

Review Paper

Impact of War-induced Stress on the Immune System: Mechanisms and Clinical Consequences

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ABSTRACT

Background: Armed conflicts expose populations to physical, chemical, and psychosocial stressors that disrupt immune homeostasis through neuroendocrine dysregulation and inflammation. The recent US-Israel war against Iran in the Persian Gulf exemplifies this, with affected populations facing blast exposures, displacement, healthcare disruption, and chronic psychosocial stress. This narrative review aimed to synthesize evidence on molecular and clinical immune alterations induced by war-related stress.

Materials and Methods: This narrative review synthesized peer-reviewed literature using PubMed, Web of Science, and Google Scholar. We included original research, systematic reviews, and meta-analyses examining immune outcomes in conflict-exposed populations.

Results: War-related stress induces persistent immune dysregulation characterized by elevated pro-inflammatory mediators (interferon-gamma, interleukin [IL]-2, tumor necrosis factor-alpha, IL-6, C-reactive protein), reduced anti-inflammatory cytokines, altered T-cell profiles, diminished natural killer (NK) cell numbers, and neuroinflammation with blood-brain barrier disruption. Post-traumatic stress disorder involves dysregulated T-helper1 differentiation, altered chemokine expression, and downregulated suppressive microRNAs. Chronic stress drives hypothalamic-pituitary-adrenal axis activation, producing low-grade inflammation with glucocorticoid resistance. Accessible biomarkers including neutrophil-to-lymphocyte ratio (NLR), systemic immune-inflammation index (SII), and C-reactive protein show promise for risk stratification. Sex-specific differences and transgenerational effects warrant further investigation.

Conclusion: War-induced immune dysfunction is multidimensional, affecting both military and civilian populations. Integrated biomarker monitoring, nutritional support, stress management, and targeted immunomodulatory therapies are priority interventions. Longitudinal studies and validated biomarkers across diverse populations are urgently required.

Keywords:

Immune dysregulation,
Inflammation, Gulf war
illness (GWI),
Persian Gulf

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Introduction

Armed conflicts expose military personnel and civilians to a combination of extreme environmental, chemical, physical, and psychosocial stressors. Jayasinghe (2024) proposed the “twelve dimensions of health impacts of war” (12-D) framework, which categorizes these consequences into direct health outcomes, social determinants, and environmental determinants [1]. These stressors converge through interconnected biological pathways—including sustained immune dysregulation, oxidative stress, and epigenetic reprogramming—as conceptualized by the “war exposome” framework [2]. In conflict settings, the war exposome includes not only direct combat-related exposures (blast waves, chemical agents, radiation) but also secondary stressors, such as displacement, food insecurity, healthcare disruption, and chronic psychosocial trauma. Therefore, understanding the war exposome is essential for comprehensive risk assessment and the development of targeted interventions.

The immune system, a complex network of innate and adaptive components, maintains host defense through a delicate balance. Disruption of this homeostasis—whether toward insufficient or excessive responses—can precipitate infections, autoimmunity, or chronic inflammation [3]. In conflict settings, such dysregulation is increasingly recognized as a critical pathway linking war exposure to long-term morbidity. For instance, Tarnas et al. (2025) demonstrated that war-related stressors in northern Syria—including compromised living conditions, malnutrition, and disrupted healthcare—significantly increase susceptibility to infectious diseases, suggesting underlying immune dysfunction [4]. Similarly, civilian survivors of the 2013 Ghouta sarin attack reported persistent multisystem symptoms, such as recurrent infections and prolonged recovery, indicative of chronic immune impairment, and highlighted the urgent need for integrated medical and psychosocial support in post-conflict settings [5].

