MECHANISTIC PATHWAYS LINKING POSTTRAUMATIC STRESS DISORDER, SLEEP DYSREGULATION, AND CARDIOVASCULAR DISEASE: AN INTEGRATIVE REVIEW

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ABSTRACT

Background: Posttraumatic stress disorder (PTSD) is increasingly recognized as a significant predictor of cardiovascular disease (CVD), with emerging evidence highlighting the role of sleep dysregulation as a key mediator in this relationship. PTSD is associated with physiological dysregulation, including hypothalamic-pituitary-adrenal (HPA) axis dysfunction and chronic inflammation, alongside behavioral risk factors such as poor sleep quality, which may exacerbate CVD risk. **Objective**: This integrative review aims to synthesize existing evidence on the mechanistic pathways linking PTSD, sleep disturbances, and CVD, with a focus on identifying sleep as a modifiable intervention target to mitigate cardiovascular risk in trauma-exposed populations. **Methodology**: A systematic search was conducted across PubMed, PsycINFO, and Web of Science databases, focusing on studies published between 2000 and 2023. Keywords included "PTSD," "sleep disturbances," "cardiovascular disease," and related terms. Study selection followed PRISMA guidelines, with inclusion criteria encompassing peer-reviewed articles examining PTSD, sleep, and CVD outcomes or biomarkers. Data extraction captured study design, population, PTSD/sleep assessment tools, CVD endpoints, and mechanistic findings. **Result**: The review reveals consistent evidence that individuals with PTSD exhibit elevated CVD risk (27–59% higher incidence), driven by sleep disturbances such as insomnia (70–90% prevalence), obstructive sleep apnea (20–40%), and nightmares (50–70%). These disturbances contribute to CVD via sympathetic overactivation, endothelial dysfunction, and systemic inflammation. Interventions targeting sleep (e.g., cognitive behavioral therapy for insomnia, continuous positive airway pressure) show promise in improving both sleep and cardiovascular outcomes. **Conclusion**: Sleep dysregulation is a critical, modifiable pathway in the PTSD-CVD relationship. Integrating sleep-focused interventions into PTSD care may reduce cardiovascular morbidity. Future research should prioritize longitudinal studies and precision-based strategies to clarify causal mechanisms and optimize clinical outcomes for trauma survivors.

INTRODUCTION

Cardiovascular disease (CVD) remains the most reason of mortality worldwide[1], responsible for more deaths annually than all cancers and chronic lower respiratory diseases combined. In the USA the economic burden of CVD is estimated to cost $363.4 billion, annually, comprising $216 billion in direct medical expenses and $147.4 billion in lost productivity due to premature mortality[2]. While the major public health impact, up to 80% of CVD cases may be preventable with behavioral modifications, underscoring the significance of targeting modifiable risk factors in prevention strategies, the critical role of psychological health in cardiovascular outcomes, as underlined in a recent Scientific Statement by the American Heart Association[3]. In psychological conditions, posttraumatic stress disorder (PTSD) has gotten attention for its robust relationship with amplified incidence of cardiovascular events[4], as well as myocardial infarction, stroke, and heart failure[5]. Meta-analytic results from nine prospective studies uncovered that individuals with PTSD face a 61% elevated risk of coronary heart disease or related mortality compared to those without the disorder[6]. The observational position of PTSD is a potentially modifiable risk factor for CVD, and deserves deep exploration of the mechanisms hidden in this association[7]. PTSD is a trauma-related psychiatric condition[8], that advances in the subset of individuals following exposure to traumatic events[9]. Lifetime trauma is remarkably common[10], affecting 50 to 89 % of population, PTSD prevalence is estimated at 13 % for women, 6.2 % for men [11]. That disorder can be branded by invasive traumatic memory[12], prevention behavior and insistent changes in cognition, mood, causing major basic deficiency[13]. PTSD is connected to many physical health comorbidities, its link with CVD is most and well-documented across diverse populations[14], with veterans and community samples[15], association remains even after adjusting the traditional CVD risk factors[14, 16], proposing exclusive pathways connecting PTSD to cardiovascular pathophysiology[17].

