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Psychosomatic Consequences of War: Impact of War-stress on the Cardiovascular System

Reyhane Najmi-Afshord[1, 2], Mohammad-Reza Vaez-Mahdavi[1, 2], Hossein Hassanpour[1, 3*], Leila Nasiri[1]

1. Health Equity Research Institute, Shahed University, Tehran, Iran.
2. Department of Physiology, Faculty of Medicine, Shahed University, Tehran, Iran.
3. Department of Basic Sciences, Faculty of Veterinary Medicine, Shahrekord University, Shahrekord, Iran.

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ABSTRACT

Background: War-related stress profoundly affects cardiovascular health; however, its psychosomatic consequences remain underrecognized.

Materials and Methods: This narrative review synthesizes evidence from longitudinal studies and mechanistic research examining war stress, post-traumatic stress disorder (PTSD), and cardiovascular disease (CVD) across military and civilian populations.

Results: War stress damages the cardiovascular system through chronic sympathetic activation, hypothalamic-pituitary-adrenal (HPA) axis dysregulation, systemic inflammation (including NOD-like receptor family, pyrin domain-containing 3 (NLRP3) inflammasome activation), endothelial dysfunction, and metabolic dysregulation. Longitudinal studies confirm persistent risk: Vietnam veterans show elevated heart disease risk two decades post-service, and Gaza civilians exhibit trauma-linked blood pressure increases. PTSD is associated with up to 80% increased cardiovascular risk. Evidence in the reverse direction is considerably weaker, derived primarily from cardiac patients rather than war-trauma contexts, and may be confounded by shared risk factors. Therefore, the bidirectional framework requires further validation.

Conclusion: War stress produces lasting cardiovascular injury through multiple interconnected pathways. The bidirectional PTSD-CVD link mandates integrated psycho-cardiac care with bidirectional screening. Current evidence overrepresents male Western veterans, with critical gaps from active conflict zones in Asia, Africa, and the Middle East. Future research requires longitudinal civilian cohort studies and randomized trials of combined cardiopsychological interventions.

*** Corresponding Author:**
Hossein Hassanpour, PhD.
Address: *Health Equity Research Institute, Shahed University, Tehran, Iran.*
Phone: *+98 (912) 5434989*
E-mail: *hassanpourh@yahoo.com*

Introduction

The psychological toll of war has been recognized for centuries; however, its physiological consequences, particularly on the cardiovascular system, remain underappreciated in both clinical practice and public health policy. While the visible destruction of armed conflict is well documented, the invisible, long-term biological embedding of war-related stress continues to affect millions of veterans, civilians, refugees, and health and care workers (HCWs) long after active hostilities cease [1].

The cardiovascular system is uniquely sensitive to psychological stress. Acute stress triggers adaptive autonomic and neuroendocrine responses mediated by the sympathetic nervous system and the hypothalamic-pituitary-adrenal (HPA) axis, which increase heart rate, blood pressure, and cardiac output. However, when stress becomes severe, recurrent, or chronic, as is the case in war zones or in the aftermath of trauma, these same mechanisms become maladaptive. Chronic sympathetic activation, HPA-axis dysregulation, systemic inflammation, endothelial dysfunction, and metabolic disturbances collectively contribute to hypertension, atherosclerosis, myocardial infarction, stroke, and stress-induced cardiomyopathies, such as Takotsubo syndrome [2, 3].

Among the most common psychiatric consequences of war trauma is post-traumatic stress disorder (PTSD). PTSD is not merely a mental health condition; it is increasingly recognized as a systemic disorder that targets the cardiovascular system through the brain–heart axis. Emerging evidence indicates that PTSD increases the risk of incident cardiovascular disease (CVD) by up to 80%. Conversely, a major cardiovascular event, such as a myocardial infarction, can itself serve as a traumatic trigger for secondary PTSD, establishing a bidirectional, self-reinforcing psychosomatic cycle [2]. This requires qualification. Evidence that PTSD increases cardiovascular risk is robust, but the reverse direction rests on weaker evidence from cardiac populations, not war-trauma contexts. Shared risk factors and genetic vulnerabilities may confound the association, and reverse causation cannot be excluded. The bidirectional framework therefore remains a hypothesis requiring further validation.

Despite growing awareness, critical gaps remain. Most existing research focuses on male, Western military veterans, with limited longitudinal data from civilian populations in active conflict zones, such as Gaza, Syria, Ukraine, and Iran. Women, children, HCWs, and survivors of chemical or environmental attacks remain underrepresented. Furthermore, the underlying molecular mechanisms, including the role of the NOD-like receptor family, pyrin domain-containing 3 (NLRP3) inflammasome, vagal tone, and neuroimmune crosstalk, are still being elucidated.

This narrative review synthesizes evidence from longitudinal studies and mechanistic research examining the psychosomatic consequences of war stress on the cardiovascular system. Specifically, it aims to:

1) Describe the acute, subacute, and chronic cardiovascular responses to war-related stress. 2) examine the bidirectional relationship between PTSD and CVD. 3) identify at-risk populations beyond military personnel. 4) outline the key biological pathways linking war stress to cardiovascular injury, including autonomic, neuroendocrine, inflammatory, endothelial, and metabolic mechanisms. 5) highlight evidence gaps and propose directions for future research and integrated psycho-cardiac care.