The recent escalation of military hostilities involving the United States and Israel against Iran in the Persian Gulf region exemplifies a contemporary conflict scenario with profound implications for immune health. Since early 2026, heightened airstrikes, naval confrontations, and the threat of chemical or radiological exposure have placed both military personnel and civilian populations along the Gulf coastline under extreme duress. Populations in affected Iranian provinces, including Hormozgan and Bushehr, have experienced repeated blast exposures, displacement, disruption of healthcare services,

and persistent psychosocial stress from continuous air raid sirens and media coverage of casualties. These compounding stressors, physical trauma, environmental pollutants from munitions, and chronic anxiety are predicted to drive the same immune dysregulation pathways documented in other war-exposed cohorts. Early reports from regional health authorities indicate rising cases of unexplained fatigue, recurrent respiratory infections, and autoimmune-like symptoms among civilians, paralleling the patterns observed in Gulf war illness (GWI) and Syrian conflict survivors [5-8]. As this conflict unfolds, it presents both a humanitarian challenge and an urgent opportunity to prospectively study war-induced immune alterations in a new, diverse population, while implementing evidence-based interventions to mitigate long-term health consequences.

Understanding these immune alterations is crucial for mitigating both immediate and long-term health consequences in war-affected populations. This narrative review synthesizes current evidence on the molecular, cellular, and clinical immune alterations induced by war-related stress.

Materials and Methods

This narrative review synthesized peer-reviewed literature published between 2000 and 2026. We searched PubMed, Web of Science, and Google Scholar using combinations of the following keywords: “war,” “conflict,” “stress,” “immune system,” “immune dysregulation,” “inflammation,” “Gulf war illness,” “PTSD,” “cytokines,” “neuroinflammation,” and “HPA axis.” Additional articles were identified through citation searching of retrieved papers and review articles. We included original research, systematic reviews, and meta-analyses examining immune outcomes in military personnel or civilians exposed to war-related stressors. Case reports, opinion pieces, and non-English articles were excluded.

GWI as a model of war-induced immune dysregulation: Immune and inflammatory profiles in GWI

The clinical features of Gulf war illness (GWI), as the most well-documented and prominent model of war-induced immune dysregulation, indicate that persistent immune dysfunction is a fundamental contributor to its chronic pathophysiology [9, 10]. A growing body of evidence points to the involvement of both the innate and adaptive immune compartments in the sustained pathology of this condition [9, 11, 12]. Accumulating evidence indicates that persistent immune dysregulation is a core

feature of GWI, with significantly higher circulating counts of T and B lymphocytes, dysregulated T-cell profiles, diminished natural killer (NK) cell numbers, elevated serum levels of pro-inflammatory mediators (interferon-gamma [IFN- γ], interleukin [IL]-2, tumor necrosis factor-alpha [TNF- α], IL-6, C-reactive protein), and reduced anti-inflammatory cytokines (IL-4, IL-13), alongside evidence of neuroinflammation and blood-brain barrier disruption [9, 13].

Patients with GWI exhibit sex-specific transcriptomic changes in peripheral blood mononuclear cells under stress. Female patients with GWI show dysregulation in pro-inflammatory and profibrotic pathways, while male patients display altered IL-12 signaling and lymphocyte activation. During recovery from exertion, female patients with GWI exhibit changes in neuroinflammatory and microglial activation pathways, whereas male patients show altered apoptotic signaling. These findings highlight sex-dependent immune dysregulation in GWI [10].

Ashbrook et al. (2018) employed a murine model of GWI and demonstrated marked upregulation of innate immune-related genes, including pro-inflammatory cytokines (IL-1 β , TNF- α , IL-6) and Toll-like receptor 2, in the frontal cortex and hippocampus—pointing to microglial activation as a central driver of sickness-like behaviors [14]. Using a long-term rat model of GWI, Cheng and colleagues found significantly elevated mRNA levels of pro-inflammatory cytokines (TNF- α , IL-6, C-C motif chemokine ligand [CCL-2], IL-1 β) in the cortex and hippocampus, alongside increased activation of microglia and astrocytes, supporting a key role for neuroinflammation in GWI pathophysiology [15].

Exposure to Gulf War chemicals (e.g. permethrin, pyridostigmine bromide) can form hapten-protein adducts, triggering adaptive immune responses including elevated cluster of differentiation 4 positive T-cells, memory B cells, and mixed T-helper (Th)1/Th2/Th17 cytokine profiles. These peripheral immune changes, along with blood-brain barrier disruption and brain-reactive autoantibodies (e.g. against glial fibrillary acidic protein, myelin basic protein, tau), suggest crosstalk between systemic adaptive immunity and central nervous system pathology [9].

