

Psychopathology and Its Moderators in a Highly Traumatized Population Living in Kurdistan

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Abstract

Background: The Kurdistan Region has experienced major human rights violations, including the 1988 chemical attacks during the Anfal Campaign carried out by the Iraqi regime, and the 2014 genocide of Yazidis perpetrated by the Islamic State (IS). These events have had long-term effects on survivors' mental and physical health.

Objective: This study examined genetic, trauma-related, and psychosocial factors associated with post-traumatic stress disorder (PTSD) and related symptoms in 892 Kurdish trauma survivors: chemical gas survivors ($n = 534$) and Yazidi survivors of IS violence ($n = 358$). We assessed whether PTSD polygenic risk scores (PRS) are associated with symptom severity, whether this varies with cumulative trauma exposure, and whether trauma-related factors explain additional variance beyond demographic and psychosocial variables. Differences between the two cohorts were also examined.

Methods: Data were collected between March and September 2023 from survivors of the chemical gas attacks and between May and September 2024 from Yazidi survivors of ISIS attacks. Participants completed the PTSD Checklist for DSM-5 (PCL-5), the Patient Health Questionnaire-15 (PHQ-15), the Perceived Stress Scale-14 (PSS-14), and the Hopkins Symptom Checklist-25 (HSCL-25). Deoxyribonucleic acid (DNA) was extracted from saliva samples and genotyped using genome-wide single nucleotide polymorphism (SNP) arrays to calculate PTSD polygenic risk scores (PRS). Hierarchical regression models were used to examine demographic, psychosocial, and trauma-related predictors of psychopathology. Linear regression and mixed-effects models were subsequently applied to assess the main effects of PRS and PRS $\times$ trauma interactions, adjusting for age, gender, education, population structure, and relatedness. In addition, this study contributed to the translation and cultural adaptation of several psychometric instruments (PHQ-15, PCL-5, HSCL-25, and PSS-14) into Kurdish. Internal consistency and reliability were evaluated using Cronbach's alpha and McDonald's omega.

Results: Demographic variables explained 10-38% of the variance in mental health outcomes. In the chemical gas cohort, cumulative trauma ($\beta = 0.21$ - 0.24), lower education ($\beta = -0.17$ to -0.21), and female gender ($\beta = 0.03$ - 0.35) were consistently associated with higher symptom levels. Psychotropic medication use was also positively associated with symptoms. In the Yazidi cohort, cumulative trauma ($\beta = 0.19$ - 0.37), lower education, and female gender were the main predictors, while social support and medication had limited additional effects beyond demographics and cumulative trauma. Across both cohorts, cumulative trauma exposure and education were the most stable predictors of PTSD, depression/anxiety, somatic symptoms, and perceived stress.

In chemical gas survivors, PTSD PRS showed a small but significant main effect ($B = 1.76$, $p = .010$) and a significant interaction with trauma exposure ($B = 1.44$, $p = .032$), indicating that genetic risk became more apparent at higher levels of cumulative trauma. In Yazidi survivors, PRS showed no significant association with symptoms and no interaction with trauma exposure.

Conclusions: The results suggest that PTSD PRS is modestly associated with symptom severity and interacts with trauma exposure among chemical gas survivors, but not among Yazidi survivors. Across both cohorts, cumulative trauma exposure, female gender, and lower education consistently emerged as key predictors, highlighting the central role of trauma and socioeconomic context. Psychotropic medication and psychosocial factors showed stronger associations in the chemical gas cohort, whereas social support appeared more protective among Yazidi survivors, underscoring the importance of context-specific environmental and structural factors. Overall, the findings highlight the importance of considering ancestry and population structure in genomic prediction models and support the need for context-sensitive interventions in long-term trauma-exposed Kurdish populations.

Key words: Psychopathology, Depression, Psychometrics, War, Catastrophe, PTSD, Genetics, genomics, PRS, polygenic

Zusammenfassung

Hintergrund: In der Region Kurdistan kam es zu schweren Menschenrechtsverletzungen, darunter die chemischen Angriffe von 1988 sowie der Völkermord an den Jesiden im Jahr 2014. Diese Ereignisse haben die psychische und physische Gesundheit der Überlebenden nachhaltig beeinträchtigt.

Zielsetzung: Diese Studie untersuchte genetische, traumabezogene und psychosoziale Faktoren, die mit posttraumatischer Belastungsstörung (PTBS) und verwandten Symptomen bei $N = 892$ kurdischen Trauma-Überlebenden zusammenhängen: Überlebende von Chemiewaffenangriffen ($n = 534$) und jesidische Überlebende der Gewalt durch den Islamischen Staat ($n = 358$). Es wurde geprüft, ob polygenetische Risikoscores (PRS) für PTBS mit der Symptomschwere assoziiert sind, ob dieser Zusammenhang von der kumulativen Traumabelastung abhängt und ob traumabezogene Faktoren zusätzliche Varianz über demografische und psychosoziale Merkmale hinaus erklären. Unterschiede zwischen den beiden Kohorten wurden ebenfalls untersucht.

Methoden: Die Datenerhebung erfolgte von März bis September 2023 bei den Überlebenden der Chemiewaffenangriffe und von Mai bis September 2024 bei den jesidischen Überlebenden. Die Teilnehmenden füllten den PTSD Checklist for DSM-5 (PCL-5), den Patient Health Questionnaire-15 (PHQ-15), die Perceived Stress Scale-14 (PSS-14) und die Hopkins Symptom Checklist-25 (HSCL-25) aus. Desoxyribonukleinsäure (DNA) wurde aus Speichelproben extrahiert und mittels genomweiter Single-Nucleotide-Polymorphism-(SNP-)Arrays genotypisiert, um polygenetische Risikoscores (PRS) für PTBS zu berechnen. Hierarchische Regressionsmodelle prüften demografische, psychosoziale und traumabezogene Prädiktoren. Anschließend wurden lineare Regressionsmodelle sowie gemischte Modelle zur Untersuchung der Haupteffekte der PRS und der $PRS \times$ Trauma-Interaktionen durchgeführt, jeweils unter Kontrolle von Alter, Geschlecht, Bildung, Populationsstruktur und genetischer Verwandtschaft. Diese Studie trug zudem zur Übersetzung und kulturellen Adaptation mehrerer psychometrischer Instrumente (PHQ-15, PCL-5, HSCL-25 und PSS-14) ins Kurdische bei. Die interne Konsistenz wurde mittels Cronbachs Alpha und McDonalds Omega bewertet.

Ergebnisse: Demografische Variablen erklärten 10–38 % der Varianz in den psychischen Gesundheitsoutcomes. In der Chemiewaffen-Kohorte waren Traumabelastung ($\beta = 0,21\text{--}0,24$), niedrigere Bildung ($\beta = -0,17$ bis $-0,21$) und weibliches Geschlecht ($\beta = 0,03\text{--}0,35$) konsistent mit höheren Symptomwerten assoziiert. Auch die Einnahme psychotroper Medikamente war positiv mit Symptomen verbunden. In der jesidischen Kohorte waren Traumabelastung ($\beta = 0,19\text{--}0,37$), niedrigere Bildung und weibliches Geschlecht die wichtigsten Prädiktoren, während soziale Unterstützung und psychotrope Medikation über demografische und traumabezogene Faktoren hinaus nur einen geringen zusätzlichen Beitrag leisteten. In beiden Kohorten waren kumulative Traumabelastung und Bildung die stabilsten Prädiktoren für PTBS, Depression/Angst, somatische Symptome und wahrgenommenen Stress. In der Chemiewaffen-Kohorte zeigten die PTBS-PRS einen kleinen, aber signifikanten Haupteffekt ($B = 1,76$, $p = .010$)

sowie eine signifikante Interaktion mit der Traumabelastung ($B = 1,44$, $p = .032$), was darauf hinweist, dass sich genetisches Risiko bei höherer kumulativer Traumabelastung stärker zeigt. In der jesidischen Kohorte zeigte sich kein signifikanter Zusammenhang zwischen PRS und Symptomen, und es bestand keine Interaktion mit der Traumabelastung.

Schlussfolgerungen: Die Ergebnisse legen nahe, dass der PTBS-PRS nur geringfügig mit der Symptomschwere assoziiert ist und mit der Traumabelastung bei Überlebenden chemischer Gasangriffe interagiert, jedoch nicht bei jesidischen Überlebenden. In beiden Kohorten traten kumulative Traumabelastung, weibliches Geschlecht und niedrigere Bildung konsistent als zentrale Prädiktoren hervor und unterstreichen die zentrale Rolle von Trauma und sozioökonomischem Kontext. In der chemischen- Gas- Kohorte zeigten sich stärkere Zusammenhänge mit psychotropen Medikamenten und bestimmten psychosozialen Faktoren, während soziale Unterstützung bei jesidischen Überlebenden eher schützend wirkte, was die Bedeutung kontextspezifischer Umwelt- und Strukturfaktoren unterstreicht. Insgesamt unterstreichen die Befunde die Notwendigkeit, Herkunft und Populationsstruktur in genomischen Vorhersagemodellen zu berücksichtigen, und unterstützen die Notwendigkeit kontextsensitiver Interventionen bei langfristig traumatisierten kurdischen Bevölkerungsgruppen.

Schlüsselwörter: Psychopathology, Depression, Psychometrics, War, Catastrophe, PTSD, Genetics, genomics, PRS, polygenic

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List of Abbreviations

Abbreviation	Full Form
AIC	Akaike Information Criterion
B	Unstandardized Regression Coefficient
β	Standardized Regression Coefficient
CRH	Corticotrophin- Releasing Hormone
DNA	Deoxyribonucleic Acid
DSM- 5- TR	Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision
GWAS	Genome- Wide Association Study
HPA	Hypothalamic- Pituitary- Adrenal
HSCL- 25	Hopkins Symptom Checklist- 25
ICC	Intraclass Correlation
LMM	Linear Mixed Model
MDD	Major Depressive Disorder

Abbreviation	Full Form
NE	Norepinephrine
OLS	Ordinary Least Squares
PCL- 5	PTSD Checklist for DSM- 5
PHQ- 15	Patient Health Questionnaire- 15
PSS- 14	Perceived Stress Scale- 14
PRS	Polygenic Risk Score
SAM	Sympathetic- Adrenal- Medullary
SD	Standard Deviation
SE	Standard Error
SNP	Single Nucleotide Polymorphism
SSD	Somatic Symptom Disorder
t	t statistic
ΔR^2	Change in Explained Variance
VIF	Variance Inflation Factor
vmPFC	Ventromedial Prefrontal Cortex
WHO	World Health Organization

Chapter 1: Background

The Anfal were operations by the Iraqi regime, the term refers to a series of brutal military offensives carried out against Kurdish opposition groups and civilians in the countryside areas of Iraqi Kurdistan. The Anfal operation took place in seven months, from February 23 to September 6, 1988 in eight stages (Kirmanj, 2020). Around 182,000 Kurd civilians were buried alive in mass graves and destroyed many Kurdish villages in just the 1980s alone. These operations stand in stark contrast to the Kurdish cultural history, which is characterized by resilience and a long struggle for autonomy, one of struggle and a push for recognition and autonomy (McDowall, 2004; Human Rights Watch, 1993). Around 70% of the victims were males ranging from 15 to 50 years old. Millions of civilians were displaced, and tens of thousands were forcibly transported to concentration camps. Thousands disappeared, and numerous villages were razed to the ground. The confiscation of Kurdish property and mass killings have been recurrent features of campaigns of violence against Kurdish populations since the 1960s, including the burning of villages, chemical warfare, ground attacks, and large-scale aerial bombardment (McDowall, 2004; Hiltermann, 2007; Salih, 2023).

Halabja massacre

In 1988, Halabja city experienced one of the most significant chemical attacks in the history of the Kurdish genocide (Human Rights Watch, 1993). Halabja has a population of about more than 170,000 and is considered an affluent area commercially, lying approximately 25 kilometers from the Iran and Iraq borders (Valadbigi & Ghobadi, 2010). Officially, around 5,000 of its people are estimated to have died in the attack, and thousands are still living with lifelong consequences (Human Rights Watch, 1993). An internal report of the unreleased findings by The Kurdistan Regional Government (KRG) Ministry of Martyrs and Anfal Affairs (MMAA) claims the presence of about 6,000 survivors with many problems affecting their health in Kurdistan, Iraq (Moradi et al., 2019).

The Iraqi regime was using a wide array of chemical warfare agents (CWA) for combat, among them nerve agents, cyanide, and mustard gas. Nerve agents have a high mortality rate, while sulfur mustard gas has a low mortality rate but a high morbidity rate (Moradi et al., 2019). Mustard and cyanide gas poisoning signs include dizziness, restlessness, respiratory irritation, nausea, headache, heartbeat irregularities, convulsions, blindness, and erosion of the cornea. The effects of nerve agents on the central nervous system and associated behavioral activation, such as agitation, easy fatigability, poor judgment, hallucinations, disorientation, and forgetfulness, are caused by nerve agents. They can last up to months or years following exposure (Gupta, 2009; Lenhart, 2008).

Despite the Halabja genocide, research on related psychological and physiological impacts regarding exposure to chemical gases has remained scant over the years. Still, some studies are now starting to show the far-reaching consequences of that heinous crime on survivors. Dworkin et al (2008) conducted a study to evaluate the post-traumatic symptoms in the victims of chemical gas in Halabja city, and they reported significantly higher levels of PTSD in the group of victims exposed to chemical gas as compared to the unexposed group. Moradi et al (2019) conducted a comprehensive study on the physical and psychological well-being of victims, highlighting a wide range of post-exposure debilitating symptoms expressed by the study population. These findings illustrate the urgency for additional research and the need for focused measures to address the multiple problems faced by victims of chemical attacks.

Yazidi Genocide

Kurdistan has been witnessing many wars since the attack with chemicals in Halabja; among these, the second and largest one in Kurdish history took place in Sinjar, and mainly populated by the Yazidi people (Sissakian & Al-Ansari, 2023). The Yazidi are Kurdish-speaking peoples who have lived for thousands of years in Turkey, Syria, Iraq, and the old Soviet Union. They also suffered the same fate as the Kurds when these regions were converted to Islam in the seventh century. The majority of the Kurds were coerced into becoming Muslims. The significant population of the Yazidis is found today in Northern Iraq. They are estimated to be members worldwide between 800,000 and 1,000,000 (Kizilhan, 2017). On August 3, 2014, ISIS (Islamic State in Iraq and al-Sham), called Da'esh, militants invaded the area. Within days, there were stories of men and boys being killed, women and children becoming victims of abductions and were sold into slavery and prostitution, raped and beaten, and forced to work, and boys were taken far from home to ISIS training camps (Cetorelli & Ashraph, 2019). Since the genocide, there have been numerous studies looking into the mental health of Yazidi survivors, with all the studies showing a high level of mental disorder. For instance, Kizilhan et al. (2020) reported staggering figures on the study of 194 Yazidi women diagnosed with PTSD and depression. Precisely, the study found a PTSD prevalence of 97.9% and a prevalence of depression of 88.1%, with the two disorders co-occurring in 86.6% of the individuals who participated. A sample of 348 was covered in the study of women caught in captivity who had been exposed to violence by Taha and Younan (2020). The study showed astounding rates of severe depression at 36.7% and generalized anxiety at 37.4%. Taken together, the studies underscore the profound psychological impact

Yazidi survivors have to face as part of the aftermath of genocide and the urgent need to put in place targeted interventions and mechanisms of support to address their mental health needs.

While the consequences of the Halabja chemical attack and the Yazidi genocide have been both psychological and physiological, but studies that simultaneously target the psychological and physiological aspects of trauma and genocide survivors in this population are scarce. This study addresses that by examining genetic risk (PRS), trauma predictors, and scale reliability simultaneously, revealing genocide's lasting multidisciplinary impacts on Kurdish survivors.

Cumulative Trauma Exposure

Survivors of chemical gas and the Yazidi genocide are often exposed to multiple traumatic events rather than a single traumatic incident (Hiltermann, 2007; Kizilhan et al., 2023). These traumatic experiences may include forced displacement, loss of family members, captivity, sexual violence, destruction of homes and communities, and long-term insecurity. For this reason, this study applies a cumulative trauma framework, which suggests that the total number of different traumatic events, often called “trauma load,” is a stronger predictor of chronic post-traumatic stress disorder (PTSD) than one single traumatic event (Kira et al., 2018; Sacchi et al., 2020).

Traditional trauma models often focus on one major traumatic event, but the cumulative trauma approach considers repeated exposure to different types of trauma over time. This is especially important in populations affected by war, persecution, and displacement, where poly-victimization is common (Kira et al., 2018). This framework is suitable for survivors of chemical attacks and genocide because their psychological distress is often related to repeated and prolonged traumatic experiences. In Chapter 3, hierarchical regression analysis is used to examine the effect of cumulative trauma exposure on PTSD symptom severity after controlling for demographic and psychosocial factors such as age, gender, social support, and socioeconomic status.

Significance and Rationale of the Study

Despite prolonged and widespread trauma exposure in Iraqi Kurdistan, research on mental health in this region remains limited. To our knowledge, no study has examined PTSD polygenic risk scores in Kurdish populations, leaving a critical gap in understanding genetic contributions to PTSD. Moreover, most prior studies in Kurdish and similar trauma-exposed populations have adopted a PTSD-centric framework, often neglecting comorbid conditions such as depression, anxiety, chronic stress, and somatic symptoms. This narrow focus has limited insight into the multidimensional impact of trauma across symptom domains.

The present study addresses these gaps by integrating genetic, psychosocial, and trauma exposure data from 892 participants across two distinct Kurdish cohorts: survivors of the chemical attacks ($n = 534$) and Yazidi genocide survivors ($n = 358$). This represents the largest genetic investigation of mental health in Kurdish populations to date. PTSD polygenic risk scores were analyzed using linear mixed models to account for familial and genomic relatedness. Phenotypic analyses of depression, anxiety, somatic symptoms, and perceived stress were conducted using hierarchical regression, and sensitivity analyses employing linear mixed models confirmed the robustness of the findings. This approach allows for accurate estimation of associations between genetic and environmental predictors while appropriately accounting for relatedness among the analyzed individuals.

This research extends previous studies by examining mental health outcomes beyond PTSD alone and by considering the broader range of trauma-related psychological symptoms. It also explores the combined role of genetic vulnerability, cumulative trauma exposure, and psychosocial factors in shaping mental health outcomes. By comparing two trauma-exposed Kurdish cohorts, the study aims to identify both shared and cohort-specific patterns of psychological distress. The findings may contribute to a better understanding of trauma-related psychopathology in post-conflict populations and may provide evidence relevant for future research and culturally appropriate mental health interventions.

Chapter 2 Literature Review

PTSD, Depression, and Somatic Symptoms in Trauma Survivors

This chapter reviews stress, PTSD, depression, and somatic symptoms in trauma-exposed populations, with a focus on their prevalence and etiology. Psychological, neurobiological, and genetic factors are discussed, with particular attention to how these processes interact to influence individual vulnerability and resilience. The aim is to provide a comprehensive and integrated framework for understanding mental and somatic health among survivors of prolonged trauma, including Kurdish populations affected by chemical attacks and genocide.

Stress and its Impact on Mental Health

Stress refers to the physiological and psychological consequences of events that pose a significant challenge to homeostasis (Selye, 1976). The presence of an actual or perceived threat, known as a stressor, activates a coordinated stress response (Chrousos, 2009). While acute stress can be adaptive by facilitating rapid responses to danger, prolonged or chronic stress is associated with a wide range of adverse psychological and physical outcomes. These include disturbances across multiple biological systems, particularly the cardiovascular, immune, gastrointestinal, and reproductive systems, and increased risk of conditions such as hypertension, coronary heart disease, stroke, diabetes, and obesity (Schneiderman, Ironson, & Siegel, 2005).

Chronic stress exerts particularly adverse effects on immune functioning, leading to increased susceptibility to infections and delayed recovery from illness (Cohen, Janicki-Deverts, & Miller, 2007). Sustained activation of the sympathetic nervous system results in persistently elevated blood pressure and vascular inflammation, thereby contributing to the development of cardiovascular disease (Chrousos & Gold, 1992).

Responses to stressors vary between individuals. Difficult temperament and heightened physiological reactivity (Caspi et al., 2002; Ellis et al., 2011), may increase sensitivity to environmental stressors, producing prolonged stress responses that heighten depression risk (Belsky & Pluess, 2009).

Physiologically, stress activates two major systems: the sympathetic-adrenal-medullary (SAM) system and the hypothalamic-pituitary-adrenal (HPA) axis (McEwen, 2007; O'Connor et al., 2021). Threat perception engages the autonomic nervous system through the amygdala and hypothalamus, resulting in the release of noradrenaline and adrenaline from the adrenal medulla, which produces the classic fight-or-flight response characterized by increased heart rate, rapid respiration, pupil dilation, suppression of digestive activity, and redistribution of blood flow toward skeletal muscles (McEwen, 2007). Concurrently, stress exposure stimulates corticotropin-releasing factor (CRF) release, triggering adrenocorticotrophic hormone (ACTH) secretion and subsequent cortisol release from the adrenal cortex. Cortisol facilitates energy mobilization by promoting glucose availability from hepatic and muscular stores, supporting cognitive and physical stress demands (O'Connor et al., 2021).

These hormonal and autonomic changes also have important cardiovascular implications. Stress-induced alterations in coronary vasoreactivity, platelet activation, and endothelial integrity may precipitate myocardial ischemia, arrhythmias, and infarction, particularly among individuals with underlying atherosclerosis. Persistent sympathetic activation further contributes to vascular inflammation and elevated blood pressure, accelerating the progression of cardiovascular disease (Vancheri et al., 2022).

Although cortisol and adrenaline are essential for survival during acute threats, their prolonged elevation is associated with substantial long-term health consequences (McEwen, 2007). Chronic stress has been linked to gastrointestinal mucosal damage and peptic ulcers (Yehuda, 2002), reduced bone density predisposing to osteoporosis, dysregulation of reproductive functioning, and elevated risk of metabolic, respiratory, and cardiovascular disorders (McEwen, 2007). Immune suppression associated with chronic stress increases vulnerability to infections and malignancies (Dhabhar, 2014). Neurobiologically, sustained stress exposure leads to structural and functional brain changes, including hippocampal atrophy, particularly within the CA3 region observed in individuals with trauma exposure and PTSD, medial prefrontal cortex impairments, and heightened amygdala activity due to diminished inhibitory control (Bremner, 2005).

Different stressors elicit distinct psychological and biological responses, and individual coping capacity plays a critical role in shaping outcomes. When active coping strategies are unavailable, individuals may adopt a vigilance response, characterized by behavioral inhibition and altered peripheral blood flow. Initially, stress-related immune changes may be adaptive; however, prolonged neuroendocrine activation disrupts immune regulation (Dhabhar, 2014). Immune cells including natural killer cells, B cells, and T cells possess adrenergic receptors, rendering them highly sensitive to HPA-axis activity (Padgett & Glaser, 2003). Consequently, chronic stress suppresses immune functioning and increases susceptibility to infectious and neoplastic diseases (Segerstrom & Miller, 2006; Allen et al., 2014).

Exposure to severe stressors during critical developmental periods, such as childhood maltreatment or sexual abuse, has profound and lasting consequences. Such experiences are associated with attachment disturbances, affect dysregulation, maladaptive interpersonal behaviors, altered neurodevelopment, and increased risk of personality disorders (Cicchetti & Toth, 2005). High rates of PTSD, anxiety and depression have been documented among war-exposed children, and adults. (Schneiderman, Ironson, & Siegel, 2005; McEwen & Gianaros, 2011).

Post-Traumatic Stress Disorder (PTSD)

The main features of PTSD comprise symptom clusters after being exposed to traumatic events e.g., exposure to life threatening events, serious injury and/or sexual abuse. However, according to the revised fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5-TR), the symptoms are classified into four essential categories: (intrusive symptoms, avoidance, Negative alterations in cognitions and mood associated with the traumatic event and marked alterations in arousal and reactivity associated with the traumatic event) (American Psychiatric Association, 2022).

Prevalence, Risk Factors, and Psychological Correlates

PTSD prevalence is high among Yazidi survivors and correlated with several demographics. Women have a higher probability to develop PTSD and other psychological disorder after exposure to traumatic event compared to men. Younger age, being unmarried, being less educated, having lower income, and being unemployed are associated with increased risk of PTSD (Table 1) (Koenen et al., 2017; Mahmood et al. 2019; Richa et al., 2020; Al Shawi & Hassen, 2022; Taha et al., 2021; Rofo et al., 2023). PTSD exhibits high comorbidity with depression (34%), other anxiety disorders (53%), and alcohol-related disorders (7%). Comorbid anxiety disorders preceded PTSD onset in 90% of cases (Longo et al., 2020). This comorbidity might be because of a common risk factors or common vulnerability. The vulnerability could be attributed to biological or psychosocial factor, like genetic, cognitive and behavioral factors or lack of skills like social skills. Lastly, the diagnostic criteria used to identify and distinguish between these two disorders may not be precise enough. As a result, individuals who actually have one disorder may

end up being diagnosed with both disorders due to overlapping symptoms or criteria (Radell, Abo Hamza and Moustafa, 2020).

The meta-analysis conducted by Lim et al. (2022) in conflict settings supports these findings. They investigated the prevalence of depression, anxiety, and post-traumatic stress among civilian and military populations in war- and conflict-afflicted areas. They included 70 studies published between 1982 and 2021, reporting on a total of 80,130 participants for depression, 36,948 participants for anxiety, and 67,153 participants for post-traumatic stress. The aggregate prevalence of depression was 28.9%, with a higher prevalence among civilians (33.3%) compared to military personnel (24.0%). Anxiety prevalence was 30.7%, with civilians exhibiting a higher prevalence (38.6%) than military personnel (16.2%). Post-traumatic stress prevalence was 23.5%, with no significant difference between civilians (25.7%) and military personnel (21.3%). Subgroup analyses revealed higher prevalence rates during wars compared to post-war periods and significant differences between civilian and military populations.

Table 1 shows prevalence of PTSD across different populations and probable risk factors.

Study	Population	PTSD Prevalence	Key Risk Factors
World Mental Health	Global	3.9%	Female sex, low income
Mahmood et al. (2019)	Syrian Kurdish refugees in Kurdistan Region of Iraq (KRI)	60%	Female sex, time in camp, trauma count
Richa et al. (2020)	Yazidi, Christian, Muslim refugees	48.7%	Ethno-religious identity (Yazidis: 70%, Muslims: 44%, Christians: 32%)
Al Shawi & Hassen (2022)	Yazidi IDPs in Chammshko camp	79%	Female sex, non-workers, widowed
Taha et al. (2021)	Yazidi IDPs in KRI camps	34%	High trauma exposure, widowhood, unmet needs
Rofo et al. (2023)	Internally displaced Christians in KRI	20.3%	Low economic status, trauma count, secondary displacement

While these studies demonstrate high PTSD prevalence among conflict-affected Kurdish populations, most rely on cross-sectional self-report assessments and do not examine biological or genetic vulnerability (Mahmood et al. 2019; Richa et al., 2020; Al Shawi & Hassen, 2022; Taha et al., 2021; Rofo et al., 2023; Kizilhan et al., 2019). Moreover, few investigations integrate PTSD with broader symptom domains such as somatic distress or perceived stress, limiting understanding of persistent psychiatric burden mechanisms in these populations.

