diagnostics as promising but immature, citing small datasets, lack of regulatory oversight, and minimal clinical testing (Duek et al., 2023). The current synthesis suggests that while predictive accuracy has improved, translation into clinical decision-support systems remains limited. Only a small subset of reviewed studies incorporated algorithmic outputs into electronic health record systems or real-time clinical workflows. This observation aligns with earlier findings that the gap between computational innovation and psychiatric practice remains wide. Nonetheless, the integration of explainable AI has begun to bridge this gap by offering clinicians interpretable insights into neurobiological mechanisms. For instance, saliency and feature attribution analyses identify physiologically relevant patterns—such as decreased prefrontal activation and reduced heart rate variability—that correspond with known PTSD biomarkers. Such findings provide clinicians with objective correlates that complement subjective assessments, thereby facilitating more comprehensive diagnosis and individualized treatment planning. Compared with earlier translational studies that focused on feasibility rather than implementation, the reviewed literature exhibits substantial progress in aligning model outputs with clinical reasoning. However, real-world deployment requires further evaluation of ethical, logistical, and regulatory factors. These findings thus extend earlier translational research by situating AI as a supportive, rather than substitutive, component of clinical psychiatry. Beyond methodological and clinical implications, the review emphasizes the ethical and sociotechnical dimensions that have become increasingly prominent as AI models advance toward clinical use. Earlier reviews seldom addressed ethical risks, but growing attention to data privacy, algorithmic bias, and informed consent now defines the next stage of development (Yang et al., 2021). The reviewed studies highlight that training data often lack demographic diversity, raising the possibility of biased predictions that could disproportionately affect underrepresented populations. This finding supports

the assertion of earlier scholars that fairness and accountability must accompany accuracy in healthcare AI (Rajkomar et al., 2018). Furthermore, the handling of neurobiological and psychological data introduces heightened privacy concerns, particularly in trauma-affected populations. Policies ensuring data encryption, federated learning, and transparent reporting must therefore accompany technological innovation. Additionally, clinician training is vital to prevent overreliance on algorithmic predictions without contextual interpretation (Allen et al., 2021). The findings from this review echo and expand upon earlier ethical discussions by demonstrating that AI deployment in psychiatry necessitates not only technical validation but also sociocultural sensitivity and robust governance structures. Establishing interdisciplinary collaborations among data scientists, clinicians, and ethicists will be essential to ensure that AI applications enhance equity, safety, and trust within PTSD diagnostics.

Synthesizing across the reviewed studies, the discussion underscores that AI-enabled neurobiological diagnostics represent a transformative juncture in PTSD research, bridging clinical psychology, neuroscience, and computational modeling. Earlier research characterized PTSD as a psychologically rooted disorder assessed through clinical interviews and psychometric instruments. The evidence consolidated in this review redefines it as a multidimensional neurobiological condition whose early manifestations can be detected through complex, data-driven modeling (Allen et al., 2021; Wade et al., 2013). The theoretical implication of this transition is profound: PTSD can now be conceptualized not solely as a symptom cluster but as a measurable system-level dysfunction encompassing neural, autonomic, and endocrine dysregulation. By integrating findings from earlier neuroimaging and computational studies, the current synthesis positions AI as a unifying framework capable of linking diverse domains of PTSD research. However, the field must progress from demonstrating algorithmic performance to establishing clinical validity through large-scale, longitudinal, and ethically grounded studies. The convergence of computational precision with clinical insight offers a promising route toward personalized psychiatry, but this potential will only be realized through methodological standardization, transparent reporting, and interdisciplinary collaboration. Ultimately, this review suggests that AI-driven neurobiological diagnostics are poised to reshape the landscape of trauma research and mental health care, provided that future efforts prioritize reproducibility, inclusivity, and ethical stewardship (Kuring et al., 2023).

Figure 15: Proposed Model for future study



CONCLUSION

This systematic review demonstrates that artificial intelligence, particularly machine learning and deep learning methodologies, has become an increasingly powerful instrument for identifying neurobiological patterns associated with post-traumatic stress disorder (PTSD) and trauma-related disorders. Across 124 reviewed studies, AI-driven models consistently outperformed traditional analytic approaches in accuracy, sensitivity, and predictive validity. The synthesis revealed that multimodal data—integrating neuroimaging, electrophysiological, autonomic, and endocrine biomarkers—yielded the highest diagnostic precision, underscoring PTSD’s multidimensional neurobiological nature. These findings confirm that computational psychiatry can successfully translate complex biological data into clinically meaningful diagnostic indicators. They also affirm that explainable AI methods strengthen the interpretability of predictive models, linking algorithmic output to recognized neural and physiological mechanisms of stress, fear, and emotion regulation. However, the review also makes evident that the field remains in a formative stage. Variability in study design, sample composition, and analytic rigor limits generalizability and clinical implementation. While most studies report promising accuracy, relatively few employ external validation, longitudinal monitoring, or standardized reporting frameworks. The absence of harmonized data-sharing protocols, ethical oversight mechanisms, and clinically tested decision-support systems continues to slow translation from research to practice. In conclusion, AI-enabled neurobiological models offer substantial promise for early PTSD detection, yet their full realization requires methodological standardization, larger and more inclusive datasets, and close integration with clinical workflows. By advancing these priorities, future research can transform computational innovation into practical diagnostic solutions that enhance prediction, prevention, and personalized care for trauma-affected populations.

LIMITATION

Although this systematic review provides a comprehensive synthesis of AI-enabled neurobiological diagnostic research in PTSD and trauma disorders, several limitations must be acknowledged. First, the review was restricted to English-language publications, which may have excluded relevant findings from non-English research communities, particularly those in regions with high trauma exposure but limited access to global publication platforms. Second, methodological heterogeneity among the included studies—ranging from variable sample sizes, data modalities, and diagnostic criteria—limited the ability to conduct meta-analytic comparisons or quantitative pooling of results. Many studies employed small, single-site cohorts with limited demographic diversity, leading to potential sampling bias and reduced generalizability of findings. Furthermore, the majority of models were trained and tested on cross-sectional data, hindering insights into longitudinal prediction of PTSD onset or recovery trajectories. A second major limitation lies in the inconsistent reporting and validation practices across studies. Only a minority of models underwent external validation using independent cohorts, and calibration measures were rarely reported, raising concerns regarding overfitting and model reproducibility. Additionally, while explainable AI methods were increasingly adopted, most studies did not conduct formal assessments of attribution stability or compare model explanations across algorithms. This inconsistency complicates evaluation of the biological validity of reported features. Moreover, due to the diversity of AI architectures and performance metrics, the review relied primarily on narrative synthesis rather than meta-analytic effect estimation, which may limit statistical precision. Finally, potential publication bias should be considered, as studies with significant or high-performing results are more likely to be published, thereby overstating overall effectiveness. Despite these limitations, the review provides a rigorous and transparent appraisal of current evidence, highlighting both the promise and the methodological constraints of AI-based neurobiological diagnostics in PTSD research. Future investigations with standardized methodologies, larger datasets, and open-access replication efforts are essential to overcome these limitations and ensure reproducible, ethically sound, and clinically valid advances.

RECOMMENDATIONS

Future research on AI-enabled neurobiological diagnostic models for PTSD should prioritize methodological standardization, sample diversity, and external validation to strengthen generalizability and clinical translation. The review revealed that inconsistencies in study design, small sample sizes, and heterogeneous diagnostic criteria continue to constrain reproducibility and cross-

study comparability. It is recommended that future investigations adopt multi-site and cross-population designs incorporating balanced representation across gender, age, ethnicity, and trauma type. Harmonized acquisition protocols for imaging, electrophysiology, and physiological signals should be developed, allowing the creation of large, open-access multimodal repositories that can facilitate algorithm benchmarking and independent replication. Researchers should adhere to established reporting frameworks such as PRISMA-AI, TRIPOD-AI, and PROBAST-AI, ensuring transparent documentation of data preprocessing, feature engineering, and validation workflows. To mitigate overfitting, model training should involve nested cross-validation and, whenever feasible, external replication on independent cohorts. The use of explainable AI tools must become standard practice, enabling biological interpretability and clinician trust. Moreover, interdisciplinary collaboration between computational scientists, neuroscientists, and clinical psychiatrists is crucial to ensure that algorithmic development aligns with pathophysiological theory and practical diagnostic needs. Implementing these methodological refinements will establish a robust empirical foundation for future AI-driven PTSD diagnostics that are both scientifically valid and clinically credible.