This review seeks to advance a more comprehensive, trauma-informed approach to cardiovascular health in conflict-affected populations by bridging the disciplines of cardiology, psychiatry, immunology, and public health.

Materials and Methods

A systematic literature search was conducted in PubMed/MEDLINE, PsycINFO, Scopus, and Web of Science from inception to June 2026. Search terms combined three domains: (1) war/conflict exposure (e.g. “war,” “combat,” “refugee,” “chemical attack”), (2) psychological stress (e.g. “PTSD,” “traumatic stress”), and (3) cardiovascular outcomes (e.g. “myocardial infarction,” “hypertension,” “endothelial dysfunction”). Two independent reviewers screened titles/abstracts, then full texts. Disagreements were resolved by consensus with a third reviewer. Data extraction captured study design, population, exposure measures, outcomes, and key findings. Reference lists of included articles were manually screened for additional studies.

Studies were included if they: 1) examined war-related stress exposure (military or civilian); reported cardiovascular outcomes (clinical events, biomarkers, or imaging); 2) used longitudinal, cross-sectional, mechanistic, or systematic review designs; and 3) were peer-reviewed English-language publications. Studies were excluded if they: 1) focused on non-war-related stress (e.g. natural disasters, general occupational stress); 2) lacked cardiovascular outcome measures; 3) were case reports, conference abstracts, or non-systematic reviews.

Cardiovascular responses to war stress: From acute adaptation to chronic maladaptation

Stress is an adaptive response to environmental challenges, but severe or prolonged stress may contribute to cardiovascular injury [4, 5]. Evidence indicates that stress, whether in its acute form (such as the battlefield) or its chronic form (such as PTSD), can target the cardiovascular system through neuro-immune-endocrine pathways [6].

The first systematic description of such effects dates back to 1871, when Jacob Mendes Da Costa described "soldier's heart" in veterans of the American Civil War. This syndrome, characterized by palpitations, chest pain, and fatigue, suggested that wartime exposure could produce persistent cardiac symptoms [7].

Acute cardiovascular responses to stress are primarily mediated by the autonomic nervous system (ANS) and the HPA axis. Sympathetic activation increases heart rate, myocardial contractility, and blood pressure through the release of catecholamines (epinephrine and norepinephrine), while parasympathetic activity modulates these effects through the vagus nerve and slows heart rate and regulates blood pressure [8, 9]. In parallel, stress activates the HPA axis through corticotropin-releasing hormone (CRH), which stimulates adrenocorticotropic hormone (ACTH) release and subsequent cortisol production. Although these responses are adaptive in the short term, repeated or prolonged activation may contribute to cardiovascular damage [10].

The response to acute, short-term stress is adaptive and protective. However, when stress becomes recurrent, severe, or prolonged, the same protective mechanisms can lead to cardiac injury [11]. Figure 1 shows a schematic diagram of physiological responses to acute stress.

Repeated activation of stress responses may produce cumulative physiological strain, the "wear and tear" of physiological systems, a process known as allostatic load [12]. Chronic stress is associated with sustained sympathetic and HPA pathway activation, reduced vagal tone, and systemic inflammation [13]. Chronic stress, by reducing vagal tone and increasing sympathetic dominance, decreases heart rate variability (HRV). HRV is an independent predictor of cardiovascular events and sudden cardiac death [14]. Inflammatory markers, such as interleukin-6 and C-reactive protein, have also been associated with psychological stress and coronary artery disease. Mental stress-induced myocardial ischemia (MSIMI) represents another important example of how psychological stress may precipitate cardiac dysfunction, even in the absence of exercise-induced ischemia. MSIMI is associated with sympathetic activation, increased blood pressure, and coronary vasoconstriction [15].

War stress refers to traumatic experiences associated with armed conflict, organized violence, and war-related existential threats [16]. These may include direct combat exposure, witnessing violence, injury or death, threats to personal safety, loss of home, loss of social networks or livelihood, and siege, bombing, or terrorist attacks [17].

War stress can be divided into three time-based categories: a) acute stress (minutes to days after the traumatic event): In this phase, the fight-or-flight response predominates. The acute stress reaction is a transient, expected response to extreme stress. At the peak of combat, the cardiovascular system is intensely activated. This response