In general, risk factors for PTSD can be divided into three domains: preexisting, event-related, and post-event factors. Preexisting factors include genetic vulnerability, gender (greater prevalence among women) and prior psychopathology or cumulative trauma history. Event-related factors such as trauma severity, perceived life threat, and peritraumatic dissociation also increase PTSD risk (Koenen et al., 2008; Kizilhan, Friedl, & Steger, 2019; Brewin et al., 2000; Koenen et al., 2017). Post-event factors like social support play a protective role, with poor support linked to greater symptom persistence. Psychological models highlight negative cognitive appraisals following trauma as maintaining symptoms (Ehlers & Clark, 2000). Environmental and contextual influences including chronic or repeated exposure to interpersonal violence further contribute to vulnerability (Hu, 2024)

Genetic Influences on PTSD

Not all individuals exposed to trauma develop PTSD. Epidemiological surveys indicate that although most adults experience at least one potentially traumatic event during their lifetime, only a minority meet diagnostic criteria for PTSD (Kilpatrick et al., 2013; Kessler et al., 2017). This variability in response to trauma has led to sustained investigation of factors that may confer vulnerability or resilience, including genetic influences.

Twin studies consistently demonstrate that PTSD is moderately heritable. In male vietnam era veterans, approximately half of the variance in PTSD symptoms was attributable to additive genetic factors after accounting for combat exposure (True et al., 1993; Wolf et al., 2017). Across studies, heritability estimates typically range from 30% to 50%, with some samples showing higher estimates when broader traumatic stress phenotypes are examined (Koenen et al., 2008; Sartor et al., 2012; Wolf et al., 2017). More recent findings from a large, nationally representative twin cohort suggest that heritability may be somewhat higher in women than in men, consistent with a quantitative gender difference in genetic liability (Bevilacqua et al., 2024). These findings indicate that genetic factors account for a substantial proportion of individual differences in PTSD risk, even when trauma exposure is considered.

At the same time, environmental and demographic factors remain central determinants of PTSD. Women develop PTSD at approximately twice the rate of men, even after accounting for differences in trauma exposure, and individuals from

minority or marginalized groups often face elevated risk, partly due to greater cumulative exposure to violence and chronic stress (Kessler et al., 2017; Kilpatrick et al., 2013). The type and timing of trauma are also important. Interpersonal traumas, including rape, sexual assault, and severe physical violence, are associated with a higher likelihood of developing PTSD compared with many other forms of trauma. Childhood adversity, particularly when severe or prolonged, is also a strong predictor of later PTSD (Koenen et al., 2017; Sareen et al., 2007). Genetic influences therefore operate within environmental contexts rather than independently of them.

At the molecular level, FKBP5 has been one of the most extensively studied genes in PTSD research. FKBP5 encodes a co-chaperone protein involved in regulating glucocorticoid receptor sensitivity and hypothalamic–pituitary–adrenal (HPA) axis function (Zannas & Binder, 2014). Variants in FKBP5 do not determine PTSD directly but appear to influence biological responses to stress. E.g., a meta-analysis of gene-environment interaction studies by Hawn et al. (2019) examined four common FKBP5 single nucleotide polymorphisms (rs1360780, rs3800373, rs9296158, rs9470080) and reported significant interactions between these variants and traumatic life events in predicting PTSD, including a gene-level FKBP5 effect. These findings provide support for earlier reports suggesting that FKBP5 risk alleles, when combined with childhood maltreatment or severe trauma exposure, are associated with increased PTSD symptoms and altered glucocorticoid feedback regulation (Klengel et al., 2013; Zannas & Binder, 2014). However, as with many candidate gene findings, effect sizes are modest and replication has not always been consistent.

More broadly, research on the genetics of PTSD has progressed from candidate gene approaches to genome-wide association studies (GWAS). Early candidate gene studies focused on biologically plausible systems, including dopaminergic (e.g., DRD2), serotonergic (e.g., SLC6A4), and HPA-axis pathways, and reported associations between selected variants and PTSD outcomes (Koenen et al., 2017; Sheerin et al., 2017). Many early genetic findings could not be replicated in larger samples, partly due to low statistical power (Banerjee, Morrison, & Ressler, 2017). In contrast, recent large-scale GWAS have used hypothesis-free approaches, scanning the entire genome to identify variants associated with PTSD without focusing on a priori specified genes, identifying multiple loci of small effect and demonstrating that PTSD has a highly polygenic architecture (Nievergelt et al., 2019; Stein et al., 2021). These studies estimate SNP-based heritability in the range of approximately 5-20%, varying by gender and ancestry. They also indicate substantial genetic overlap between PTSD and other internalizing and externalizing disorders, while suggesting that some loci may be relatively specific to PTSD (Koenen et al., 2017; Nievergelt et al., 2019; Stein et al., 2021). Overall, this body of work supports a model in which PTSD reflects the combined influence of trauma exposure and numerous common genetic variants with individually small effects.

Building on GWAS findings, polygenic risk scores (PRS) have been developed to summarize genome-wide genetic liability for PTSD. These scores aggregate the effects of thousands of common variants into a single quantitative index. In independent European-ancestry samples, PTSD PRS explain approximately 2-5% of variance in outcomes (Nievergelt et al., 2019). Although modest, these effects are consistent with expectations for complex psychiatric traits and provide a scalable way to assess genetic risk. In the Veteran Program (N = 146,660), higher PTSD PRS predicted diagnostic status and symptom severity, even after adjusting for trauma exposure, highlighting their relevance for understanding differences in symptom levels across individuals (Nievergelt et al., 2021; Waszczuk et al., 2020; Misganaw et al., 2023). Emerging gene–environment research further suggests that polygenic liability may moderate the impact of adverse experiences. For example, PRS interacted with childhood neglect to predict elevated PTSD risk in civilian women (Lee et al., 2024), and among 9/11 responders, PRS was associated with long-term symptom trajectories beyond baseline severity and trauma exposure (Ressler et al., 2025).

Despite these advances, important limitations remain. Most GWAS and PRS for PTSD have been derived predominantly from European-ancestry cohorts, often enriched for military samples. Predictive performance declines when these scores are applied to ancestrally diverse populations, partly due to differences in linkage disequilibrium structure and allele frequencies (Mostoufi-Rad et al., 2021). A South African cohort study, for example, reported minimal predictive utility of European-derived PTSD PRS ($R^2 < 1\%$) (Swart et al., 2021). These findings raise concerns about the generalizability of current polygenic models.

In addition, little is known about how polygenic liability operates in the context of extreme, adult-onset mass trauma in non-Western settings. To date, no study has examined PTSD polygenic risk scores (PRS) in Kurdish trauma survivors, including survivors of chemical warfare or ISIS-related violence. Polygenic risk scores (PRS) derived primarily from European-ancestry genome-wide association studies may not fully capture genetic vulnerability in such contexts, and their interaction with severe, collective trauma exposure remains unclear. Addressing this gap is necessary for evaluating the cross-population applicability of existing genetic risk models and for advancing understanding of PTSD in underrepresented populations.

Neurobiology of PTSD

During threat exposure, humans and animals exhibit rapid survival responses. The locus coeruleus (LC) activates to modulate stress responses via connections to the amygdala, hippocampus, hypothalamus, and spinal cord, coordinating emotional, cognitive, and motor reactions (Southwick et al., 1999). LC activation triggers norepinephrine (NE) secretion, which regulates stress responses; repeated stress exposure can deplete NE, leading to reduced exploration, appetite loss, and impaired

learned skills resembling "learned helplessness" after uncontrollable stressors (Southwick et al., 1999; Vedantham et al., 2000).

Beyond NE, PTSD involves dysregulation of serotonin, dopamine, GABA, CRH, and other neurotransmitters. Elevated dopamine/NE correlates with hyperarousal and startle responses, while low serotonin disrupts amygdala-hippocampus connectivity, contributing to flashbacks and intrusive memories (Sherin & Nemeroff, 2011). Reduced GABA impairs stress regulation, and elevated CRH/NE drives hyperarousal and dissociation (Bremner, 2006).

Structural changes include reduced hippocampal volume, amygdala hyperactivity, anterior cingulate cortex (ACC) abnormalities, insula, and orbitofrontal cortex (OFC) alterations (Sakellariou & Stefanatou, 2017; Shin et al., 2006). These correlate with sleep disruption, nightmares, hypervigilance, memory deficits, and HPA axis dysregulation characteristic of PTSD (Ressler et al., 2022).

Depression

Definitions, Epidemiology, and Risk Factors

Depression is a common and chronic mental illness; the DSM-5-TR outlines criteria for diagnosing Major Depressive Disorder (MDD) based on specific symptoms persisting for at least 2 weeks, including feelings of depressed mood, loss of interest, changes in appetite and sleep patterns, low self-esteem, guilt, and suicidal thoughts (American Psychiatric Association, 2022).

Dysthymic Disorder, characterized by chronic symptoms lasting at least 2 years but less severe than MDD, is also described. The DSM-5 maintains these diagnostic criteria, renaming dysthymic disorder as Chronic Depressive Disorder (CDD).

In contrast, the World Health Organization's defines depressive episodes with similar symptom criteria but a lower threshold for diagnosis compared to the DSM-IV-TR. Despite some differences, there's significant agreement between these classification systems for depressive disorders (Richards, 2011).

In the DSM-V-TR depressive disorders including disruptive mood dysregulation disorder, major depressive disorder (including major depressive episode), persistent depressive disorder, premenstrual dysphoric disorder, substance/medication-induced depressive disorder, depressive disorder due to another medical condition, other specified depressive disorder, and unspecified depressive disorder. Feeling sadness, irritable or empty mood are the core feature of all types of depression. Here we focus on major depressive disorder (APA, 2022).

For diagnosis of major depressive disorder, patients must fulfill five or more of these symptoms for at least two weeks, and at least one of the symptoms should be 1) depressed mood or 2) loss of interest or pleasure. 1) depressed mood; the individual has this symptom almost every day, and most of the days. Either he/she reports it or is

observed by others. 2) Lack of interest or pleasure: marked diminishing of interest in almost all activities in almost all days. 3) Weight lost or gaining 5% of body weight in a month. 4) Insomnia or hypersomnia nearly every day. 5) Psychomotor agitation or retardation, almost every day. 6) Fatigue or diminished of energy. 7) Feeling guilt or worthlessness almost every day. 8) lack of concentration and problem with making decisions almost every day, 9) Recurrent thoughts of death, suicidal ideation, plans, or attempts. These symptoms result impairment in social, occupational or other important area. and these symptoms must not happen due to physiological effect of substance or any medical condition (APA, 2022).

Several large-scale meta-analyses and systematic reviews have examined the global prevalence of depression across different populations. Shorey et al. (2022) reviewed studies from 2001 to 2020 and found that the worldwide point prevalence of depression among adolescents was 34%. They also reported an increase in depressive symptoms over time, from 24% in 2001-2010 to 37% in 2011–2020. The highest prevalence was observed in the Middle East, Africa, and Asia.

Hu et al. (2022) conducted a systematic review of 48 studies published between 2000 and 2021, focusing on older adults (60 years and above). They found that the pooled global prevalence of depression in this age group was 28.4%. Most of the studies were conducted in Asia and used self-report screening tools, with the Geriatric Depression Scale (GDS-15) being the most common. Prevalence estimates varied by screening tool (15.6% for DASS-21 to 31.5% for GDS-15) and by continent, with Africa showing the highest prevalence (41.1%) and Europe the lowest (21.1%).

Another meta-analysis by Lim et al. (2018), which included 90 studies from 1994 to 2014 and more than 1.1 million adults, reported an aggregate point prevalence of 12.9%, a one-year prevalence of 7.2%, and a lifetime prevalence of 10.8%. Prevalence was higher among women (14.4%) and in countries with medium Human Development Index scores (29.2%).

These reviews highlight considerable variation in depression prevalence across age groups, regions, and populations, as well as differences related to study design, sample characteristics, and measurement tools.

Reports from world health organization WHO estimate depression to be the greatest burden in the world by 2030. Depression runs in families, having first degree with depression increase risk of depression with an odds ratio of 2.84. Additionally, twins studies suggest the heritability of depression as 37% (Sullivan et al., 2000). And in a meta-analysis study which 807553 people were recruited, 102 loci have been determined which were associated with the depression (Howard et al, 2019).

Although there is strong evidence that depression partially develops due to genetic factors, there are other factors that also result in the development of depression, for example, environmental factors like stressful life events have role in developing depression (Nestler et al., 2002) and stressful life event results activation of HPA axis.

Approximately 70% of individuals with depression exhibit HPA axis dysregulation. Brain imaging reveals diminished grey matter in the hippocampus, reduced neural activity in the medial prefrontal cortex, and hippocampal neuronal atrophy. These changes are accompanied by elevated cortisol levels (damaging hippocampal neurons), increased glutamate, and decreased serotonin (Yang et al., 2015).

Psychological and Environmental Contributors

A behavioral theory of depression suggests that depression develops due to a lack of pleasant activities and positive reinforcements. Lack of reinforcement can happen as a result of losing a loved one, losing a job, a breakup, relocating to a new environment, or aging. Also, lack of skills, especially social skills, is crucial in developing depression. Lack of skills results in a lack of positive reinforcement, and this leads to depression symptoms (Kanter, Busch, Cautilli, & Baruch, 2011).

Environmental factors have a key role in developing depression, stressful life events and traumatic events (such as divorce and break up, the death of a loved one, a job loss, or a serious medical illness and life-threatening events) can result in a depressive episode. Around 80% of individuals with depression report a stressful event before onset. Additionally, the meaning and interpretation of an event are also important (National Research Council & Institute of Medicine, 2009), dysfunctional cognitive patterns such as negative self-concept (e.g., worthlessness and uselessness), rumination, negative interpretation, attentional bias and neuroticism are associated with depression. Additionally, maladaptive coping strategies, negative body image, maladaptive perfectionism, low self-esteem, shame, or worthlessness are make individuals more susceptible to depressive episodes (Remes, Mendes, & Templeton, 2021).

Dysfunctional attitudes, such as negative and rigid beliefs about oneself, the world and the future and negative core beliefs, assumptions, or schemas interact with stressful life events (Beck, 1976). This interaction gives rise to negative thoughts, including generalization of the problems, minimization of success, jumping to conclusions, mind reading, black-and-white or absolutistic thinking, and labeling, and according to the cognitive model of depression, this leads to negative emotion; for instance, if someone thinks he is a loser, this may make him/her feel sad or helpless. Once the person feels depressed, then there will be a feedback process, which means negative mood results in further negative thoughts and reinforces core beliefs and schemas (Leahy, 2017). Additionally, avoidance behavior and isolation, which are depression symptoms, can result in even more negative events such as social rejection or losing a job (Dobson, 2008; Fu et al., 2021).

The social Origins of Depression model suggested by Brown & Harris (1978) explains how social factors contribute to depression development, lack of protective factors such as negative relationships, early loss, and lack of employment, and this leads to low self-esteem and increasing susceptibility to develop depression.

Additionally, Stressful life events can trigger grief and negative emotion, leading to depression, especially in those with low self-esteem and poor coping mechanisms. However, strong coping skills can help avoid depression.

Adverse Life Events and Trauma

A large body of evidence demonstrates that early adverse experiences have lasting negative effects on both mental health and physical health (Felitti et al., 1998; Norman et al., 2012; Hughes et al., 2017). Childhood adversity, including maltreatment, neglect, separation, or abuse, is linked not only to depression and anxiety but also to harmful behaviors such as alcohol and drug use, smoking, and to medical conditions like diabetes, cardiovascular disease, obesity, and cancer (Felitti et al., 1998; Hughes et al., 2017). Experiencing adverse events in childhood can triple the risk of developing depression in adulthood. Adults with depression who experienced early adversity are also more likely to have comorbid conditions, particularly anxiety and antisocial personality disorder (Nanni et al., 2012).

Severe childhood adversity is associated with more chronic and recurrent depressive episodes and a lower likelihood of responding to standard treatments (Nanni et al., 2012; Mandelli et al., 2015). It is also linked to psychotic and atypical depression and a higher risk of suicide attempts (Mandelli et al., 2015; Krause et al., 2021). Individuals with a history of childhood trauma often require hospitalization and intensive therapeutic interventions due to the severity and chronicity of their symptoms (Krause et al., 2021).

Genetic Influences on Depression

Twin, adoption, and population studies indicate that depression is moderately heritable, with genetic factors accounting for approximately 30-40% of the risk (Sullivan, Neale, & Kendler, 2000; Polderman et al., 2015). Genome-wide association studies (GWAS) have identified many single nucleotide polymorphisms (SNPs) associated with depression, although individual variants generally have small effects (Howard et al., 2019). Large meta-analyses, combining data from resources such as the UK Biobank and the Psychiatric Genomics Consortium (PGC), have identified dozens of risk loci (Howard et al., 2019; Wray et al., 2018). The largest GWAS to date, including over 800,000 participants, identified 102 genetic variants associated with depression (Howard et al., 2019; McIntyre et al., 2024).

Genetic vulnerability interacts with environmental factors. For example, polygenic risk scores for depression show stronger effects on MDD risk in individuals exposed to childhood trauma compared to those without trauma (Peyrot et al., 2014). Functional imaging studies show that these individuals often exhibit hyperactivity in the amygdala, a brain region involved in emotion regulation, which is also observed in depression (Sheline et al., 2001; Kempton et al., 2011). Other neuroimaging studies have identified structural and functional differences in individuals with depression,

including reduced hippocampal and caudate volumes, hyperactive amygdala, and hypoactive prefrontal cortex (Kempton et al., 2011; Schmaal et al., 2016).

Overall, depression develops through a combination of genetic predisposition and environmental exposures. Dysregulation of neurotransmitter systems (serotonin, norepinephrine, dopamine) and stress-response systems, such as the HPA axis, likely contribute to the disorder (Saveanu & Nemeroff, 2012). Evidence from gene-environment studies suggests that **stressful early-life experiences can interact with genetic vulnerability to produce structural and functional brain changes, increasing the risk of depression (Mann et al., 2013; Peyrot et al., 2014)

Diathesis-Stress Models and Resilience

This model proposes that psychological disorders develop through interactions between predispositional factors and environmental stressors and integrates genetic, neurobiological, psychological, and environmental influences. This means that if an individual carries a high genetic load for depression that increases vulnerability, exposure to a stressor can substantially raise the likelihood of developing a disorder (Peyrot et al., 2014). For example, in depression, when the person has genetic predisposition to develop depression when he faces a stressor the interaction between genetic and environmental stressor precipitate depressive disorder (Kring & Johnson, 2012).

As mentioned before, genetic liability of depression ranges between 30-40%, therefore both environment and genetics are crucial for developing the disorder. Additionally, beyond gene-environment interaction, recent studies suggest gene-environment correlation, meaning that genetic predispositions may be associated with particular environmental exposures that individuals experience; in other words, certain genetic factors influence the likelihood of encountering specific environments. For example, genetic factors that predispose an individual to depression may create certain adverse life events, like engaging in risky behaviors or conflicts (Jaffee & Price, 2007; Feurer et al., 2022).

There are three types of gene - environment correlation.; 1) passive correlation: this can happen when parents provide a particular environment to their offspring, for instance , when parent both have predisposition in playing musical instruments at the same time they created environment that at the same time motivate children to play and lean instruments. 2) reactive or evocative: this happens when the persons genetic predisposition evoke specific response from other people who live around him. For example when the child is shy this evokes parents to be more protective. 3) active: this happens when the person search to find anything from environment that matches his predispositions, for example when a person has an athletic body, she or he chooses to join a sport team (Jaffee & Price, 2007; Rende & Plomin, 1992).

Within the diathesis-stress framework, resilience is a process which a person can actively adapt or overcome a stressful situation, or the individual is able to recover

rapidly from the consequences of the stressor. It is hard to fully define resilience because of diverse interpretation of the term, some scholars view the resilience as a personality trait, and some others believe it is a copying strategy (Snijders et al., 2018).

Studies of resilience suggest that individuals with higher resilience scores report lower levels of anxiety and depression after stress exposure and recover more quickly from symptoms of disorders such as depression, anxiety, and PTSD (S. Dhungana et al., 2022). Usually individual when face a traumatic event, they suffer from many symptoms of anxiety, depression or PTSD, but these symptoms go away after few weeks or month, therefore resilience is most common response after facing a stressor, but only few suffer from mental disorder after facing a stressor. One important aspect to understand the resilience is neuroimaging data, how brain regions work pre traumatic event, during traumatic event and after the traumatic event can be very helpful why some people show psychopathology symptoms after exposing to a stressor and others show resilience instead. For example, individuals with lower amygdala activity after showing them a threatening stimuli tend to show great resilience. Which in some cohort this investigated, for example young adults before military deployment with lower amygdala reactivity showed lower level of stress symptoms after exposure to stressful event, similarly, children before terrorist event, those who had lower amygdala reactivity prior to the attack they showed less PTSD symptoms following the exposure. Additionally, the vmPFC region which helps to adapt in stressful events and implements top-down control over some brain regions like amygdala which is responsible for fear and help to regulate in response to fearful situations, studies found grater activation of (vmPFC) before exposing to stressful event. Further, individual with greater grey matter size had fewer symptoms after exposure, and also greater volume of hippocampus was associated with fewer symptoms after exposure (Roeckner et al., 2021).

Southwick and Charney (2012) categorized ten resilience factors for dealing with stressors: 1) optimistic and realistic viewpoint. 2) Looking for social support. 3) Having a reliable person to depend on or having developed sense of what is right and what is wrong which can guide you what you should do in certain situation. 4) Participating in religious rituals. 5) Accepting anything from your life that cannot be changed. 6) Taking care of physical and mental health, like doing exercises. 7) having problem solving skill. 8) Having sense of humor. 9) Taking care of emotional well-being. 10) Utilizing the traumatic experience to improve one's skills and character. Studies postulated that resilience can be built during early life, for example, parents who treat their children in a good way and provide support for their children are important to dealing with stressful events in life. Children who have good relationships with their parents and have secure attachments are more resilient (Masten, 2001). On the other hand, overprotective parenting is associated with a lack of resilience development. Additionally, parent's well-being, self-efficacy, family

functioning, and social bonding are sign of parental resilience, which can be a positive factor in enhancing offspring resilience (Friedberg & Malefakis, 2018).

Studies have asserted that resilience is a protective factor against developing mental disorders and is crucial for adaptation. And people with lower level of resilience are more at risk of developing mental illness. However, childhood trauma especially, emotional abuse and neglect are associated with lower level of resilience and developing psychopathologies like depression and anxiety. (Park et al., 2023).

Somatic Symptoms and Somatic Symptom Disorder

Definitions and Diagnostic Features

Somatic symptoms are associated with significant impairment in psychological and physical functioning and in quality of life with this condition (D'Souza & Hooten, 2023). In addition to having somatic symptoms, according to DSM-5-TR in order to diagnose someone with a somatic symptoms, he/she require the presence of “excessive thoughts, feelings, behaviors related to the somatic symptoms or associated health concerns” which are “disproportionate and persistent,” with a high level of anxiety or with “excessive time and energy devoted to the symptoms” (Smakowski et al., 2024; APA, 2022) .

The findings from the systematic review and meta-analysis reveal that individuals diagnosed with somatic symptom disorder (SSD) demonstrate elevated levels of depression, anxiety, health anxiety, and alexithymia when compared to healthy counterparts (Smakowski, et al, 2024).

Epidemiology and Prevalence

The occurrence of SSD shows variability across different groups and healthcare environments (Lehmann et al., 2022; Löwe et al., 2021). In studies involving the general population, the estimated frequency of proxy diagnosis for SSD ranged from approximately 6.7% to 17.4%. Within non-specialized medical settings, rates varied from 3.5% to 45.5%, with the highest prevalence noted among individuals experiencing somatic symptom burden. In specialized medical fields like pulmonology or cardiology, SSD frequency was found to fluctuate between 5.8% and 52.9%. Remarkably, within mental health facilities tailored for SSD treatment, the frequency ranged from approximately 40.3% to 77.7%. However, most prevalence estimates are based on medical records or self-report, and there is a lack of studies using standardized diagnostic interviews. This limits the precision of current SSD prevalence estimates (Löwe et al., 2021).

Association with Trauma and PTSD

Many survivors of trauma report both PTSD and somatic symptoms, and these often occur together (Sharp & Harvey, 2001). Co-occurring somatic symptoms are linked with greater functional impairment and psychological distress (Asmundson, 2014).

Studies suggest that individuals with PTSD are more likely to report multiple somatic complaints compared to those without PTSD (Dehghani et al., 2018). This justifies PHQ-15 assessment in trauma-exposed populations.

Somatic Symptom Disorder (SSD), as classified in the DSM-5-TR under Somatic Symptom and Related Disorders, is defined by the presence of one or more somatic symptoms that cause significant distress or interfere with daily functioning. In addition to the physical symptoms themselves, individuals with SSD show excessive thoughts, feelings, or behaviors related to these symptoms. These can include a persistent preoccupation with the severity of the symptoms, heightened anxiety about health or symptom-related concerns, and spending considerable time and energy attending to symptoms or health worries. Although symptoms may fluctuate over time, the condition is considered persistent, typically lasting six months or longer.

SSD is further categorized into subtypes to capture variations in symptom presentation:

- With predominant pain: when pain is the primary symptom.
- Persistent: when symptoms are chronic, severe, and substantially impair daily functioning (American Psychiatric Association, 2022).

Among populations exposed to prolonged or severe trauma, such as survivors of mass violence or chemical attacks, somatic symptoms frequently reflect dysregulation across multiple physiological systems, including neuroendocrine, immune, and autonomic pathways (Bugge et al., 2017). These manifestations illustrate the close relationship between chronic stress, traumatic experiences, and physical health (McFarlane, 2010). This underscores the importance of assessing somatic symptomatology in highly trauma-exposed groups (Pace & Heim, 2011).

Etiology: Biopsychological and Genetic Factors

The etiology of somatic symptom disorder is complex and multifactorial, including biopsychological factors.

Genetic and Neurobiological Factors

Genetics play a role in developing somatic symptoms; twin studies estimate heritability at 30% for somatic distress, with 33% genetic variance specific to somatic distress independent of anxiety/depression (Gillespie et al., 2000). Abnormalities in brain networks including default mode/sensorimotor regions (Lee et al., 2022) and reduced GMV in vmPFC, insula, somatosensory cortex contribute to exaggerated bodily perception (Vermeulen et al., 2020). Serotonin/noradrenergic dysfunction contributes to somatic symptoms (Liu et al., 2019). Functional syndromes like IBS/CFS share genetic factors (Holliday et al., 2010).