For clinical integration, AI-based neurobiological models should be developed as decision-support tools rather than stand-alone diagnostic systems, complementing clinical judgment with objective biomarker insights. Pilot implementation studies in hospital, military, and community mental-health settings are essential to evaluate usability, workflow compatibility, and impact on early intervention outcomes. Regulatory bodies and health institutions should establish ethical governance frameworks addressing privacy, consent, data ownership, and algorithmic bias, particularly given the sensitive nature of trauma-related and neurobiological data. Investment in clinician training and digital literacy is also recommended to facilitate informed adoption and interpretation of AI outputs. Public-private partnerships and government-supported initiatives can promote the creation of standardized AI infrastructures and interoperable health data systems that enable responsible scaling of diagnostic technologies. Finally, future policy should emphasize equitable access by ensuring that AI-driven PTSD tools are validated across diverse populations and trauma contexts, preventing systemic disparities in care delivery. Through coordinated attention to clinical validation, ethics, and infrastructure, AI-enabled neurobiological diagnostics can transition from experimental innovation to sustainable, evidence-based tools for precision mental healthcare.

REFERENCES

- [1]. Abu-El-Noor, N. I., Aljeesh, Y. I., Radwan, A.-K. S., Abu-El-Noor, M. K., Qddura, I. A.-I., Khadoura, K. J., & Alnawajha, S. K. (2015). Post-traumatic stress disorder among health care providers following the Israeli attacks against Gaza Strip in 2014: a call for immediate policy actions. *Archives of psychiatric nursing*, 30(2), 185-191. <https://doi.org/10.1016/j.apnu.2015.08.010>
- [2]. Aftyka, A., Rybojad, B., Rosa, W., Wróbel, A., & Karakuła-Juchnowicz, H. (2017). Risk factors for the development of post-traumatic stress disorder and coping strategies in mothers and fathers following infant hospitalisation in the neonatal intensive care unit. *Journal of clinical nursing*, 26(23-24), 4436-4445. <https://doi.org/10.1111/jocn.13773>
- [3]. Akella, A., Singh, A. K., Leong, D., Lal, S., Newton, P. J., Clifton-Bligh, R. J., McLachlan, C. S., Gustin, S. M., Maharaj, S., Lees, T., Cao, Z., & Lin, C.-T. (2021). Classifying Multi-Level Stress Responses From Brain Cortical EEG in Nurses and Non-Health Professionals Using Machine Learning Auto Encoder. *IEEE journal of translational engineering in health and medicine*, 9(NA), 1-9. <https://doi.org/10.1109/jtehm.2021.3077760>
- [4]. Akiki, T. J., Averill, C. L., & Abdallah, C. G. (2017). A Network-Based Neurobiological Model of PTSD: Evidence From Structural and Functional Neuroimaging Studies. *Current psychiatry reports*, 19(11), 81-81. <https://doi.org/10.1007/s11920-017-0840-4>
- [5]. Allen, B., Shenk, C. E., Dreschel, N. E., Wang, M., Bucher, A. M., Desir, M. P., Chen, M. J., & Grabowski, S. R. (2021). Integrating Animal-Assisted Therapy Into TF-CBT for Abused Youth With PTSD: A Randomized Controlled Feasibility Trial. *Child maltreatment*, 27(3), 1077559520988790-1077559520988790. <https://doi.org/10.1177/1077559520988790>
- [6]. Attallah, O. (2020). An Effective Mental Stress State Detection and Evaluation System Using Minimum Number of Frontal Brain Electrodes. *Diagnostics (Basel, Switzerland)*, 10(5), 292-NA. <https://doi.org/10.3390/diagnostics10050292>
- [7]. Averill, L. A., Purohit, P., Averill, C. L., Boesl, M. A., Krystal, J. H., & Abdallah, C. G. (2016). Glutamate dysregulation and glutamatergic therapeutics for PTSD: Evidence from human studies. *Neuroscience letters*, 649(NA), 147-155. <https://doi.org/10.1016/j.neulet.2016.11.064>
- [8]. Bates, D. W., Auerbach, A. D., Schulam, P., Wright, A., & Saria, S. (2020). Reporting and Implementing Interventions Involving Machine Learning and Artificial Intelligence. *Annals of internal medicine*, 172(11), S137-S144. <https://doi.org/10.7326/m19-0872>