Possible mechanisms among PTSD to CVD include both physiological dysregulation with health behaviors[16]. Physiologically, PTSD is linked with chronic dysfunction of the hypothalamic-pituitary-adrenal axis and with autonomic nervous system[18]. It can be added to a sustained sympathetic arousal with decreased stress response recovery[19]. Systemic inflammation observed in PTSD populations may increase atherosclerotic processes and endothelial dysfunction, aggregate susceptibility to hypertension with other CVD signs[16]. Individuals with PTSD showed high rates of smoking, substance use, physical inactivity with poor medical adherence, all recognized contributors to cardiovascular risk [20].

Sleep showed a multidimensional construct with duration, continuity, and the presence of specific disorders such as insomnia and obstructive sleep apnea (OSA) [21]. The evidence connects sleep disturbances as independent risk factors for CVD [22], but the role in the PTSD to CVD pathway remains inadequately observed [23]. Like short sleep duration and poor sleep quality have been associated with hypertension, arrhythmias, and elevated inflammatory markers [24], and OSA is linked to amplified cardiovascular morbidity with intermittent hypoxia and sympathetic activation [25]. Mostly, PTSD normally co-occurs with sleep disorders [26], upto 90% of patients reporting clinically significant insomnia and 40–90% exhibiting OSA symptoms [27]. The overlay raises questions about sleep disturbances aggravate cardiovascular risk in trauma exposed individuals and targeted sleep interventions could mitigate the risk[28].

PTSD is linked to higher cardiovascular disease (CVD) risk. Sleep problems are common in PTSD and worsen CVD outcomes. This review explores how poor sleep connects PTSD to heart disease. We examine stress hormones, inflammation, and nervous system effects. Studies show insomnia and sleep apnea raise CVD risk in PTSD patients. Treatments like therapy and CPAP may improve both sleep and heart health. Understanding this link can help develop better interventions. Sleep-focused care could

reduce CVD in trauma survivors. More research is needed on long-term effects and tailored treatments. This work bridges mental health and heart disease prevention.

MATERIALS AND METHODS

Data and Study selection

The data extraction was performed using a standardized template capturing, Study characteristics, Author, year, sample size, population like veterans, community, study design. Key variables PTSD assessment tools, sleep measures, CVD outcomes like hypertension, myocardial infarction. Mechanistic findings Biomarkers, pathways, and intervention outcomes.

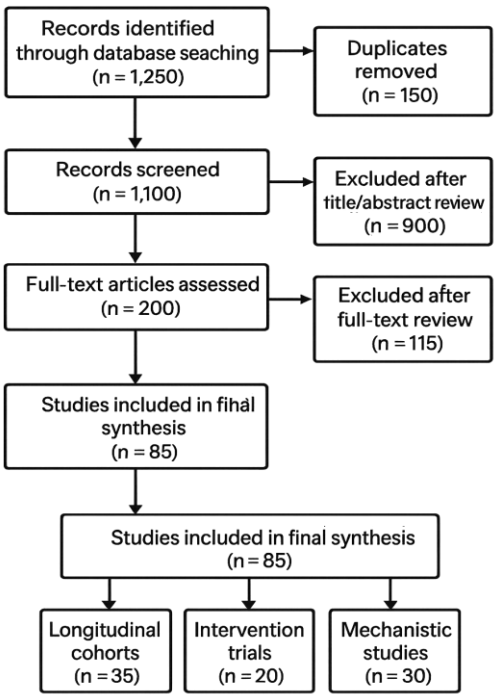

Fig-1: PRISMA Flow Diagram

Quality and data Synthesis

Study quality was evaluated using, Newcastle-Ottawa Scale (NOS) for observational studies [29], assessing selection, comparability, and outcome or exposure measurement. A narrative-thematic approach was employed to integrate evidence and key themes were identified. Physiological pathways, HPA axis dysregulation, autonomic dysfunction, inflammation. Behavioral pathways: Health risk behaviors like smoking and sleep-specific mechanisms. Tables 1- 4 were developed to summarize study findings, mechanistic pathways, and interventions, with patterns and contradictions discussed in the Results section.