Psychological and Anxiety-Related Traits

Individuals with somatic symptoms often interpret bodily sensations as signs of serious illness. This misinterpretation and catastrophizing lead to excessive worry and repeated medical consultations (Löwe et al., 2021). Additionally, people with somatic symptoms are excessively attentive to physical sensations. According to learning theory, attention and secondary gains from illness (e.g., sympathy or escape from responsibilities) can reinforce somatic complaints. Furthermore, certain personality patterns such as neuroticism and obsessive compulsiveness predispose individual to develop somatic symptoms (D'Souza & Hooten, 2023).

Negative life events like neglect, sexual abuse or disorganized family environment, history of childhood serious medical illness, unemployment, or bereavement predispose individual to develop somatic symptoms, additionally, some cultures expressing emotional distress through physical symptoms is more acceptable than having psychological symptoms and this can lead to somatic symptoms (Kurlansik & Maffei, 2016; D'Souza & Hooten, 2023; Fobian & Elliott, 2019).

These psychological processes overlap substantially with anxiety-related traits, particularly anxiety sensitivity (AS), defined as the fear of anxiety-related sensations and their perceived harmful consequences. In general, AS comprises three domains: physical concerns (fear that bodily symptoms reflect serious illness), psychological concerns (fear that anxiety sensations indicate mental instability), and social concerns (fear of negative social consequences of visible anxiety) (Lang, Kennedy, & Stein, 2002).

Additionally, trait anxiety, the tendency to perceive situations as threatening plays a role in the development of somatic symptoms.

People high in trait anxiety are more attentive to bodily arousal and changes and tend to amplify these sensations, leading them to report somatic complaints more often and to interpret pain as the onset of serious illness (Hensley & Varela, 2008).

Study Objectives

Overall Aim

This study examines how genetic risk (PTSD PRS), cumulative trauma exposure, and sociodemographic factors relate to PTSD and other mental health symptoms among Kurdish survivors of mass trauma.

Specific Aims

1. Genetic analysis

To assess whether PTSD polygenic risk scores (PRS) predict PTSD symptom severity, whether this association is moderated by cumulative trauma exposure, and whether

these effects remain after controlling for demographic covariates and familial relatedness.

2. Associations with Mental Health Outcomes

To examine the associations and relative contributions of sociodemographic, psychosocial, and trauma-related factors to PTSD, depression/anxiety, somatic symptoms, and perceived stress across Kurdish trauma survivor cohorts.

3. Hierarchical prediction

To examine whether cumulative trauma exposure explains additional variance in mental health symptoms beyond demographic and psychosocial factors after accounting for prior psychiatric diagnosis, using sequential regression models.

4. Cohort comparison

To evaluate whether the associations between trauma exposure, PTSD polygenic risk scores, and their interaction with PTSD symptoms differ between survivors of the chemical attacks and Yazidi genocide survivors, and whether cohort-specific sociodemographic and contextual factors are related to these differences.

Chapter 3: Methods

Ethical Review and Approval

The study protocol was reviewed and approved by the Ethics Committees of the University of Duhok and the University of Halabja. All procedures were conducted in accordance with the ethical standards of the responsible institutional research committees and the principles of the Declaration of Helsinki.

Ethical approval was obtained from the Ethics Committee of the University of Duhok on 3 October 2021 (Approval No. 141) and from the University of Halabja (Approval No. 01/2024/1). Additional administrative approval was obtained from the Halabja Governorate in April 2022 (Reference No. 10/158) and the Duhok Governorate in June 2022 (Reference No. 1024), including approval from the General Directorates of Security and Police. Such approvals are mandatory in the Kurdistan Region before conducting research involving survivors of conflict and displacement, in order to ensure participant safety and facilitate secure access to the study population.

Permission was granted to contact survivors attending the Halabja Hospital for Treatment of Chemical Victims and individuals residing in several refugee camps. All participants were fully informed about the aims and procedures of the study and were recruited on a voluntary basis. Participants who declined participation faced no negative consequences. Confidentiality was assured for all participants, and written informed consent was obtained before data collection. Due to the high rate of illiteracy among some participants, consent was documented using a thumbprint in the presence of a witness.

Participant Recruitment Process

Participants were recruited through a structured, multistage process in collaboration with local institutions and humanitarian organizations serving survivors of chemical weapons attacks and survivors of ISIS-related atrocities residing in displacement camps in Duhok city. These collaborations facilitated access to eligible participants and helped ensure engagement with the community throughout the recruitment period. For the chemical gas survivors cohort, recruitment was coordinated with the Halabja Hospital for Treating Chemical Victims and the Society of Victims of Chemical Weapons. For the Yazidi survivors cohort, administrative authorities and service providers in Sharia, Khanke, Qadia, Bersive, and Chamshko camps in Duhok city assisted in identifying potential participants. Institutional approval was obtained prior to accessing participant records or personal information. Potential participants were initially contacted by telephone using contact details provided by collaborating organizations. During these calls, trained research staff explained the aims and procedures of the study, clarified that participation was voluntary, and explained the steps taken to protect confidentiality, such as pseudonymizing participant data by replacing names with unique ID codes, storing coded data files on password-protected computers accessible only to the research team, securely locking original paper scales and interviews in restricted-access cabinets, and keeping a separate password-

protected list matching codes to real names in a different secure location. Individuals who expressed interest were invited to take part in an in-person meeting at a location of their choice, most commonly their homes. At the in-person visit, the study procedures were explained in detail and any questions were addressed. Written informed consent was obtained before data collection commenced, and participants were informed of their right to withdraw from the study at any time without consequences. To support recruitment beyond individuals initially identified through institutional records, enrolled participants were asked whether they were willing to inform other eligible survivors within their social networks about the study. This referral process allowed inclusion of additional participants who may not have been reachable through formal records and helped increase the demographic and socioeconomic diversity within the sample.

Sampling method

A combination of convenience sampling and snowball sampling was employed in light of the vulnerability of the study population and the ethical and practical constraints inherent in recruiting survivors of chemical weapons exposure and ISIS-related violence. Under these conditions, probability-based sampling was not feasible. Convenience sampling enabled recruitment of individuals who were accessible through partner organizations and willing to participate. Snowball sampling was subsequently used to extend recruitment by allowing enrolled participants to refer other eligible survivors. This approach increased the overall sample size and supported inclusion across a broad range of demographic and socioeconomic backgrounds.

Sample Size and Recruitment

For the chemical gas survivors cohort, lists of identified survivors were obtained from the Society of Victims of Chemical Weapons and clinical records at Halabja Hospital for Treating Chemical Victims. Recruitment was limited to individuals with valid contact information. Of the 679 individuals contacted, 145 were unable to participate, including 102 who had relocated or were living in areas that were difficult to access because of distance and logistical barriers, 28 with unreachable telephone numbers, and 15 who refused participation after explanation of the study.

Participants were recruited through multiple pathways to ensure representation across different levels of formal recognition. The largest group (n = 353) was drawn from official registries maintained by the Kurdistan Regional Government's Ministry of Martyrs and Anfal Affairs. These individuals had undergone formal evaluation for survivor status and were eligible for financial compensation. Additional participants who had completed formal government evaluations but were not formally registered were recruited through non-governmental organizations (NGOs) providing medical and psychosocial services (n = 102) and through snowball sampling (n = 79). Survivor status for these participants was confirmed using documented exposure histories, clinical assessments available in NGO records, supporting organizational

documentation, and structured trauma exposure questionnaires administered during interviews. Non-participation is a common challenge when studying vulnerable or hard-to-reach populations. In this study, 21% of contacted individuals did not participate. This proportion is consistent with previous reports, where non-response rates in similar populations ranged from 20% to 40% (East Tracy, 2007; Martikainen et al., 2007). Such rates are considered acceptable and reflect the practical difficulties of reaching survivors who were dispersed across different locations.

For the Yazidi survivor cohort, recruitment focused on adults aged 18 years or older who had survived the 2014 ISIS genocide in Shingal and were residing in internally displaced persons (IDP) camps in Duhok Governorate at the time of the study. Eligible participants had directly witnessed attacks or experienced captivity, displacement, loss of relatives, or other war-related trauma. Eligibility was confirmed through phone verification of residence in Shingal during the attacks and subsequent displacement. A total of 358 participants were successfully recruited from IDP camps, including Sharia, Khanke, Qadia, Bersive, and Chamshko (Figure 1).

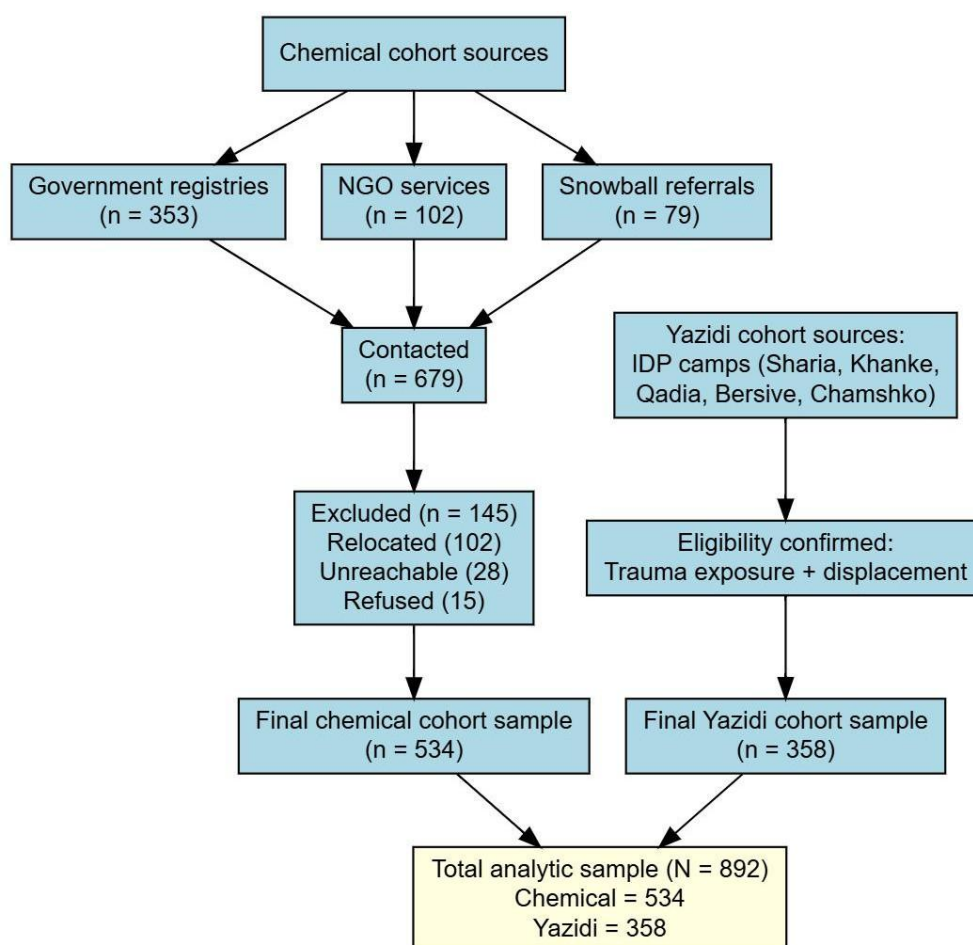


Figure 1. The figure presents the recruitment pathways for both cohorts. Chemical survivors were identified through government registries, NGOs, and snowball referrals, followed by

screening and exclusion procedures. Yazidi survivors were recruited from IDP camps following eligibility verification based on trauma exposure and displacement history. The final analytic sample comprised N = 892 participants (chemical cohort n = 534; Yazidi cohort n = 358).

Inclusion and Exclusion Criteria

For both cohorts, inclusion criteria were confirmed exposure to the relevant traumatic events, age ≥ 18 years, provision of informed consent, and sufficient physical and mental capacity to complete all assessments. Exclusion criteria included: (1) severe physical illness preventing communication or completion of assessments (e.g., advanced respiratory failure due to chemical exposure or late-stage cancer), and (2) severe cognitive impairment or dementia preventing questionnaire completion.

Data Collection Procedures

Data collection for the chemical gas survivors cohort was conducted in two phases between March and September 2023. Phase 1 (March-April) included 234 participants (recorded as Kurd1 in the genomic dataset), and Phase 2 (July-September) included 300 participants (recorded as Kurd2 in the genomic dataset).

For the Yazidi cohort, 358 participants were interviewed between May and September 2024 (recorded as Kurd3 in the genomic dataset). Interviews were conducted in the Kurmanji dialect using instruments that were translated, back-translated, and pretested prior to field administration. Because many participants had limited literacy, trained interviewers administered the questionnaires and recorded responses.

Participation was voluntary across both cohorts, and no financial or material compensation was provided. Participants were fully informed about the study objectives, procedures, and potential risks. Each assessment required approximately 60-90 minutes. Confidentiality and the right to withdraw at any time were emphasized. Participants who showed signs of distress were referred to local NGOs for psychological support, with follow-up coordinated through relevant authorities.

Formal diagnostic interviews were not conducted; instead, data collection relied on standardized symptom screening instruments, including the PCL-5, HSCL-25, PHQ-15, PSS-14, and trauma exposure checklists.

Biomaterial Collection

Following completion of the interviews, saliva samples were collected for genetic analyses using Oragene DNA collection kits. Participants were instructed to refrain from eating, drinking, or using tobacco products for at least 30 minutes prior to sample collection to ensure sample integrity. All samples were labeled using pseudonymized codes and stored and transported in accordance with manufacturer and laboratory protocols.

Quality Assurance and Interviewer Training

All interviews were conducted by trained psychologists who completed a one-week structured training program covering study protocols, ethical procedures, standardized administration of assessment instruments, and management of participant distress. Data collection was supervised through regular field monitoring and spot checks to support adherence to study procedures. After transferring the data to an electronic format, data were screened using statistical software to ensure that values fell within valid ranges, to identify potential outliers, and to assess consistency across variables. Interviewer self-care and support were provided throughout the study period.

Assessment Instruments

PTSD Checklist for DSM-5 (PCL-5)

The PCL-5 is a 20-item widely recognized, and extensively validated instrument used to assess symptoms of PTSD over the past month. Items are rated on a 5-point Likert scale (0 = not at all to 4 = extremely). It has demonstrated acceptable internal consistency, test-retest reliability, and convergent validity across diverse populations, including survivors of trauma and conflict (Weathers et al., 2013; Forkus et al., 2022). Total symptom severity scores are calculated by summing item responses, with a cutoff score ≥ 23 indicating probable PTSD (Ibrahim et al., 2018). Given the participants' exposure to multiple overlapping traumatic experiences during the chemical attack period, including injury, deprivation, loss of family members, respiratory distress, displacement, and witnessing death, the standard single-index trauma approach was not used. Instead, this study adopted a cumulative trauma framework, in which trauma exposure was conceptualized as trauma load reflecting repeated and overlapping traumatic experiences rather than one single traumatic event. Participants completed the PCL-5 based on their overall trauma exposure during the chemical attack period rather than identifying one index trauma. This approach is supported by previous psychometric studies and population-based research showing that cumulative trauma exposure is a stronger predictor of chronic PTSD symptoms in post-conflict populations (Kira et al., 2018; Sacchi et al., 2020; Priebe et al., 2018; Jayasuriya et al., 2025; Priebe et al., 2018).

Hopkins Symptom Checklist (HSCL-25)

The HSCL is a scale designed to assess symptoms of depression and anxiety on a 4-point Likert scale (not at all to extremely). It has been developed from the Symptom Checklist-90 (SCL-90), and its factor structure varies by population (Derogatis et al., 1974; Sivertsen et al., 2022). It has been extensively validated in various cultural contexts, demonstrating strong psychometric properties and sensitivity to changes in mental health status over time. Mean scores are used, with a recommended cutoff of 1.75 for clinically significant symptoms validated in prior studies with chemical attack survivors (Fares et al., 2021; Neldner et al., 2023).

Perceived Stress Scale (PSS-14)

The PSS-14 is a widely used measure of perceived stress levels, assessing individual subjective perceptions of stress and their ability to cope with life stressors. PSS-14 is a 14-items measuring subjective stress levels on a 5-point Likert scale from (0 = never to 4 = very often) over the past month. It has demonstrated good reliability and validity across diverse populations and has been utilized in several studies examining stress and coping mechanisms among trauma-exposed populations (Santiago et al., 2020).

Patient Health Questionnaire-15 (PHQ-15)

The PHQ-15 is a widely used instrument for assessing somatic symptoms associated with psychological distress, including pain, fatigue, and gastrointestinal complaints. It has been shown to be reliable and valid in both clinical and community samples and is appropriate for use in trauma-exposed populations. Items are rated on a 3-point scale (0 = not bothered at all, 1 = bothered a little, 2 = bothered a lot) (Kroenke et al., 2002).

War and Adversity Exposure Checklist-26 (WAE-26)

WAE-26 (binary 0/1 scoring) administrated (Ibrahim et al., 2018), plus 11 extra questions tailored to chemical attack experiences measured traumatic events such as (e.g., 'did your family member die during the chemical attack?', 'did you lose your sight or faint?', 'were you injured and bleeding?', 'did you witness bodies?', 'did you feel unable to think clearly?', 'did your family member go missing during the chemical attack', 'did you harm someone else or leave behind any family members during the attack in order to survive? '). Items scored yes/no, with total trauma score as sum of all endorsed responses.

In addition to the standardized psychiatric scales (PHQ-15, PCL-5, HSCL-25, PSS-14), we collected detailed demographic and clinical variables through structured interview questions administered during data collection. These covariates account for important factors that could influence the results of our genetic and regression analyses, such as taking psychotropic medication, having chronic medical conditions, or having a family history of mental illness.

Current psychotropic medication status ascertained via two sequential questions: (1) "Are you currently taking any psychotropic or psychiatric medications?" (yes/no); (2) if yes, "What specific medication(s) are you taking?" Responses coded dichotomously (any psychotropic use vs. none) for primary analyses for descriptive purposes.

Chronic medical conditions were assessed using two questions: (1) "Do you have any ongoing chronic diseases or medical conditions?" (yes/no), and (2) if yes, "Which specific condition(s)?" The most reported conditions included diabetes mellitus, hypertension, autoimmune disorders, cardiovascular disease, and respiratory conditions. For analyses, these were coded as dichotomous indicators (presence/absence of any chronic disease).

Family history of mental illness captured via: (1) "Has any family member ever been diagnosed with or treated for a mental health condition?" (yes/no); (2) if yes, and what condition?". Positive family history dichotomized for analyses.

Social support was assessed using a brief item assessing perceived support. Participants were asked "Do you feel that you have people you can rely on for emotional or practical support?" and responses were coded dichotomously (yes/no).

Translation and Adaptation Procedures

All self-report measures employed in this study were carefully adapted into Kurdish using a structured forward-backward translation process following Beaton et al (2000) guidelines. This included the Patient Health Questionnaire-15 (PHQ-15), the PTSD Checklist for DSM-5 (PCL-5), the Hopkins Symptom Checklist-25 (HSCL-25), and the Perceived Stress Scale-14 (PSS-14). For each measure, two independent bilingual translators, both native Kurdish speakers, produced initial forward translations. One translator had a background in clinical psychology to ensure conceptual fidelity, while the other had no formal medical training to capture more natural, everyday language. The research team reviewed both forward translations, resolved discrepancies, and generated a single reconciled version for each instrument.

Each reconciled Kurdish version was then backtranslated into English by an independent bilingual translator who had not participated in the forward translation. The back-translations were compared with the original instruments to identify discrepancies and to ensure semantic and conceptual equivalence. Minor revisions were made as needed to preserve the intended meaning of each item while maintaining cultural relevance.

The pre-final versions were evaluated by a panel of clinical psychologists experienced in trauma assessment and cross-cultural research. They assessed clarity, cultural appropriateness, and relevance of all items. Pilot testing was conducted with 18 trauma-exposed participants to confirm comprehension and acceptability. Participants were asked to explain items in their own words and provide feedback on clarity. No major revisions were required following the pilot, indicating that the translations were both linguistically and conceptually sound.

Statistical Analyses

Psychometric Analyses

The internal consistency of the PHQ-13K scale was measured using McDonald's omega (ω) for the overall scale and Cronbach's alpha (α) for individual items, using the psych package in R (Revelle, 2025). Item-total correlations were also examined. Analyses that required complete data were done using listwise deletion. Reliability and descriptive analyses for other measures, including the PCL-5 (20 items), HSCL-

25 (25 items), and PSS-14 (14 items), were based on complete cases. Detailed results are presented in Appendix Tables A1-A9.

Prior to analysis, all questionnaire items were screened for missing data and applicability. Items with more than 30% missing responses or those not applicable to all participants were reviewed for potential exclusion (van Buuren & Groothuis-Oudshoorn, 2011).

Within the PHQ instrument, PHQ-4 (menstrual pain) was excluded from all analyses as it was not applicable to male participants, and its inclusion would have reduced the available sample size. PHQ-11 (pain during sexual intercourse) showed 34.8% missing responses (186 of 534 cases) in the chemical gas survivor cohort. Multiple imputation using predictive mean matching (MICE) was tested to assess whether the item could be retained, but imputation did not improve data quality, and the item was therefore excluded in this cohort.

As a result, a 13-item version of the PHQ (PHQ-13K; excluding PHQ-4 and PHQ-11) was used for chemical gas survivors and for combined analyses. In the Yazidi survivor group, a 14-item version (PHQ-14; excluding PHQ-4 but keeping PHQ-11) was used for group-specific analyses, because PHQ-4 does not apply to male participants and including it would have reduced the sample size for complete-case analyses.

The psychometric validation of the PHQ-13K, including the ESEM factor structure (CFI = .992), invariance testing, and the rationale for item exclusion, is reported in detail in my first-author paper in *BMC Psychiatry* (Mohammed et al., 2026; see Appendix C).

Hierarchical Multiple Regression Analyses

Hierarchical multiple regression was used to examine factors predicting mental health outcomes across both cohorts and in the combined sample. This method was chosen because it allows us to evaluate the contribution of different sets of predictors in a structured way, entering variables in theoretically ordered blocks to understand how each layer adds to the explanation of outcomes (Greenland, 1994). In trauma research, background and prior risk factors are often entered first, followed by more immediate psychological or trauma-related variables, allowing researchers to examine the additional contribution of each set (Dolan et al., 2020). Using this approach allow estimation the unique variance explained by each set of variables (ΔR^2) while controlling for confounding from earlier blocks, and avoids the issues associated with purely data-driven stepwise methods that can obscure theoretically meaningful relationships (Greenland, 1994).

The chemical gas survivor cohort (n=534) was previously analyzed via multivariate GLM in my first-author publication (Appendix B; Mohammed et al., 2025), establishing overall associations. The current hierarchical approach extends these findings by testing sequential predictor entry, revealing incremental variance (ΔR^2) patterns not captured by simultaneous entry.

The two cohorts were analyzed jointly to examine overall associations in the combined sample. To account for differences between cohorts, separate analyses were also conducted within each group, and cohort membership and cohort-by-predictor interaction terms were included in the pooled models. Significant interaction terms indicate that the effect of a predictor differs between cohorts, whereas non-significant interactions suggest similar effects across groups. This approach allows for the assessment of pooled associations while accounting for cohort heterogeneity and is consistent with recommendations for the analysis of combined individual-level data from multiple populations (Blettner et al., 1999).

Four continuous outcomes were modeled using ordinary least-squares (OLS) regression: PTSD symptom severity (PCL-5 total score), depression and anxiety (HSCL-25 total score), somatic symptoms (PHQ13K total score), and perceived stress (PSS-14 total score). Variables were entered in three conceptually ordered blocks across all models to assess how each domain is associated with outcome variance beyond the preceding blocks.

Block 1: Demographic variables. Age, gender, years of education, occupation, place of residence, social status, and number of children were included. These factors represent basic participant characteristics that could influence mental health and provide a baseline against which other variables are assessed.

Block 2: Psychosocial and general health factors. This block added social support, current psychotropic medication use, family history of mental disorders, and chronic physical illness. These variables reflect the social and health context, which may reduce or exacerbate symptoms, and allow assessment of their contribution beyond demographic factors.

Block 3: Trauma exposure and clinical status variables. Cumulative trauma exposure was included as the primary exposure variable. Self-reported psychiatric diagnosis (e.g., history of professionally diagnosed depression, PTSD, or anxiety disorder) was included as a clinical covariate to adjust for pre-existing mental health conditions. These variables were entered in the final block to examine the incremental contribution of trauma exposure to mental health outcomes beyond demographic and psychosocial factors, while accounting for baseline clinical status. Because both psychiatric diagnosis and outcome measures were assessed cross-sectionally, findings represent statistical associations and do not imply causality.

Given the presence of familial relatedness in the sample, we conducted additional sensitivity analyses using linear mixed-effects models incorporating a genetic relatedness matrix (GRM) to account for non-independence among participants. These models were compared with the primary ordinary least-squares (OLS) regression results. The pattern of findings remained consistent across both approaches, indicating that the observed associations were robust to family clustering (see Genetic Relatedness section).

This block structure was applied consistently across three sets of models: (a) the pooled sample ($N = 892$), (b) chemical gas survivors only ($n = 534$), and (c) Yazidi survivors only ($n = 358$). Using the same structure for all cohorts allowed direct comparison of patterns while maintaining theoretical consistency.

For each model and outcome, the coefficient of determination (R^2) was reported for all blocks, along with the change in explained variance (ΔR^2) for each new block. Cohen's f^2 was calculated to assess the incremental effect size of Blocks 2 and 3 ($f^2 = \Delta R^2 / (1 - R^2_{\text{total}})$), where ΔR^2 is the increase in explained variance when the block is added, and $1 - R^2_{\text{total}}$ is the variance still unexplained by the model. This shows how much each block contributes relative to the remaining unexplained variance. According to conventional guidelines, f^2 values of 0.02, 0.15, and 0.35 indicate small, medium, and large effects, respectively (Cohen, 1988).

Standardized regression coefficients (β) were reported with 95% confidence intervals for unstandardized estimates to indicate both magnitude and precision. Model diagnostics included variance inflation factors ($VIF < 5$) to check for multicollinearity, Durbin-Watson statistics to assess residual autocorrelation, and tests for residual normality and homoscedasticity. Robust standard errors were applied when OLS assumptions were not fully met, particularly in cases of non-normal residuals or heteroscedasticity.