While GWI offers the most comprehensive longitudinal dataset on war-related immune dysregulation, several factors limit its generalizability to other conflict settings. First, GWI exposures, particularly pyridostigmine bromide (an anti-nerve agent pretreatment), permethrin-treated uniforms, and oil well fire smoke, are specific

to the 1990–1991 Gulf War theater and do not reflect the chemical, radiological, or blast exposures common in contemporary conflicts (e.g. Syria, Ukraine, Gaza). Second, GWI cohorts are predominantly male U.S. and U.K. military personnel, limiting insights into civilian populations, women, and children who constitute the majority of war-affected individuals globally. Third, the decades-long latency between exposure and systematic study in GWI may not capture the acute and subacute immune changes occurring in active conflict zones. Fourth, the relatively controlled deployment setting of the Gulf War differs substantially from the protracted, urban, and asymmetric warfare seen in modern conflicts, where repeated blast exposures, displacement, and healthcare collapse compound immune stressors [16–18]. Despite these limitations, GWI remains a valuable starting point for understanding chronic immune dysregulation, provided its findings are interpreted within these contextual boundaries.

Figure 1 represents a schematic overview of GWI pathophysiology.

War-related immune dysregulation beyond GWI: Evidence from other populations

Epidemiological evidence indicates a higher prevalence of autoimmune disorders among individuals with post-traumatic stress disorder (PTSD). Chronic stress in PTSD disrupts immune homeostasis, resulting in either suppression or overactivation of immune responses and altered immune cell function and cytokine release [19].

Bam et al. demonstrated that war-related PTSD involves immune dysregulation at multiple molecular levels. The Th cell differentiation pathway was altered, with upregulation of signal transducer and activator of transcription 4 (STAT4) and T-box transcription factor 21 (TBX21)—key transcription factors driving Th1-type pro-inflammatory responses and IFN- γ production. Additionally, expression of pro-inflammatory chemokines (CCL4, CCL5, C-X-C motif chemokine ligand [CXCL1]–3, CXCL6, CXCL8) and their receptors (CXCR1, CXCR2) was altered, suggesting impaired leukocyte migration. The dendritic cell maturation pathway was also dysregulated. Notably, suppressive microRNAs (miRNAs) (miR-125a, miR193a5p) were down-regulated, correlating with increased expression of their pro-inflammatory targets, including IFN- γ , IL-12B, T-box transcription factor 21 (TBX21), signal transducer and activator of transcription 4 (STAT4), and the aforementioned chemokines [20]. These findings suggest novel avenues for pathway-specific studies and tar-