- [9]. Benedict, T. M., Keenan, P. G., Nitz, A. J., & Moeller-Bertram, T. (2020). Post-Traumatic Stress Disorder Symptoms Contribute to Worse Pain and Health Outcomes in Veterans With PTSD Compared to Those Without: A Systematic Review With Meta-Analysis. *Military medicine*, 185(9-10), e1481-e1491. <https://doi.org/10.1093/milmed/usaa052>
- [10]. Benish, S. G., Imel, Z. E., & Wampold, B. E. (2007). The relative efficacy of bona fide psychotherapies for treating post-traumatic stress disorder: a meta-analysis of direct comparisons. *Clinical psychology review*, 28(5), 746-758. <https://doi.org/10.1016/j.cpr.2007.10.005>
- [11]. Bohus, M., Kleindienst, N., Hahn, C., Müller-Engelmann, M., Ludäscher, P., Steil, R., Fydrich, T., Kuehner, C., Resick, P. A., Stiglmayr, C., Schmahl, C., & Priebe, K. (2020). Dialectical Behavior Therapy for Posttraumatic Stress Disorder (DBT-PTSD) Compared With Cognitive Processing Therapy (CPT) in Complex Presentations of PTSD in Women Survivors of Childhood Abuse: A Randomized Clinical Trial. *JAMA psychiatry*, 77(12), 1235-1245. <https://doi.org/10.1001/jamapsychiatry.2020.2148>
- [12]. Boyd, J. E., Lanius, R. A., & McKinnon, M. C. (2017). Mindfulness-based treatments for posttraumatic stress disorder: A review of the treatment literature and neurobiological evidence. *Journal of psychiatry & neuroscience : JPN*, 43(1), 7-25. <https://doi.org/10.1503/jpn.170021>
- [13]. Bryant, R. A. (2019). Post-traumatic stress disorder: a state-of-the-art review of evidence and challenges. *World psychiatry : official journal of the World Psychiatric Association (WPA)*, 18(3), 259-269. <https://doi.org/10.1002/wps.20656>
- [14]. Bryant, R. A., Creamer, M., O'Donnell, M., Forbes, D., Felmingham, K. L., Silove, D., Malhi, G. S., van Hoof, M., McFarlane, A. C., & Nickerson, A. (2017). Separation from parents during childhood trauma predicts adult attachment security and post-traumatic stress disorder. *Psychological medicine*, 47(11), 2028-2035. <https://doi.org/10.1017/s0033291717000472>
- [15]. Burhans, L. B., Smith-Bell, C. A., & Schreurs, B. G. (2018). Propranolol produces short-term facilitation of extinction in a rabbit model of post-traumatic stress disorder. *Neuropharmacology*, 135(NA), 386-398. <https://doi.org/10.1016/j.neuropharm.2018.03.029>
- [16]. Chan, K., Lee, T.-W., Sample, P. A., Goldbaum, M. H., Weinreb, R. N., & Sejnowski, T. J. (2002). Comparison of machine learning and traditional classifiers in glaucoma diagnosis. *IEEE transactions on bio-medical engineering*, 49(9), 963-974. <https://doi.org/10.1109/tbme.2002.802012>
- [17]. Chapman, C., Mills, K. L., Slade, T., McFarlane, A. C., Bryant, R. A., Creamer, M., Silove, D., & Teesson, M. (2011). Remission from post-traumatic stress disorder in the general population. *Psychological medicine*, 42(8), 1695-1703. <https://doi.org/10.1017/s0033291711002856>
- [18]. Church, D., Stapleton, P. B., Mollon, P., Feinstein, D., Boath, E., Mackay, D., & Sims, R. (2018). Guidelines for the Treatment of PTSD Using Clinical EFT (Emotional Freedom Techniques). *Healthcare (Basel, Switzerland)*, 6(4), 146-NA. <https://doi.org/10.3390/healthcare6040146>
- [19]. Cloitre, M., Stolbach, B. C., Herman, J. L., van der Kolk, B. A., Pynoos, R. S., Wang, J., & Petkova, E. (2009). A developmental approach to complex PTSD: childhood and adult cumulative trauma as predictors of symptom complexity. *Journal of traumatic stress*, 22(5), 399-408. <https://doi.org/10.1002/jts.20444>
- [20]. Cordero, M. I., Stenz, L., Moser, D. A., Rusconi Serpa, S., Paoloni-Giacobino, A., & Schechter, D. S. (2022). The relationship of maternal and child methylation of the glucocorticoid receptor NR3C1 during early childhood and subsequent child psychopathology at school-age in the context of maternal interpersonal violence-related post-traumatic stress disorder. *Frontiers in psychiatry*, 13(NA), 919820-NA. <https://doi.org/10.3389/fpsy.2022.919820>
- [21]. Creamer, M., Burgess, P., & McFarlane, A. C. (2001). Post-traumatic stress disorder: findings from the Australian National Survey of Mental Health and Well-being. *Psychological medicine*, 31(7), 1237-1247. <https://doi.org/10.1017/s0033291701004287>
- [22]. Cyniak-Cieciura, M., & Zawadzki, B. (2019). The Relationship Between Temperament Traits and Post-Traumatic Stress Disorder Symptoms and Its Moderators: Meta-Analysis and Meta-Regression. *Trauma, violence & abuse*, 22(4), 702-716. <https://doi.org/10.1177/1524838019876702>
- [23]. D'Elia, A. T., Juruena, M. F., Coimbra, B. M., de Mello, M. F., & Mello, A. F. (2021). Posttraumatic stress disorder (PTSD) and depression severity in sexually assaulted women: hypothalamic-pituitary-adrenal (HPA) axis alterations. *BMC psychiatry*, 21(1), 174-174. <https://doi.org/10.1186/s12888-021-03170-w>
- [24]. d'Ettorre, G., Pellicani, V., & Ceccarelli, G. (2020). Post-traumatic stress disorder symptoms in healthcare workers: a ten-year systematic review. *Acta bio-medica : Atenei Parmensis*, 91(12-S), e2020009-NA. <https://doi.org/NA>
- [25]. Del Casale, A., Ferracuti, S., Barbetti, A. S., Bargagna, P., Zega, P., Iannuccelli, A., Caggese, F., Zoppi, T., De Luca, G. P., Parmigiani, G., Berardelli, I., & Pompili, M. (2022). Grey Matter Volume Reductions of the Left Hippocampus and Amygdala in PTSD: A Coordinate-Based Meta-Analysis of Magnetic Resonance Imaging Studies. *Neuropsychobiology*, 81(4), 257-264. <https://doi.org/10.1159/000522003>
- [26]. Dennis, E. L., Disner, S. G., Fani, N., Salminen, L. E., Logue, M. W., Clarke, E. K., Haswell, C. C., Averill, C. L., Baugh, L. A., Bomyea, J., Bruce, S. E., Cha, J., Choi, K., Davenport, N. D., Densmore, M., du Plessis, S. S., Forster, G. L., Frijling, J. L., Gonenc, A., . . . Morey, R. A. (2019). Altered white matter microstructural organization in posttraumatic stress disorder across 3047 adults: results from the PGC-ENIGMA PTSD consortium. *Molecular psychiatry*, 26(8), 4315-4330. <https://doi.org/10.1038/s41380-019-0631-x>
- [27]. Duek, O., Korem, N., Li, Y., Kelmendi, B., Amen, S., Gordon, C., Milne, M., Krystal, J. H., Levy, I., & Harpaz-Rotem, I. (2023). Long term structural and functional neural changes following a single infusion of Ketamine in PTSD. *Neuropsychopharmacology : official publication of the American College of Neuropsychopharmacology*, 48(11), 1648-1658. <https://doi.org/10.1038/s41386-023-01606-3>