To explore potential differences between cohorts, interaction analyses were conducted in the pooled sample. Analyses were hypothesis-driven and based on a priori specified predictors and interaction terms; therefore, formal correction for multiple testing was not applied for these pre-specified interaction analyses, in line with confirmatory modeling approaches. All predictors were entered into the regression models along with their interaction with cohort membership (chemical gas survivors = 1, Yazidi survivors = 0), producing coefficients for both main effects and interaction terms. These findings should be interpreted with caution, as the absence of correction for multiple testing increases the risk of Type I error. However, consistent patterns across outcomes and adequate model fit ($R^2 = .22 - .44$) support the interpretation of these interactions as reflecting meaningful cohort differences.

Overall, hierarchical regression provided a structured approach to evaluate the incremental contributions of demographics, psychosocial factors, and trauma-related variables. Sensitivity analyses using linear mixed-effects models confirmed that the observed associations were robust to familial relatedness, indicating that results were not driven by non-independence among related participants.

Genotyping and Quality Control

Genotyping was performed using Illumina Global Screening Array 3.0 SNP arrays (Illumina, San Diego, USA) across three subsamples: Kurd1 (n = 234) and Kurd2 (n = 300), which included chemical gas survivors, and Kurd3 (n = 355), which included Yazidi survivors. Three Yazidi participants did not provide saliva samples and were excluded from the genetic analysis. All of the QC procedures detailed below were carried out by Dr. Sergi Papiol.

Initial processing with the PLINK software (Purcell et al., 2007) involved standardizing SNP identifiers and removing duplicate markers, leaving approximately 726,303 SNPs per batch. Samples with low call rates (<98%) were removed (1 from Kurd1, 20 from Kurd2, and 10 from Kurd3). Variants with low call rates (<98%) or low minor allele frequency (<0.005) were also excluded, as these variants are less reliable in genetic analyses. Genetic sex was verified using X-chromosome homozygosity (F statistic) to detect discrepancies with the phenotypic sex.

After these quality control steps, the final datasets included 233 samples with 475,424 SNPs for Kurd1, 280 samples with 483,871 SNPs for Kurd2, and 345 samples with 541,266 SNPs for Kurd3. The final merged dataset included 858 participants and 371,872 SNPs.

Pairwise relatedness was assessed using LD-pruned SNPs (70,000–100,000 markers) in PLINK. Duplicate samples (PI-HAT = 1) were removed, resulting in the exclusion of nine individuals and leaving 849 unique participants. Heterozygosity checks identified three additional outliers, likely due to sample contamination, which were removed.

First- and second-degree relatives were identified but retained, as their relatedness was accounted for in downstream analyses using a genomic relationship matrix (GRM). Approximately 23,000 SNPs showed allele frequencies differing by more than 10% from European reference populations; these variants were noted but retained given the Kurdish-specific context. Allele frequencies were also compared to the Haplotype Reference Consortium (HRC) panel before exporting the data to VCF format for imputation.

Population structure was examined using multidimensional scaling (MDS) based on LD-pruned autosomal SNPs (70,000–90,000 variants). To contextualize the sample globally, the dataset was merged with reference populations from the 1000 Genomes

Project, including African, Admixed American, East Asian, European, and South Asian groups, and MDS was recalculated. MDS was also performed on the study sample alone to identify potential ancestry outliers. The first two MDS components were plotted for visual inspection, and no individuals were removed based on these analyses.

Analyses focused on autosomal and X-chromosome variants, with Y chromosome, pseudo-autosomal regions, and mitochondrial DNA excluded. SNPs violating Hardy-Weinberg equilibrium ($p < 10^{-4}$), having MAF $< 0.5\%$, or featuring palindromic alleles (A/T, C/G) were filtered out to avoid ambiguity. Variants with large deviations ($>10\%$) from European allele frequencies were retained due to the Kurdish-specific context.

After quality control, the cleaned dataset included 846 samples and 358,382 SNPs. The data were exported from PLINK binary files to VCF format for processing via the Michigan Imputation Server, imputing non-genotyped SNPs. Genotype-phenotype matching was conducted using unique FID/IID identifiers, and six individuals ($<1\%$) who could not be matched across datasets were excluded to ensure accurate alignment.

Population Stratification Adjustment Using Principal Components

To account for population structure, regression models including one to ten ancestry principal components (PCs) were compared within each cohort. In both cohorts, models including ten PCs demonstrated the best fit based on Akaike Information Criterion (AIC), and inclusion of additional PCs did not meaningfully alter PTSD PRS effect estimates. Correlations among PCs were low, and variance inflation factors indicated minimal multicollinearity, supporting the inclusion of ten PCs as covariates in primary analyses (Figure 2).

Analytic Sample and Phenotypic Processing

The final analytic samples consisted of 499 Kurdish survivors of chemical gas exposure and 342 Yazidi survivors of ISIS-related trauma. Following quality control and alignment with genomic data, included participants had complete phenotypic and genotypic information. Inclusion required available PCL-5 total scores, cumulative trauma exposure, and valid PTSD PRS, along with genomic data aligned with the genomic relationship matrix (GRM). Continuous covariates, including age, cumulative trauma exposure, and PTSD PRS, were z-score standardized within each cohort to facilitate interpretation and comparison of effect sizes. Genetic principal components (PC1-PC10), derived from genome-wide data, were included as covariates to control for population stratification. Categorical variables were factor-coded with reference levels of male gender and lowest education level. PTSD PRS were derived from the latest European-ancestry GWAS (Nievergelt et al., 2024), using linkage disequilibrium clumping ($r^2 < 0.1$ within 500 kb windows), and were standardized within each cohort for subsequent analyses. All analyses were conducted separately within each cohort.

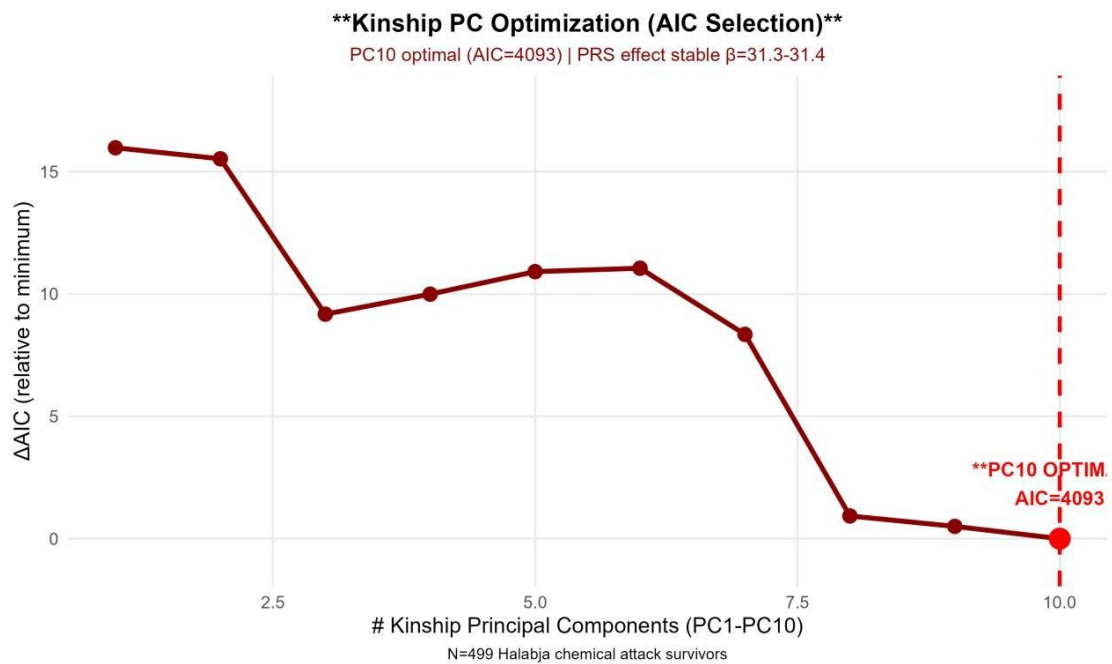


Figure 2. Kinship principal factor optimization for the chemical gas cohort (N = 499), showing change in Akaike Information Criterion (Δ AIC) across models including 1-10.

Genetic Relatedness

Genetic relatedness among participants was assessed using a genomic relationship matrix (GRM). Pairwise relatedness coefficients were calculated from genome-wide SNP data, and standard thresholds were used to classify relationships: $\text{GRM} \geq 0.25$ for first-degree relatives, $0.125 \leq \text{GRM} < 0.25$ for second-degree relatives, and $\text{GRM} < 0.125$ for unrelated individuals (Yang et al., 2011). Because related individuals were present in the dataset, subsequent association analyses were conducted using linear mixed models that incorporated the GRM to account for non-independence among participants.

Statistical Modeling Rationale and Sensitivity Analysis

For analyses involving the PTSD polygenic risk score (PRS), we used linear mixed-effects models (LMMs) and included the genomic relationship matrix (GRM) as a random effect. This accounts for the fact that genetically similar individuals may have more similar outcomes, reducing potential bias in estimates. Using LMMs with the GRM is standard practice in genetic research when family structure or relatedness is present.

For outcomes that did not involve genetic predictors, such as depression, anxiety, somatic symptoms, and perceived stress, we used standard hierarchical regression models. To ensure that ignoring relatedness did not bias these results, we conducted sensitivity analyses by repeating the regressions using LMMs with the GRM as a

random effect. The results were essentially unchanged, indicating that genetic relatedness did not meaningfully affect these estimates.

Linear Mixed Model Analysis

We examined how trauma exposure and genetic risk influence PTSD symptom severity using linear mixed-effects models (LMMs). This analysis was conducted separately from the hierarchical phenotypic models to evaluate the contribution of genetic liability while accounting for genetic ancestry and relatedness. The models included PTSD polygenic risk score (PRS), trauma exposure, and their interaction term (PRS $\times$ trauma) as predictors of total PCL-5 scores.

All models were adjusted for age, gender, education, and genomic principal components (PCs) to control for population stratification, PCs 1-10 were included in all models.

Because the sample included biologically related individuals, we incorporated the SNP-derived genomic relationship matrix (GRM) as a random effect (see also above). This approach accounts for genetic relatedness by modeling covariance between individuals as a function of genome-wide similarity. Including the GRM reduces bias in standard errors that can arise when familial structure is ignored.

All continuous predictors were standardized prior to analysis to facilitate interpretation and improve model stability. Gender and education were entered as categorical covariates.

The primary model therefore estimated:

1. The main effect of trauma exposure,
2. The main effect of PTSD PRS,
3. The interaction between trauma exposure and genetic risk.

The interaction term tested whether the association between trauma exposure and PTSD symptoms differed as a function of genetic liability.

Models were estimated using restricted maximum likelihood (REML) in R (version 4.4.3) with the sommer package. Model assumptions (linearity, homoscedasticity, and normality of residuals) were evaluated by visual inspection of diagnostic plots.

To quantify variance explained, we calculated marginal pseudo- R^2 (variance attributable to fixed effects) and conditional pseudo- R^2 (variance attributable to fixed plus random effects). The proportion of variance attributable to the GRM random effect was interpreted as familial clustering due to genetic relatedness, rather than as a direct estimate of SNP-based heritability.

Interaction effects were visualized by plotting predicted PCL-5 scores across the observed range of PTSD PRS at low (-1 SD), mean, and high (+1 SD) levels of trauma exposure, holding covariates constant.

Sensitivity Analyses

To ensure the robustness of our findings in both the Yazidi and chemical gas survivor cohorts, we conducted several additional checks. In the chemical gas cohort, we assessed multicollinearity among predictors using variance inflation factors (VIF). The main predictors, total trauma (VIF = 1.39), PTSD polygenic risk score (PRS; VIF = 1.15), and their interaction (VIF = 1.10), showed no evidence of problematic collinearity. A few genetic principal components had elevated VIF values, so we re-ran the models using a reduced set of PCs (PC1-PC5). Results remained consistent, and the variance components for the genomic random effect and residuals were stable across models.

In the Yazidi cohort, we examined whether results were influenced by gender or extreme observations. Separate models for males and females revealed similar effects of trauma exposure, while the PRS and its interaction with trauma remained small and non-significant. Removing influential cases with extreme values did not materially alter the estimates. Across both cohorts, trauma exposure consistently predicted PTSD symptom severity, indicating that our main findings are robust and not driven by outliers, gender differences, or modeling choices.

Separate and Combined Analyses of Kurdish Cohorts

The study included two distinct Kurdish cohorts: chemical gas survivors and Yazidi survivors. Although both groups share Kurdish ethnicity, they differ in key demographic, cultural, and environmental factors that may influence mental health outcomes. Chemical gas survivors were more often married, lived in urban areas, and showed different patterns of social support, whereas Yazidi survivors were more likely to live in camps or rural settings and were more often unmarried. It should also be noted that the Yazidi population is largely endogamous, with marriage typically occurring within the group, which further distinguishes their social structure. In addition, differences were observed between the cohorts in chronic disease prevalence, occupation, and access to resources.

These differences justify analyzing each cohort separately, as combining them without accounting for these factors could mask cohort-specific patterns. Our findings support this: genetic analyses involving PTSD polygenic risk scores (PRS) were conducted separately within each cohort and showed different patterns across groups, with significant PRS effects observed in chemical gas survivors but not in Yazidi survivors. In pooled phenotypic analyses, psychosocial predictors such as social support and psychotropic medication use showed evidence of cohort-specific moderation for PTSD and anxiety/depression outcomes.

At the same time, cumulative trauma and, despite differing residential distributions, residential context showed broadly similar directional effects across both cohorts.

Somatic symptom outcomes showed fewer cohort-specific differences, suggesting similar patterns across groups. This supports the use of combined analyses to identify predictors that generalize beyond a single cohort. By including cohort as a covariate and testing cohort $\times$ predictor interactions in hierarchical regression models, we accounted for systematic differences while maximizing statistical power to detect shared effects.

This analytic strategy aligns with best practices in trauma research, where heterogeneity in demographics, culture, and environment can produce cohort-specific effects. Reporting both separate and combined analyses provides transparency about unique cohort patterns while allowing identification of predictors that apply across populations.

Cohort Interaction Analyses

To determine whether associations between predictors and mental health outcomes differed across the two Kurdish cohorts, we conducted pooled analyses including cohort as a moderator. Fully interacted pooled models tested whether the effects of phenotypic predictors, including trauma exposure, social support, psychotropic medication use, demographic factors, and other clinical variables, varied by cohort. PTSD polygenic risk score (PRS) analyses were conducted separately within each cohort because differences in genetic relatedness structure and population stratification made pooled PRS interaction models less appropriate.

Chapter 4: Results

Reliability Analyses (chemical gas survivors)

Internal consistency was examined for all questionnaire scales using listwise deletion for scale-level missing data, and item exclusion where missingness exceeded 30%. Reliability coefficients were measured using coefficient omega (ω) and Cronbach's alpha (α).

For the PHQ-13K somatic symptom, internal consistency was examined in chemical gas survivors ($n = 534$), (excluding PHQ-4 menstrual pain and PHQ-11 sexual pain due to >30% missing data) showed strong reliability ($\omega = .894$; $\alpha = .863$). Item-total correlations ranged from .30-.62.

PHQ-13K validation (ESEM: CFI=.992, RMSEA=.046; full invariance across gender) detailed in my first-author publication (Appendix B).

The war adversity exposure checklist- 26, combined with the 11 additional questions, showed internal consistency of $\omega = .85$ using tetrachoric correlations, appropriate for dichotomous indicators.

The PCL-5 ($\omega = .909$; $\alpha = .888$), HSCL-25 ($\omega = .944$; $\alpha = .936$), and PSS-14 ($\omega = .908$; $\alpha = .875$) also showed strong internal consistency. Item deletion for each scale did not produce a clinically meaningful change in reliability, suggesting stable item performance across measures (Table 2). Full item statistics are provided in Appendix Tables A1-S9.

Table 2. Chemical gas survivors scale-level reliability summary

Scale	N	Items	McDonald's ω	Cronbach's α
PHQ-13K	534	13	.894	.863
PCL-5	534	20	.909	.888
HSCL-25	534	25	.944	.936
PSS-14	534	14	.908	.875

Reliability Analyses (Yazidi survivors)

Internal consistency was examined for all questionnaire scales in the Yazidi survivors cohort ($n = 358$) using listwise deletion for missing cases. Reliability coefficients were quantified using coefficient omega (ω) and Cronbach's alpha (α). The full PHQ-15 (including PHQ4) yielded strong reliability among complete cases ($\omega = .87$; $\alpha = .84$). To maximize sample size and avoid listwise deletion bias, PHQ-14 (excluding

PHQ4 due to data unavailability) was analyzed across the full sample ($n = 358$) and showed comparable strong reliability ($\omega = .896$; $\alpha = .880$). Item-total correlations ranged from moderate to high ($r = .30-.69$), with no single item removal substantially improving scale performance (α remained .87-88). The internal consistency of the War adversity exposure checklist-26 was $\omega = .80$, using tetrachoric correlations.

The remaining scales demonstrated robust internal consistency: PCL-5 ($\omega = .907$; $\alpha = .890$), HSCL-25 ($\omega = .917$; $\alpha = .905$), and PSS-14 ($\omega = .850$; $\alpha = .805$). These findings align with results of chemical gas survivors (Table 3).

Table 3. Yazidi survivors cohort scale reliability summary ($n = 358$)

Scale	Items	McDonald's ω	Cronbach's α
PHQ-14	14	.896	.880
PCL-5	20	.907	.890
HSCL-25	25	.917	.905
PSS-14	14	.850	.805

Group 1: Victims of Chemical Gas Exposure (Demographic Profile)

Total participants were 534, 300 of them were female (56.2%) and 234 were male (43.8%). The mean age was 53.66 years ($SD = 11.16$). The average income was 419.26 US\$ ($SD = 269.49$). Regarding education levels, 22.8% had no formal education, 44.0% had completed elementary school, 14.4% had finished secondary school, and 18.7% had attended institution or university (Figure 3).

In terms of employment status, 4.5% were unemployed, 42.7% were housewives, 29.6% were governmental employees, 12.4% were self-employed, 1.3% were unable to work, and 9.6% were retired. The mean number of children per participant was 4.4 ($SD = 2.6$).

The marital status distribution was as follows: 9.0% were single, 83.1% were married, 2.4% were divorced, and 5.4% were widowed. A total of 68.7% had a chronic disease. The majority of the sample lived in urban areas (92.1%), while 5.6% lived in suburban areas and 2.2% lived in villages. Regarding social factors, 59.7% reported having social support, and 16.8% were taking psychotropic medication.

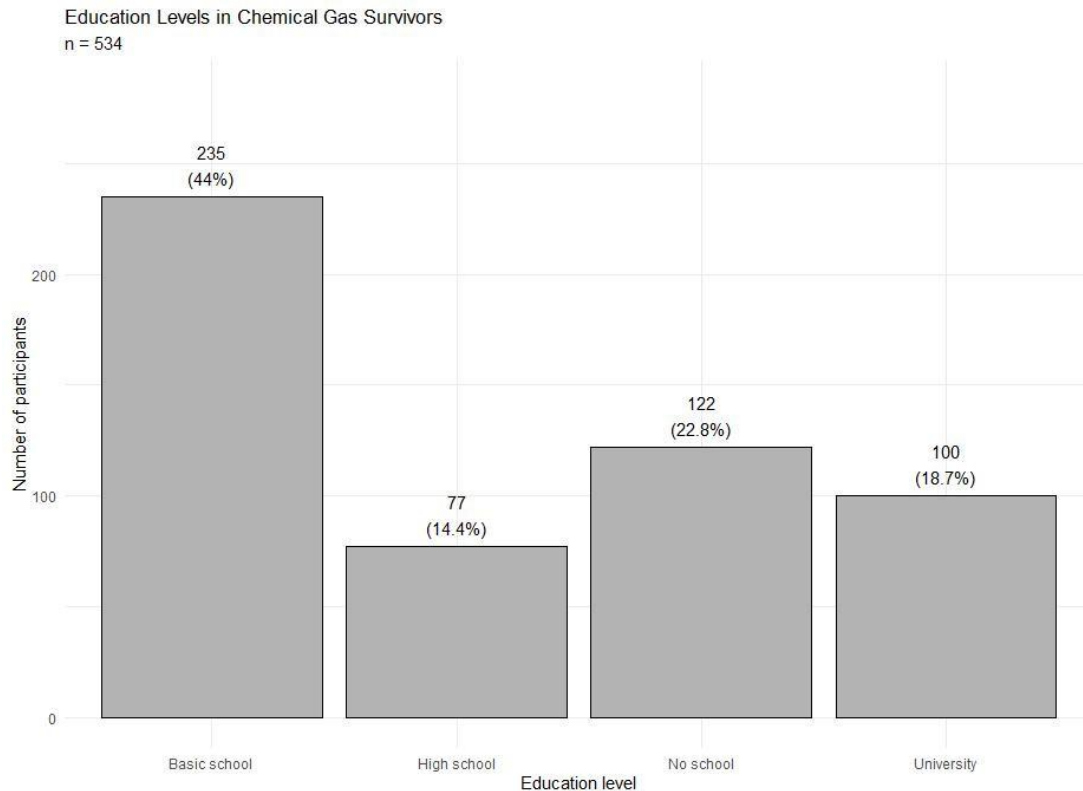


Figure 3 shows education levels (n = 534). Distribution shows 44.0% basic school, 22.8% no formal education, 18.7% university graduates

Symptom Profiles

The mean score for the PCL-5 was (M=36.14, SD = 16.05). PHQ-13K (M=13.62, SD = 6.36), HSCL25 (M=2.07, SD=.66). PSS14 (M=24.30, SD = 11.14). Combined Trauma Checklist (M= 14.33, SD = 4.66), ranged from 2 to 30 traumatic events, trauma exposure levels were defined as Low = 9.64 (-1 SD), Mean = 14.33, and High = 19.00 (+1 SD). The most frequently reported symptoms as extreme were for the PHQ-13K “Pain in your arms, legs, or joints (knees, hips, etc.) ” 74.7% of the total participants. “Feeling tired or having low energy” by 66.1% of participants, and “back pain” reported by 64.6% participants. In the PCL-5, “ Repeated, disturbing, and unwanted memories of the stressful experience “ reported by 38% of the participants, and “Feeling very upset when something reminded you of the stressful experience?” reported by 23.8% of participants and “Trouble falling or staying asleep “reported by 30.5% of the participants . Regarding HSCL-25, “Feeling tense or keyed up” was experienced by 26.4% of the sample. Similarly, “Difficulty falling asleep, staying asleep” 23.2% and “Loss of sexual interest or pleasure” 26.8%. and the most frequently reported traumatic events are demonstrated in (Figure 4).

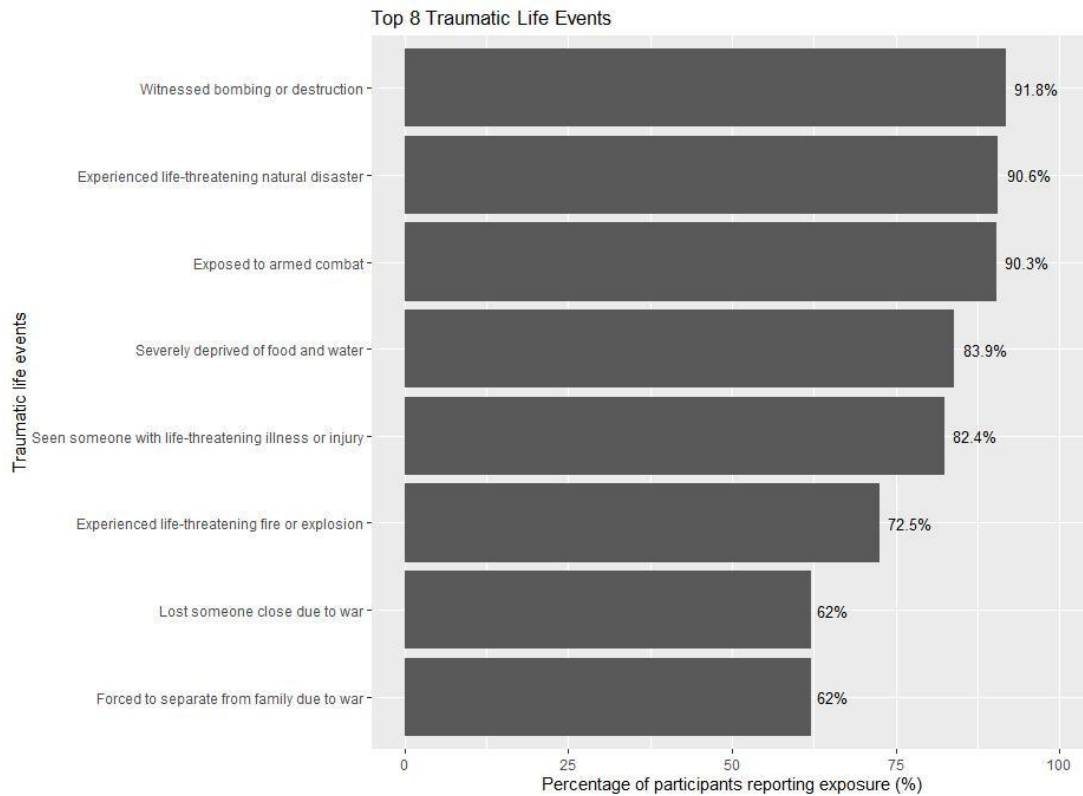


Figure 4: Most common traumatic events that experienced by participants.

Related to clinically relevant cut-off scores, we found for PCL5 78.8% participants scored equal or higher than ≥ 23 and 21.2% scored below this threshold. For HSCL-25, we found 65.2% had score equal or higher than ≥ 1.75 , while 34.8% scored below this cut-off. 62.5 % of participants had PTSD and depression/anxiety symptoms, 45.3% have PTSD and somatic symptoms. However, only 16.8% of Participants took psychiatric medication during the time of the data collection (Figure 5).