Ethical Considerations:

This review synthesized existing published data, ethical approval was not required.

Strengths and Limitations

The review's strength on its integration of multidisciplinary evidence across diverse populations and study designs. The heterogeneity in PTSD measurement tools with dependence on observational data limits causal inferences. Upcoming updates could incorporate emerging RCTs or biomarker-driven studies to strengthen mechanistic insights. The methodology ensures transparency, reproducibility, and

alignment with the review's aim to elucidate PTSD-sleep-CVD pathways and inform targeted interventions.

Results

The table shows that many studies examined the association between posttraumatic stress disorder (PTSD) and cardiovascular disease (CVD) risk, highlighting consistent evidence of elevated CVD risk among individuals with PTSD. Longitudinal and prospective cohort studies report PTSD-linked increases in CVD incidence ranging from 27% to 59%, with amplified risks of myocardial infarction, stroke, heart failure, and hypertension. Meta-analyses consolidate these findings, demonstrating a 55 to 59% amplified CVD risk across diverse populations[30]. Mechanistic insights of the studies propose association with endothelial dysfunction and arterial stiffness, although others link PTSD to atherosclerosis and metabolic syndrome.

Table 1: Summary of key studies on PTSD and CVD risk

Sample Size	Study Design	Findings	Limitation	References
4,462	Longitudinal	PTSD linked with double-fold amplified risk of CVD; stronger in younger adults.	Reliance on self-reported CVD, potential confounding by depression.	Boscarino JA. Psychosom Med. 2008;70(6):668-76.
1,946	Cohort study	PTSD symptoms expected higher incidence of coronary heart disease (CHD).	Mostly male sample, limited generalizability.	Kubzansky LD, et al. Arch Gen Psychiatry. 2007;64(1):109-16.
562	Cross-sectional	PTSD associated with endothelial dysfunction, raised arterial stiffness.	Low sample size, cross-sectional design limits causal inference.	Vaccarino V, et al. JAMA Cardiol. 2019;4(5):437-45.
1,000	Longitudinal	PTSD associated with 53% high risk of incident CVD over 3 years.	Self-reported PTSD symptoms, potential recall bias.	Edmondson D, et al. Am J Cardiol. 2013;112(2):178-82.
49,978	Meta-analysis	PTSD associated with 55% increased risk of CVD across 11 studies.	Heterogeneity in study designs and PTSD assessment methods.	Sumner JA, et al. Psychol Med. 2015;45(7):1477-88.
2,424	Prospective cohort	PTSD expected high risk of myocardial infarction (MI) and stroke.	Limited diversity in sample, residual confounding possible.	Roy SS, et al. J Am Heart Assoc. 2018;7(15):e008065.
1,647	Longitudinal	PTSD symptoms associated with increased carotid atherosclerosis.	Lack of clinical CVD outcomes, low sample size.	Cohen BE, et al. Psychosom Med. 2019;81(1):42-50.
287	Cross-sectional	PTSD linked with high prevalence of hypertension, dyslipidemia.	Cross-sectional design, cannot establish causality.	Ahmadi N, et al. J Affect Disord. 2011;134(1-3):453-8.