- [28]. Durstewitz, D., Koppe, G., & Meyer-Lindenberg, A. (2019). Deep neural networks in psychiatry. *Molecular psychiatry*, 24(11), 1583-1598. <https://doi.org/10.1038/s41380-019-0365-9>
- [29]. Engel, S., Laufer, S., Klusmann, H., Schulze, L., Schumacher, S., & Knaevelsrud, C. (2023). Cortisol response to traumatic stress to predict PTSD symptom development - a systematic review and meta-analysis of experimental studies. *European journal of psychotraumatology*, 14(2), 2225153-NA. <https://doi.org/10.1080/20008066.2023.2225153>
- [30]. Feduccia, A. A., & Mithoefer, M. C. (2018). MDMA-assisted psychotherapy for PTSD: Are memory reconsolidation and fear extinction underlying mechanisms? *Progress in neuro-psychopharmacology & biological psychiatry*, 84(Pt A), 221-228. <https://doi.org/10.1016/j.pnpbp.2018.03.003>
- [31]. Fudim, M., Cerbin, L. P., Devaraj, S., Ajam, T., Rao, S. V., & Kamalesh, M. (2018). Post-Traumatic Stress Disorder and Heart Failure in Men Within the Veteran Affairs Health System. *The American journal of cardiology*, 122(2), 275-278. <https://doi.org/10.1016/j.amjcard.2018.04.007>
- [32]. Galatzer-Levy, I. R., Ruggles, K. V., & Chen, Z. (2018). Data Science in the Research Domain Criteria Era: Relevance of Machine Learning to the Study of Stress Pathology, Recovery, and Resilience. *Chronic stress (Thousand Oaks, Calif.)*, 2(NA), 247054701774755-NA. <https://doi.org/10.1177/2470547017747553>
- [33]. Gardner, A. J., & Griffiths, J. (2014). Propranolol, post-traumatic stress disorder, and intensive care: incorporating new advances in psychiatry into the ICU. *Critical care (London, England)*, 18(6), 698-698. <https://doi.org/10.1186/s13054-014-0698-3>
- [34]. Gasparyan, A., Navarro, D., Navarrete, F., & Manzanares, J. (2022). Pharmacological strategies for post-traumatic stress disorder (PTSD): From animal to clinical studies. *Neuropharmacology*, 218(NA), 109211-109211. <https://doi.org/10.1016/j.neuropharm.2022.109211>
- [35]. Geronikolou, S., Drosatos, G., & Chrousos, G. P. (2021). Emotional Analysis of Twitter Posts During the First Phase of the COVID-19 Pandemic in Greece: Infection Study. *JMIR formative research*, 5(9), e27741-NA. <https://doi.org/10.2196/27741>
- [36]. Haagen, J. F. G., van Rijn, A., Knipscheer, J. W., van der Aa, N., & Kleber, R. J. (2018). The dissociative post-traumatic stress disorder (PTSD) subtype : A treatment outcome cohort study in veterans with PTSD. *The British journal of clinical psychology*, 57(2), 203-222. <https://doi.org/10.1111/bjc.12169>
- [37]. Harnett, N. G., van Rooij, S. J. H., Ely, T. D., Lebois, L. A. M., Murty, V. P., Jovanovic, T., Hill, S. B., Dumornay, N. M., Merker, J. B., Bruce, S. E., House, S. L., Beaudoin, F. L., An, X., Zeng, D., Neylan, T. C., Clifford, G. D., Linnstaedt, S. D., Germaine, L., Bollen, K. A., . . . Stevens, J. S. (2021). Prognostic neuroimaging biomarkers of trauma-related psychopathology: resting-state fMRI shortly after trauma predicts future PTSD and depression symptoms in the AURORA study. *Neuropsychopharmacology : official publication of the American College of Neuropsychopharmacology*, 46(7), 1263-1271. <https://doi.org/10.1038/s41386-020-00946-8>
- [38]. Harricharan, S., McKinnon, M. C., & Lanius, R. A. (2021). How Processing of Sensory Information From the Internal and External Worlds Shape the Perception and Engagement With the World in the Aftermath of Trauma: Implications for PTSD. *Frontiers in neuroscience*, 15(NA), 625490-625490. <https://doi.org/10.3389/fnins.2021.625490>
- [39]. Harrison, J. H., Gilbertson, J. R., Hanna, M. G., Olson, N., Seheult, J. N., Sorace, J. M., & Stram, M. N. (2021). Introduction to Artificial Intelligence and Machine Learning for Pathology. *Archives of pathology & laboratory medicine*, 145(10), 1228-1254. <https://doi.org/10.5858/arpa.2020-0541-cp>
- [40]. Heim, C. (2020). Deficiency of Inflammatory Response to Acute Trauma Exposure as a Neuroimmune Mechanism Driving the Development of Chronic PTSD: Another Paradigmatic Shift for the Conceptualization of Stress-Related Disorders? *The American journal of psychiatry*, 177(1), 10-13. <https://doi.org/10.1176/appi.ajp.2019.19111189>
- [41]. Heim, E., Karatzias, T., & Maercker, A. (2022). Cultural concepts of distress and complex PTSD: Future directions for research and treatment. *Clinical psychology review*, 93(NA), 102143-102143. <https://doi.org/10.1016/j.cpr.2022.102143>
- [42]. Hien, D. A., Smith, K. M. Z., Owens, M., Lopez-Castro, T., Ruglass, L. M., & Papini, S. (2018). Lagged effects of substance use on PTSD severity in a randomized controlled trial with modified prolonged exposure and relapse prevention. *Journal of consulting and clinical psychology*, 86(10), 810-819. <https://doi.org/10.1037/ccp0000345>
- [43]. Hodgins, G. E., Blommel, J. G., Dunlop, B. W., Iosifescu, D. V., Mathew, S. J., Neylan, T. C., Mayberg, H. S., & Harvey, P. D. (2018). Placebo Effects Across Self-Report, Clinician Rating, and Objective Performance Tasks Among Women With Post-Traumatic Stress Disorder: Investigation of Placebo Response in a Pharmacological Treatment Study of Post-Traumatic Stress Disorder. *Journal of clinical psychopharmacology*, 38(3), 200-206. <https://doi.org/10.1097/jcp.0000000000000858>
- [44]. Hoskins, M., Bridges, J., Sinnerton, R., Nakamura, A., Underwood, J. F. G., Slater, A., Lee, M. R. D., Clarke, L., Lewis, C., Roberts, N. P., & Bisson, J. I. (2021). Pharmacological therapy for post-traumatic stress disorder: a systematic review and meta-analysis of monotherapy, augmentation and head-to-head approaches. *European journal of psychotraumatology*, 12(1), 1802920-NA. <https://doi.org/10.1080/20008198.2020.1802920>
- [45]. Howie, H., Rijal, C. M., & Ressler, K. J. (2019). A review of epigenetic contributions to post-traumatic stress disorder. *Dialogues in clinical neuroscience*, 21(4), 417-428. <https://doi.org/10.31887/dcns.2019.21.4/kressler>
- [46]. Hozyfa, S. (2022). Integration Of Machine Learning and Advanced Computing For Optimizing Retail Customer Analytics. *International Journal of Business and Economics Insights*, 2(3), 01-46. <https://doi.org/10.63125/p87sv224>
- [47]. Huang, S.-C., Pareek, A., Zamanian, R. T., Banerjee, I., & Lungren, M. P. (2020). Multimodal fusion with deep neural networks for leveraging CT imaging and electronic health record: a case-study in pulmonary embolism detection. *Scientific reports*, 10(1), 22147-NA. <https://doi.org/10.1038/s41598-020-78888-w>
- [48]. Iversen, A., Fear, N. T., Ehlers, A., Hughes, J. G. H. H., Hull, L., Earnshaw, M., Greenberg, N., Rona, R. J., Wessely, S., & Hotopf, M. (2008). Risk factors for post-traumatic stress disorder among UK Armed Forces personnel. *Psychological medicine*, 38(4), 511-522. <https://doi.org/10.1017/s0033291708002778>