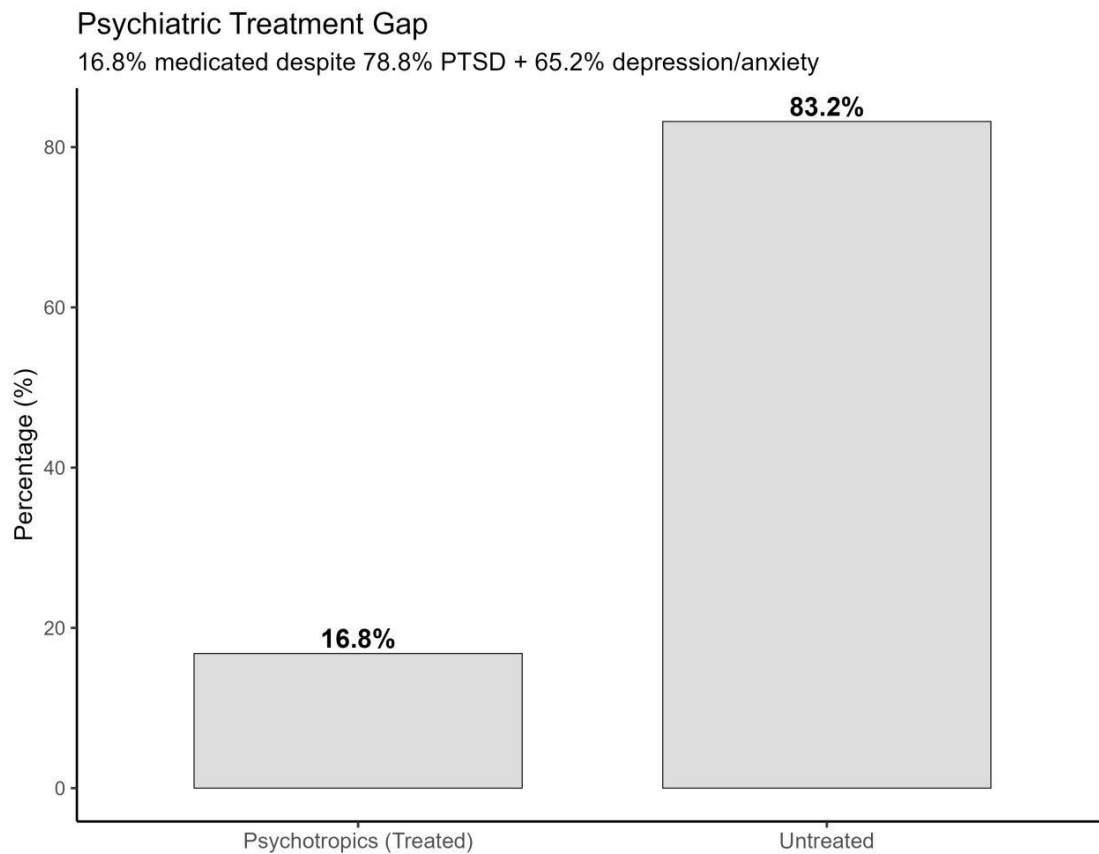


Figure 5. Shows Psychiatric treatment utilization gap. Despite extensive clinical symptomatology, only 16.8% of participants received psychotropic medications at time of assessment, represents profound disparity between symptom burden and treatment access.

Group 2: Yazidi Survivors group (Demographic Profile)

Table 4 shows the descriptive statistics for the primary psychological variables among the participants (N = 358). Almost half of participants have not formal education (Figure 6).

The sample consisted mainly of women and had a relatively young average age. Participants reported generally low income and a high proportion had no formal education. Most participants lived in camps or villages, reflecting their displacement. Employment levels were low, with many participants unemployed or working as housewives. Most were married and had several children. Around three-quarters reported having social support, and about one-fifth reported a chronic disease.

Table 4. Descriptive statistics for demographic and psychological variables among participants (N = 358).

Demographic Variables	Category	Frequency
Gender	Female	256 (71.5%)
Age	Mean (SD)	32.24 (12.62)
Income in US\$	Mean (SD)	234.99 (126.10)
Level of education	No formal Education	157 (43.9%)
	Elementary School	81 (22.6%)

	Secondary School	77 (21.5%)
	Institution or University	43(12.0%)
Work Status	Unemployed	133 (37.2%)
	Student	49 (13.7)
	Housewife	125 (34.9%)
	Governmental employee	10 (2.8%)
	NGO/Company	39 (10.9%)
	Unable to Work	2 (.6%)
	Retired	51 (14.2%)
Marital Status	Single	85 (23.7%)
	Married	267 (74.6%)
	Divorced	6 (1.7%)
Number of Children	Mean (SD)	4.4(2.6)
Living Location	City	3 (0.8 %)
	Around The City	7 (2.8 %)
	Village	131 (36.6%)
	Camp	217 (60.6%)
Social Support	Having social support	267 (74.6%)
Chronic Disease	Having Chronic Disease	77 (21.5%)

Yazidi Survivors: Education Levels

n = 358 | 44% No School

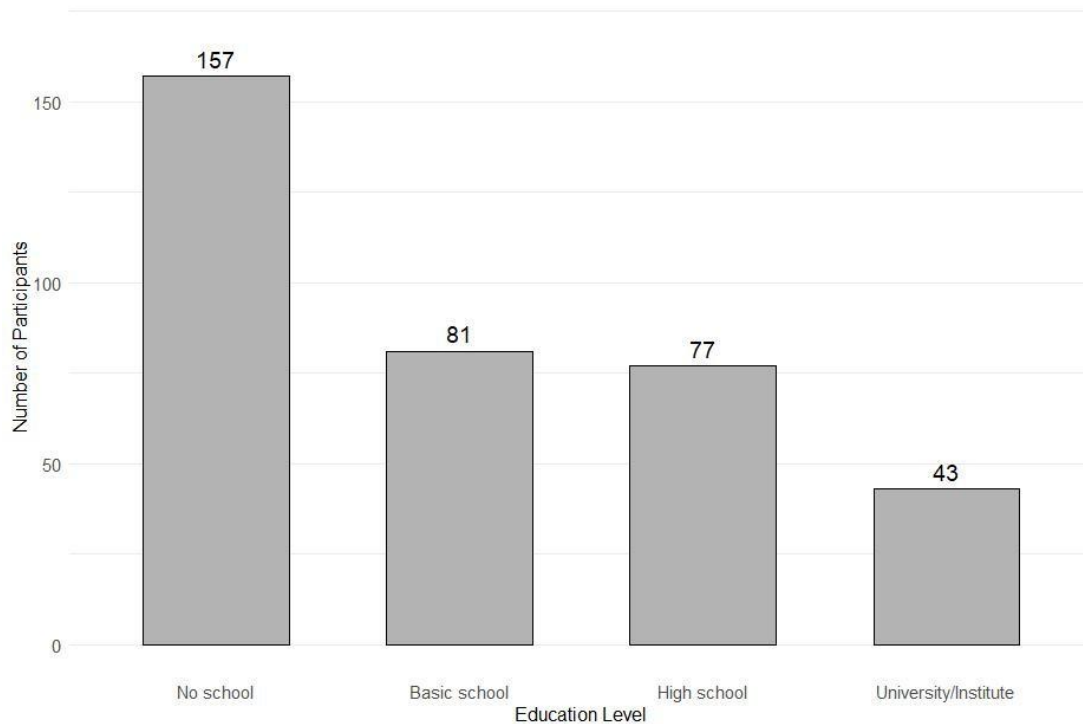


Figure 6. Education levels (n = 358). No schooling most common (43.9%), basic school 22.6%. Five educational categories

Symptom Profiles

Somatic symptom severity measured by the PHQ-14 showed a mean score of 14.33 (SD = 7.16) when the item “menstrual cramps” was excluded. When this item was included, the mean increased to 15.79 (SD = 7.71), indicating a higher level of reported physical symptoms. Perceived stress (PSS-14) was elevated, with a mean score of 34.66 (SD = 9.22). PTSD symptom severity (PCL-5) was also high, with a mean of 51.08 (SD = 15.56). Participants reported exposure to a relatively large number of traumatic events ($M = 8.09$, $SD = 4.13$; range 0-22). The HSCL-25 mean score was 2.68 (SD = 0.60), reflecting elevated symptoms of anxiety and depression (Table 5).

Table 5. Psychological symptoms summary

Variable	Minimum	Maximum	Mean	Std. Deviation
Traumatic Events	0	22	8.09	4.13
PCL-5	2	77	51.08	15.56
PHQ-14	0	36	14.33	7.16
PHQ-15	0	36	15.79	7.71
PSS-14	5	55	34.66	9.22
HSCL-25	1	3.88	2.68	0.60

Figure 7 presents most frequently endorsed traumatic events among participants. The most common events included severe deprivation of food and water due to war or flight (81%), witnessing someone suffer a life-threatening illness or injury (74.9%), and exposure to armed combat (57.5%). Several other experiences, including witnessing bombings and separation from family due to war, were also reported by nearly half or more of the sample.

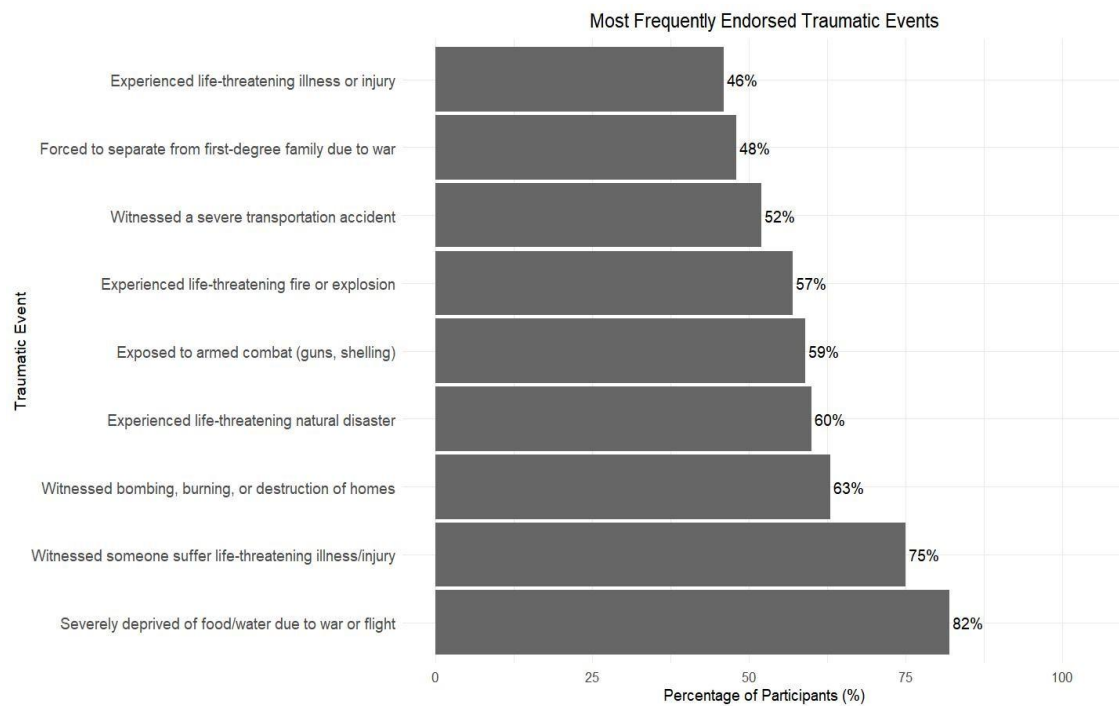


Figure 7. most common traumatic events experienced by participants

Figure 8 shows the distribution of PTSD based on the PCL-5 cutoff score of 23 (Ibrahim et al., 2018). 94.4% of participants meet the criteria for probable PTSD (PCL-5 score ≥ 23). The colors represent these groups, with green indicating probable PTSD and red indicating no PTSD.

Similarly, 91.3% of participants met or exceeded the HSCL-25 cutoff score of 1.75.

The most prevalent symptoms include feeling tired or having low energy (67.3%), upset by reminders of trauma (64%), feeling low in energy or slowed down (57.5%), and headaches (57.5%). Other common symptoms include intrusive memories (58.7%), blaming you for things (53.9%), and worrying too much (53.1%). Figure 9 shows that symptoms related to depression/anxiety (HSCL), somatic complaints (PHQ), and PTSD (PCL) were among the most commonly reported symptoms across participants.

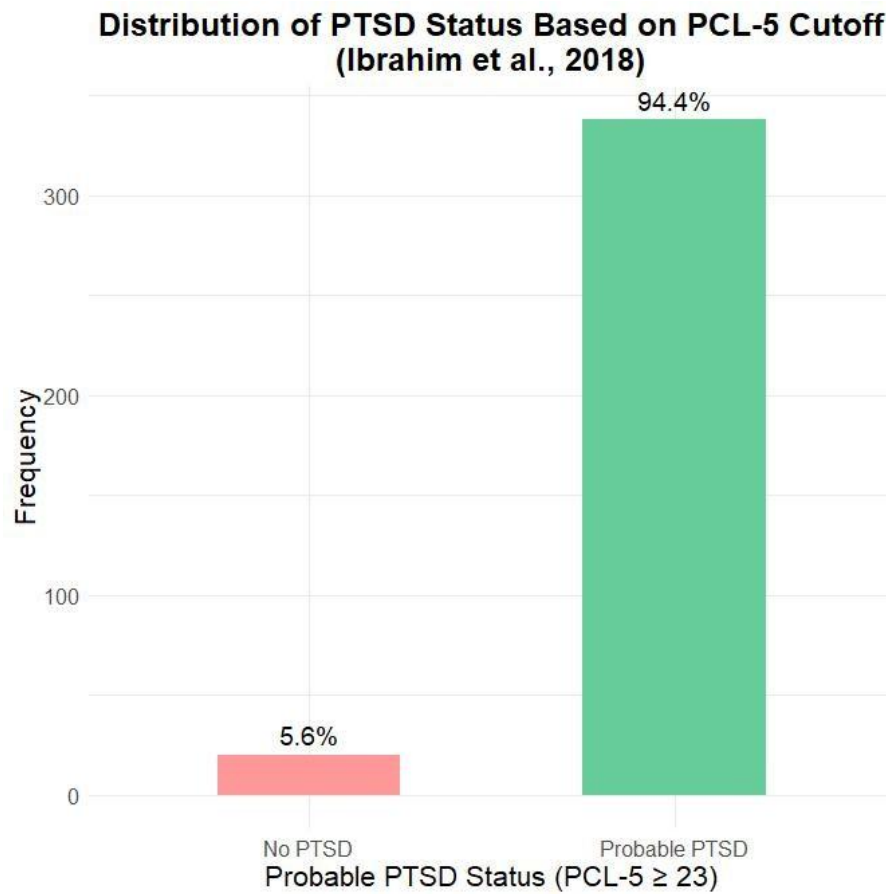


Figure 8. The distribution of PTSD based on the PCL-5 cutoff score of 23.

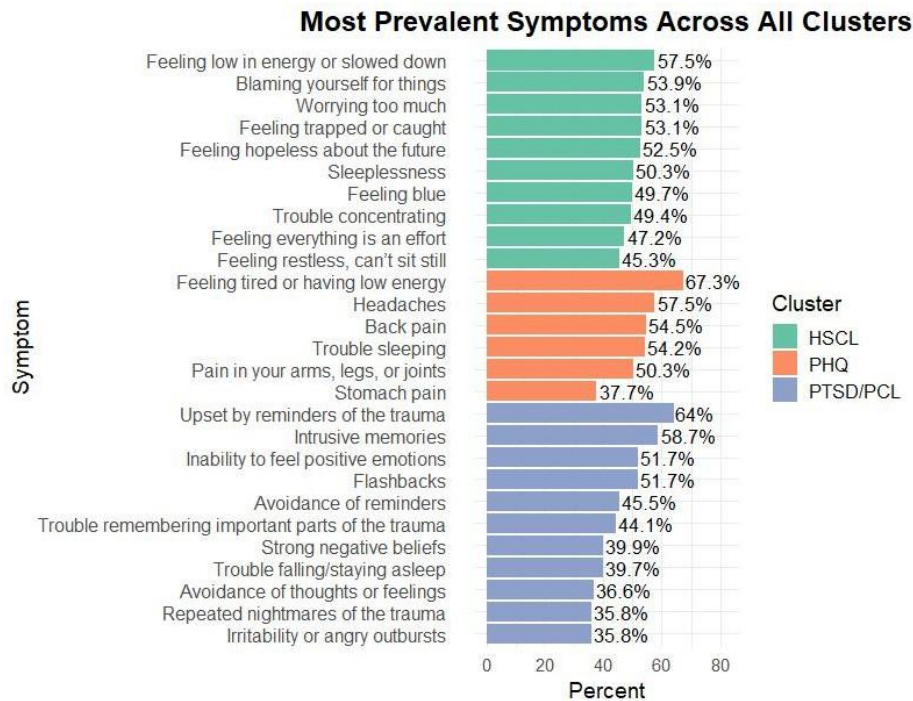


Figure 9 shows the prevalence of symptoms across all scales.

Hierarchical Regression Analyses (chemical gas survivors, N = 534)

Extending my first-author publication's multivariate GLM analysis (Appendix B; Mohammed et al., 2025; trauma $\beta=0.85$, $\eta^2=0.38$ across outcomes), hierarchical regression examined sequential predictor entry among chemical survivors (N=534).

We conducted hierarchical regression to examine the factors contributing to mental health outcomes. In the first step, demographic variables alone explained between 10% and 38% of the variance across outcomes. The highest variance explained was for PHQ-13K ($R^2 = 0.378$), while the lowest was for PSS-14 ($R^2 = 0.102$), indicating that basic demographic characteristics associated meaningfully with mental health differences in this sample.

When psychosocial factors were added in Block 2, the models improved meaningfully. The increase in explained variance (ΔR^2) ranged from 0.041 to 0.109, with the largest improvement seen for HSCL-25. This suggests that psychosocial resources, such as social support, provide additional explanatory power beyond demographics. Effect sizes for this block were generally small to moderate ($f^2 = 0.071$ -0.167), with the largest effect again for HSCL-25.

Finally, including trauma exposure and health-related variables in Block 3 further increased the variance explained. Total R^2 values ranged from 0.219 for PSS-14 to 0.462 for PHQ13K. The incremental variance added by this block (ΔR^2) was small to moderate (0.024-0.051, $f^2 = 0.031$ -0.085), showing that trauma and health factors are meaningfully associated with mental health outcomes even after accounting for

demographics and psychosocial variables. Overall, these results indicate that mental health in chemical gas survivors is influenced by a combination of demographic, psychosocial, and trauma-related factors (Table 6).

Table 6. Model Fit Across Blocks

Outcomes	Block 1 R²	Block 2 R²	ΔR² Block 2	f² Block 2	Block 3 R²	ΔR² Block 3	f² Block 3
PHQ13K	0.378	0.420	0.041	0.071	0.462	0.042	0.078
PCL5	0.213	0.284	0.072	0.100	0.332	0.047	0.071
HSCL25	0.238	0.346	0.109	0.167	0.397	0.051	0.085
PSS14	0.102	0.194	0.093	0.115	0.219	0.024	0.031

Note: R² = variance explained by each block; ΔR² = incremental variance from new block; f² = Cohen's effect size for Blocks 2-3 (0.02 small, 0.15 medium, 0.35 large) (Cohen, 1988).

In the final regression models (Block 3), several variables showed consistent stronger associations with symptom severity across mental health outcomes. Gender, income, education, psychotropic medication use, and cumulative trauma consistently showed meaningful associations. Women reported higher symptom levels than men, particularly on the PHQ-13K and PCL-5. Lower income and lower educational attainment were linked to worse mental health outcomes across all measures, highlighting the role of socioeconomic disadvantage in exacerbating symptoms.

Use of psychotropic medication and higher cumulative trauma were associated with increased symptom severity across the four outcomes, suggesting that participants with greater exposure to traumatic events or those requiring medication experienced more pronounced mental health difficulties. Effect sizes varied across measures, with gender showing the strongest effect for PHQ13K ($\beta = 0.35$), and income the strongest negative association ($\beta = -0.34$) on the same outcome. Overall, these results indicate that both demographic and trauma-related factors play a substantial role in shaping mental health among chemical gas survivors exposure (Table 7).

Table 7. Key Block 3 Predictors (Standardized β)

Predictor	PHQ13K	PCL-5	HSCL-25	PSS-14
Gender	0.35	0.26	0.24	0.03

Income	-0.34	-0.24	-0.24	-0.12
Education	-0.21	-0.17	-0.20	-0.20
Psychotropics	0.17	0.24	0.27	0.29
Cumulative trauma	0.21	0.24	0.24	0.18

Note: Table shows strongest predictors, full coefficients in Appendix table A10.

Diagnostic checks were performed to assess model assumptions. Multicollinearity was low across all models (maximum VIF = 2.30). Durbin–Watson statistics ranged from 1.82 to 1.92, indicating no meaningful autocorrelation. Shapiro–Wilk tests suggested that residuals were approximately normally distributed for PHQ13K and PCL-5, whereas HSCL-25 and PSS-14 showed deviations from normality ($p < .001$). Given this, robust standard errors were applied for models where normality was not met (Table 8).

Table 8. Model Diagnostics (Block 3)

Outcome	Max VIF	Durbin-Watson	Shapiro p
PHQ13K	2.30	1.88	0.493
PCL-5	2.30	1.85	0.260
HSCL-25	2.30	1.82	<0.001
PSS-14	2.30	1.92	<0.001

Sensitivity Analyses: Linear Mixed Models Accounting for Familial Relatedness

Because some participants were biologically related, additional analyses were conducted to check whether the primary hierarchical regression results were influenced by non-independence among relatives. Linear mixed models (LMMs) were estimated including a genetic relatedness matrix (GRM) as a random effect. The GRM encodes genome-wide genetic similarity between participants and allows the model to account for clustering among related individuals while estimating associations between demographic, psychosocial, and trauma-related predictors and mental health outcomes.

Variance Components

The random effect associated with the GRM captures variance in symptom levels due to clustering among related participants. For PTSD symptoms (PCL-5) and depression/anxiety (HSCL-25), the intraclass correlation (ICC) captured by the GRM was moderate (PCL-5: 0.45; HSCL-25: 0.43). Somatic symptoms (PHQ13K) showed similar clustering (0.45), whereas perceived stress (PSS-14) showed weaker clustering (0.20). These results indicate that related individuals tend to report more similar symptom levels, particularly for PTSD and internalizing symptoms.

Marginal R^2 values, representing the variance explained by fixed predictors alone, ranged from 0.10 (PSS-14) to 0.30 (PHQ13K), closely matching the primary hierarchical regression results. Conditional R^2 values, which include the GRM random effect, increased substantially (e.g., PHQ13K: 0.30-0.62), showing that accounting for relatedness improved overall model fit (Table 9).

Table 9. Variance Components and Explained Variance Across Outcomes (Sensitivity LMMs)

Outcome	ICC	R^2_{marginal}	$R^2_{\text{conditional}}$
HSCL-25	0.43	0.239	0.569
PHQ13K	0.45	0.303	0.617
PSS-14	0.20	0.100	0.283
PCL-5	0.45	0.207	0.564

Fixed Effects

The pattern of fixed effects in the mixed models was highly consistent with the primary hierarchical regression analyses. Total trauma exposure remained a strong and statistically significant predictor across all outcomes. Female gender was associated with higher levels of PTSD, depression/anxiety, and somatic symptoms, while higher educational attainment remained protective. Social support showed a modest negative association with symptoms, though it did not consistently reach statistical significance. Age and social status associated minimally once trauma and socioeconomic factors were included (Table 10).

The magnitude and direction of these effects were largely unchanged compared to the hierarchical regression models, and no substantial reversals or attenuation of key predictors were observed after accounting for clustering among related participants. These findings indicate that the primary regression results are robust to familial clustering.

Although the GRM random effect captured additional variance in symptom levels, these estimates should be interpreted cautiously. The GRM reflects overall relatedness between participants and may also include shared environmental influences, such as family environment or shared trauma experiences. Therefore, these variance components do not represent direct estimates of heritability.

Table 10. Fixed Effects for PTSD Symptoms (PCL-5)

Predictor	Estimate	SE	t	95% CI	p
Age	-0.06	0.07	-0.91	-0.19-0.07	.364
Gender (female)	8.09	1.56	5.17	5.01-11.16	< .001
Education	-3.25	0.73	-4.47	-4.68– -1.82	< .001
Social Status	-0.98	1.16	-0.85	-3.26-1.29	.396
Total Trauma	1.23	0.16	7.78	0.92-1.54	< .001
Social Support	-2.34	1.35	-1.73	-4.98-0.31	.084

Note. Coefficients represent the expected change in PCL-5 scores per unit change in the predictor, holding other variables constant.

Hierarchical Regression Analyses (Yazidi survivors, N = 358)

Hierarchical multiple regression analyses were conducted to examine predictors of mental health outcomes in the Yazidi cohort. Predictors were entered in three theoretically informed blocks: demographics (Block 1: gender, age, education, occupation, residential location, social status, family history of disorder), psychosocial factors (Block 2: social support, psychotropic medication use), and trauma/health variables (Block 3: total cumulative trauma, chronic disease, diagnosis). Ordinary least squares models were estimated, and standardized beta coefficients were reported from the final Block 3 models.

In Block 1, demographic variables alone explained between 9% and 26% of the variance across outcomes, with the highest explained variance observed for PHQ-14 and the lowest for PSS-14. This suggests that basic demographic characteristics, particularly gender, education, and occupation, associated meaningfully to differences in mental health symptoms in this sample.

Adding psychosocial factors in Block 2 led to only minimal increases in explained variance ($\Delta R^2 = 0.003-0.010$, $f^2 < 0.02$), indicating that once demographic differences

are accounted for, social support and psychotropic medication use contribute relatively little additional predictive power.

In Block 3, trauma and health-related variables produced the largest increases in explained variance ($\Delta R^2 = 0.012-0.117$, $f^2 = 0.013-0.181$), primarily driven by cumulative trauma. The final models explained between 11% and 35% of variance across outcomes, with the strongest effects observed for PCL-5 and HSCL-25. These results highlight that, while demographics shape baseline mental health differences, cumulative trauma exposure has the most substantial impact on symptom severity, emphasizing the importance of trauma-informed interventions in this population (Table 11).

Table 11. Hierarchical Regression Model Fit (Yazidi survivors Cohort). Model fit across blocks, demographics explained between 9% (PSS-14) and 26% (PHQ-14) of variance. Psychosocial factors associated minimally once demographics were accounted for ($\Delta R^2 = 0.003-0.010$, $f^2 < 0.02$). And trauma and health variables added the largest increase in explained variance ($\Delta R^2 = 0.012-0.117$, $f^2 = 0.013-0.181$), primarily driven by cumulative trauma.

Outcome	Block 1 R²	Block 2 R²	ΔR^2 Block 2 (f^2)	Block 3 R²	ΔR^2 Block 3 (f^2)
PCL-5	0.232	0.238	0.006 (0.007)	0.354	0.117 (0.181)
HSCL-25	0.229	0.233	0.004 (0.005)	0.344	0.111 (0.170)
PHQ-14	0.263	0.273	0.010 (0.014)	0.314	0.041 (0.060)
PSS-14	0.094	0.096	0.003 (0.003)	0.108	0.012 (0.013)

Cumulative trauma consistently showed the strongest association across outcomes, indicating that higher cumulative trauma is linked to worse mental health. Education showed protective effects; higher years of education predicted lower symptom levels. Gender, age, social support, and prior diagnosis had weaker effects.

In the final models, cumulative trauma emerged as the strongest predictor across all outcomes, highlighting that higher cumulative trauma exposure is consistently associated with more severe mental health symptoms. Participants with greater trauma reported higher scores on PCL-5, HSCL-25, PHQ-14, and PSS-14, with the largest standardized effects observed for PCL-5 ($\beta = 0.37$) and HSCL-25 ($\beta = 0.36$).