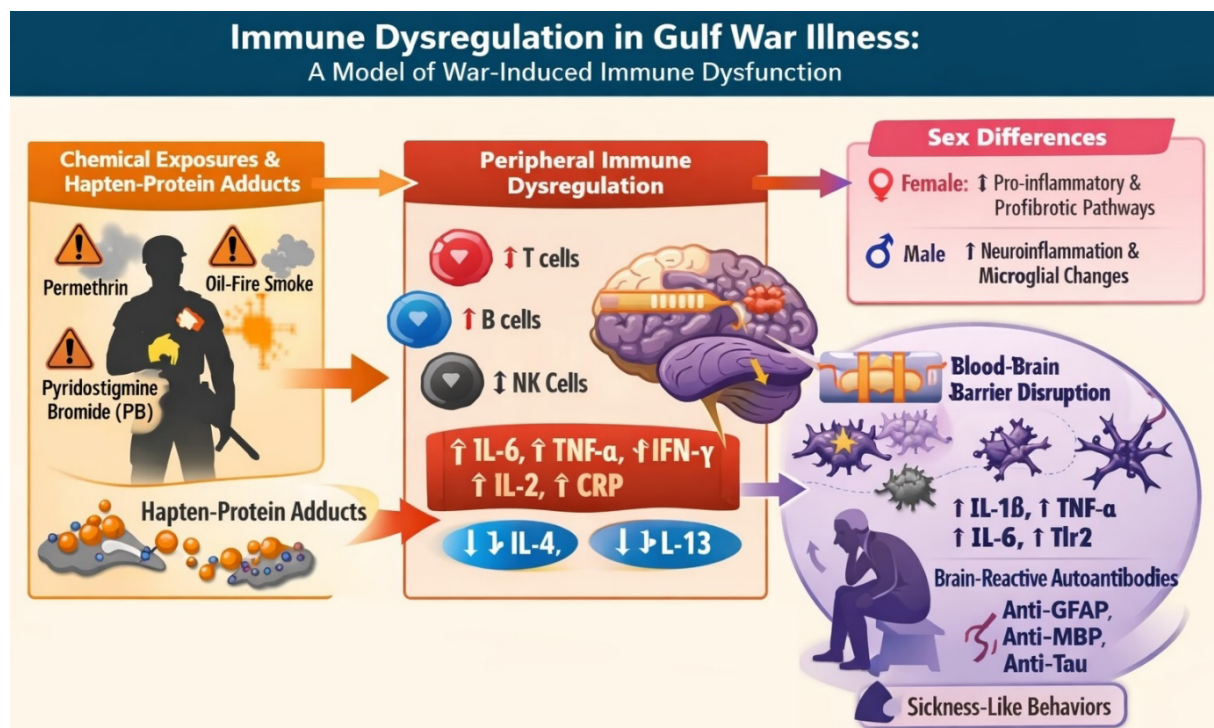


Figure 1. Schematic overview of GWI pathophysiology

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Note: This diagram summarizes the chemical exposures (permethrin, pyridostigmine bromide, oil well fire smoke) implicated in GWI. The full pathophysiological model, as discussed in the text, integrates these chemical factors with chronic psychological stress and neuroinflammation, leading to immune dysregulation, blood-brain barrier disruption, and central nervous system pathology, ultimately manifesting as the persistent symptoms characteristic of GWI.

geted therapeutic strategies—including epigenetic and miRNA-based interventions—to control inflammation in PTSD patients, both in veterans and the general population.

Korinek et al. (2024) found that war-related trauma—particularly killing/injury and witnessing atrocities—was associated with PTSD and arthritis in elderly Vietnamese civilians. This links psychological war stress to chronic inflammatory disease, supporting the immune-dysregulation pathway discussed in this review [21].

Mohammed et al. (2026) assessed long-term mental health outcomes among 534 Kurdish survivors of the 1988 Halabja chemical attacks. They found that 78.8% met PTSD criteria, and 46.3% had comorbid PTSD, somatic symptoms, and depression/anxiety. Women, individuals with lower education, and those with greater trauma exposure showed significantly higher symptom severity. Common somatic complaints included joint pain, fatigue, and back pain—symptoms often linked to chronic inflammation. This study demonstrates that chemical warfare leads to persistent psychological and physical morbidity decades after exposure, highlighting the need to address both mental and physical health in war-affected popula-

tions. Importantly, the high prevalence of somatic symptoms suggests that trauma-related stress may contribute to long-term inflammatory processes [22].

In a study of Croatian combat veterans with chronic PTSD, total lymphocyte counts were significantly elevated compared to healthy controls. A positive correlation was found between time since trauma and glucocorticoid receptor expression on lymphocytes. However, no significant differences were observed in lymphocyte subset proportions, NK cytotoxicity, or serum cortisol, suggesting that chronic war-related PTSD selectively alters lymphocyte counts and receptor expression in a time-dependent manner [23].

A case-control study of 698 young adults in war-affected Ukraine found that individuals with PTSD were more likely to adhere to restrictive dietary patterns and exhibit disordered eating. Conversely, adherence to the Mediterranean diet was associated with lower PTSD probability. Haematologically, PTSD was linked to reduced haemoglobin and red blood cell levels, and elevated neutrophil-to-lymphocyte ratio (NLR). Machine learning identified NLR, white blood cell, and haemoglobin as the strongest predictors of PTSD status [24].

Mechanisms of war-induced immune dysregulation

Chronic war stress leads to persistent activation of the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system. This results in a paradoxical state of low-grade chronic inflammation (elevated IL-6, TNF- α , CRP) together with relative glucocorticoid resistance. Additionally, the release of damage-associated molecular patterns from injured tissues triggers systemic inflammatory response syndrome (SIRS), followed by a compensatory anti-inflammatory response syndrome (CARS). Persistent imbalance between these two phases leads to reduced monocyte human leukocyte antigen – DR isotype expression, lymphocyte apoptosis, a shift from Th1 to Th2, elevated IL-10 and TGF- β , and expansion of myeloid-derived suppressor cells [25]. Immunophenotyping studies in veterans and refugees have consistently shown reduced numbers of naive T-cells, decreased cytotoxic activity of NK cells, and accumulation of senescent/exhausted T-cells. These changes correlate with shorter telomeres and epigenetic modifications (e.g. DNA methylation of stress-related genes) [23, 26, 27].