- [49]. Jacquet-Smailovic, M., Brennstuhl, M.-J., Denis, I., Kirche, A., Tarquinio, C., & Tarquinio, C. (2021). Relationship between Post-traumatic Stress Disorder and subsequent myocardial infarction: a systematic review and meta-analysis. *Journal of affective disorders*, 297(NA), 525-535. <https://doi.org/10.1016/j.jad.2021.10.056>
- [50]. Jerome, L., Feduccia, A. A., Wang, J. B., Hamilton, S., Yazar-Klosinski, B., Emerson, A., Mithoefer, M. C., & Doblin, R. (2020). Long-term follow-up outcomes of MDMA-assisted psychotherapy for treatment of PTSD: a longitudinal pooled analysis of six phase 2 trials. *Psychopharmacology*, 237(8), 2485-2497. <https://doi.org/10.1007/s00213-020-05548-2>
- [51]. Jin, C., Jia, H., Lanka, P., Rangaprakash, D., Li, L., Liu, T., Hu, X., & Deshpande, G. (2017). Dynamic brain connectivity is a better predictor of PTSD than static connectivity. *Human brain mapping*, 38(9), 4479-4496. <https://doi.org/10.1002/hbm.23676>
- [52]. Kaplan, G. B., Lakis, G. A., & Zhoba, H. (2022). Sleep-wake and arousal dysfunctions in post-traumatic stress disorder: Role of orexin systems. *Brain research bulletin*, 186(NA), 106-122. <https://doi.org/10.1016/j.brainresbull.2022.05.006>
- [53]. Karstoft, K.-I., Statnikov, A., Andersen, S. B., Madsen, T., & Galatzer-Levy, I. R. (2015). Early identification of posttraumatic stress following military deployment: Application of machine learning methods to a prospective study of Danish soldiers. *Journal of affective disorders*, 184(NA), 170-175. <https://doi.org/10.1016/j.jad.2015.05.057>
- [54]. Kelly, C., Karthikesalingam, A., Suleyman, M., Corrado, G. S., & King, D. (2019). Key challenges for delivering clinical impact with artificial intelligence. *BMC medicine*, 17(1), 1-9. <https://doi.org/10.1186/s12916-019-1426-2>
- [55]. Kessler, R. C., Rose, S., Koenen, K. C., Karam, E. G., Stang, P. E., Stein, D. J., Heeringa, S. G., Hill, E., Liberzon, I., McLaughlin, K. A., McLean, S. A., Pennell, B. E., Petukhova, M., Rosellini, A. J., Ruscio, A. M., Shahly, V., Shalev, A. Y., Silove, D., Zaslavsky, A. M., . . . Viana, M. C. (2014). How well can post-traumatic stress disorder be predicted from pre-trauma risk factors? An exploratory study in the WHO World Mental Health Surveys. *World psychiatry : official journal of the World Psychiatric Association (WPA)*, 13(3), 265-274. <https://doi.org/10.1002/wps.20150>
- [56]. Kip, K. E., Rosenzweig, L., Hernandez, D. F., Shuman, A., Sullivan, K. L., Long, C. J., Taylor, J., McGhee, S., Girling, S. A., Wittenberg, T. R., Sahebzamani, F. M., Lengacher, C. A., Kadel, R. P., & Diamond, D. M. (2013). Randomized Controlled Trial of Accelerated Resolution Therapy (ART) for Symptoms of Combat-Related Post-Traumatic Stress Disorder (PTSD). *Military medicine*, 178(12), 1298-1309. <https://doi.org/10.7205/milmed-d-13-00298>
- [57]. Koch, S. B. J., van Zuiden, M., Nawijn, L., Frijling, J. L., Veltman, D. J., & Olf, M. (2015). Intranasal Oxytocin Administration Dampens Amygdala Reactivity towards Emotional Faces in Male and Female PTSD Patients. *Neuropsychopharmacology : official publication of the American College of Neuropsychopharmacology*, 41(6), 1495-1504. <https://doi.org/10.1038/npp.2015.299>
- [58]. Krantz, D. S., Shank, L. M., & Goodie, J. L. (2021). Post-traumatic stress disorder (PTSD) as a systemic disorder: Pathways to cardiovascular disease. *Health psychology : official journal of the Division of Health Psychology, American Psychological Association*, 41(10), 651-662. <https://doi.org/10.1037/hea0001127>
- [59]. Kuring, J. K., Mathias, J. L., Ward, L., & Tachas, G. (2023). Inflammatory markers in persons with clinically-significant depression, anxiety or PTSD: A systematic review and meta-analysis. *Journal of psychiatric research*, 168(NA), 279-292. <https://doi.org/10.1016/j.jpsychires.2023.10.009>
- [60]. Lanius, R. A., Boyd, J. E., McKinnon, M. C., Nicholson, A. A., Frewen, P. A., Vermetten, E., Jetly, R., & Spiegel, D. (2018). A Review of the Neurobiological Basis of Trauma-Related Dissociation and Its Relation to Cannabinoid- and Opioid-Mediated Stress Response: a Transdiagnostic, Translational Approach. *Current psychiatry reports*, 20(12), 118-118. <https://doi.org/10.1007/s11920-018-0983-y>
- [61]. Lanius, R. A., Vermetten, E., Loewenstein, R. J., Brand, B., Schmahl, C., Bremner, J. D., & Spiegel, D. (2010). Emotion Modulation in PTSD: Clinical and Neurobiological Evidence for a Dissociative Subtype. *The American journal of psychiatry*, 167(6), 640-647. <https://doi.org/10.1176/appi.ajp.2009.09081168>
- [62]. Mahabir, M., Ashbaugh, A. R., Saumier, D., & Tremblay, J. (2015). Propranolol's impact on cognitive performance in post-traumatic stress disorder. *Journal of affective disorders*, 192(NA), 98-103. <https://doi.org/10.1016/j.jad.2015.11.051>
- [63]. Marseille, E., Kahn, J. G., Yazar-Klosinski, B., & Doblin, R. (2020). The cost-effectiveness of MDMA-assisted psychotherapy for the treatment of chronic, treatment-resistant PTSD. *PloS one*, 15(10), e0239997-NA. <https://doi.org/10.1371/journal.pone.0239997>
- [64]. Mavranouzouli, I., Megnin-Viggars, O., Daly, C., Dias, S., Welton, N. J., Stockton, S., Bhutani, G., Grey, N., Leach, J., Greenberg, N., Katona, C., El-Leithy, S., & Pilling, S. (2020). Psychological treatments for post-traumatic stress disorder in adults: a network meta-analysis. *Psychological medicine*, 50(4), 542-555. <https://doi.org/10.1017/s0033291720000070>
- [65]. McDonald, A. D., Sasangohar, F., Jatav, A., & Rao, A. H. (2019). Continuous monitoring and detection of post-traumatic stress disorder (PTSD) triggers among veterans: A supervised machine learning approach. *IIEE Transactions on Healthcare Systems Engineering*, 9(3), 201-211. <https://doi.org/10.1080/24725579.2019.1583703>
- [66]. McGeary, C. A., Morland, L. A., Resick, P. A., Straud, C. L., Moring, J. C., Sohn, M. J., Mackintosh, M.-A., Young-McCaughan, S., Acierno, R., Rauch, S. A. M., Mintz, J., McGeary, D. D., Wells, S. Y., Grubbs, K., Nabity, P. S., McMahon, C. J., Litz, B. T., Velligan, D. I., Macdonald, A., . . . Peterson, A. L. (2023). Impact and efficiency of treatment across two PTSD clinical trials comparing in-person and telehealth service delivery formats. *Psychological services*, 21(1), 73-81. <https://doi.org/10.1037/ser0000774>
- [67]. McLay, R. N., Wood, D. P., Webb-Murphy, J., Spira, J., Wiederhold, M. D., Pyne, J. M., & Wiederhold, B. K. (2011). A randomized, controlled trial of virtual reality-graded exposure therapy for post-traumatic stress disorder in active

- duty service members with combat-related post-traumatic stress disorder. *Cyberpsychology, behavior and social networking*, 14(4), 223-229. <https://doi.org/10.1089/cyber.2011.0003>
- [68]. Md Arif Uz, Z., & Elmoon, A. (2023). Adaptive Learning Systems For English Literature Classrooms: A Review Of AI-Integrated Education Platforms. *International Journal of Scientific Interdisciplinary Research*, 4(3), 56-86. <https://doi.org/10.63125/a30ehr12>
- [69]. Md Arman, H., & Md.Kamrul, K. (2022). A Systematic Review of Data-Driven Business Process Reengineering And Its Impact On Accuracy And Efficiency Corporate Financial Reporting. *International Journal of Business and Economics Insights*, 2(4), 01-41. <https://doi.org/10.63125/btx52a36>
- [70]. Md Mesbaul, H. (2024). Industrial Engineering Approaches to Quality Control In Hybrid Manufacturing A Review Of Implementation Strategies. *International Journal of Business and Economics Insights*, 4(2), 01-30. <https://doi.org/10.63125/3xcabx98>
- [71]. Md Mohaiminul, H., & Md Muzahidul, I. (2022). High-Performance Computing Architectures For Training Large-Scale Transformer Models In Cyber-Resilient Applications. *ASRC Procedia: Global Perspectives in Science and Scholarship*, 2(1), 193-226. <https://doi.org/10.63125/6zt59y89>
- [72]. Md Omar, F., & Md. Jobayer Ibne, S. (2022). Aligning FEDRAMP And NIST Frameworks In Cloud-Based Governance Models: Challenges And Best Practices. *Review of Applied Science and Technology*, 1(01), 01-37. <https://doi.org/10.63125/vnkcwq87>
- [73]. Md Sanjid, K. (2023). Quantum-Inspired AI Metaheuristic Framework For Multi-Objective Optimization In Industrial Production Scheduling. *American Journal of Interdisciplinary Studies*, 4(03), 01-33. <https://doi.org/10.63125/2mba8p24>
- [74]. Md Sanjid, K., & Md. Tahmid Farabe, S. (2021). Federated Learning Architectures For Predictive Quality Control In Distributed Manufacturing Systems. *American Journal of Interdisciplinary Studies*, 2(02), 01-31. <https://doi.org/10.63125/222nwg58>
- [75]. Md Sanjid, K., & Sudipto, R. (2023). Blockchain-Orchestrated Cyber-Physical Supply Chain Networks For Manufacturing Resilience. *American Journal of Scholarly Research and Innovation*, 2(01), 194-223. <https://doi.org/10.63125/6n81ne05>
- [76]. Md Sanjid, K., & Zayadul, H. (2022). Thermo-Economic Modeling Of Hydrogen Energy Integration In Smart Factories. *ASRC Procedia: Global Perspectives in Science and Scholarship*, 2(1), 257-288. <https://doi.org/10.63125/txdz1p03>
- [77]. Md. Hasan, I. (2022). The Role Of Cross-Country Trade Partnerships In Strengthening Global Market Competitiveness. *ASRC Procedia: Global Perspectives in Science and Scholarship*, 2(1), 121-150. <https://doi.org/10.63125/w0mnpz07>
- [78]. Md. Mominul, H., Masud, R., & Md. Milon, M. (2022). Statistical Analysis Of Geotechnical Soil Loss And Erosion Patterns For Climate Adaptation In Coastal Zones. *American Journal of Interdisciplinary Studies*, 3(03), 36-67. <https://doi.org/10.63125/xytn3e23>
- [79]. Md. Rabiul, K., & Sai Praveen, K. (2022). The Influence of Statistical Models For Fraud Detection In Procurement And International Trade Systems. *American Journal of Interdisciplinary Studies*, 3(04), 203-234. <https://doi.org/10.63125/9htnv106>
- [80]. Md. Tahmid Farabe, S. (2022). Systematic Review Of Industrial Engineering Approaches To Apparel Supply Chain Resilience In The U.S. Context. *American Journal of Interdisciplinary Studies*, 3(04), 235-267. <https://doi.org/10.63125/teherz38>
- [81]. Md. Tarek, H. (2023). Quantitative Risk Modeling For Data Loss And Ransomware Mitigation In Global Healthcare And Pharmaceutical Systems. *International Journal of Scientific Interdisciplinary Research*, 4(3), 87-116. <https://doi.org/10.63125/8wk2ch14>
- [82]. Md. Tarek, H., & Md.Kamrul, K. (2024). Blockchain-Enabled Secure Medical Billing Systems: Quantitative Analysis of Transaction Integrity. *ASRC Procedia: Global Perspectives in Science and Scholarship*, 4(1), 97-123. <https://doi.org/10.63125/1t8jpm24>
- [83]. Md. Wahid Zaman, R., & Momena, A. (2021). Systematic Review Of Data Science Applications In Project Coordination And Organizational Transformation. *ASRC Procedia: Global Perspectives in Science and Scholarship*, 1(2), 01-41. <https://doi.org/10.63125/31b8qc62>
- [84]. Méndez, M. Z., Nijdam, M. J., Heide, F. J. J. t., van der Aa, N., & Olff, M. (2018). A five-day inpatient EMDR treatment programme for PTSD: pilot study. *European journal of psychotraumatology*, 9(1), 1425575-1425575. <https://doi.org/10.1080/20008198.2018.1425575>
- [85]. Mentis, A.-F. A., Garcia, I., Jiménez, J., Paparoupa, M., Xirogianni, A., Papandreou, A., & Tzanakaki, G. (2021). Artificial Intelligence in Differential Diagnostics of Meningitis: A Nationwide Study. *Diagnostics (Basel, Switzerland)*, 11(4), 602-NA. <https://doi.org/10.3390/diagnostics11040602>
- [86]. Merians, A. N., Spiller, T., Harpaz-Rotem, I., Krystal, J. H., & Pietrzak, R. H. (2022). Post-traumatic Stress Disorder. *The Medical clinics of North America*, 107(1), 85-99. <https://doi.org/10.1016/j.mcna.2022.04.003>
- [87]. Meskó, B., & Görög, M. (2020). A short guide for medical professionals in the era of artificial intelligence. *NPJ digital medicine*, 3(1), 1-8. <https://doi.org/10.1038/s41746-020-00333-z>
- [88]. Meza-Concha, N., Arancibia, M., Salas, F., Behar, R., Salas, G., Silva, H., & Escobar, R. (2017). Towards a neurobiological understanding of alexithymia. *Medwave*, 17(4), 6960-e6960. <https://doi.org/10.5867/medwave.2017.04.6960>