Education demonstrated a protective effect, with higher educational attainment associated with lower symptom levels across all outcomes. The effects of gender, age, social support, psychotropic medication use, and prior diagnosis were smaller and varied across outcomes. For example, gender was positively associated with symptom severity for PCL-5, HSCL-25, and PHQ-14, but showed little effect on perceived stress (PSS-14). Age and psychotropic medication use had modest associations, while social support associated minimally once trauma and demographics were accounted for. Prior diagnosis showed small negative associations, indicating that participants with a previous mental health diagnosis did not consistently report higher symptom levels in the models (Table 12).

Table 12. Key Block 3 Predictors (Standardized β)

Predictor	PCL-5	HSCL-25	PHQ-14	PSS-14
Cumulative trauma	0.37	0.36	0.19	0.11
Education	-0.23	-0.23	-0.19	-0.08
Gender	0.13	0.20	0.21	-0.08
Age	0.10	0.08	0.22	0.16
Social Support	0.10	0.08	0.08	0.06
Psychotropics	0.07	0.07	0.11	-0.02
Prior Diagnosis	-0.19	-0.15	-0.15	-0.01

Note: Table displays

, full coefficients in Appendix table A11.

Variance Inflation Factor (VIF) values remained low (1.09-1.50), indicating no multicollinearity. Durbin-Watson values ranged between 1.85 and 1.92 and were within acceptable limits.

Model diagnostics indicated that the main regression assumptions were generally satisfied. VIF values ranged from 1.09 to 1.50, suggesting no evidence of problematic multicollinearity among predictors. Durbin-Watson statistics ranged from 1.85 to 1.92, indicating no meaningful autocorrelation in the residuals.

Shapiro-Wilk tests suggested deviations from normality for PCL-5, HSCL-25, and PSS-14 ($p < .05$), whereas PHQ-14 did not significantly deviate from normality. Given the sample size and the known robustness of regression to moderate non-normality, robust standard errors were applied to ensure reliable inference (Table 13).

Table 13. Block 3 Model Diagnostic it is overall clean .

Outcome	Max VIF	Durbin-Watson	Shapiro-Wilk p
PCL-5	1.50	1.85	<0.001
HSCL-25	1.50	1.85	0.018
PHQ-14	1.50	1.85	0.187
PSS-14	1.50	1.92	0.017

Sensitivity Analyses Accounting for Familial Relatedness

Sensitivity analyses were conducted using linear mixed models with a genetic relatedness matrix (GRM) to account for non-independence among biologically related participants. The results were consistent with the primary regression analyses, indicating that relatedness did not meaningfully influence the observed associations.

Separate models were fitted for four outcomes: PTSD symptoms measured by the PCL-5, anxiety and depression symptoms measured by the HSCL-25, somatic symptoms measured by the PHQ-14, and perceived stress measured by the PSS-14.

Variance Components and Clustering

Intraclass correlation coefficients (ICCs) indicated moderate clustering of symptom levels among related individuals, with ICCs ranging from 0.48-0.49 for PCL-5 and HSCL-25, 0.41 for PHQ-14, and 0.23 for PSS-14 (Table 14). The corresponding Z-ratios for the random effects were 2.77 (PCL-5), 2.82 (HSCL-25), 2.02 (PHQ-14), and 1.43 (PSS-14), suggesting statistically significant clustering for PTSD and depression/anxiety outcomes, while clustering for perceived stress was weaker and not statistically significant.

For somatic symptoms (PHQ-14) and perceived stress (PSS-14), the variance components associated with relatedness were smaller (PHQ-14: 15.68; PSS-14: 4.70) compared with the residual variance (PHQ-14: 22.92; PSS-14: 15.89), showing weaker clustering for these outcomes.

Overall, the relative magnitude of the random-effect and residual variance components suggests that clustering among related individuals was substantial for PTSD and anxiety/depression, but weaker for somatic symptoms and perceived stress.

These estimates should be interpreted cautiously: the GRM captures overall similarity between participants and may also reflect shared environmental exposures, such as shared trauma experiences or living conditions. Therefore, these values should not be interpreted as direct measures of genetic heritability.

Table 14. Random Effect Variance Components (LMM) for Mental Health Outcomes in the Yazidi survivors cohort.

Outcome	GRM Random Effect Variance	Residual Variance	Z-ratio
PCL-5	76.97	84.20	2.77
HSCL-25	74.74	79.13	2.82
PHQ-14	15.68	22.92	2.02
PSS-14	4.70	15.89	1.43

Fixed Effects and Comparison With Hierarchical Regression

The marginal R^2 values from the mixed models, representing variance explained by fixed predictors alone, ranged from 0.094 to 0.342. These values are highly consistent with the final R^2 values obtained from the hierarchical regression analyses (0.108-0.354).

This consistency indicates that the explanatory contribution of cumulative trauma, education, gender, and other predictors remained stable after accounting for genetic relatedness. cumulative trauma continued to show the strongest association across outcomes, particularly for PCL-5 ($\beta = 0.37$) and HSCL-25 ($\beta = 0.36$). Education retained its protective effect, while gender showed moderate outcome-specific associations. Psychosocial variables showed smaller effects once demographics and trauma exposure were included..

The GRM random effect increased conditional R^2 values to 0.30-0.65. This 0.30 increase reflects clustering due to relatedness (genetic and shared environmental factors), rather than narrow-sense heritability.

However, these higher conditional R^2 values should not be interpreted as indicating that genetic factors explain most of the symptom severity. Rather, they indicate that symptom levels cluster within genetically similar individuals in this specific cohort (Table 15).

Overall, the mixed model analyses support the robustness of the primary hierarchical regression findings. The magnitude and direction of key predictors remained largely unchanged after adjusting for genetic relatedness, and no meaningful changes in the results were observed.

These results suggest that the associations between cumulative trauma exposure and mental health outcomes are not an artifact of familial clustering. At the same time, the findings highlight that related individuals share similarities in symptom levels, underscoring the importance of accounting for non-independence in genetically informed samples.

Table 15. Marginal and Conditional R^2 for LMM Sensitivity Analyses

Outcome	R^2_{marginal} (fixed only)	$R^2_{\text{conditional}}$ (fixed + random)
PCL-5	0.338	0.654
HSCL-25	0.331	0.656
PHQ-14	0.342	0.609
PSS-14	0.094	0.301

Genetic ancestry and population structure

The Multidimensional scaling (MDS) analysis with 1000 Genomes reference populations revealed clear population structure within the study sample. As shown in Figure 10, the Kurdish samples clustered closest to the European (EUR) reference group, while remaining distinct from African, East Asian, South Asian, and admixed American populations. This clustering indicates that, genetically, the samples are most similar to Europeans among the superpopulations included, but they still form a separate, cohesive group.

Clear clustering differences were observed between the Yazidi survivors and chemical gas samples, consistent with underlying population structure. Within-group variation across genotyping batches (Kurd1, Kurd2, and Kurd3) was small and may reflect technical batch effects. These differences do not appear to meaningfully alter the overall clustering pattern.

The internal MDS plot of the study sample alone (Figure 11) confirmed that all participants fell within the expected range for the Kurdish cohorts, and no extreme ancestry outliers were identified. Together, these findings indicate that the sample is appropriate for downstream analyses without the need to exclude individuals because of extremely divergent ancestry. Nevertheless, distinct ancestral heterogeneity is present between the Yazidi group and the chemical gas survivors group.

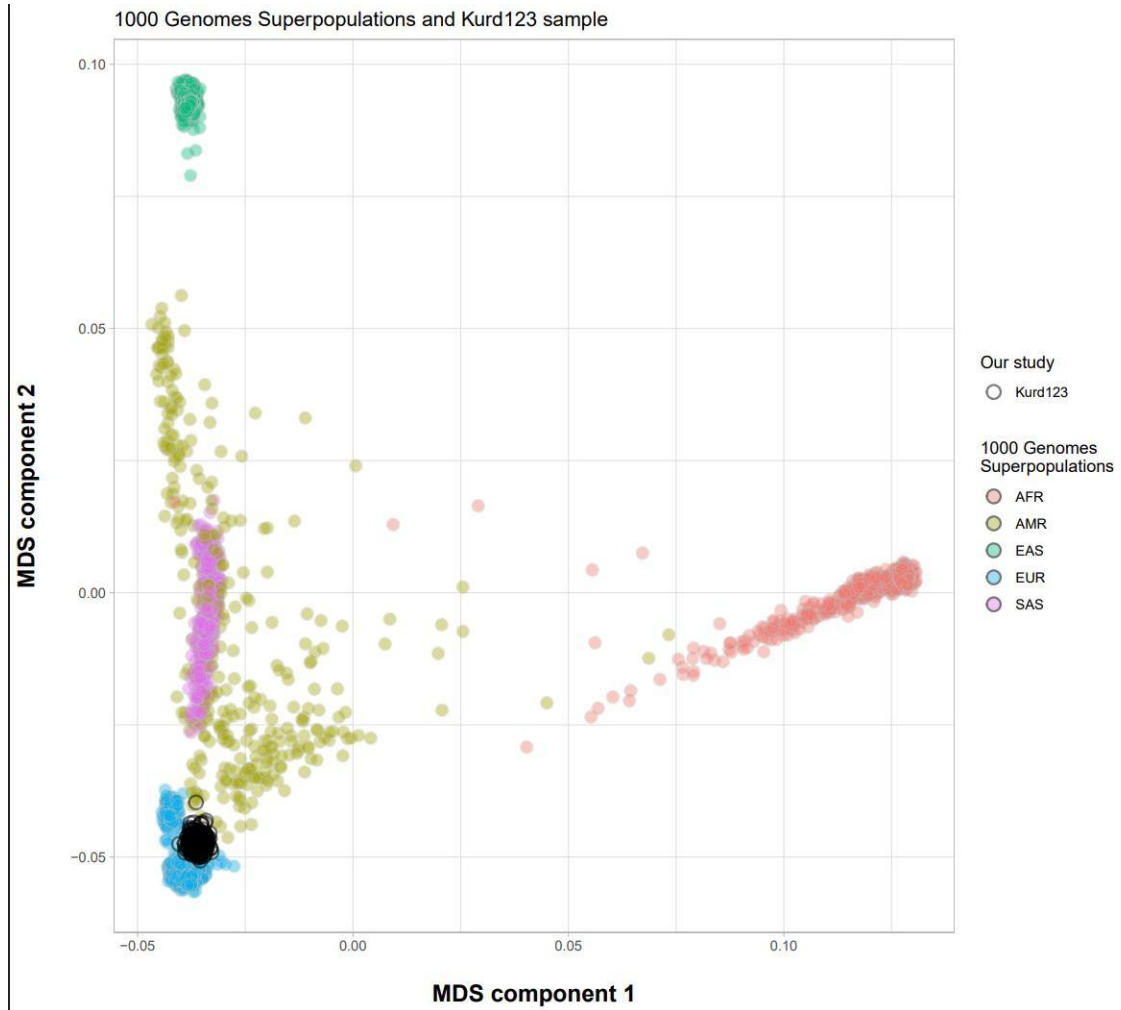


Figure 10. Multidimensional scaling (MDS) plot showing the genetic ancestry of the study sample relative to reference populations from the 1000 Genomes Project. Each point represents an individual, colored by cohort or batch. The Kurdish samples cluster closest to the European (EUR) superpopulation while remaining distinct from African (AFR), East Asian (EAS), South Asian (SAS), and Admixed American (AMR) populations, illustrating overall population structure and ancestry composition.

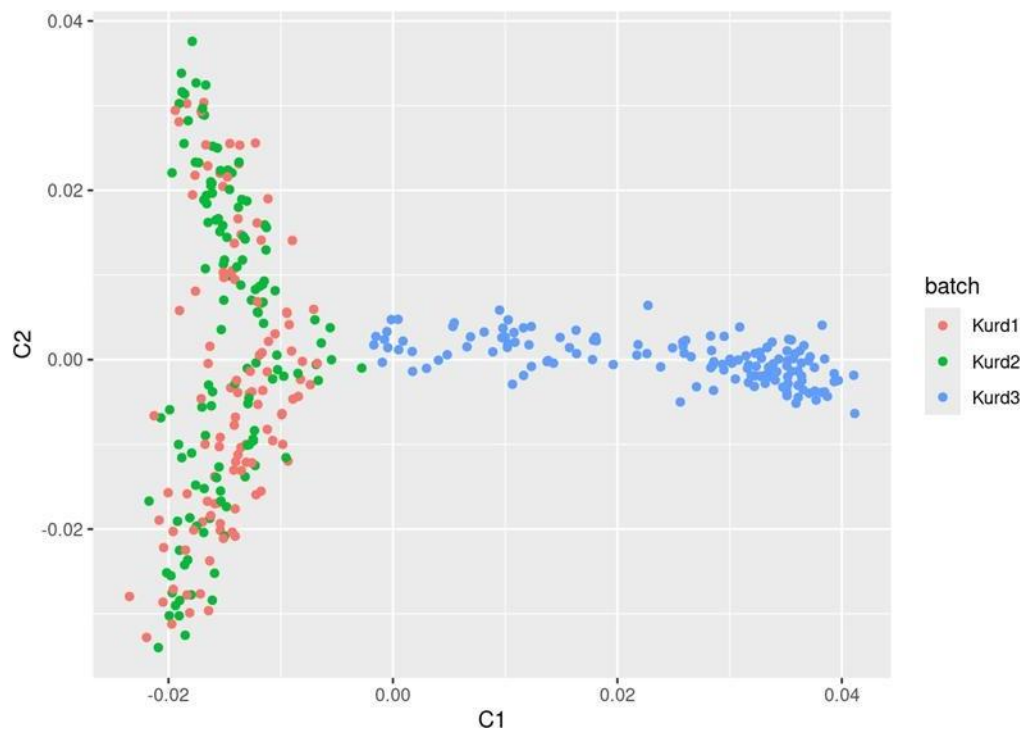


Figure 11. MDS plot showing population structure within the Kurdish cohorts. chemical gas survivors exposure (red/green) and Yazidi survivors (blue) are plotted along the first two components (C1 and C2). The plot illustrates genetic clustering across cohorts.

Polygenic Risk Score Analyses (chemical gas survivors group)

The analytic sample consisted of 499 participants from the chemical gas survivors cohort who had complete phenotypic and genotypic data and were included in the genomic relationship matrix (GRM). Key variables included total trauma exposure (standardized cumulative trauma score), PTSD symptom severity (PCL-5 total score, range 0-80), PTSD polygenic risk score (PRS; standardized based on 2024 European-ancestry GWAS, clumped at $r^2 < 0.1$ within 500 kb), age (standardized), gender (coded as 1 = male, 2 = female), and education level (1 = none, 2 = primary, 3 = secondary, 4 = university). The first 10 genomic principal components (PC1-PC10) were included to account for population stratification. All continuous predictors were standardized to z-scores to allow comparison of effect sizes, and categorical variables were factor-coded with reference levels (Male for gender, Level 1 for education).

Linear Mixed Model Analysis

A linear mixed-effects model was used to predict PTSD symptom severity (PCL-5 total score) from total trauma exposure, PTSD polygenic risk score (PRS), and their interaction, while controlling for age, gender, education, and the first 10 genomic principal components. A random effect structured by the genetic relatedness matrix (GRM) was included to account for relatedness among participants. The model was fitted using the *mmer* function from the *sommer* package (version 4.4.3) and

converged after five iterations. Model assumptions of linearity and homoscedasticity were evaluated using residual diagnostics (all $P > 0.10$), and no convergence issues were observed.

Fixed Effects

Table 16 presents the fixed-effect estimates from the linear mixed model predicting PCL-5 scores. Trauma exposure was a strong predictor of symptom severity. PTSD PRS showed a smaller but significant main effect, and the interaction between trauma and PRS indicated that genetic risk moderated the association between trauma and PTSD symptoms (Figure 12). Females reported higher symptom levels, while higher education was associated with lower scores. Age and population principal components were not significant predictors.

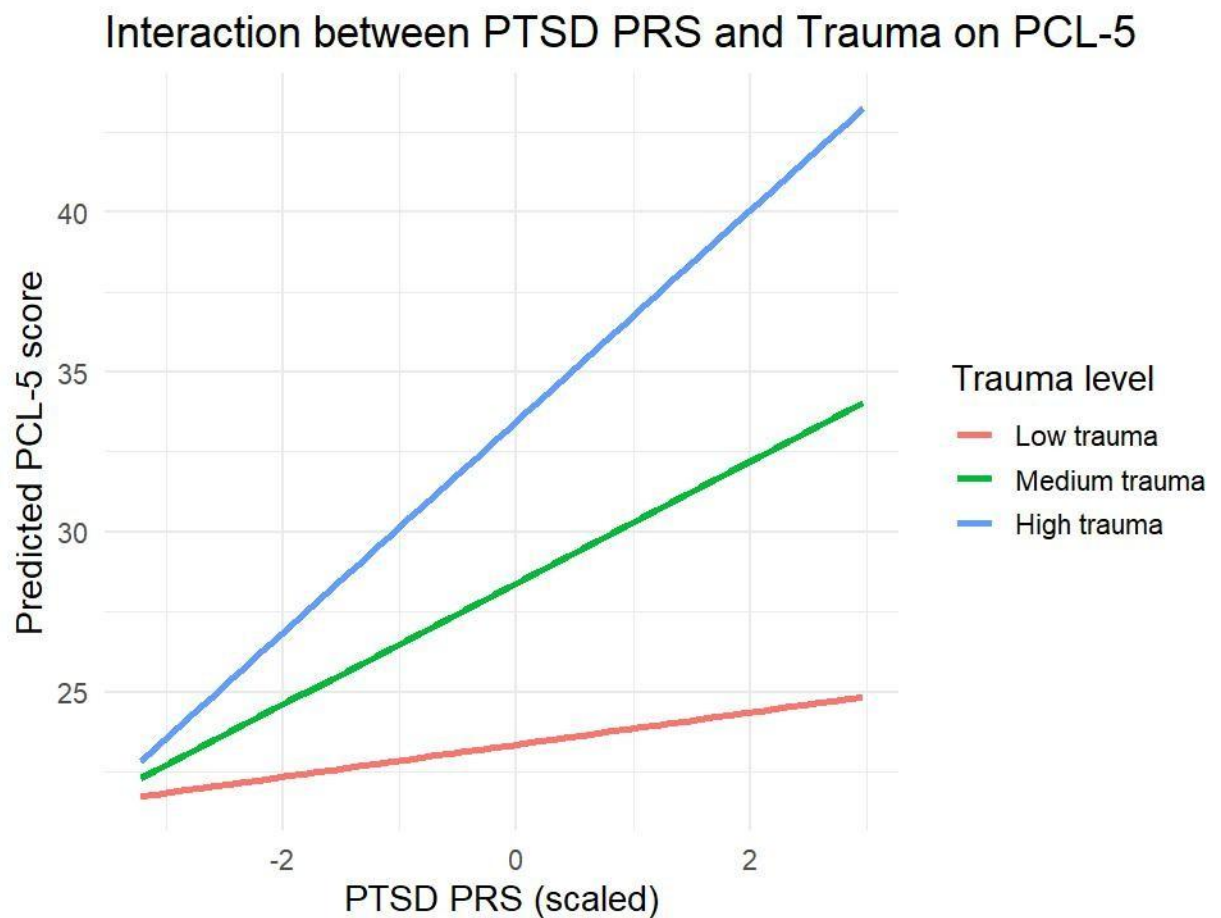
Table 16. Fixed-Effect Estimates from LMM Predicting PCL-5 Total Scores (n = 499)

Effect	Estimate	Std. Error	t-value	p-value	95% CI Lower	95% CI Upper
Cumulative trauma	5.20	0.76	6.88	<0.001	3.71	6.68
PTSD PRS	1.91	0.73	2.61	0.009	0.47	3.34
Age	-0.81	0.73	-1.10	0.271	-2.25	0.63
Gender_Female	8.03	1.54	5.21	<0.001	5.00	11.05
Basic Education	-3.80	1.82	-2.09	0.037	-7.37	-0.24
High School Education	-8.36	2.46	-3.40	<0.001	-13.17	-3.55
University	-11.31	2.52	-4.50	<0.001	-16.24	-6.38
PC1	2.68	2.61	1.02	0.305	-2.44	7.80
PC2	8.52	5.98	1.43	0.154	-3.20	20.24
PC3	-3.38	4.96	-0.68	0.496	-13.10	6.35
PC4	0.85	1.14	0.75	0.455	-1.38	3.09
PC5	-2.03	1.33	-1.53	0.126	-4.62	0.57

PC6	-2.82	2.31	-1.22	0.222	-7.35	1.70
PC7	-7.08	4.58	-1.55	0.122	-16.06	1.89
PC8	-7.25	4.11	-1.77	0.077	-15.30	0.80
PC9	-1.06	1.48	-0.72	0.475	-3.95	1.84
PC10	0.26	1.06	0.24	0.808	-1.83	2.35
Trauma and PTSD PRS interaction	1.46	0.71	2.04	0.039	0.06	2.86

Note. β = unstandardized (except standardized predictors). PCs summarized (full table in Supplement Y). $p < .001$ reported as such.

Figure 12. Interaction between PTSD polygenic risk score (PRS) and trauma exposure predicting PTSD symptom severity (PCL-5). Predicted PCL-5 scores are shown across levels of PTSD PRS for low, medium, and high trauma exposure.



Random Effects and Familial Clustering

A linear mixed model examined PTSD symptom severity (PCL-5 total score), accounting for trauma exposure, PTSD polygenic risk score (PRS), and covariates, with a random effect for participant-level genetic relatedness based on the GRM. Variance components from the model are presented in Table 17. The variance attributable to the random effect based on the genetic relationship matrix (GRM) was 93.93 (SE = 31.72, $Z = 2.96$), while the residual variance was 109.78 (SE = 28.40, $Z = 3.87$), yielding an intraclass correlation (ICC) of 0.46 (Table 17).

The ICC (0.46) indicates moderate clustering of PCL-5 scores among related individuals, reflecting shared genetic and environmental similarity captured by the GRM, not narrow SNP-based heritability.

Table 17. Random Effects from the Linear Mixed Model Predicting PTSD Symptom Severity

Random Effect	Variance (σ^2)	Standard Error (SE)	Z-value
GRM	93.93	31.72	2.96
Residual	109.78	28.40	3.87

Variance Explained (Pseudo- R^2)

Table 18 summarizes the proportion of total variance in PTSD symptom severity explained by different components of the genomic linear mixed model. Fixed effects (trauma exposure, PRS, and covariates) accounted for 25.6% of total variance in PTSD symptoms (marginal $R^2 = 0.256$). The GRM-based intraclass correlation (ICC = 0.46) indicates moderate clustering of PCL-5 scores among related individuals, reflecting shared genetic and environmental similarity captured by the model. The PTSD PRS explained an additional 1.8% of variance beyond trauma and covariates (partial $R^2 = 0.018$, $p = 0.009$). The remaining 54.0% of variance was unexplained residual variation. These results indicate that both measured environmental exposures and genetic factors are meaningfully associated with individual differences in PTSD symptoms in this cohort.

Table 18. Total variance in PTSD symptom severity explained by different components of the genomic linear mixed model.

Component	$R^2_{\text{mar,g}}$	% of Total Variance
Fixed Effects	0.256	25.6%
Genetic Relatedness	0.46%	46.0%

PTSD PRS	0.018	1.8%
Residual	—	54.0%

Note. The percentages represent how much of the total variation in PTSD symptom scores is explained by different parts of the model. Fixed effects (trauma, PTSD PRS, their interaction, and covariates) accounted for 25.6% of the total variation. The GRM-based ICC (0.46) indicates moderate clustering of PCL-5 scores within related individuals. The PTSD PRS contributed an additional 1.8% of variance beyond trauma and covariates, while 54.0% of the variance remained unexplained.

Sensitivity analysis

We assessed multicollinearity using variance inflation factors (VIF). The main predictors total trauma (VIF = 1.39), PTSD PRS (VIF = 1.15), and their interaction (VIF = 1.10) showed no evidence of problematic collinearity. Some genetic principal components had elevated VIF values, so we reran the model using a reduced set of PCs (PC1-PC5). The main results remained consistent, and variance components for the genetic random effect and residuals were stable across models (82-89 and 112-116, respectively).

Sample Characteristics and Genetic Structure (Yazidi Survivors)

Genetic relatedness screening of the 342 genotyped individuals revealed substantial familial clustering typical of a community-ascertained Kurdish cohort (Table 19). Of these, 124 individuals (36.3%) had no first- or second-degree relatives and were classified as independent. Twenty-three individuals (6.7%) had only second-degree relatives, 100 (29.2%) had only first-degree relatives, and 95 (27.8%) had both first- and second-degree relatives, such as in multi-generational families.

Table 19. Sample Composition by Relatedness Category (N = 342)

Category	N People	% of Sample
Independent	124	36.3
2nd-degree only	23	6.7
1st-degree only	100	29.2
Both 1st & 2nd	95	27.8

Note. Thresholds: first-degree > 0.25; second-degree > 0.125. Percentages rounded to one decimal place.

Overall, 218 individuals (63.7%) had at least one close relative, consistent with pedigree structure in the Yazidi population. No pairs exceeded relatedness of 0.9, indicating absence of sample duplicates or monozygotic twins.

Linear Mixed Model Analysis (Main-Effects Model)

To examine the influence of genetic liability and trauma exposure on PTSD symptom severity, a linear mixed model (LMM) was fitted with PCL-5 total score as the outcome variable. Fixed effects included standardized PTSD polygenic risk score (PRS), standardized total trauma exposure, age, gender, and education level, along with the first ten genetic principal components (PC1-PC10) to control for population stratification. Genetic relatedness among participants was accounted for using a genomic relationship matrix (GRM) as a random effect.

The model converged successfully and produced stable parameter estimates (Table 20). Trauma exposure emerged as the strongest predictor of PTSD symptom severity. Specifically, a one-standard-deviation increase in trauma exposure was associated with an increase of 4.61 points in PCL-5 total score ($B = 4.61$, $SE = 0.96$, $t = 4.80$, $p < .001$, 95% CI [2.73, 6.49]).

Educational attainment was significantly associated with lower PTSD symptoms. Each unit increase in education level was linked to a reduction of 3.93 points in PCL-5 score ($B = -3.93$, $SE = 0.87$, $t = -4.53$, $p < .001$, 95% CI [-5.63, -2.23]). Gender was also a significant predictor, with females showing higher PTSD symptom severity compared to males ($B = 4.96$, $SE = 1.81$, $t = 2.74$, $p = .006$, 95% CI [1.42, 8.51]). Age showed a positive but non-significant association with PTSD symptoms ($B = 1.49$, $SE = 0.88$, $t = 1.70$, $p = .089$).

The PTSD polygenic risk score did not significantly predict PTSD symptom severity ($B = 0.96$, $SE = 0.85$, $t = 1.13$, $p = .259$, 95% CI [-0.71, 2.64]). None of the genetic principal components (PC1-PC10) showed significant associations with PCL-5 scores, indicating that population stratification did not meaningfully influence the results.