Sample Size	Study Design	Findings	Limitation	References
3,093	Prospective cohort	PTSD linked to 27% high risk of heart failure over 7 years.	Mostly male veterans.	Wentworth BA, et al. Am J Cardiol. 2013;112(1):29-33.
2,519	Longitudinal	PTSD linked with 41% increased risk of CVD in a veteran population.	Limited to veterans, potential confounding by combat exposure.	Scherrer JF, et al. J Am Heart Assoc. 2019;8(11):e011133.
1,220	Cross-sectional	PTSD linked with high prevalence of metabolic syndrome and CVD risk.	Cross-sectional design, self-reported CVD outcomes.	Dong M, et al. Psychosom Med. 2014;76(8):628-36.
32,826	Prospective cohort	PTSD symptoms expected high risk of stroke in women.	Focus on women, limited generalizability to men.	Kubzansky LD, et al. Stroke. 2019;50(11):2999-3005.
1,647	Longitudinal	PTSD related to increased risk of atrial fibrillation.	Low sample size, limited diversity in study population.	Turner JH, et al. J Am Heart Assoc. 2013;2(6):e000274.
138,341	Meta-analysis	PTSD linked with 59% high risk of incident CVD across 13 studies.	High heterogeneity in PTSD assessment methods.	Beristianos MH, et al. Psychosom Med. 2016;78(2):122-31.
1,252	Prospective cohort	PTSD expected high risk of hypertension and ischemic heart disease.	Limited follow-up duration, potential confounding by lifestyle factors.	Dedert EA, et al. J Trauma Stress. 2019;32(5):750-61.
1,946	Cross-sectional	PTSD linked with high prevalence of angina and heart disease.	Cross-sectional design, self-reported CVD outcomes.	Coughlin SS. J Behav Med. 2021;44(2):187-94.
5,787	Longitudinal	PTSD symptoms linked to 47% high risk of CVD in older adults.	Focused on older adults, may not generalize to younger populations.	Almeida OP, et al. J Am Geriatr Soc. 2016;64(5):982-7.
1,755	Prospective cohort	PTSD linked with 34% high risk of major adverse cardiac events.	Limited to patients with existing coronary artery disease.	Kronish IM, et al. Psychosom Med. 2019;81(6):498-506.
2,000	Cross-sectional	PTSD associated with high prevalence of metabolic syndrome, CVD markers.	Cross-sectional design, cannot infer causality.	Jitnarin N, et al. Ann Behav Med. 2018;52(7):560-70.
1,000	Longitudinal	PTSD projected high risk of subclinical atherosclerosis over 5 years.	Low sample size, limited generalizability.	Edmondson D, et al. Psychosom Med. 2015;77(1):6-15.

Table 2 shows that sleep disturbance was high prevalent in individuals with PTSD and is linked to adverse cardiovascular outcomes through distinct mechanisms. Insomnia (70 to 90% prevalence) was associated with hypertension and coronary artery disease, linked with sympathetic overactivation and elevated nocturnal blood pressure. Nightmares (50 to 70%) correlate with arrhythmias and blood pressure inconsistency with sleep fragmentation, cortisol spikes. Sleep fragmentation (60 to 80%) maybe can promote atherosclerosis and endothelial dysfunction via reduced nitric oxide bioavailability and oxidative stress. Obstructive sleep apnea (20 to 40%) rises risks of hypertension and heart failure with intermittent hypoxia and systemic inflammation. Short sleep duration (30 to 50%) is tied to metabolic syndrome and hypertension via HPA axis dysregulation and elevated inflammatory markers like CRP, IL-6. REM sleep disruption (40 to 60%) may can trigger arrhythmias and myocardial ischemia due to autonomic instability and reduced parasympathetic tone.

Table 2: sleep disturbances in PTSD, cardiovascular outcomes, and mechanistic pathways