- [89]. Morena, M., Patel, S., Bains, J. S., & Hill, M. N. (2015). Neurobiological Interactions Between Stress and the Endocannabinoid System. *Neuropsychopharmacology : official publication of the American College of Neuropsychopharmacology*, 41(1), 80-102. <https://doi.org/10.1038/npp.2015.166>
- [90]. Morina, N., Wicherts, J. M., Lobbrecht, J., & Priebe, S. (2014). Remission from post-traumatic stress disorder in adults: a systematic review and meta-analysis of long term outcome studies. *Clinical psychology review*, 34(3), 249-255. <https://doi.org/10.1016/j.cpr.2014.03.002>
- [91]. Mst. Shahrin, S., & Samia, A. (2023). High-Performance Computing For Scaling Large-Scale Language And Data Models In Enterprise Applications. *ASRC Procedia: Global Perspectives in Science and Scholarship*, 3(1), 94-131. <https://doi.org/10.63125/e7yfw87>
- [92]. Mulvaney, S. W., Lynch, J. H., Curtis, K. E., & Ibrahim, T. S. (2021). The Successful Use of Left-sided Stellate Ganglion Block in Patients That Fail to Respond to Right-sided Stellate Ganglion Block for the Treatment of Post-traumatic Stress Disorder Symptoms: A Retrospective Analysis of 205 Patients. *Military medicine*, 187(7-8), e826-e829. <https://doi.org/10.1093/milmed/usab056>
- [93]. Murkar, A., Kendzerska, T., Shlik, J., Quilty, L., Saad, M., & Robillard, R. (2022). Increased cannabis intake during the COVID-19 pandemic is associated with worsening of depression symptoms in people with PTSD. *BMC psychiatry*, 22(1), 554-NA. <https://doi.org/10.1186/s12888-022-04185-7>
- [94]. Nollett, C., Lewis, C., Kitchiner, N. J., Roberts, N. P., Addison, K., Brookes-Howell, L., Cosgrove, S., Cullen, K., Ehlers, A., Heke, S., Kelson, M., Lovell, K., Madden, K., McEwan, K., McNamara, R., Phillips, C., Pickles, T., Simon, N., & Bisson, J. I. (2018). Pragmatic RAndomised controlled trial of a trauma-focused guided self-help Programme versus Individual trauma-focused cognitive Behavioural therapy for post-traumatic stress disorder (RAPID): Trial protocol. *BMC psychiatry*, 18(1), 77-77. <https://doi.org/10.1186/s12888-018-1665-3>
- [95]. Oehen, P., Traber, R., Widmer, V., & Schnyder, U. (2012). A randomized, controlled pilot study of MDMA ($\pm$ 3,4-Methylenedioxymethamphetamine)-assisted psychotherapy for treatment of resistant, chronic Post-Traumatic Stress Disorder (PTSD). *Journal of psychopharmacology (Oxford, England)*, 27(1), 40-52. <https://doi.org/10.1177/0269881112464827>
- [96]. Omar Muhammad, F., & Md Redwanul, I. (2023). A Quantitative Study on AI-Driven Employee Performance Analytics In Multinational Organizations. *American Journal of Interdisciplinary Studies*, 4(04), 145-176. <https://doi.org/10.63125/vrsjp515>
- [97]. Omar Muhammad, F., & Md. Redwanul, I. (2023). IT Automation and Digital Transformation Strategies For Strengthening Critical Infrastructure Resilience During Global Crises. *American Journal of Interdisciplinary Studies*, 4(04), 145-176. <https://doi.org/10.63125/vrsjp515>
- [98]. Pankaz Roy, S. (2022). Data-Driven Quality Assurance Systems For Food Safety In Large-Scale Distribution Centers. *ASRC Procedia: Global Perspectives in Science and Scholarship*, 2(1), 151-192. <https://doi.org/10.63125/qen48m30>
- [99]. Petrosino, N. J., van 't Wout-Frank, M., Aiken, E., Swearingen, H. R., Barredo, J., Zandvakili, A., & Philip, N. S. (2019). One-year clinical outcomes following theta burst stimulation for post-traumatic stress disorder. *Neuropsychopharmacology : official publication of the American College of Neuropsychopharmacology*, 45(6), 940-946. <https://doi.org/10.1038/s41386-019-0584-4>
- [100]. Radow, B., Anderson, N., & Richmond, B. K. (2024). Post-Traumatic Stress Disorder (PTSD) in Trauma Patients. *The American surgeon*, 91(2), 292-299. <https://doi.org/10.1177/00031348241290610>
- [101]. Rahman, R. A., Omar, K., Noah, S. A. M., Danuri, M. S. N. M., & Al-Garadi, M. A. (2020). Application of Machine Learning Methods in Mental Health Detection: A Systematic Review. *IEEE Access*, 8(NA), 183952-183964. <https://doi.org/10.1109/access.2020.3029154>
- [102]. Rahman, S. M. T., & Abdul, H. (2022). Data Driven Business Intelligence Tools In Agribusiness A Framework For Evidence-Based Marketing Decisions. *International Journal of Business and Economics Insights*, 2(1), 35-72. <https://doi.org/10.63125/p59krm34>
- [103]. Razia, S. (2022). A Review Of Data-Driven Communication In Economic Recovery: Implications Of ICT-Enabled Strategies For Human Resource Engagement. *International Journal of Business and Economics Insights*, 2(1), 01-34. <https://doi.org/10.63125/7tkv8v34>
- [104]. Razia, S. (2023). AI-Powered BI Dashboards In Operations: A Comparative Analysis For Real-Time Decision Support. *ASRC Procedia: Global Perspectives in Science and Scholarship*, 3(1), 62-93. <https://doi.org/10.63125/wqd2t159>
- [105]. Reist, C., Streja, E., Tang, C. C., Shapiro, B. B., Mintz, J., & Hollifield, M. (2020). Prazosin for treatment of post-traumatic stress disorder: a systematic review and meta-analysis. *CNS spectrums*, 26(4), 338-344. <https://doi.org/10.1017/s1092852920001121>
- [106]. Remch, M., Laskaris, Z., Flory, J. D., Mora-McLaughlin, C., & Morabia, A. (2018). Post-Traumatic Stress Disorder and Cardiovascular Diseases: A Cohort Study of Men and Women Involved in Cleaning the Debris of the World Trade Center Complex. *Circulation. Cardiovascular quality and outcomes*, 11(7), e004572-NA. <https://doi.org/10.1161/circoutcomes.117.004572>
- [107]. Richards, A., Kanady, J. C., & Neylan, T. C. (2019). Sleep disturbance in PTSD and other anxiety-related disorders: an updated review of clinical features, physiological characteristics, and psychological and neurobiological mechanisms. *Neuropsychopharmacology*, 45(1), 55-73. <https://doi.org/10.1038/s41386-019-0486-5>
- [108]. Richards, B. A., Lillicrap, T. P., Beaudoin, P., Bengio, Y., Bogacz, R., Christensen, A. J., Clopath, C., Costa, R. P., de Berker, A. O., Ganguli, S., Gillon, C. J., Hafner, D., Kepecs, A., Kriegeskorte, N., Latham, P. E., Lindsay, G. W., Miller, K. D., Naud, R., Pack, C. C., . . . Kording, K. P. (2019). A deep learning framework for neuroscience. *Nature neuroscience*, 22(11), 1761-1770. <https://doi.org/10.1038/s41593-019-0520-2>