Gene-Environment Interaction

To test whether genetic risk moderated the effect of trauma exposure on PTSD symptoms, an interaction term between PTSD PRS and trauma exposure was included in the model. The interaction was not statistically significant ($B = 0.02$, $SE = 0.79$, $t = 0.02$, $p = .981$, 95% CI [-1.57, 1.54]), indicating that genetic risk did not modify the relationship between trauma exposure and PTSD symptom severity.

Variance Components and Familial Clustering

Variance components from the mixed model indicated substantial variation attributable to genetic relatedness. The estimated variance for the random effect was

104.48 and the residual variance was 80.65, yielding an intraclass correlation (ICC) of 0.56. This indicates substantial clustering of PCL-5 scores among related individuals captured by the GRM. This estimate does not represent SNP-based heritability. Fixed effects accounted for 25% of total variance.

Table 20. Linear Mixed Model Results for PTSD Symptom Severity (PCL-5)

Effect	Estimate	SE	<i>t</i>-value	<i>p</i>-value	95% CI Lower	95% CI Upper
PTSD PRS	0.96	0.85	1.13	.259	−0.71	2.64
Cumulative trauma	4.61	0.96	4.80	< .001	2.73	6.49
Age	1.49	0.88	1.70	.089	−0.23	3.21
Gender (female vs male)	4.96	1.81	2.74	.006	1.42	8.51
Education	−3.93	0.87	−4.53	< .001	−5.63	−2.23
PC1	−2.71	3.97	−0.68	.494	−10.48	5.06
PC2	12.20	9.73	1.25	.209	−6.86	31.27
PC3	10.72	7.51	1.43	.153	−3.99	25.43
PC4	1.71	1.58	1.08	.280	−1.39	4.81
PC5	0.04	2.03	0.02	.983	−3.92	4.02
PC6	−2.97	3.54	−0.84	.401	−9.88	3.96
PC7	3.89	6.94	0.56	.575	−9.72	17.50
PC8	2.17	6.47	0.33	.738	−10.52	14.86
PC9	−0.36	2.41	−0.15	.881	−5.09	4.37
PC10	−1.00	1.65	−0.61	.544	−4.23	2.23

Effect	Estimate	SE	t-value	p-value	95% CI Lower	95% CI Upper
PTSD PRS × cumulative trauma	0.02	0.79	0.02	.981	-1.57	1.54

Sensitivity Analysis

To assess the robustness of these findings, several sensitivity analyses were conducted:

Outlier Removal: Two participants with the highest Cook’s distances were removed to assess the impact of extreme values. Re-fitting the linear mixed model without these cases yielded results nearly identical to the main analysis (Trauma: $B = 4.78$, $SE = 0.93$; PRS: $B = 0.80$, $SE = 0.84$), indicating that outliers did not influence the overall findings.

2. Gender-Specific Analyses: Separate models were fitted for males ($N = 100$) and females ($N = 241$). Trauma exposure remained a strong predictor of PTSD symptoms in both groups (males: $B = 4.73$, $SE = 1.27$; females: $B = 3.99$, $SE = 0.88$). The PTSD polygenic risk score (PRS) showed a larger effect in males ($B = 3.98$, $SE = 1.78$) compared to females ($B = -0.51$, $SE = 0.51$), suggesting possible sex-specific differences in genetic influence. No interaction between PRS and trauma exposure was included in the sex-stratified models, consistent with the non-significant interaction in the main model.

3. OLS Benchmark: A standard linear regression without the genomic relationship matrix was also fitted. Estimates for both trauma exposure ($B = 4.87$, $SE = 0.94$) and PRS ($B = 0.81$, $SE = 0.82$) were similar to the main mixed model, supporting the robustness of the findings.

Across all specifications, trauma exposure consistently predicted higher PTSD symptom severity, while the PRS effect was generally small and non-significant, except for a moderate effect in males. The intraclass correlation from the original model ($ICC = 0.56$) remained stable across sensitivity analyses (Table 21).

Table 21. Sensitivity analyses for key fixed effects on PCL-5 total scores

Analysis	N	PRS ($B \pm SE$)	Trauma ($B \pm SE$)
Main LMM	342	0.87 ± 0.83	4.68 ± 0.92 ***
OLS (no GRM)	342	0.81 ± 0.82	4.87 ± 0.94 ***

Outliers removed	339	0.80 ± 0.84	4.78 ± 0.93 ***
Males only	100	3.98 ± 1.78	4.73 ± 1.27 **
Females only	242	-0.51 ± 0.51	3.99 ± 0.88 ***

B = unstandardized regression coefficient; SE = standard error.

* Significance levels: *** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$.

Polygenic Risk Score Analyses Across Cohorts

To compare genetic influences on PTSD symptoms across the two Kurdish trauma- exposed cohorts, we ran parallel PRS models in chemical gas survivors attacks and Yazidi survivors. In both cohorts, higher trauma exposure was strongly associated with higher PTSD symptoms, with each standard deviation increase in trauma linked to an increase of about 5 points on the PCL- 5 ($p < .001$). In contrast, the PTSD polygenic risk score (PRS) and its interaction with trauma were significant only in the chemical gas cohort (PRS: $\beta = 1.8-1.9$, $p = .01$; PRS $\times$ trauma: $\beta = 1.4-1.5$, $p = .039$), but not in the Yazidi cohort (PRS: $p = .29-.80$; PRS $\times$ trauma: $p = .66-.91$). The total variance explained by the models was similar across cohorts ($R^2 = 0.24-0.28$), showing that while trauma exposure was a strong and consistent predictor of PTSD, the effects of genetic risk and its interaction with trauma were not the same across the two groups (Table 22, Figure 13).

Table 22. PRS effects across cohorts

Predictor	chemical gas survivors (N = 499)	Yazidi Survivors (N = 342)
PTSD PRS	1.76 ($p = 0.010$)*	3.51 ($p = 0.803$)
Cumulative trauma	4.99 ($p < 0.001$)**	5.31 ($p < 0.001$)**
PRS $\times$ cumulative trauma	1.44 ($p = 0.039$)*	6.80 ($p = 0.665$)
Model R^2	0.245	0.278

* $p < 0.05$, ** $p < 0.001$

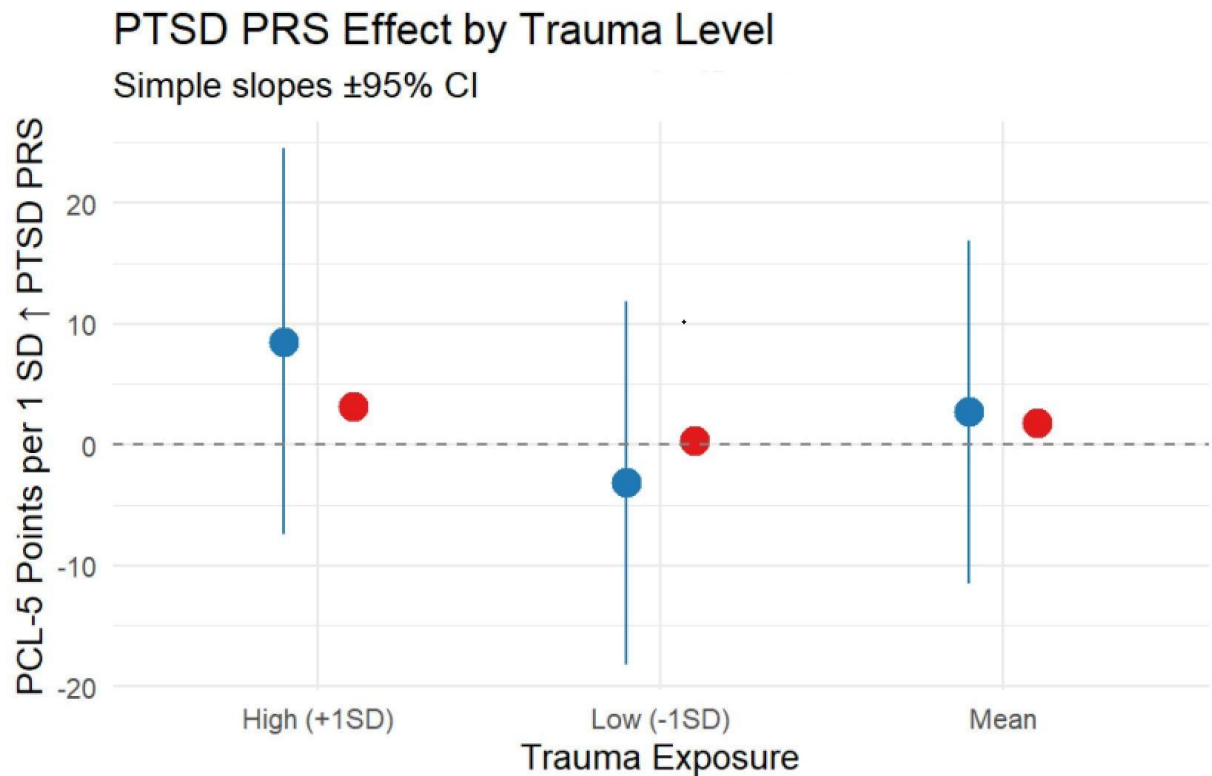


Figure 13. Simple slopes analysis showing the association between PTSD polygenic risk score (PRS) and PTSD symptom severity (PCL-5) at different levels of trauma exposure in two Kurdish cohorts. Red points represent the chemical gas survivor cohort, where the PRS $\times$ trauma interaction was significant ($p = .039$), indicating that the association between PRS and PTSD symptoms varied across trauma levels. Blue points represent the Yazidi survivor cohort, where the interaction was not significant ($p = .665$). Points indicate estimated effects of PRS on PCL-5 scores, and error bars represent 95% confidence intervals.

Demographic and Clinical Differences Between Cohorts

PTSD polygenic risk score (PRS) was not included in the following analyses.

- **Occupation**

There were substantial differences in occupation between groups, $\chi^2(6) = 491.87$, $p < .001$, Cramer's $V = .743$. For example, 42.7% of chemical gas victims were housewives compared to 13.7% among Yazidi survivors, while 34.9% of Yazidi survivors were students versus 0% in the chemical group.

- **Residential location**

Residential location was different between the two groups, $\chi^2(3) = 810.21$, $p < .001$, Cramer's $V = .953$. Almost all chemical gas victims lived inside the city (92.1%), whereas 60.6% of Yazidi survivors lived in camps and 36.6% in villages.

- **Marital Status**

Marital status showed large group differences, $\chi^2(3) = 408.65$, $p < .001$, Cramer's $V = .677$. The majority of Yazidi survivors were single (74.6%), while 83.1% of chemical gas victims were married.

- **Social Support**

A significant difference in social support availability was observed, $\chi^2(2) = 287.06$, $p < .001$, Cramer's $V = .567$. 40.3% of chemical victims reported no social support, while none of the ISIS survivors did.

- **Family Mental Disorder**

There was no significant difference between groups in terms of having a family member with a mental disorder, $\chi^2(1) = .003$, $p = .954$, Cramer's $V = .002$.

- **Chronic Disease**

Chronic disease prevalence differed significantly, $\chi^2(2) = 200.11$, $p < .001$, Cramer's $V = .474$. Most chemical victims (68.7%) reported chronic illness, compared to only 21.5% of ISIS survivors (Table 23).

Table 23. summarizes key demographic differences between chemical gas victims and Yazidi survivors. Significant differences were found in occupation, residential location, marital status, social support and chronic disease prevalence (higher in chemical victims).

Variable	Chi-Square	p-value	Cramer's V	Notable Finding
Gender	21.45 (1)	< .001	.155	More women in Yazidi group
Occupation	491.87 (6)	< .001	.743	Chemical: more housewives, Yazidi: more students
Residential location	810.21 (3)	< .001	.953	Chemical: city; Yazidi: camps/villages
Marital Status	408.65 (3)	< .001	.677	Chemical: married; Yazidi: single
Social Support	287.06 (2)	< .001	.567	Chemical: more reported no support
Family Mental Disorder	0.003 (1)	.954	.002	No significant difference
Chronic Disease	200.11 (2)	< .001	.474	Chemical: more with chronic illness

Given the substantial baseline differences between cohorts, predictor $\times$ cohort interactions were tested to examine whether the effects of key demographic and psychosocial variables differed across trauma types (Appendix Table A13).

Hierarchical Regression Analyses (combined sample)

Hierarchical regression analyses were conducted to examine predictors of four mental health outcomes in the full combined sample (N = 892): PTSD symptoms (PCL-5 total), anxiety/depression symptoms (HSCL-25 total), perceived stress (PSS-14 total), and somatic symptoms (PHQ-13K total). Predictors were entered sequentially in three blocks to assess their incremental contributions:

Block 1: Demographics- age, gender, education, occupation, and residential location.

Block 2: Psychosocial factors- social status, social support, and psychotropic medication use.

Block 3: Trauma and health-related factors, cumulative trauma, psychiatric diagnosis, family history of mental disorders, and chronic disease.

All models used ordinary least squares (OLS) estimation. This hierarchical approach allowed evaluation of how much variance in mental health outcomes was explained by demographics alone, and the additional predictive value contributed by psychosocial and trauma/health variables.

Model Fit and Variance Explained

Demographics alone (Block 1) explained a substantial portion of variance across outcomes, with R^2 values ranging from 0.129 for perceived stress to 0.318 for anxiety/depression symptoms. Adding psychosocial factors in Block 2 provided modest but meaningful additional variance ($\Delta R^2 = 0.043$ -0.062). The largest increases occurred after including trauma and health-related variables in Block 3, particularly for PTSD and anxiety/depression outcomes, reflecting the strong influence of trauma and health factors on mental health (final $R^2 = 0.203$ -0.443; Table 24).

These results indicate that while demographics and psychosocial resources are important, cumulative trauma and health-related risk factors are the most powerful predictors of symptom severity in this population.

Hierarchical Model Fit

Table 24. Hierarchical Regression Results (Combined Sample, N = 892)

Outcome	Block 1 R^2	Block 2 R^2	ΔR^2 Block 2	f^2 Block 2	Block 3 R^2	ΔR^2 Block 3	f^2 Block 3
PCL-5	0.302	0.346	0.044	0.068	0.402	0.056	0.093

HSCL-25	0.318	0.380	0.062	0.100	0.443	0.063	0.112
PSS-14	0.129	0.190	0.061	0.075	0.203	0.014	0.017
PHQ-13K	0.223	0.276	0.053	0.074	0.308	0.032	0.046

Note: R^2 indicates the proportion of variance explained by each block of predictors. ΔR^2 represents the incremental variance explained by adding that block. f^2 is Cohen's effect size for each block, with larger values indicating stronger contributions.

Model Coefficients (Block 3)

Table 25 presents the standardized regression coefficients (β) from the final hierarchical regression models including all predictors (demographics, psychosocial factors, and trauma/health variables).

Residential location emerged as the strongest predictor for PTSD symptoms (PCL-5; $\beta = 0.457$, $p < .001$) and anxiety/depression (HSCL-25; $\beta = 0.416$, $p < .001$), suggesting that individuals' living context, such as village, camp, or urban setting, significantly influenced symptom severity. This highlights the importance of environmental and contextual factors in mental health outcomes.

Cumulative trauma showed consistent, significant effects across all four outcomes ($\beta = 0.112$ – 0.265 , $p < .01$), confirming that greater cumulative exposure to traumatic events is associated with higher symptom severity.

Psychotropic medication use was positively associated with symptoms ($\beta = 0.204$ – 0.223 , $p < .001$), likely reflecting that individuals with more severe symptoms are more likely to be on medication, rather than suggesting a causal effect of medication on symptom severity.

Education demonstrated a protective effect, with lower symptom severity observed in participants with higher educational attainment ($\beta = -0.133$ to -0.225 , $p < .001$). This finding aligns with evidence that education may buffer the impact of stress and trauma, possibly through enhanced coping resources or social advantages.

Gender effects were also observed: female participants had higher PTSD and anxiety/depression scores ($\beta = 0.163$ – 0.197 , $p < .001$), consistent with well-documented gender differences in mental health outcomes.

Overall, these results suggest that both environmental factors (residential location, trauma exposure) and individual characteristics (education, gender) play important

roles in predicting mental health outcomes. Psychotropic use appears as a marker of symptom severity rather than a causal influence. Full hierarchical regression coefficients for all predictors are available in Appendix Table A12.

Table 25. Key Standardized Betas from Final Models (Block 3)

Predictor	PCL-5	HSCL-25	PSS-14	PHQ-13K
Residential location	0.457*	0.416*	0.178**	0.120*
Cumulative trauma	0.265*	0.257*	0.112	0.207*
Psychotropics	0.204*	0.223*	0.206*	0.210*
Gender	0.163***	0.197***	-0.002	0.301*
Education	-0.189***	-0.207***	-0.133***	-0.225***

Note: ***p < .001, **p < .01, *p < .05. Complete Hierarchical Regression Coefficients presented in Appendix Table A12

Model Diagnostics

Regression diagnostics indicated that the models were generally well-specified and free from major statistical violations.

Multicollinearity was low across predictors, with the maximum variance inflation factor (VIF) observed for residential location at 2.69, well below the commonly used threshold of 5, suggesting that predictor variables were not highly correlated.

Autocorrelation of residuals was assessed using Durbin-Watson statistics, which ranged from 1.82 to 1.92 across outcomes. These values indicate only mild positive residual dependence, likely due to clustering of participants by village or camp, but not at a level that would bias regression estimates.

Residual normality was evaluated with Shapiro-Wilk tests. PCL-5 residuals did not significantly deviate from normality ($p = 0.520$), whereas minor deviations were observed for HSCL-25, PSS-14, and PHQ-13K ($p < 0.01$). To account for potential effects of non-normality, robust standard errors were applied in all models (Table 26).

These diagnostics confirm that the assumptions of ordinary least squares regression were largely met and that the models provide reliable estimates of the relationships between predictors and mental health outcomes.

Table 26. Regression Diagnostics (Block 3 Models)

Outcome	Max VIF (residential location)	Durbin-Watson	Shapiro-Wilk p
PCL-5	2.688	1.85	0.520
HSCL-25	2.688	1.82	0.007
PSS-14	2.688	1.92	<0.001
PHQ-13K	2.688	1.88	0.009

Note: VIF = variance inflation factor; values <5 indicate low multicollinearity. Durbin-Watson values close to 2 suggest minimal autocorrelation. Shapiro-Wilk $p < .05$ indicates slight deviations from normality; robust standard errors were used to mitigate this.

Cohort Interaction Effects

To examine whether associations between key predictors and mental health outcomes differed between chemical gas survivors ($n = 534$) and Yazidi survivors ($n = 358$), we tested 12 interaction terms between cohort membership and selected predictors across four outcomes: PTSD symptoms (PCL-5), anxiety/depression (HSCL-25), perceived stress (PSS-14), and somatic symptoms (PHQ-13K). Of these, five interactions reached conventional significance ($p < .05$), and three additional interactions approached significance ($p < .10$), suggesting potential cohort differences in specific predictor–outcome associations (Table 27).

Table 27. Hierarchical Regression Results (cohort interaction effects)

Outcome	Predictor	β Interaction	SE	t- value	p- value	Direction
PTSD	Psychotropics	-0.384	0.183	-2.10	.036	Yazidi stronger
PTSD	Social Support	0.242	0.106	2.28	.023	Yazidi protective
PTSD	Prior Diagnosis	-0.058	0.024	-2.46	.014	Yazidi weaker
PTSD	Family mental illness	0.303	0.120	2.52	.012	Yazidi stronger

HSCL-25	Psychotropics	-0.482	0.131	-3.68	<.001	Yazidi stronger
PSS-14	Education	-0.084	0.036	-2.37	.018	Yazidi protective
PSS-14	Gender	0.270	0.083	3.25	.001	Yazidi stronger

Note: Positive interaction coefficients indicate a stronger association in chemical gas survivors (coded 1), whereas negative coefficients indicate a stronger association in Yazidi survivors (reference group, coded 0).

PTSD Symptoms (PCL-5; $R^2 = .42$)

Cohort differences were most evident for PTSD outcomes. Psychotropic medication use showed a stronger association with PTSD symptoms in chemical gas survivors compared with Yazidi survivors ($\beta = -0.384$, $p = .036$). Social support was more strongly protective among Yazidi survivors ($\beta = 0.242$, $p = .023$), while family history of mental illness showed a stronger association in chemical gas survivors ($\beta = 0.303$, $p = .012$). Other predictors, including cumulative trauma and residential location, did not show statistically significant cohort moderation, indicating broadly comparable effects across groups.

Anxiety/Depression (HSCL-25; $R^2 = .44$)

Cohort moderation effects were limited and primarily observed for psychotropic medication use, which showed a stronger association in chemical gas survivors compared with Yazidi survivors ($\beta = -0.482$, $p < .001$). All other predictors, including cumulative trauma and residential location, did not show significant cohort differences, suggesting largely consistent associations across groups.

Perceived Stress (PSS-14; $R^2 = .22$)

Significant cohort differences were observed for education and gender. Higher education showed a stronger protective association among Yazidi survivors ($\beta = -0.084$, $p = .018$), while the association between female gender and perceived stress was stronger in Yazidi survivors ($\beta = 0.270$, $p = .001$). Psychotropic medication showed a marginal cohort difference ($\beta = -0.084$, $p = .087$). Other predictors did not show evidence of cohort moderation.

Somatic Symptoms (PHQ-13K; $R^2 = .30$)

No significant cohort interactions were observed for somatic symptoms; all predictors demonstrated equivalent effects across chemical gas and Yazidi survivors (all $p > .20$). This indicates that, unlike PTSD or anxiety/depression, somatic complaints may be influenced similarly by individual and trauma-related factors across these populations. Overall, cohort-specific moderation was most pronounced for PTSD and

anxiety/depression, with psychosocial and treatment-related predictors showing variable effects across groups. In contrast, cumulative trauma, residential location, and somatic symptoms were broadly consistent between cohorts. These findings underscore the importance of contextual factors such as living conditions, treatment access, and community support in shaping mental health outcomes in highly trauma-exposed populations. Importantly, no multicollinearity concerns were detected (all VIF < 3), and the full models maintained excellent fit ($R^2 = .40-.44$) despite inclusion of multiple interaction terms.

Discussion

This dissertation had four main aims. The first was to examine whether PTSD polygenic risk scores predict symptom severity and whether this association is moderated by cumulative trauma exposure. The second and third aims focused on the associations of sociodemographic, psychosocial, and trauma-related factors with PTSD, depression/anxiety, somatic symptoms, and perceived stress, and on whether trauma exposure adds unique explanatory value beyond these factors. The fourth aim was to compare these patterns between chemical gas survivors attacks and Yazidi genocide survivors. The following discussion interprets these findings in relation to existing theory and empirical literature.

Across the chemical survivor cohort, PTSD polygenic risk scores showed a small but consistent association with PTSD symptoms. The association became stronger at higher levels of cumulative trauma exposure, suggesting that genetic vulnerability may become more visible under conditions of greater environmental stress. At the same time, trauma exposure and broader environmental conditions remained substantially stronger predictors of mental health outcomes than genetic risk alone. These findings are in line with results from larger European-ancestry cohorts, where polygenic risk scores typically explain only about 2-5% of the variance in PTSD symptoms or diagnosis (Nievergelt et al., 2019). To our knowledge, this is the first study to examine PTSD polygenic risk among Kurdish survivors of both the chemical attacks and Yazidi survivors of IS-related violence, and it underscores the need to investigate genetic and environmental influences in populations that are largely underrepresented in genomic research.

In contrast, among Yazidi survivors, PTSD polygenic risk scores were not significantly associated with PTSD symptoms, and the interaction between PRS and cumulative trauma exposure was also not significant. Several factors may help explain this difference between cohorts. Current PTSD polygenic risk scores are primarily derived from genome-wide association studies conducted in populations of European ancestry. As a result, their predictive performance is often reduced when applied to populations with different genetic backgrounds. Reduced portability of polygenic scores has been widely documented in psychiatric and other complex traits and is generally attributed to differences in linkage disequilibrium structure, allele

frequencies, and gene-environment correlations across populations (Duncan et al., 2019). In this context, the modest signal observed among chemical attack survivors and the absence of a detectable signal among Yazidi survivors are consistent with broader findings in psychiatric genetics, which show that polygenic scores tend to perform less reliably outside the populations in which they were developed. These findings suggest that the predictive value of existing PTSD polygenic risk scores varies across populations and is currently uncertain in Middle Eastern groups due to limited genomic representation. In the present study, dose-dependent associations were observed in the chemical gas cohort, suggesting that PRS may capture a modest component of genetic liability in this cohort. In contrast, no association was found in the Yazidi cohort. This may be related to differences in genetic structure in the Yazidi population, including higher levels of endogamy, which could affect how well PRS developed in other populations apply to this group. Overall, these results highlight the importance of including more diverse populations in genomic research to better capture regional genetic differences.

Differences in statistical power may also explain part of the observed pattern. The chemical gas cohort ($N = 499$) was larger than the Yazidi cohort ($N = 342$), which may have given slightly more power to detect small genetic effects. At the same time, gene-environment interactions are generally hard to detect, and studies with even larger samples have often failed to identify or replicate them (Waszczuk et al., 2021). Because polygenic risk scores usually have small effect sizes, the smaller Yazidi sample may have reduced the chance of finding a significant association or interaction. This is in line with previous work in psychiatric genetics showing that large samples are usually needed for reliable gene-environment findings.

The two cohorts also differed substantially in the nature and context of their trauma experiences. Survivors in the chemical gas cohort were exposed to chemical attacks and probable long-term environmental consequences of toxic exposure, while Yazidi survivors experienced genocide, captivity, displacement, and prolonged insecurity. These contrasting trauma histories suggest that different forms of mass violence may involve distinct psychological and biological stress pathways and may interact differently with genetic vulnerability. Existing PTSD genome-wide association studies have focused largely on combat-related or Western disaster-related trauma, which may capture only part of the biological mechanisms relevant to genocide-related or war induced trauma (Stein et al., 2016). As a result, genetic variants identified in current polygenic risk scores may not fully reflect the biological pathways involved in the trauma experiences of these populations. Together, these factors suggest that the influence of genetic vulnerability is likely shaped by ancestry, trauma characteristics, and the broader social and environmental context.

The interaction observed between polygenic risk scores and cumulative trauma exposure in the chemical survivor cohort supports the diathesis-stress framework, which proposes that genetic vulnerability becomes more apparent under conditions of

severe environmental stress. In this cohort, PRS showed only a weak association with PTSD symptoms at lower levels of trauma exposure, but its predictive value increased among individuals who reported higher cumulative trauma. Similar patterns have been reported in studies of PTSD after large-scale disasters, where genetic risk tends to be more detectable in highly traumatized populations while environmental factors continue to play the dominant role in influencing outcomes (Waszczuk et al., 2021; Campbell-Sills et al., 2023; Kondratenko et al., 2021; Mann et al., 2025). These findings reinforce the idea that genetic risk does not act in isolation but interacts with difficult environmental conditions, especially in populations exposed to extreme and long-lasting trauma.