A pilot study of 178 military personnel exposed to extreme operational stress reported a significant post-deployment increase in the NLR, SII, systemic immune inflammation index (SII), and inflammatory index cumulative (IIC), reflecting complex immune activation [28].

War disrupts children's development through chronic activation of the hypothalamic-pituitary-adrenal (HPA) axis, leading to neurobiological impairments (memory, attention, executive function) and increased risk of PTSD, anxiety, and depression. Physiologically, prolonged stress weakens immune function, disturbs sleep, and elevates the risk of chronic diseases. These findings link childhood war trauma to long-term immune dysregulation and inflammatory pathways [29].

Persistent immune dysregulation predisposes affected individuals to a higher risk for respiratory infections, reactivation of latent viruses (e.g. Epstein–Barr virus, cytomegalovirus), and autoimmune disorders, such as rheumatoid arthritis and multiple sclerosis. In injured individuals, the imbalance between SIRS and CARS can lead to delayed wound healing, late-onset sepsis, multi-organ dysfunction syndrome, acute respiratory distress syndrome, and persistent inflammation-immunosuppression-catabolism syndrome [25]. Emerging data from the Ukrainian conflict confirm similar immune dysregulation patterns even in young civilians exposed to bombings and displacement.

Clinical implications and therapeutic opportunities

The immune alterations described above have direct clinical implications in war-affected populations. Several immune parameters show promise as accessible biomarkers for identifying at-risk individuals in conflict settings. The NLR and SII are inexpensive markers calculable from routine complete blood counts, while CRP is widely available in basic laboratory settings. In the Ukrainian study, NLR emerged as one of the strongest predictors of PTSD status, suggesting utility in mental health risk stratification [24]. Table 1 summarizes key immune biomarkers with established or emerging utility in war-related stress.

The association between adherence to the Mediterranean diet and lower PTSD probability in the Ukrainian cohort suggests that dietary modifications may have immunomodulatory benefits. The Mediterranean diet is rich in components with anti-inflammatory properties, including omega 3 fatty acids, polyphenols, and antioxidants. These nutrients may counteract chronic stress-induced inflammation [30, 31]. Similarly, stress reduction interventions including cognitive-behavioral therapy and trauma-focused therapies have demonstrated secondary immunological benefits in non-conflict populations and warrant implementation in post-conflict recovery programs [32].

Table 1. Immune biomarkers in war-related stress: Clinical utility

Biomarker	Change	Associated Outcomes	Clinical Utility
NLR	↑	PTSD, systemic inflammation	Low-cost; widely available
CRP	↑	Chronic inflammation	Low-cost; available
IL-6	↑	Fatigue, depression	Requires lab capacity
SII	↑	Systemic inflammation	Low-cost; calculable from CBC
NK cell count	↓	Viral reactivation risk	Requires flow cytometry

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Abbreviations: NLR: Neutrophil-to-lymphocyte ratio; CRP: C-reactive protein; IL-6: Interleukin-6; SII: Systemic immune-inflammation index; NK: Natural killer; CBC: Complete blood count; PTSD: Post-traumatic stress disorder.

The identification of specific immune pathways altered in war-related stress opens avenues for targeted pharmacotherapy. Anti-cytokine therapies (e.g. TNF- α inhibitors and IL-6 receptor antagonists), glucocorticoid receptor modulators, and microRNA-based interventions represent emerging approaches requiring investigation in war-affected populations.

Realizing these clinical opportunities faces substantial barriers in conflict settings, including disrupted healthcare infrastructure, limited availability of specialized agents, competing priorities, and stigma. Addressing these barriers requires multi-sectoral collaboration between ministries of health, humanitarian organizations, and academic partners.