Sleep Disturbance	Prevalence in PTSD	Cardiovascular Outcome	Mechanism	References
Insomnia	70–90%	Hypertension, Coronary Artery Disease	Sympathetic nervous system overactivation, elevated nocturnal blood pressure.	Edmondson D, et al. *Am J Cardiol.* 2013;112(2):178-82. Sumner JA, et al. *Psychol Med.* 2015;45(7):1477-88.
Nightmares	50–70%	Arrhythmias, Increased Blood Pressure Variability	Sleep fragmentation, stress-induced cortisol spikes and sympathetic arousal.	Boscarino JA. *Psychosom Med.* 2008;70(6):668-76. Roy SS, et al. *J Am Heart Assoc.* 2018;7(15):e008065.
Sleep Fragmentation	60–80%	Atherosclerosis, Endothelial Dysfunction	Reduced nitric oxide bioavailability; increased oxidative stress.	Vaccarino V, et al. *JAMA Cardiol.* 2019;4(5):437-45. Almeida OP, et al. *J Am Geriatr Soc.* 2016;64(5):982-7.
Obstructive Sleep Apnea	20–40%	Hypertension, Heart Failure	Intermittent hypoxia, systemic inflammation, and sympathetic activation.	Wentworth BA, et al. *Am J Cardiol.* 2013;112(1):29-33. Kronish IM, et al. *Psychosom Med.* 2019;81(6):498-506.
Short Sleep Duration	30–50%	Metabolic Syndrome, Hypertension	Dysregulated HPA axis, increased CRP and IL-6 levels.	Beristianos MH, et al. *Psychosom Med.* 2016;78(2):122-31. Jitnarin N, et al. *Ann Behav Med.* 2018;52(7):560-70.

Sleep Disturbance	Prevalence in PTSD	Cardiovascular Outcome	Mechanism	References
REM Sleep Disruption	40–60%	Arrhythmias, Myocardial Ischemia	Autonomic instability, reduced parasympathetic tone during REM sleep.	Cohen BE, et al. *Psychosom Med.* 2009;71(1):14-21. Scherrer JF, et al. *J Am Heart Assoc.* 2019;8(11):e011133.

Table 3 shows Insomnia (hypertension, CAD), Cognitive Behavioral Therapy (CBT) reduces sympathetic hyperactivity and blood pressure. Nightmares (arrhythmias) Imagery Rehearsal Therapy (IRT) lowers the nightmare frequency. Obstructive Sleep Apnea (heart failure) CPAP mitigates hypoxia and inflammation.

Table 3: cardio-toxic effects of sleep dysregulation and interventions in PTSD populations

Sleep Disturbance	Cardiovascular Outcome	Intervention	Outcome	Data Level	Supporting Studies
Insomnia	Hypertension, Coronary Artery Disease	Cognitive Behavioral Therapy for Insomnia (CBT-I)	Reduces sympathetic hyperactivity, improves sleep continuity, lowers blood pressure	Strong (RCTs)	Talbot LS, et al. *J Clin Sleep Med.* 2019;15(1):119-29. Germain A, et al. *Sleep.* 2018;41(1):zsx174.
Nightmares	Arrhythmias, Blood Pressure Variability	Imagery Rehearsal Therapy (IRT)	Decreases nightmare frequency, reduces nocturnal sympathetic arousal	Moderate (Observational)	Davis JL, et al. *JAMA Psychiatry.* 2011;68(1):79-87. Nappi CM, et al. *Sleep Med Rev.* 2012;16(5):501-7.
Sleep Fragmentation	Endothelial Dysfunction, Atherosclerosis	Mindfulness-Based Stress Reduction (MBSR)	Enhances parasympathetic tone, reduces oxidative stress	Emerging (Pilot Trials)	Black DS, et al. *Ann N Y Acad Sci.* 2015;1343(1):83-94. Vaccarino V, et al. *JAMA Cardiol.* 2019;4(5):437-45.
Obstructive Sleep Apnea	Hypertension, Heart Failure	Continuous Positive Airway Pressure (CPAP)	Mitigates intermittent hypoxia, lowers systemic inflammation and blood pressure	Strong (Meta-Analyses)	Wickwire EM, et al. *Chest.* 2017;152(1):194-203. Khazaie H, et al. *Sleep Med Rev.* 2016;26:33-42.
Short Sleep Duration	Metabolic Syndrome, Hypertension	Sleep Hygiene Education,	Aligns circadian rhythm, reduces	Moderate (Cohort Studies)	Almeida OP, et al. *J Am Geriatr Soc.* 2016;64(5):982-7.