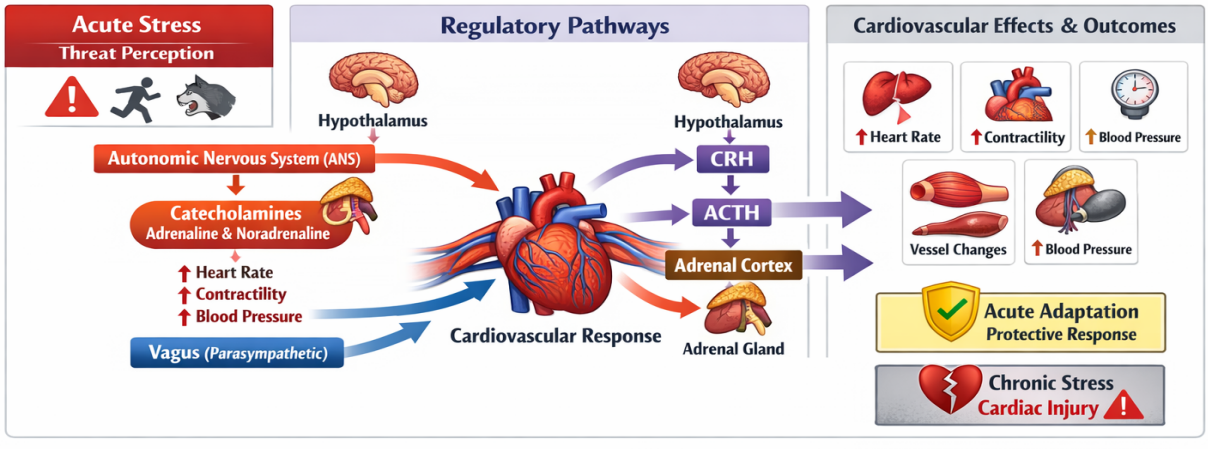

Figure1. Schematic diagram of physiological responses to acute stress

can contribute to arrhythmias and myocardial ischemia, and in rare cases it may trigger stress-induced cardiomyopathy, such as Takotsubo syndrome [18]. One cardiac consequence of acute stress is Takotsubo cardiomyopathy (TCM), also known as “broken heart syndrome”. In this syndrome, severe emotional or physical stress causes sudden, reversible left ventricular dysfunction that mimics myocardial infarction, but without coronary artery obstruction [19]. Cases of TCM have been reported in Ukrainian war survivors and even in individuals exposed to war-related media coverage, suggesting that the effects of war stress may extend beyond direct geographic exposure [20]. b) subacute Stress (days to weeks): In this phase, stress responses remain active and may be accompanied by early symptoms of PTSD. Acute stress disorder is characterized by intrusion, negative mood, dissociation, avoidance, and arousal symptoms, beginning after trauma exposure and lasting from 3 days to 1 month [21].

Persistent HPA-axis and sympathetic activation characterize subacute stress. Basal cortisol levels may remain elevated, blood pressure becomes chronically elevated, and inflammatory markers may stay raised. During this phase, although the initial traumatic event has passed, the individual may remain exposed to ongoing war conditions, including repeated bombardment and persistent threat, which may exacerbate and prolong the stress response. c) chronic/post-conflict stress (months to years after the event): This phase is often associated with PTSD, depression, and anxiety disorders, leading to long-term cardiovascular and systemic consequences [22]. Figure 2 shows a schematic diagram of physiological responses to chronic stress and CVD.

Definition and epidemiology of PTSD

PTSD is among the most common psychiatric disorders in individuals with a history of war trauma. PTSD is defined by symptoms, such as re-experiencing, avoidance, negative alterations in cognition and mood, and hyperarousal [23]. In a longitudinal study from Gaza (2006–2021), PTSD trajectories following chronic war trauma were classified into four groups: Acute, remitting, delayed, and resilient [24]: Acute: High PTSD symptoms at both time points; remitting: High PTSD symptoms in 2006 but not in 2021; Delayed: no PTSD symptoms in 2006 but significant symptoms in 2021; and Resilient: no PTSD symptoms at either time point. These findings suggest that PTSD has systemic consequences, including effects on cardiovascular health.

Bidirectional psychosomatic cycle: PTSD and CVD

The relationship between war-related stress and CVD is bidirectional. War-induced PTSD is associated with CVD, and cardiovascular events may, in turn, contribute to secondary PTSD, creating a self-reinforcing psychosomatic cycle, trapping individuals in a loop of psychosomatic illness [25]. Several biological mechanisms may explain this bidirectional link [26]: a) autonomic dysfunction: Individuals with PTSD show reduced HRV and increased sympathetic activity. b) HPA-axis dysregulation: In PTSD, cortisol secretion is altered, with lower basal levels in some patients and enhanced negative feedback, which may lead to a flat diurnal slope. This dysregulation is associated with insulin resistance and visceral obesity. c) chronic inflammation: PTSD is associated