Discussion

War-induced immune dysfunction is a multidimensional process involving neuroendocrine dysregulation, persistent inflammation, accelerated immune aging, and epigenetic reprogramming. The convergence of physical, chemical, and psychological stressors—as captured by the war exposome framework—creates a unique pathological signature that cannot be fully understood by examining any single factor in isolation. The evidence reviewed here demonstrates that both military personnel and civilians are susceptible to long-lasting immune alterations, with GWI providing the most comprehensive and well-validated model for studying these effects.

Several gaps remain. First, most studies are cross-sectional, limiting causal inference. Longitudinal studies are urgently needed to track immune trajectories from exposure to disease development. Second, the relative contribution of psychological versus chemical stressors is difficult to disentangle, and future research should employ multi-exposure models. Third, while biomarkers, such as NLR, CRP, and cytokine panels show promise (Table 1), they require validation across diverse populations and settings before widespread clinical implementation. Fourth, the potential for transgenerational effects—via epigenetic inheritance—warrants investigation. Finally, the development of preventive and therapeutic strategies—ranging from stress reduction programs and nutritional support (e.g. Mediterranean diet) to targeted immunomodulatory agents—should be prioritized and tested in high-risk groups.

Conclusion

War induced immune dysfunction is a multidimensional process involving neuroendocrine dysregulation, persistent inflammation, accelerated immune aging, and epigenetic reprogramming. These alterations affect both military and civilian populations, predisposing them to infections, autoimmune conditions, and chronic multi-system morbidity. The clinical implications are three-fold: accessible biomarkers (NLR, CRP, and SII) offer practical risk stratification tools, nutritional and stress management interventions may provide immunomodulatory benefits, and emerging pathway-specific evidence suggests potential for targeted pharmacotherapies. Despite these insights, critical knowledge gaps remain. Longitudinal studies, validation of biomarkers across diverse populations, and transgenerational epigenetic investigations are urgently required. Realizing the translational potential of these findings requires integrated monitoring, interdisciplinary collaboration, and sustained investment in research and healthcare delivery in conflict-affected regions.

Ethical Considerations

Compliance with ethical guidelines

There were no ethical considerations to be considered in this research.

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Authors' contributions

Methodology, review, and editing Hossein Hassanpour; Investigation and writing the original draft: Ensie Sadat Mirsharif; Data interpretation and final approval: All authors.

Conflicts of interest

The authors declared no conflict of interest.