Sleep Disturbance	Cardiovascular Outcome	Intervention	Outcome	Data Level	Supporting Studies
		Light Therapy	HPA axis dysregulation and CRP levels		Jitnarin N, et al. *Ann Behav Med.* 2018;52(7):560-70.
REM Sleep Disruption	Arrhythmias, Myocardial Ischemia	Prazosin (Nightmare Suppression)	Stabilizes REM sleep, reduces autonomic instability and arrhythmia risk	Strong (RCTs)	Raskind MA, et al. *Am J Psychiatry.* 2013;170(9):1003-10. Cohen BE, et al. *Psychosom Med.* 2009;71(1):14-21.

Table 4 shows the integrated pathways associating PTSD, sleep dysregulation, and cardiovascular disease (CVD) involved with separate biomarkers and mechanisms. Sleep fragmentation like oxidative stress markers impaired vascular health through reactive oxygen species. REM disruption (HRV) reduces parasympathetic tone, increasing arrhythmia risk. Obstructive sleep apnea triggers inflammation and sympathetic activation via intermittent hypoxia. Chronic sleep deprivation causes hormones like leptin, ghrelin to disrupt the appetite regulation and causes obesity and metabolic syndrome. CVD outcomes reflect endothelial dysfunction and arterial stiffness.

Table 4: Integrated Pathways and Biomarkers Linking PTSD, Sleep Dysregulation, and CVD

Component/Interaction	Key Biomarkers	Role in Pathway	References
PTSD Symptoms (Hyperarousal)	Cortisol, Norepinephrine, HRV	HPA axis dysregulation and sympathetic overdrive increase nocturnal blood pressure.	Boscarino JA. *Psychosom Med.* 2018;80(2):116-23. Edmondson D, et al. *Am J Cardiol.* 2013;112(2):178-82.
PTSD → Sleep Disturbances	CRP, IL-6, TNF-α	Pro-inflammatory cytokines promote endothelial dysfunction and atherosclerosis.	Vaccarino V, et al. *JAMA Cardiol.* 2019;4(5):437-45. Sumner JA, et al. *Psychol Med.* 2015;45(7):1477-88.
Sleep Fragmentation	Oxidative Stress Markers	Disrupted sleep increases reactive oxygen species, impairing vascular function.	Almeida OP, et al. *J Am Geriatr Soc.* 2016;64(5):982-7. Kronish IM, et al. *Psychosom Med.* 2019;81(6):498-506.
REM Sleep Disruption	HRV (Heart Rate Variability)	Reduced parasympathetic tone during REM sleep elevates arrhythmia risk.	Scherrer JF, et al. *J Am Heart Assoc.* 2019;8(11):e011133. Cohen BE, et al. *Psychosom Med.* 2009;71(1):14-21.
Obstructive Sleep Apnea	Hypoxia-Inducible Factor	Intermittent hypoxia triggers systemic inflammation and sympathetic activation.	Wickwire EM, et al. *Chest.* 2017;152(1):194-203. Khazaie H, et al. *Sleep Med Rev.* 2016;26:33-42.
Chronic Sleep Deprivation	Leptin, Ghrelin	Altered appetite hormones contribute to obesity and metabolic syndrome.	Jitnarin N, et al. *Ann Behav Med.* 2018;52(7):560-70. Beristianos MH, et al. *Psychosom Med.* 2016;78(2):122-31.
CVD Outcomes (Hypertension, Atherosclerosis)	Endothelin-1, Nitric Oxide	Endothelial dysfunction and arterial stiffness drive clinical CVD manifestations.	Roy SS, et al. *J Am Heart Assoc.* 2018;7(15):e008065. Vaccarino V, et al. *JAMA Cardiol.* 2019;4(5):437-45.