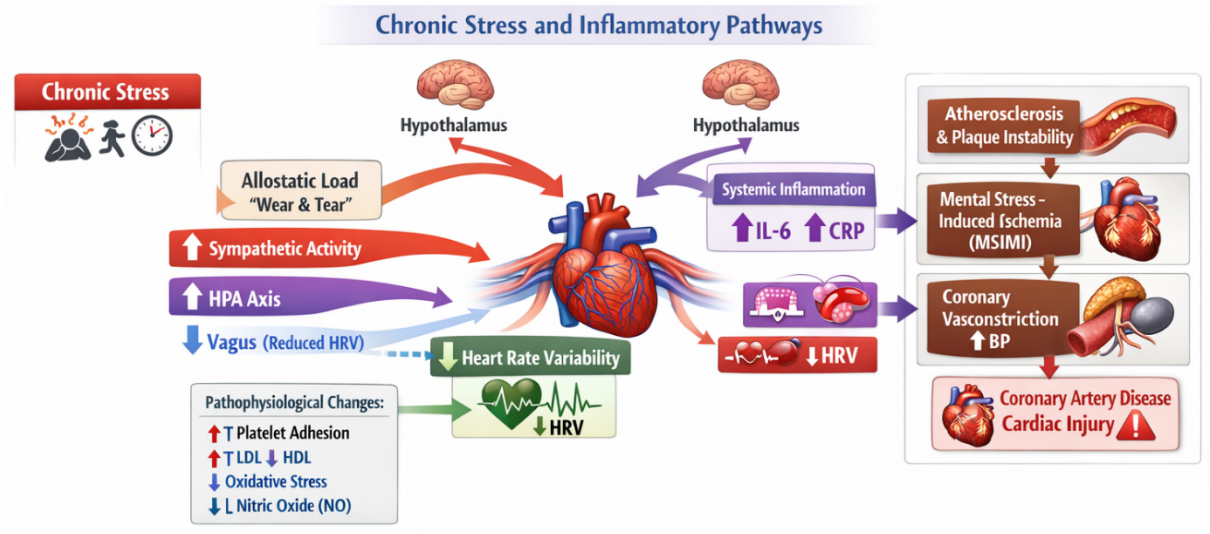

Figure 2. Schematic diagram of physiological responses to chronic stress

with elevated interleukin (IL)-6, tumor necrosis factor-alpha (TNF-α), and C-reactive protein (CRP). Activation of the NLRP3 inflammasome is a novel pathway linking traumatic stress, neuroinflammation, and vascular injury. d) endothelial dysfunction: Reduced flow-mediated dilation (FMD) and decreased nitric oxide biomarkers are observed in PTSD. e) Metabolic dysregulation: PTSD is also associated with increased total cholesterol, low-density lipoprotein (LDL), and triglycerides, as well as decreased high-density lipoprotein (HDL). Metabolic syndrome has been reported to be 1.53 times more common in women with PTSD. Figure 3 shows a schematic diagram of physiological responses to war stress.

The evidence base for bidirectionality is markedly asymmetrical. The association between PTSD and subsequent cardiovascular risk is supported by robust longitudinal evidence, including a 20-year follow-up of Vietnam veterans and meta-analyses showing an 80% increased risk. In contrast, evidence that cardiovascular events trigger secondary PTSD derives almost exclusively from cardiac patient populations (e.g. post-myocardial infarction cohorts). It has not been systematically examined in war-trauma contexts. Moreover, shared risk factors including smoking, physical inactivity, poor diet, and low socioeconomic status as well as genetic vulnerabilities may confound both directions of this association, and reverse causation cannot be ruled out. Given these limitations, the bidirectional framework should be viewed as a working hypothesis requiring further empirical validation rather than an established clinical reality.

At-risk populations for war-related cardiovascular harm

War stress is not limited to soldiers on battlefields. A wide range of populations is affected.

1) military personnel and veterans: This group is the most studied. A review of 26 studies found that combat exposure was associated with an 80% increased risk of CVD (hazard ratio=1.80) [27]. A 20-year cohort study of Vietnam War veterans showed a 1.48-fold higher risk of heart disease [28]. 2) civilians in war zones: Civilian populations in conflict zones are often overlooked. A study in Gaza following 1,000 adults from 2013 to 2019 showed that 22.4% had an ascending blood-pressure trajectory linked to war-related traumatic experiences, including airstrikes, witnessing violence, and loss of family [29]. 3) refugees and displaced persons: War refu-

Figure 3. Schematic diagram of physiological responses to war stress

gees face not only war trauma but also migration stress, homelessness, and socio-economic deprivation [30, 31]. 4) children: Children and students constitute one of the most vulnerable segments of society and, during armed conflicts, are exposed to psychological and physical trauma that may result in long-term sequelae [32, 33]. In Iran's recent conflict, following the direct American airstrike on the Shajareh Tayyebeh School in Minab, children and educators were killed and caused irreparable and indelible harm to their families and community. Furthermore, surviving students were exposed to trauma resulting from the loss of their peers and their school. The distress associated with bereavement, encompassing family members, friends, and school, and the pervasive fear of mortality during bombardment may also extend to other children through secondary traumatic exposure [33]. 5) healthcare workers (HCWs): HCWs play a central role in delivering essential medical services and promoting public health. HCWs are particularly vulnerable in conflict settings, where war-related stress, limited resources, and system overload may adversely affect multiple dimensions of health, including physical and mental well-being [34]. Research on the physical and psychological health of HCWs in conflict-affected regions remains limited. During the recent war in Iran, HCWs not only treated severe war injuries but were also directly targeted by repeated attacks on medical centers [35]. 6) Survivors of chemical and biological attacks: A study on the short and longterm consequences of the Sarin attack in Eastern Ghouta, Syria (2013) showed that survivors suffered longterm cardiovascular risks in addition to acute pulmonary and neurological damage [36]. In the recent attacks by the US-Israeli regime on oil depots in Tehran during the early days of the war [37], the combination of severe pollution from burning oil products and rain led to acid rain; the consequences of attacks on fuel depots and resulting environmental contamination may also contribute to long-term health risks [38].