References

- [1] Jayasinghe S. The 12 dimensions of health impacts of war (the 12-D framework): A novel framework to conceptualise impacts of war on social and environmental determinants of health and public health. *BMJ Global Health*. 2024; 9(5):e014749. [DOI:10.1136/bmjgh-2023-014749] [PMID] [PMCID]
- [2] Kovalyov O. The war exposome: Environmental, chemical, and psychosocial determinants of health. *Exposome*. 2026; 6(1):osag003. [DOI:10.1093/exposome/osag003]
- [3] Sompayrac LM. How the immune system works. Hoboken: John Wiley & Sons; 2022. [Link]
- [4] Tarnas MC, Almhawish N, Ratnayake R, Elferruh Y, Aladhan I, Alhaffar MBA, et al. Climate change, armed conflict, forced displacement, and epidemic-prone diseases: An exploratory study in northern Syria. *BMC Public Health*. 2025; 25(1):2642. [DOI:10.1186/s12889-025-23918-3] [PMID] [PMCID]
- [5] Alhaffar MBA, Zarzar L, Eriksson A, Namour S. Short and long-term health consequences of the 2013 Sarin attack in Ghouta, Syria: A retrospective descriptive study of civilian survivors. *Scientific Reports*. 2026; 16(1):11379. [DOI:10.1038/s41598-026-47135-z] [PMID] [PMCID]
- [6] Pirzad JG. Psychological Trauma from a Neuroscience Perspective Following Israel's 12-day War against Iran. *Journal of Military Medicine*. 2025; 27(1):96-101. [Link]
- [7] Kumari S, Fathima Haseena H, Jaber Amin MH. A humanitarian crisis in healthcare: The Israel-Iran conflict. *Annals of Medicine and Surgery*. 2026; 88(1):961-2. [DOI:10.1097/MS9.0000000000004317] [PMID] [PMCID]
- [8] Everson MP, Shi K, Aldrige P, Bartolucci AA, Blackburn Jr WD. Is there immune dysregulation in symptomatic Gulf War veterans?. *Zeitschrift für Rheumatologie*. 2000; 59(Suppl 2):124-6. [DOI:10.1007/s003930070007] [PMID]
- [9] Nkiliza A, Joshi U, Evans JE, Ait-Ghezala G, Parks M, Crawford F, et al. Adaptive immune responses associated with the central nervous system pathology of Gulf War illness. *Neuroscience Insights*. 2021; 16:26331055211018458. [DOI:10.1177/26331055211018458] [PMID] [PMCID]
- [10] Frank J, Tehrani L, Gamer J, Van Booven DJ, Ballarin S, Rossman R, et al. Gulf war illness induced sex-specific transcriptional differences under stressful conditions. *International Journal of Molecular Sciences*. 2025; 26(8):3610. [DOI:10.3390/ijms26083610] [PMID] [PMCID]
- [11] Dickey B, Madhu LN, Shetty AK. Gulf War illness: Mechanisms underlying brain dysfunction and promising therapeutic strategies. *Pharmacology & Therapeutics*. 2021; 220:107716. [DOI:10.1016/j.pharmthera.2020.107716] [PMID] [PMCID]
- [12] Trageser KJ, Sebastian-Valverde M, Naughton SX, Pasinetti GM. The innate immune system and inflammatory priming: Potential mechanistic factors in mood disorders and Gulf War illness. *Frontiers in Psychiatry*. 2020; 11:704. [DOI:10.3389/fpsy.2020.00704] [PMID] [PMCID]
- [13] Burzynski HE, Reagan LP. Exposing the latent phenotype of Gulf War Illness: Examination of the mechanistic mediators of cognitive dysfunction. *Frontiers in Immunology*. 2024; 15:1403574. [DOI:10.3389/fimmu.2024.1403574] [PMID] [PMCID]
- [14] Ashbrook DG, Hing B, Michalovicz LT, Kelly KA, Miller JV, de Vega WC, et al. Epigenetic impacts of stress priming of the neuroinflammatory response to sarin surrogate in mice: A model of Gulf War illness. *Journal of Neuroinflammation*. 2018; 15(1):86. [DOI:10.1186/s12974-018-1113-9] [PMID] [PMCID]
- [15] Cheng CH, Guan Y, Chiplunkar VP, Mortazavi F, Medalla ML, Sullivan K, et al. Nerve agent exposure and physiological stress alter brain microstructure and immune profiles after inflammatory challenge in a long-term rat model of Gulf War Illness. *Brain, Behavior, & Immunity-Health*. 2024; 42:100878. [DOI:10.1016/j.bbih.2024.100878] [PMID] [PMCID]
- [16] Kerr KJ. Gulf War illness: An overview of events, most prevalent health outcomes, exposures, and clues as to pathogenesis. *Reviews on Environmental Health*. 2015; 30(4):273-86. [DOI:10.1515/reveh-2015-0032] [PMID]
- [17] Coughlin SS, Krengel M, Sullivan K, Pierce PF, Heboyan V, Wilson LC. A review of epidemiologic studies of the health of Gulf War women veterans. *Journal of Environment and Health Sciences*. 2017; 3(2):10-5436. [DOI:10.15436/2378-6841.17.1551] [PMID] [PMCID]
- [18] Hotopf M, Wessely S. Can epidemiology clear the fog of war? Lessons from the 1990-91 gulf war. *International Journal of Epidemiology*. 2005; 34(4):791-800. [DOI:10.1093/ije/dyi102] [PMID]
- [19] Song H, Fang F, Tomasson G, Arnberg FK, Mataix-Cols D, Fernández de la Cruz L, et al. Association of stress-related disorders with subsequent autoimmune disease. *JAMA*. 2018; 319(23):2388-400. [DOI:10.1001/jama.2018.7028] [PMID] [PMCID]
- [20] Bam M, Yang X, Zumbrun EE, Zhong Y, Zhou J, Ginsberg JP, et al. Dysregulated immune system networks in war veterans with PTSD is an outcome of altered miRNA expression and DNA methylation. *Scientific Reports*. 2016; 6(1):31209. [DOI:10.1038/srep31209] [PMID] [PMCID]
- [21] Korinek K, Loebach P, Teerawichitchainan B. Physical and mental health consequences of war-related stressors among older adults: An analysis of posttraumatic stress disorder and arthritis in northern Vietnamese war survivors. *Journals of Gerontology Series B*. 2017; 72(6):1090-102. [DOI:10.1093/geronb/gbv157] [PMID]
- [22] Mohammed I, Ahmed H, Hasin B, Kizilhan J, Nariman H, Ahmed S, et al. A study on long-term trauma-related mental health outcomes among Kurdish survivors of chemical attacks. *Frontiers in Psychiatry*. 2025; 16:1693072. [DOI:10.3389/fpsy.2025.1693072] [PMID] [PMCID]
- [23] Vidović A, Vilibić M, Sabioncello A, Gotovac K, Rabić S, Folnegović-Šmalc V, et al. Circulating lymphocyte subsets, natural killer cell cytotoxicity, and components of hypothalamic-pituitary-adrenal axis in Croatian war veterans with posttraumatic stress disorder: Cross-sectional study. *Croatian Medical Journal*. 2007; 48(2):198-206. [Link]
- [24] Halabitska I, Petakh P, Buchynskiy M, Kamyshna I, Lushchak O, Kamyshnyi O. War, diet, and PTSD in Ukrainian youth. *Scientific Reports*. 2025; 16(1):1422. [DOI:10.1038/s41598-025-31138-3] [PMID] [PMCID]