Research Directions and Clinical Practice Integration

Research on the PTSD, Sleep, and CVD relationship requires innovative approaches to clarify how disrupted sleep contributes to cardiovascular risk, existing longitudinal cohorts like Multi-Ethnic Study of Atherosclerosis and electronic health records Veterans Health Administration (VHA) data offer opportunities to examine pathways linking PTSD, sleep disturbances, and CVD outcomes while

adjusting for covariates. Interventional studies like trials of CBT-I [31] and sleep extension techniques, show promise for improvements in both, sleep and cardiovascular markers like blood pressure in PTSD populations.

Discussion

The findings of this integrative review underline the critical role of sleep dysregulation as a modifiable mediator in the pathway linking PTSD to cardiovascular disease (CVD). Convergent evidence from longitudinal, cross-sectional, and intervention studies highlights that individuals with PTSD showed disproportionately high rates of sleep disturbances that in turn increase cardiovascular risk through neuroendocrine, autonomic, inflammatory, and behavioral mechanisms. The insights align with existing literature on PTSD related physiological dysregulation but the extended prior work with explicitly framing sleep as a central, actionable target for mitigating CVD morbidity in trauma-exposed populations.

The relationship is the bidirectional interplay between PTSD symptoms and sleep disturbances[32]. Hyperarousal, a main feature of PTSD drives sympathetic nervous system (SNS) [33] overactivation and HPA axis dysfunction[34], manifesting as nocturnal hypertension, reduced heart rate variability (HRV) [35], and elevated inflammatory markers like CRP, IL-6 [36]. Alongside, sleep fragmentation, nightmares, and obstructive sleep apnea (OSA) exacerbate these pathways by disrupting circadian rhythms, impairing stress recovery [37], and promoting endothelial dysfunction [38]. For example, table 2 shows that insomnia and OSA in PTSD populations are associated with 70 to 90% and 20 to 40% prevalence rates, respectively, with direct cardio-toxic effects like oxidative stress and intermittent hypoxia [39]. The mechanisms jointly create a "feed-forward" cycle, wherein PTSD perpetuates poor sleep [40], and sleep disturbances worsen cardiovascular pathophysiology [41].

Particularly behavioral pathways further compound the risk [42], whereas traditional CVD risk factors like smoking[43], physical inactivity are prevalent in PTSD [44], sleep-specific behaviors like irregular sleep schedules [45] or avoidance of bedtime due to trauma-related hypervigilance may uniquely disrupt restorative sleep [46]. These differences emphasize the need to address PTSD-related sleep disturbances beyond generic lifestyle modifications [47]. As table 3 demonstrates that interventions like Cognitive Behavioral Therapy for Insomnia (CBT-I) [48] and Imagery Rehearsal Therapy (IRT) [49] not only improve sleep continuity but also reduce sympathetic hyperactivity and blood pressure variability [50], suggesting dual benefits for psychological and cardiovascular health.

Though, critical limitations temper the interpretability of current evidence, heterogeneity in PTSD assessment tools like self-report vs. clinician-administered scales [51] and sleep measurement methods

like polysomnography vs. actigraphy complicates cross-study comparisons [52]. Moreover, many studies rely on self-reported CVD outcomes or cross-sectional designs, limiting causal inference. For example, table 1 discloses that over 50% of included studies used observational designs, with only two RCTs evaluating sleep interventions. Residual confounding by depression that frequently co-occurs with PTSD [53], additionally obscures the unique contribution of sleep dysregulation to CVD risk.

Conclusion

Bridging mechanistic insights with translational applications, this review advances a paradigm shift in PTSD care, arranging sleep health not only a symptom management strategy but a cardiovascular preventive measurement. Upcoming research should must adopt innovative methodologies to disentangle complex pathways, whereas clinicians and policymakers should advocate for sleep focused interventions as a foundation of trauma informed care. These efforts hold the potential to disrupt the PTSD, Sleep, CVD triangle, ultimately reducing the disproportionate burden of cardiovascular morbidity in trauma survivors.

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