Longitudinal evidence of post-war cardiovascular risk

War stress targets the cardiovascular system through multiple pathways, both during combat and for years afterward. One of the strongest pieces of evidence for the post-war effect on the heart comes from longitudinal studies. A 20-year follow-up of Vietnam War veterans showed that they remained at higher risk of heart disease even decades after military service [28]. This indicates cumulative effects of war stress that do not subside over time and may intensify with age and additional risk factors. The Gaza longitudinal study (2013-2019) is important because it focuses on a civilian population [29]. For refugees, displaced persons, and survivors, post-war stress is accompanied by challenges that can worsen cardiac outcomes: Socio-economic deprivation, limited healthcare access, loss of support networks, and refugee-status-related problems. The study of Sarin attack survivors in Eastern Ghouta showed that they face long-term cardiovascular risks [36]. These findings indicate that war, even in its indirect forms (chemical attacks, sieges, sanctions), can target the cardiovascular system.

Mechanistic pathways: Autonomic, neuroendocrine, and immune links to the cardiovascular system

ANS: Acute stress activates the sympathetic nervous system, leading to the rapid release of catecholamines from the adrenal medulla and sympathetic nerve fibers. These catecholamines bind to β-adrenergic receptors in the myocardium, increasing heart rate, contractility, and electrical conduction [39]. In severe war stress, heart rate can exceed 150 bpm, greatly increasing cardiac oxygen demand [40]. The hypothalamus sends signals to the adrenal medulla, leading to the rapid release of catecholamines (adrenaline and noradrenaline). These hormones cause the following changes in the cardiovascular system: Increased heart rate (tachycardia), increased myocardial contractility, dilation of skeletal muscle vessels (to supply blood to the muscles), constriction of visceral vessels (redistributing blood), and increased blood pressure due to increased peripheral vascular resistance [41]. Chronic sympathetic activation, common in PTSD, increases renin-angiotensin-aldosterone system (RAAS) activity, causing sodium retention, increased blood volume, and elevated blood pressure [42]. Studies show that combat veterans with PTSD exhibit exaggerated sympathetic responses to even mild stressors, even after the external stressor is removed. This phenomenon, called sensitization, indicates that the nervous system in individuals with a history of war trauma overreacts even to mild stressors [43].

Reduced vagal tone and autonomic dysregulation are key mechanisms linking chronic stress and war exposure to cardiovascular complications. The parasympathetic system, via the vagus nerve, slows heart rate and modulates sympathetic activity; chronic stress reduces vagal activity and increases sympathetic dominance, thereby reducing HRV [44]. Lower HRV is an independent predictor of sudden cardiac death, dangerous arrhythmias, and cardiovascular events, and studies have shown that patients with PTSD have lower HRV and a higher risk of cardiac death [26]. In parallel, increased sympathetic activity shortens the repolarization period and predisposes

individuals to ventricular arrhythmias, while reduced vagal tone weakens one of the body's main anti-arrhythmic mechanisms [44].

The HPA axis: When a threat is perceived, the paraventricular nucleus of the hypothalamus releases CRH. CRH signals the anterior pituitary to secrete ACTH. ACTH travels via the bloodstream to the adrenal cortex, stimulating it to produce cortisol [1]. Cortisol release increases blood pressure by enhancing sensitivity to catecholamines and stimulating mineralocorticoid receptors in blood vessels. In the acute response, cortisol has beneficial cardiovascular effects by increasing adrenergic receptor sensitivity, increasing cardiac output, and regulating blood flow. However, in chronic stress, this response becomes dysregulated.

HPA dysregulation in PTSD: In individuals with PTSD, the cortisol secretion pattern shows characteristic changes. Contrary to expectation, basal cortisol levels in many PTSD patients are below normal. However, the hypothalamic-pituitary negative feedback to cortisol is more sensitive, leading to excessive suppression of the HPA-axis. This phenomenon is known as enhanced negative feedback sensitivity [45].

At the same time, some PTSD subgroups show higher cortisol levels, which are associated with increased blood pressure, insulin resistance, and visceral adiposity. This HPA dysregulation is one of the most important pathways linking PTSD to metabolic and CVD [26].

Cortisol and atherosclerosis: Cortisol contributes to atherosclerosis through several mechanisms [46]: 1) It increases blood pressure by increasing vascular sensitivity to catecholamines; 2) It alters lipid metabolism, increasing triglycerides and decreasing HDL. 3) It increases platelet aggregation and blood thrombogenicity. 4) It has pro-inflammatory effects, increasing production of pro-inflammatory cytokines from endothelial cells and macrophages.