- [25] Thompson KB, Krispinsky LT, Stark RJ. Late immune consequences of combat trauma: A review of trauma-related immune dysfunction and potential therapies. *Military Medical Research*. 2019; 6(1):11. [DOI:10.1186/s40779-019-0202-0] [PMID] [PMCID]
- [26] Jergović M, Tomičević M, Vidović A, Bendelja K, Savić A, Vojvoda V, et al. Telomere shortening and immune activity in war veterans with posttraumatic stress disorder. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*. 2014; 54:275-83. [DOI:10.1016/j.pnpbp.2014.06.010] [PMID]
- [27] Uddin M, Aiello AE, Wildman DE, Koenen KC, Pawelec G, de Los Santos R, et al. Epigenetic and immune function profiles associated with posttraumatic stress disorder. *Proceedings of the National Academy of Sciences*. 2010; 107(20):9470-5. [DOI:10.1073/pnas.0910794107] [PMID] [PMCID]
- [28] Mihai FD, Trasca ET, Radulescu D, Radulescu PM, Mercut R, Caluianu EI, et al. Dynamic assessment of the inflammatory response in military personnel: A pilot study on Δ NLR and composite markers in operational environments. *Journal of Medicine and Life*. 2025; 18(3):257. [DOI:10.25122/jml-2025-0056] [PMID] [PMCID]
- [29] Ferrara P, Zona M, Giardino I, Scaltrito F, Pettoello-Mantovani C, Cammisa I. The silent wounds of war: Psychophysical impacts and international legal implications for children in conflict zones. *Global Pediatrics*. 2025;100287. [DOI:10.1016/j.gpeds.2025.100287]
- [30] Puljiz Z, Kumric M, Borovina Marasovic T, Mastelic T, Rakusic M, Pavela G, et al. Mediterranean diet adherence, physical activity, and advanced glycation end products in complex PTSD: A comprehensive examination of lifestyle and cardiovascular risk in war veterans. *Nutrients*. 2024; 16(11):1791. [DOI:10.3390/nu16111791] [PMID] [PMCID]
- [31] Dama A, Shpati K, Daliu P, Dumur S, Gorica E, Santini A. Targeting metabolic diseases: the role of nutraceuticals in modulating oxidative stress and inflammation. *Nutrients*. 2024; 16(4). [DOI:10.3390/nu16040507] [PMID]
- [32] Shields GS, Spahr CM, Slavich GM. Psychosocial interventions and immune system function: a systematic review and meta-analysis of randomized clinical trials. *JAMA Psychiatry*. 2020; 77(10):1031-43. [DOI:10.1001/jamapsychiatry.2020.0431] [PMID] [PMCID]