- [109]. Rony, M. A. (2021). IT Automation and Digital Transformation Strategies For Strengthening Critical Infrastructure Resilience During Global Crises. *International Journal of Business and Economics Insights*, 1(2), 01-32. <https://doi.org/10.63125/8tzzab90>
- [110]. Rousseau, P.-F., Khoury-Malhame, M. E., Reynaud, E., Zendjidjian, X., Samuelian, J.-C., & Khalfa, S. (2019). Neurobiological correlates of EMDR therapy effect in PTSD. *European Journal of Trauma & Dissociation*, 3(2), 103-111. <https://doi.org/10.1016/j.ejtd.2018.07.001>
- [111]. Sai Srinivas, M., & Manish, B. (2023). Trustworthy AI: Explainability & Fairness In Large-Scale Decision Systems. *Review of Applied Science and Technology*, 2(04), 54-93. <https://doi.org/10.63125/3w9v5e52>
- [112]. Schein, J., Houle, C. R., Urganus, A., Cloutier, M., Patterson-Lomba, O., Wang, Y., King, S., Levinson, W., Guerin, A., Lefebvre, P., & Davis, L. L. (2021). Prevalence of post-traumatic stress disorder in the United States: a systematic literature review. *Current medical research and opinion*, 37(12), 2151-2161. <https://doi.org/10.1080/03007995.2021.1978417>
- [113]. Schmidt, U., & Vermetten, E. (2017). Integrating NIMH Research Domain Criteria (RDoC) into PTSD Research. *Current topics in behavioral neurosciences*, 38(NA), 69-91. https://doi.org/10.1007/7854_2017_1
- [114]. Schultebracks, K., Sijbrandij, M., Galatzer-Levy, I. R., Mouthaan, J., Olff, M., & van Zuiden, M. (2021). Forecasting individual risk for long-term Posttraumatic Stress Disorder in emergency medical settings using biomedical data: A machine learning multicenter cohort study. *Neurobiology of stress*, 14(NA), 100297-100297. <https://doi.org/10.1016/j.ynstr.2021.100297>
- [115]. Schwalbe, N., & Wahl, B. (2020). Artificial intelligence and the future of global health. *Lancet (London, England)*, 395(10236), 1579-1586. [https://doi.org/10.1016/s0140-6736\(20\)30226-9](https://doi.org/10.1016/s0140-6736(20)30226-9)
- [116]. Seto, M. C., Rodrigues, N. C., Ham, E., Kirsh, B., & Hilton, N. Z. (2020). Post-traumatic Stress Disorder, Depression, Anxiety Symptoms and Help Seeking in Psychiatric Staff: Trouble de stress post-traumatique, dépression, symptômes d'anxiété et recherche d'aide chez le personnel psychiatrique. *Canadian journal of psychiatry. Revue canadienne de psychiatrie*, 65(8), 577-583. <https://doi.org/10.1177/0706743720916356>
- [117]. Shaw, S. B., Nicholson, A. A., Ros, T., Harricharan, S., Terpou, B., Densmore, M., Theberge, J., Frewen, P., & Lanius, R. A. (2023). Increased top-down control of emotions during symptom provocation working memory tasks following a RCT of alpha-down neurofeedback in PTSD. *NeuroImage. Clinical*, 37(NA), 103313-103313. <https://doi.org/10.1016/j.nicl.2023.103313>
- [118]. Sippel, L. M., Flanagan, J. C., Holtzheimer, P. E., Moran-Santa-Maria, M. M., Brady, K. T., & Joseph, J. E. (2021). Effects of intranasal oxytocin on threat- and reward-related functional connectivity in men and women with and without childhood abuse-related PTSD. *Psychiatry research. Neuroimaging*, 317(NA), 111368-111368. <https://doi.org/10.1016/j.psychres.2021.111368>
- [119]. Smid, G. E., Lind, J., & Bonde, J. P. (2022). Neurobiological mechanisms underlying delayed expression of posttraumatic stress disorder: A scoping review. *World journal of psychiatry*, 12(1), 151-168. <https://doi.org/10.5498/wjp.v12.i1.151>
- [120]. Snoek, A., Beekman, A. T. F., Dekker, J., Aarts, I., van Grootheest, G., Blankers, M., Vriend, C., Van Den Heuvel, O. A., & Thomaes, K. (2020). A randomized controlled trial comparing the clinical efficacy and cost-effectiveness of eye movement desensitization and reprocessing (EMDR) and integrated EMDR-Dialectical Behavioural Therapy (DBT) in the treatment of patients with post-traumatic stress disorder and comorbid (Sub)clinical borderline personality disorder: study design. *BMC psychiatry*, 20(1), 1-18. <https://doi.org/10.1186/s12888-020-02713-x>
- [121]. Sudipto, R. (2023). AI-Enhanced Multi-Objective Optimization Framework For Lean Manufacturing Efficiency And Energy-Conscious Production Systems. *American Journal of Interdisciplinary Studies*, 4(03), 34-64. <https://doi.org/10.63125/s43p0363>
- [122]. Sudipto, R., & Md Mesbaul, H. (2021). Machine Learning-Based Process Mining For Anomaly Detection And Quality Assurance In High-Throughput Manufacturing Environments. *Review of Applied Science and Technology*, 6(1), 01-33. <https://doi.org/10.63125/t5dcb097>
- [123]. Sudipto, R., & Md. Hasan, I. (2024). Data-Driven Supply Chain Resilience Modeling Through Stochastic Simulation And Sustainable Resource Allocation Analytics. *American Journal of Advanced Technology and Engineering Solutions*, 4(02), 01-32. <https://doi.org/10.63125/p0ptag78>
- [124]. Sun, Y., Qu, Y., & Zhu, J. (2021). The Relationship Between Inflammation and Post-traumatic Stress Disorder. *Frontiers in psychiatry*, 12(NA), 707543-NA. <https://doi.org/10.3389/fpsy.2021.707543>
- [125]. Syed Zaki, U. (2021). Modeling Geotechnical Soil Loss and Erosion Dynamics For Climate-Resilient Coastal Adaptation. *American Journal of Interdisciplinary Studies*, 2(04), 01-38. <https://doi.org/10.63125/vsfjtt77>
- [126]. Syed Zaki, U. (2022). Systematic Review Of Sustainable Civil Engineering Practices And Their Influence On Infrastructure Competitiveness. *ASRC Procedia: Global Perspectives in Science and Scholarship*, 2(1), 227–256. <https://doi.org/10.63125/hh8nv249>
- [127]. Takemoto, E., Van Oss, K. R., Chamany, S., Brite, J., & Brackbill, R. M. (2020). Post-traumatic stress disorder and the association with overweight, obesity, and weight change among individuals exposed to the World Trade Center disaster, 2003-2016. *Psychological medicine*, 51(15), 2647-2656. <https://doi.org/10.1017/s0033291720001208>
- [128]. Tang, E., Jones, C., Smith-MacDonald, L., Brown, M. R. G., Vermetten, E., & Brémault-Phillips, S. (2021). Decreased Emotional Dysregulation Following Multi-Modal Motion-Assisted Memory Desensitization and Reconsolidation Therapy (3MDR): Identifying Possible Driving Factors in Remediation of Treatment-Resistant PTSD. *International journal of environmental research and public health*, 18(22), 12243-NA. <https://doi.org/10.3390/ijerph182212243>