Immune system activation in stress: Acute and chronic stress activate the innate immune system through several pathways [47]: 1) Catecholamines and chronic cortisol promote the release of pro-inflammatory cytokines by stimulating glucocorticoid receptors on macrophages and endothelial cells; 2) Sympathetic activation increases trafficking of immune cells (e.g. neutrophils, monocytes) to perfused tissues; 3) Stress increases oxidative stress and free radical production, damaging cell membranes.

Multiple studies have shown that PTSD is associated with elevated levels of pro-inflammatory cytokines [48]: 1) IL-6 is a pleiotropic cytokine that plays a key role in systemic inflammation by stimulating the liver to produce CRP and activating endothelial cells. IL-6 increases expression of adhesion molecules on endothelial cells, facilitating monocyte migration into the vessel wall; 2) TNF-α is a potent pro-inflammatory cytokine that increases vascular permeability, activates macrophages, and induces apoptosis in endothelial cells. TNF-α also causes insulin resistance in muscle and liver cells; 3) CRP is an acute-phase protein produced by the liver in response to IL-6. Elevated CRP indicates systemic inflammation and is independently associated with increased risk of cardiovascular events.

NLRP3 inflammasome: A recent advance in understanding the stress-inflammation link is the role of the NLRP3 inflammasome. Inflammasomes are intracellular protein complexes that regulate innate inflammatory responses. Oxidative stress, catecholamines, and cortisol activate NLRP3. NLRP3 inflammasome activation initiates a cascade that activates caspase-1 and leads to the production of IL-1β and IL-18, both potent pro-inflammatory cytokines. Excessive NLRP3 activation is associated with neurotoxicity, loss of synaptic integrity, mitochondrial damage, and increased oxidative stress, especially in brain structures vulnerable to stress, such as the hippocampus, amygdala, and prefrontal cortex. Recent studies show that NLRP3 activation is a key pathway linking traumatic stress, neuroinflammation, and vascular injury [49].

Inflammation and atherosclerosis: Chronic inflammation observed in war stress and PTSD plays a central role in atherosclerosis. The process begins with endothelial injury. Inflammation increases expression of adhesion molecules (vascular cell adhesion molecule-1, intercellular adhesion molecule-1) on endothelial cells, facilitating monocyte migration into the subendothelium [50]. Migrated monocytes become foam macrophages, forming the core of the atherosclerotic plaque. As the plaque progresses, the fibrous layer thickens, and the plaque becomes unstable, prone to rupture and thrombus formation [51].

Endothelial dysfunction as a vascular gateway to CVD

The endothelium, the single-cell layer lining the blood vessels, acts as more than a passive barrier. It is an active endocrine organ secreting various substances that regulate vascular tone, thrombogenicity, inflammation, and smooth muscle growth. The most important of these is nitric oxide (NO), produced by endothelial nitric oxide synthase [52].

Chronic stress impairs endothelial function through several mechanisms [53]: a) reduced NO production; Oxidative stress from increased sympathetic metabolism produces superoxide radicals, which react with NO to form peroxynitrite (a strong oxidant). This both reduces NO bioavailability and increases oxidative stress; b) reduced FMD; FMD is a non-invasive measure of endothelial function using duplex ultrasound. Studies show that individuals with PTSD have significantly reduced FMD, indicating endothelial dysfunction; c) increased endothelial inflammation; Stress increases expression of adhesion molecules and endothelial growth factors, setting the stage for atherosclerosis.

Arterial stiffness is another consequence of chronic stress on the vascular system. Increased sympathetic and RAAS activity causes structural remodeling of the vessel wall; increased collagen with decreased elastin in the tunica media reduces arterial flexibility [54]. Arterial stiffness, measured by pulse wave velocity, is an independent predictor of CVD, isolated systolic hypertension, and cardiovascular death [55].

Pro-thrombotic states in war stress: coagulation, platelets, and fibrinolysis

Acute and chronic stress are associated with a pro-thrombotic state, created through multiple mechanisms [56]: a) Increased coagulation factors: Catecholamines and cortisol increase production of fibrinogen, factor VIII, and von Willebrand factor from the liver and endothelial cells; b) Increased platelet activity: Stress increases expression of glycoprotein IIb/IIIa receptors on platelets, enhancing platelet adhesion and aggregation; c) Reduced fibrinolysis: Stress increases production of plasminogen activator inhibitor-1, reducing the body's ability to dissolve clots.

The pro-thrombotic state in war stress, especially in individuals with underlying atherosclerosis, can lead to acute vascular thrombosis and events, such as myocardial infarction (MI) and stroke [6]. Studies have shown that PTSD is associated with a 75% increased risk of CVD, a 42% increased risk of MI, and more than a 100% increased risk of stroke. About 25% of individuals develop PTSD after a cardiac event (e.g. MI) [2]. This vicious cycle shows that the heart and mind are closely linked in a pathogenic loop.

Metabolic dysregulation: Visceral obesity, insulin resistance, and dyslipidemia

Chronic stress is associated with several metabolic disorders, all of which are risk factors for CVD [57]: a) visceral obesity: Chronic cortisol causes fat accumulation in the abdominal region (visceral adiposity). Visceral fat is more dangerous than subcutaneous fat due to its high metabolic activity and production of pro-inflammatory adipokines; b) insulin resistance: TNF-α and other pro-inflammatory cytokines disrupt insulin signalling in muscle and liver cells, leading to elevated blood glucose and eventually type 2 diabetes; c) dyslipidemia: Stress increases total cholesterol, LDL, triglycerides, and decreases HDL – an unfavorable lipid profile that is a core component of metabolic syndrome; d) hypertension: Chronic sympathetic and RAAS activation leads to increased blood pressure [58]. One study found that PTSD is associated with a 24-46% increased risk of hypertension [59].

Metabolic syndrome, defined by the presence of at least three of five criteria (abdominal obesity, hypertension, hyperglycemia, hypertriglyceridemia, and low HDL), is more common in individuals with PTSD. One study showed that metabolic syndrome is significantly associated with PTSD (odds ratio=1.53) in women, but there was no significant association in men [60].

Brain-heart axis: Neural and immune crosstalk in PTSD-Related CVD

The brain and heart are in constant communication through neural, hormonal, and immune pathways. This bidirectional axis, the brain-heart axis, plays a key role in the link between PTSD and CVD [61].

Sympathetic pathway: The hypothalamus and amygdala (fear centers in the brain) control sympathetic fibers to the heart and vessels via brainstem nuclei. In PTSD, amygdala hyperactivity leads to increased sympathetic output [62].

Parasympathetic pathway: The nucleus ambiguous and dorsal motor nucleus of the vagus in the brainstem regulate cardiac parasympathetic activity. In PTSD, reduced activity in these nuclei leads to decreased vagal tone [62].

Neuro-immune pathways: The nervous and immune systems communicate via psychoneuroimmunology. The vagus nerve, 80% of whose fibers are afferent (sensory), carries inflammatory signals from internal organs to the brain. At the same time, vagal activation reduces TNF-α production from macrophages, a phenomenon called the cholinergic anti-inflammatory pathway. In PTSD, reduced vagal tone leads to loss of this anti-inflammatory control and increased systemic inflammation [63].

Figure 4 represents a schematic diagram of the overall effect of stress on physiological systems.

From pathophysiology to moral responsibility: war, justice, and heart

War, as examined in this article, is a collective trauma that extends far beyond the battlefield and continues long after the guns fall silent. War stress affects the cardiovascular system through chronic sympathetic activation, HPA-axis dysregulation, systemic inflammation mediated by pro-inflammatory cytokines and the NLRP3 inflammasome, endothelial dysfunction, and metabolic dysregulation. These effects are not limited to military personnel; civilians in war zones, refugees, and survivors of chemical attacks are also at risk.

The relationship between war trauma and CVD is bidirectional: War trauma harms the heart, and CVD can further worsen mental health. Breaking this cycle requires integrated psycho-cardiac care, beginning with bidirectional screening, followed by interdisciplinary intervention, and supported by public health policies that recognize cardiovascular and mental health as central components of post-war recovery.

While in today's world issues of health, health justice, stress, and quality of life are being addressed by researchers across a wide range of fields, and while the necessity of justice and stress reduction for a healthier life is undeniably proven, some politicians ridiculing such issues to satisfy their inner greed for power trample on the independence of nations and, by instigating internal and external wars, attempt to push back developing countries. Historically, Western colonialism has been clearly evident in Asia and the world. Although in the present century these scientifically proven issues ought to be applied and implemented at the highest level by politicians, and indeed are implemented in a few societies, in West Asia researchers are studying topics, such as resilience, resistance, and post-traumatic growth instead of improving quality of life because imposed wars have forcibly affected their quality of life. In a world where health awareness has advanced considerably, why do conditions, such as those in Gaza persist? Why have decades of oppression and violence continued? And why has international silence allowed such brutality to spread? Is the purpose of human life merely to consume and to aggress for personal gain? Is the purpose of researchers merely to give literary expression to the aspects of life's traumas, without seeking to bring con-