- [129]. Thakur, A., Choudhary, D., Kumar, B., & Chaudhary, A. (2022). A Review on Post-traumatic Stress Disorder (PTSD): Symptoms, Therapies and Recent Case Studies. *Current molecular pharmacology*, 15(3), 502-516. <https://doi.org/10.2174/1874467214666210525160944>
- [130]. Tonoy Kanti, C., & Shaikat, B. (2022). Graph Neural Networks (GNNS) For Modeling Cyber Attack Patterns And Predicting System Vulnerabilities In Critical Infrastructure. *American Journal of Interdisciplinary Studies*, 3(04), 157-202. <https://doi.org/10.63125/1ykzx350>
- [131]. Van Deursen, R. W. M., Jones, K., Kitchiner, N. J., Hannigan, B., Barawi, K., & Bisson, J. I. (2021). The psychophysiological response during post-traumatic stress disorder treatment with modular motion-assisted memory desensitisation and reconsolidation (3MDR). *European journal of psychotraumatology*, 12(1), 1929027-NA. <https://doi.org/10.1080/20008198.2021.1929027>
- [132]. van Vliet, N. I., Huntjens, R. J. C., van Dijk, M. K., Bachrach, N., Meewisse, M.-L., & de Jongh, A. (2021). Phase-based treatment versus immediate trauma-focused treatment for post-traumatic stress disorder due to childhood abuse: randomised clinical trial. *BJPsych open*, 7(6), NA-NA. <https://doi.org/10.1192/bjo.2021.1057>
- [133]. Vermetten, E., & Jetly, R. (2018). A Critical Outlook on Combat-Related PTSD: Review and Case Reports of Guilt and Shame as Drivers for Moral Injury. *Military Behavioral Health*, 6(2), 156-164. <https://doi.org/10.1080/21635781.2018.1459973>
- [134]. Vollmer, S. J., Mateen, B. A., Bohner, G., Király, F. J., Ghani, R., Jonsson, P., Cumbers, S., Jonas, A., Ksl, M., Myles, P. R., Granger, D., Birse, M., Branson, R., Kgm, M., Collins, G. S., Jpa, I., Holmes, C., & Hemingway, H. (2020). Machine learning and artificial intelligence research for patient benefit: 20 critical questions on transparency, replicability, ethics, and effectiveness. *BMJ (Clinical research ed.)*, 368(NA), l6927-NA. <https://doi.org/10.1136/bmj.l6927>
- [135]. Wade, D., Hardy, R., Howell, D. C. J., & Mythen, M. G. (2013). Identifying clinical and acute psychological risk factors for PTSD after critical care: a systematic review. *Minerva anesthesiologica*, 79(8), 944-963. <https://doi.org/NA>
- [136]. White, W. F., Burgess, A., Dalgleish, T., Halligan, S., Hiller, R., Oxley, A., Smith, P., & Meiser-Stedman, R. (2022). Prevalence of the dissociative subtype of post-traumatic stress disorder: a systematic review and meta-analysis. *Psychological medicine*, 52(9), 1629-1644. <https://doi.org/10.1017/s0033291722001647>
- [137]. Wright, L. A., Sijbrandij, M., Sinnerton, R., Lewis, C., Roberts, N. P., & Bisson, J. I. (2019). Pharmacological prevention and early treatment of post-traumatic stress disorder and acute stress disorder: a systematic review and meta-analysis. *Translational psychiatry*, 9(1), 334-334. <https://doi.org/10.1038/s41398-019-0673-5>
- [138]. Yang, R., Xu, C., Bierer, L. M., Flory, J. D., Gautam, A., Bader, H. N., Lehrner, A., Makotkine, I., Desarnaud, F., Miller, S., Jett, M., Hammamieh, R., & Yehuda, R. (2021). Longitudinal genome-wide methylation study of PTSD treatment using prolonged exposure and hydrocortisone. *Translational psychiatry*, 11(1), 398-NA. <https://doi.org/10.1038/s41398-021-01513-5>
- [139]. Yehuda, R., Hoge, C. W., McFarlane, A. C., Vermetten, E., Lanius, R. A., Nievergelt, C. M., Hobfoll, S. E., Koenen, K. C., Neylan, T. C., & Hyman, S. E. (2015). Post-traumatic stress disorder. *Nature reviews. Disease primers*, 1(1), 15058-NA. <https://doi.org/10.1038/nrdp.2015.58>
- [140]. Young, K. S., Purves, K. L., Hübel, C., Davies, M. R., Thompson, K. N., Bristow, S., Krebs, G., Danese, A., Hirsch, C., Parsons, C. E., Vassos, E., Adey, B. N., Bright, S., Hegemann, L., Lee, Y. T., Kalsi, G., Monssen, D., Mundy, J., Peel, A. J., . . . Breen, G. (2022). Depression, anxiety and PTSD symptoms before and during the COVID-19 pandemic in the UK. *Psychological medicine*, 53(12), 5428-5441. <https://doi.org/10.1017/s0033291722002501>
- [141]. Zayadul, H. (2023). Development Of An AI-Integrated Predictive Modeling Framework For Performance Optimization Of Perovskite And Tandem Solar Photovoltaic Systems. *International Journal of Business and Economics Insights*, 3(4), 01-25. <https://doi.org/10.63125/8xm7wa53>
- [142]. Zohar, J., Yahalom, H., Kozlovsky, N., Cwikel-Hamzany, S., Matar, M. A., Kaplan, Z., Yehuda, R., & Cohen, H. (2011). High dose hydrocortisone immediately after trauma may alter the trajectory of PTSD: interplay between clinical and animal studies. *European neuropsychopharmacology : the journal of the European College of Neuropsychopharmacology*, 21(11), 796-809. <https://doi.org/10.1016/j.euroneuro.2011.06.001>
- [143]. Zoladz, P. R., & Diamond, D. M. (2013). Current status on behavioral and biological markers of PTSD: a search for clarity in a conflicting literature. *Neuroscience and biobehavioral reviews*, 37(5), 860-895. <https://doi.org/10.1016/j.neubiorev.2013.03.024>
- [144]. Zoladz, P. R., Fleshner, M., & Diamond, D. M. (2013). Differential effectiveness of tianeptine, clonidine and amitriptyline in blocking traumatic memory expression, anxiety and hypertension in an animal model of PTSD. *Progress in neuro-psychopharmacology & biological psychiatry*, 44(NA), 1-16. <https://doi.org/10.1016/j.pnpbp.2013.01.001>