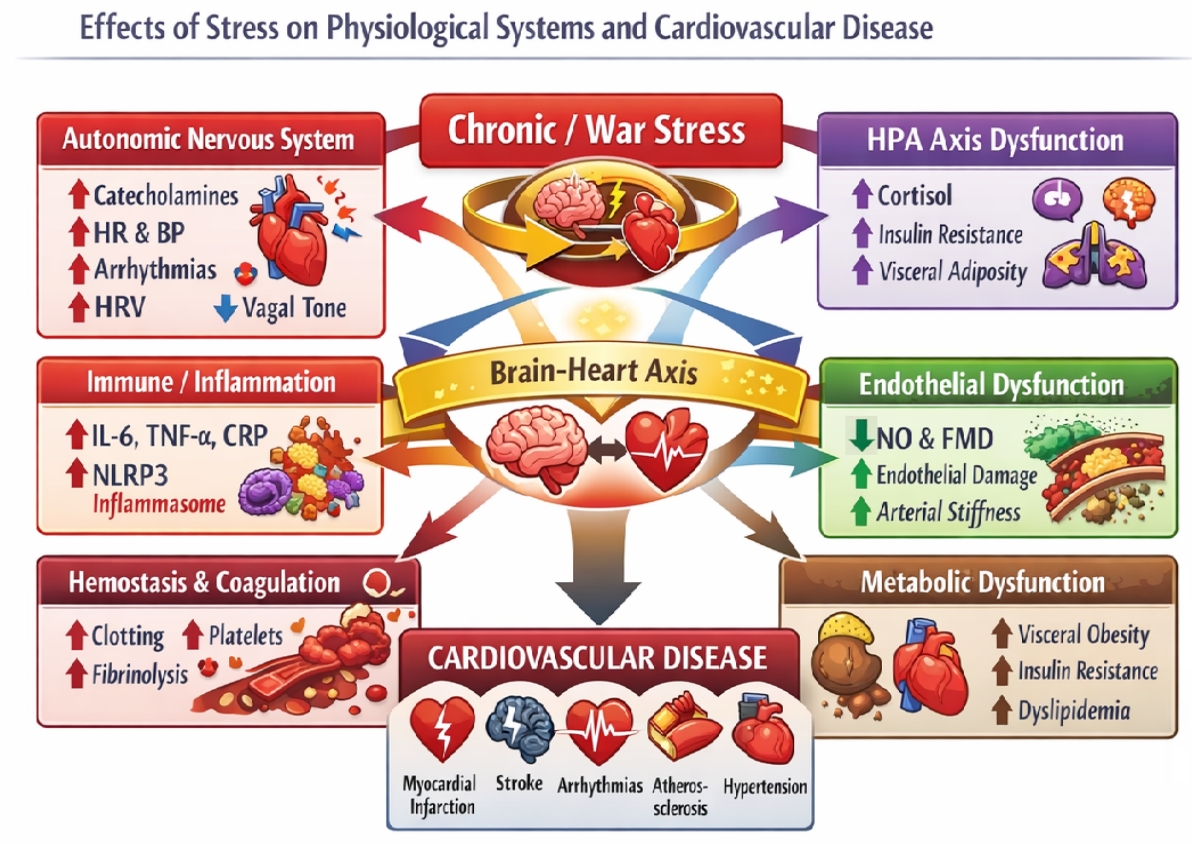

Figure 4. Schematic diagram of effect of stress on physiological systems

cepts like justice to fruition? Since the human body is ultimately mortal, what fear remains in living without submitting to tyranny?

Conclusion

This review demonstrates that war-related stress exerts significant and lasting effects on the cardiovascular system through interconnected physiological pathways. These include chronic sympathetic activation, dysregulation of the HPA axis, systemic inflammation driven by proinflammatory cytokines and the NLRP3 inflammasome, endothelial dysfunction, and metabolic dysregulation. Together, these mechanisms contribute to increased risks of hypertension, atherosclerosis, myocardial infarction, stroke, and stressinduced cardiomyopathy, such as Takotsubo syndrome. While PTSD is consistently associated with increased cardiovascular risk, evidence for the reverse direction is substantially weaker and derived from cardiac populations, not war-exposed individuals. Shared risk factors may confound these associations. Therefore, the bidirectional framework requires further validation. War stress is not limited to military personnel. Civilians in conflict zones, refugees, displaced persons, children, HCWs, and survivors of chemical or environmental attacks are all at elevated cardiovascular risk. Longitudinal studies in both veteran and civilian populations confirm that these effects persist for decades after the initial trauma and may accelerate biological aging. Current evidence reveals significant geographic and sexbased biases, with most research focused on male American or European veterans. Women remain underrepresented despite showing different biological and psychosocial responses to war stress, and data from active conflict zones in Asia, Africa, and the Middle East are still lacking. Methodological quality is often limited by reliance on selfreport and crosssectional designs.

Future research should prioritize longitudinal cohort studies in civilian waraffected populations, mechanistic studies of neuroimmune pathways (including the NLRP3 inflammasome and cholinergic antiinflammatory axis), and randomized controlled trials of integrated cardiopsychological interventions. From a clinical perspective, bidirectional screening, assessing cardiovascular risk in PTSD patients and PTSD symptoms after cardiac events, is urgently needed, alongside interdisciplinary care models that include cardiologists, psychiatrists, and immunologists.

Ethical Considerations

Compliance with ethical guidelines

This study was approved by the Ethics Committee of Shahed University, Tehran, Iran.

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Authors' contributions

Study supervision: Mohammad-Reza Vaez-Mahdavi and Hossein Hassanpour; Methodology conceptualization and project administration: Hassanpour; Literature review, original manuscript drafting, and figures' preparation: Leila Nasiri and Reyhane Najmi-Afshord; Review, editing and final approval: All authors.

Conflicts of interest

The authors declared no conflicts of interest.

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