The genomic analyses included several procedures to ensure that the observed associations were not driven by population structure or familial relatedness. The models incorporated ancestry principal components and mixed-effects approaches that accounted for genetic similarity among participants, and the main PRS effects and PRS-trauma interactions remained stable across different modelling strategies. Additional sensitivity analyses, including models with reduced numbers of ancestry principal components, robust standard errors, and mixed-effects specifications, produced consistent results. These checks support the idea that the observed associations are meaningful and not due to statistical artefacts.

Familial relatedness was an important consideration in this study because recruiting a sample of entirely unrelated individuals was not feasible in the demographic and social context of Kurdish and Yazidi communities. In many parts of the Middle East, extended family networks and inter-family marriages remain common, and individuals who consider themselves unrelated may still share substantial genetic background due to historical patterns of marriage within clans or tribes. Consanguinity has been widely documented in the region and reflects long-standing cultural and social traditions (Bittles & Black, 2010). In addition, both populations have experienced decades of displacement, migration, and population loss following mass trauma, which further limited the possibility of recruiting genetically unrelated participants through probability-based sampling.

Rather than excluding related individuals and reducing statistical power and representativeness, familial relatedness was accounted for directly in the analyses through the inclusion of a Genetic Relatedness Matrix (GRM) in linear mixed models. This approach is commonly used in human genetics to control for non-independence among related individuals while retaining the full sample (Yang et al., 2011). For the PTSD polygenic risk score analyses, LMM with GRM served as the main analytic framework. For the phenotypic outcomes, sensitivity analyses using mixed models with the same GRM confirmed that the estimates for trauma exposure, demographic variables, and psychosocial factors were largely unchanged, indicating that the main findings were robust despite the presence of related participants. The increase in conditional R^2 observed in some models may reflect shared variance attributable to

relatedness and shared environmental exposures among relatives, rather than purely genetic effects.

Across both cohorts, cumulative trauma consistently emerged as the strongest predictor of PTSD symptoms, highlighting the central role of trauma exposure in shaping mental health outcomes. Education showed a protective effect, and demographic and psychosocial factors associated to baseline differences in symptom severity, but their influence remained smaller than that of trauma exposure. The effect of polygenic risk was cohort-specific, with modest genetic influence observed in the chemical survivor cohort and no detectable effect in the Yazidi cohort. These differences illustrate that genetic vulnerability operates within broader social and environmental contexts and may only become detectable under certain conditions.

Environmental and social factors further help explain differences between cohorts. Variations in living conditions, residential environments, social support structures, occupation, and health status likely shaped the visibility of genetic effects. Previous research has shown that trauma exposure and socioeconomic disadvantage are significantly associated with poorer mental health outcomes (Brewin et al., 2000; Tang et al., 2017), and the present findings are consistent with this perspective. Genetic risk appears to contribute modestly to symptom variation, but environmental conditions remain the primary drivers of mental health outcomes in these severely traumatized populations.

Gender emerged as a consistent predictor of mental health outcomes in both cohorts. In the chemical gas- survivor group, women reported higher levels of PTSD, depression/anxiety, somatic symptoms, and perceived stress compared with men, even after adjusting for trauma exposure, education, and psychosocial factors. A similar pattern was observed among Yazidi survivors, with female gender associated with higher symptom levels across most outcomes. These findings align with epidemiological evidence showing women are approximately 2-3 times more likely than men to develop PTSD following trauma exposure (Tolin & Foa, 2006; Taha et al., 2021; Mahmood et al., 2019). Three primary mechanisms explain this pattern. Biologically, women exhibit heightened amygdala reactivity due to estrogen modulation of fear conditioning pathways and differences in hypothalamic-pituitary-adrenal (HPA) axis stress responses (Bangasser & Valentino, 2014; Ramikie & Ressler, 2018). Psychologically, women experience greater peri-traumatic dissociation during trauma and tend to use more internalizing coping strategies post-trauma (Olf, 2017; Christiansen & Elklit, 2012). Socially, women are more frequently exposed to interpersonal and sexual violence, which are among the trauma types most strongly associated with PTSD, and this differential exposure contributes to higher PTSD prevalence in women (Brewin et al., 2022).

Trauma-focused therapies such as Cognitive Processing Therapy (CPT) and Prolonged Exposure (PE) are associated with greater average reductions in PTSD

symptoms among women compared to men, suggesting potential gender differences in treatment response (Wade et al., 2016).

Education emerged as a consistent protective factor for mental health in both cohorts. In the chemical gas- survivor group, lower educational attainment was associated with higher levels of PTSD, depression/anxiety, somatic symptoms, and perceived stress, even after accounting for trauma exposure and psychosocial variables. A similar pattern was seen among Yazidi survivors, where higher levels of education were linked to lower symptom severity across outcomes.

Taken together, these findings highlight the importance of considering contextual and population-specific factors when interpreting gene-environment interactions in PTSD. Trauma exposure, genetic vulnerability, and sociodemographic conditions appear to operate jointly rather than independently, and their relative influence varies across populations and environments. While polygenic risk scores provide useful insights into biological susceptibility, their predictive value remains limited in highly traumatized and underrepresented populations. In contrast, cumulative trauma exposure consistently explains a substantial proportion of symptom variation, reinforcing the importance of trauma-focused interventions and social support systems in improving mental health outcomes among survivors of mass violence.

The findings suggest that interventions with chemical gas and Yazidi survivors should be primarily trauma- focused, but also sensitive to gender and educational differences. Women, who often carry heavy caregiving responsibilities and may have more limited access to health services, appear to be at higher risk for PTSD, depression, and related symptoms and should therefore be a priority in programming. Supporting access to education and basic literacy may contribute to long- term mental health benefits, particularly in contexts where formal mental health services are scarce. In camp- based and village- based communities, strengthening social support and community- based networks can serve as important adjuncts to individual- level treatment. In more urban or hospital- based settings, integrating psychosocial support with pharmacological treatment may be particularly relevant for the most severely affected individuals. Overall, broader social and economic policies that reduce ongoing stressors such as displacement, unemployment, and housing insecurity, while improving access to education and health care, are likely to have meaningful effects on mental health outcomes in the long term.

Overall, this study contributes to psychiatric genetics by demonstrating gene - environment ($G \times E$) interactions in Kurdish trauma survivors. The findings align with established literature showing that PTSD polygenic risk scores (PRS) explain only a modest proportion of variance even in large European cohorts (Nievergelt et al., 2019), that $G \times E$ effects may be amplified under conditions of extreme and cumulative trauma, as reflected in the present study's dose-dependent PRS-trauma interaction, and that trauma exposure remains the strongest predictor of PTSD risk across populations (Kessler et al., 2017). Critically, Kurdish and other Middle Eastern

populations remain severely underrepresented in psychiatric genomic research, despite the global burden of trauma-related disorders, underscoring the need for more inclusive and ancestry-diverse GWAS efforts (Martin et al., 2019).

Future Directions

The findings of this study point to several directions for future research aimed at improving understanding of how genetic, environmental, and psychosocial factors contribute to psychiatric distress in trauma-exposed populations.

First, the PRS findings highlight the need for population-specific genomic research. Current PTSD PRS are based on genome-wide association studies that include multiple ancestries, but they still disproportionately reflect samples with a large proportion of individuals of European descent (Duncan et al., 2019; Martin et al., 2019). Previous studies have shown that PRS tend to perform better when the discovery and target populations share similar ancestry, suggesting reduced predictive performance in non-European groups (Duncan et al., 2019). Because of this, PRS may be less accurate in Middle Eastern populations, including the Kurdish cohorts studied here, even though Kurdish samples cluster relatively close to European populations in genetic analyses, while still remaining distinct.

Future studies should focus on doing genome-wide association studies in Kurdish and other Middle Eastern populations to find variants that are specific to these groups. This would improve how PRS are built and how well they work across different populations. It would also make it easier to compare results across groups in a more reliable way. In addition, whole-genome sequencing and studies of rare variants may help identify genetic risk factors that current PRS do not capture.

Second, the cross-sectional design of the present study limits conclusions about causal relationships. Longitudinal studies are needed to examine how genetic risk interacts with trauma exposure over time and how symptoms develop or change following traumatic events. Prospective designs could help determine whether individuals with higher genetic risk show earlier onset of symptoms after trauma exposure or whether protective factors such as social support or higher education reduce symptom severity across time. Longitudinal data would also help identify periods during which interventions may be particularly effective.

Third, the present findings indicate that certain associations differed between cohorts. For example, social support and psychotropic medication showed different relationships with mental health outcomes among chemical attack survivors and Yazidi survivors. Future research should examine broader contextual factors more closely, including access to healthcare, reliance on community networks, cultural perceptions of mental health, and ongoing daily stressors that may not be fully captured by trauma exposure measures alone. Mixed-methods studies that combine

quantitative analyses with qualitative interviews may help clarify how these factors shape mental health outcomes and influence treatment responses.

Fourth, future studies should move beyond genetic risk scores alone and incorporate additional biological markers of stress, including cortisol levels, inflammatory markers, and epigenetic changes. Combining genetic data with physiological and epigenetic indicators may provide a more complete understanding of the biological mechanisms linking trauma exposure to mental health outcomes. This approach may also help identify early biological indicators of vulnerability in highly trauma-exposed populations.

Finally, replication in other conflict-affected populations will be essential to evaluate the generalizability of the present findings. Collaborative research and data sharing across regions could increase statistical power for genetic analyses and allow systematic comparisons across different trauma contexts. Such work would help distinguish between universal predictors of trauma-related distress and factors that are specific to particular populations or historical experiences.

Limitations

Several limitations should be considered when interpreting the findings of this study.

1. Main limitation relates to the sampling strategy in both cohorts. Because the study focused on highly vulnerable groups survivors of chemical weapons exposure and Yazidi survivors of ISIS-related violence probability-based sampling was not feasible. Ethical concerns, incomplete population records, population displacement, and ongoing political and economic instability made systematic random sampling very difficult in practice. As a result, recruitment relied on a mix of convenience and snowball sampling.

This approach made it possible to reach populations that are otherwise very hard to access, but it also limits how representative the sample is. Participants were recruited based on availability, existing contact information, and willingness to participate. People who were more socially connected or in contact with government or humanitarian services may therefore be overrepresented, while those who were more isolated, severely ill, or living outside the region may have been missed.

For the chemical gas survivor cohort, recruitment partly relied on official registries. However, incomplete or outdated contact details meant that only those who could be reached during the study period were able to take part. Although participants came from several districts, their distribution reflected where people were living and how reachable they were, rather than proportional representation of all regions. The sample therefore cannot be considered geographically stratified.

Similarly, the Yazidi survivor cohort was recruited from selected internally displaced

persons camps in the Duhok Governorate. Survivors who had migrated to Europe, returned to Sinjar, or resettled elsewhere were not systematically included. As a result, the findings mainly reflect the experiences of Yazidi survivors who were still living in camps at the time of data collection, rather than the wider displaced population.

Snowball sampling may also have introduced network clustering, because participants are likely to refer people from their own social circles who share similar demographic or psychosocial characteristics. Although efforts were made to include people from different backgrounds, the non-probability sampling strategy limits how far the results can be generalized beyond the study population.

Despite these challenges, using multiple recruitment sources helped strengthen verification of survivor status. Government records, documentation from non-governmental organizations, and structured trauma assessments were used to confirm exposure histories. This approach improved the reliability of participant classification, while still acknowledging the practical constraints of doing research in post-conflict settings.

Future studies using registry-based or stratified sampling designs may improve population representativeness and provide more precise estimates of prevalence and risk factors.

2. The cross-sectional design of this study also limits how the findings can be interpreted. Psychological, trauma-related, and genetic data were collected at a single time point, which prevents conclusions about causality. Although hierarchical regression models were used to examine associations between trauma exposure, psychosocial factors, genetic risk, and mental health outcomes, the results should still be understood as correlational.

3. Another important issue is the amount of time that has passed since the main traumatic events. The chemical attacks occurred in 1988, whereas the primary traumatic events for Yazidi survivors occurred in 2014. Data collection was carried out in 2023 and 2024, so the reported symptoms reflect long-term psychological outcomes rather than acute responses to trauma. This long interval may introduce recall bias in reports of trauma exposure and may also hide fluctuations in symptoms that happened in the intervening years.

4. Psychiatric symptoms were measured using standardized self-report instruments rather than clinician-administered diagnostic interviews. Although these instruments have good psychometric properties and are widely used in trauma research, self-report data can be influenced by subjective interpretation, cultural expressions of distress, and current life circumstances. The absence of structured diagnostic interviews limits the ability to confirm clinical diagnoses.

5. Test-retest reliability and longitudinal measurement invariance were not assessed. The PHQ-13K was validated for chemical survivors in my published study (Appendix B), but other scales (PCL-5, HSCL-25, PSS-14) lack formal validation in Kurdish

trauma survivors despite good internal consistency here and common use in local research. This may limit measurement precision. Longitudinal studies are needed.

6. Several limitations also apply to the genetic analyses. The polygenic risk scores used in this study were based on genome-wide association studies conducted in European-ancestry samples (EUR-only). Differences in allele frequencies and genetic structure may limit the transferability of these scores to Kurdish populations.

In addition, the proportion of variance explained by the PRS was relatively small, which suggests limited statistical power to detect subtle genetic effects. Interaction analyses involving PRS and trauma exposure should therefore be interpreted with caution. Although principal components were included to adjust for population stratification, some residual population structure may still influence the genetic associations.

7. The inclusion of biologically related individuals also required careful analytic handling. The main analyses used hierarchical regression models, and sensitivity analyses using linear mixed models incorporated the genomic relationship matrix to account for relatedness. The consistency of results across these approaches suggests that kinship did not substantially bias the findings, although shared environmental influences among relatives may still be difficult to separate from genetic effects.

Conclusion

This dissertation aimed to examine how genetic risk, cumulative trauma exposure, and sociodemographic factors relate to PTSD and other mental health symptoms among Kurdish survivors of mass trauma. The work focused on two groups: chemical gas survivors attacks and survivors of the Yazidi genocide. Across both cohorts, the central finding is that cumulative trauma exposure is the strongest and most consistent predictor of PTSD, depression/anxiety, somatic symptoms, and perceived stress. At the same time, gender, education, and social context shape how trauma translates into symptoms, and genetic vulnerability appears to play a role in the chemical gas survivor cohort. The results support a model in which trauma is central, but its effects are not automatic and depend on individual and contextual factors.

The first aim was to assess whether PTSD polygenic risk scores (PRS) predict PTSD symptom severity, whether this association is moderated by cumulative trauma exposure, and whether these effects remain after controlling for demographic covariates and familial relatedness. In the chemical gas cohort, PTSD PRS was modestly associated with symptom severity, and this effect became stronger under higher levels of trauma exposure. This suggests that genetic vulnerability does not act in isolation, but becomes more visible when environmental stress is high. After accounting for age, gender, education, population structure, and genetic relatedness, these patterns remained stable, indicating that the association is not simply an artefact of sample structure or confounding. In the Yazidi cohort, however, the same PRS did not clearly predict PTSD symptoms or interact with trauma exposure. PTSD symptom

levels were still substantially influenced by trauma and social factors, but the measured genetic risk score did not add meaningful prediction beyond these variables. This difference between cohorts may reflect limitations in the cross-ancestry portability of European-derived PRS and/or contextual differences in environmental and social exposures.

The second aim was to examine the associations and relative contributions of sociodemographic, psychosocial, and trauma-related factors to PTSD, depression/anxiety, somatic symptoms, and perceived stress. Across both groups, higher cumulative trauma exposure was consistently linked to higher symptom levels for all four outcomes. Female gender was associated with higher PTSD and depression/anxiety symptoms, even after controlling for trauma and education, whereas higher education was linked to lower symptoms. This indicates that socioeconomic position and gender shape vulnerability in meaningful ways. Psychosocial factors such as social support and psychotropic medication use also mattered, but their role differed by cohort. In the chemical gas cohort, psychotropic medication use was more strongly associated with higher symptom levels, which may reflect greater clinical severity and engagement with formal mental health services. In the Yazidi group, social support appeared more protective, and psychotropic medication showed a weaker association with symptoms, consistent with different patterns of help-seeking and community-based coping. Living conditions and residential location (city, village, camp) also played an important role, especially for PTSD and depression/anxiety, underscoring the impact of environment and access to services.

The third aim was to test whether cumulative trauma exposure explains additional variance in mental health symptoms beyond demographic and psychosocial factors using sequential regression models. In the combined sample of survivors, demographic variables alone explained a meaningful but limited proportion of symptom variance. Adding psychosocial factors increased prediction slightly, but the largest gains came from including trauma and health-related variables. Trauma exposure consistently added unique variance to PTSD, depression/anxiety, and related symptoms once background and psychosocial factors were accounted for. This finding supports the idea that, although sociodemographic and psychosocial characteristics are important, exposure to repeated trauma remains a central and consistent risk factor for poor mental health in these populations. The models showed that trauma remains a central and consistent risk factor for poor mental health in these populations.

The fourth aim was to evaluate whether the associations between trauma exposure, PTSD PRS, and their interaction with PTSD symptoms differ between survivors of the chemical attacks and Yazidi genocide survivors, and whether cohort-specific sociodemographic and contextual factors are linked to these differences. Parallel analyses in the two cohorts showed that trauma exposure was strongly associated with PTSD symptoms in both groups, and demographic patterns such as the role of gender

and education were broadly similar. However, the role of PTSD PRS and certain psychosocial predictors did differ. The PRS effect and its interaction with trauma were clear in the chemical gas cohort but not in the Yazidi cohort, suggesting that genetic risk may operate differently depending on cultural, historical, and structural context. Cohort interaction tests also revealed that psychotropic medication use, social support, education, and gender had somewhat different strengths of association with symptoms depending on living conditions (urban vs. camp/village) and social organization. For somatic symptoms, by contrast, the same predictors worked similarly across groups, indicating that some forms of distress may be less sensitive to these contextual differences.

The findings of this dissertation have several implications. Theoretically, they support an integrated, context-sensitive diathesis-stress model of PTSD and related disorders. Trauma exposure is the main driver, but genetic vulnerability, psychosocial resources, and structural factors such as education, gender, and living conditions all shape how individuals respond to trauma. The observed contribution of genetic relatedness in both cohorts, alongside differences in PRS performance, highlights the importance of developing ancestry-specific genomic tools for Kurdish and Middle Eastern populations.

Clinically, the results suggest that trauma-focused interventions should be central, but they must be adapted to local contexts, taking into account gender, education, community structure, and access to mental health services. In the Yazidi setting, strengthening social support networks and community-based care may be particularly important, while in the chemical gas exposed group, combining psychosocial support with more structured clinical and pharmacological approaches may be necessary.

In summary, this dissertation shows that among Kurdish survivors of mass trauma, cumulative trauma exposure is central to PTSD and related symptoms, but its effects are shaped by gender, education, social context, and, in some settings, genetic vulnerability. The findings support the need for more nuanced, context-sensitive models of trauma and mental health and for interventions that address both psychological and structural determinants of well-being. For these highly traumatized communities, the message is clear: trauma matters deeply, but the path from exposure to symptoms is also shaped by social, economic, and, in part, genetic factors that must be acknowledged and addressed together.

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Appendix A. Tables

Appendix Table A1: PHQ Item Statistics and Item-Total Correlations

Item	M	SD	Item-total correlation	α if Item Deleted
PHQ1	1.030	0.887	0.567	0.825
PHQ2	1.607	0.722	0.470	0.831
PHQ3	1.756	0.552	0.443	0.833
PHQ4	0.841	0.925	0.325	0.843
PHQ5	1.359	0.772	0.600	0.823
PHQ6	1.267	0.855	0.572	0.826
PHQ7	1.052	0.821	0.622	0.822
PHQ8	0.189	0.493	0.416	0.834
PHQ9	1.189	0.775	0.570	0.825
PHQ10	1.363	0.791	0.534	0.828
PHQ11_imp1	0.400	0.670	0.309	0.839

PHQ12	0.815	0.833	0.615	0.822
PHQ13	0.689	0.795	0.559	0.826
PHQ14	1.693	0.596	0.566	0.827
PHQ15	1.356	0.809	0.539	0.827

Appendix Table A2. PHQ-14 Item Statistics (Full Sample, n = 534, PHQ4 Excluded)

Item	M	SD	Item-total correlation	α if Item Deleted
PHQ1	0.895	0.882	0.570	0.854
PHQ2	1.457	0.792	0.566	0.854
PHQ3	1.652	0.647	0.548	0.855
PHQ5	1.195	0.812	0.626	0.850
PHQ6	1.006	0.882	0.635	0.850
PHQ7	0.905	0.838	0.648	0.849
PHQ8	0.126	0.412	0.344	0.863
PHQ9	1.008	0.815	0.654	0.849
PHQ10	1.150	0.858	0.655	0.849
PHQ11	0.369	0.637	0.252	0.868
PHQ12	0.757	0.829	0.511	0.857
PHQ13	0.689	0.799	0.486	0.858
PHQ14	1.552	0.682	0.659	0.850

PHQ15	1.240	0.855	0.614	0.851
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Appendix Table A3. PCL Item Statistics and Item-Total Correlations (n=534)

Item	M	SD	Item-total correlation	α if Item Deleted
PCL1	2.612	1.460	0.607	0.881
PCL2	1.755	1.554	0.627	0.880
PCL3	1.925	1.528	0.615	0.880
PCL4	2.292	1.442	0.712	0.878
PCL5	2.178	1.468	0.725	0.877
PCL6	2.300	1.355	0.249	0.892
PCL7	2.082	1.396	0.267	0.891
PCL8	0.513	1.028	0.075	0.893
PCL9	1.189	1.383	0.488	0.884
PCL10	1.255	1.353	0.490	0.884
PCL11	1.966	1.447	0.631	0.880
PCL12	2.071	1.419	0.644	0.879
PCL13	1.708	1.514	0.616	0.880
PCL14	1.423	1.439	0.614	0.880
PCL15	2.051	1.456	0.541	0.882
PCL16	0.987	1.310	0.448	0.885

PCL17	1.588	1.423	0.501	0.884
PCL18	1.858	1.537	0.611	0.880
PCL19	2.230	1.411	0.568	0.882
PCL20	2.159	1.604	0.598	0.881

Appendix Table A4. HSCL Item Statistics and Item-Total Correlations (n=534)

Item	M	SD	Item-total correlation	α if Item Deleted
HSCL1	1.723	1.069	0.701	0.932
HSCL2	2.039	1.141	0.648	0.933
HSCL3	2.131	1.051	0.600	0.933
HSCL4	2.448	1.130	0.627	0.933
HSCL5	2.264	1.126	0.709	0.932
HSCL6	1.809	1.042	0.598	0.933
HSCL7	2.693	1.064	0.740	0.931
HSCL8	2.440	1.106	0.602	0.933
HSCL9	1.622	0.935	0.655	0.933
HSCL10	1.970	1.129	0.658	0.933
HSCL11	2.421	1.040	0.701	0.932
HSCL12	1.957	1.010	0.604	0.933
HSCL13	2.341	1.200	0.633	0.933

HSCL14	2.313	1.171	0.192	0.939
HSCL15	1.888	1.017	0.528	0.934
HSCL16	2.481	1.145	0.636	0.933
HSCL17	2.129	1.162	0.570	0.934
HSCL18	2.440	1.126	0.822	0.930
HSCL19	1.955	1.114	0.666	0.932
HSCL20	1.099	0.445	0.262	0.937
HSCL21	1.483	0.924	0.370	0.936
HSCL22	2.232	1.088	0.734	0.931
HSCL23	2.039	1.113	0.701	0.932
HSCL24	2.393	1.134	0.653	0.933
HSCL25	1.481	0.870	0.543	0.934

Appendix Table A5. PSS Item Statistics and Item-Total Correlations (n=534)

Item	M	SD	Item-total correlation	α if Item Deleted
PSS1	1.860	1.542	0.598	0.865
PSS2	1.772	1.402	0.686	0.860
PSS3	1.800	1.510	0.699	0.859
PSS4	1.543	1.118	0.625	0.866
PSS5	1.320	1.094	0.680	0.864

PSS6	1.285	1.140	0.667	0.865
PSS7	2.365	1.165	0.447	0.873
PSS9	1.642	1.115	0.690	0.864
PSS10	1.661	1.111	0.737	0.862
PSS13	1.603	1.131	0.483	0.871
PSS8	1.579	1.338	0.514	0.869
PSS11	1.893	1.448	0.516	0.869
PSS12	2.380	1.338	0.195	0.884
PSS14	1.599	1.455	0.719	0.859

Appendix Table A6. Yazidi cohort PHQ-14 Item Statistics (n = 358, PHQ4 Excluded)

Item	M	SD	Item-total correlation	α if Deleted
PHQ1	1.045	0.842	0.633	0.870
PHQ2	1.327	0.811	0.621	0.871
PHQ3	1.229	0.852	0.625	0.871
PHQ5	1.385	0.742	0.468	0.877
PHQ7	0.416	0.680	0.299	0.884
PHQ6	1.556	0.695	0.618	0.872
PHQ8	1.425	0.740	0.580	0.873
PHQ9	0.827	0.825	0.611	0.871

PHQ10	0.939	0.851	0.620	0.871
PHQ11	0.774	0.835	0.617	0.871
PHQ12	0.844	0.815	0.691	0.868
PHQ13	0.877	0.814	0.625	0.871
PHQ14	0.684	0.780	0.539	0.875
PHQ15	0.846	0.811	0.665	0.869

Appendix Table A7. Yazidi cohort PCL-5 Item Statistics (n = 358)

Item	M	SD	Item-total correlation	α if Deleted
PCL1	3.207	1.158	0.632	0.883
PCL2	2.589	1.348	0.473	0.887
PCL3	3.061	1.240	0.577	0.884
PCL4	3.399	0.984	0.587	0.885
PCL5	2.726	1.298	0.627	0.883
PCL6	2.953	1.172	0.483	0.887
PCL7	2.916	1.218	0.377	0.890
PCL8	1.922	1.414	0.244	0.894
PCL9	2.556	1.536	0.668	0.881
PCL10	2.011	1.548	0.519	0.886

PCL11	2.545	1.354	0.614	0.883
PCL12	2.821	1.260	0.643	0.882
PCL13	2.218	1.515	0.496	0.886
PCL14	2.201	1.406	0.663	0.881
PCL15	2.318	1.445	0.500	0.886
PCL16	1.430	1.497	0.342	0.891
PCL17	2.436	1.406	0.616	0.883
PCL18	2.430	1.465	0.687	0.881
PCL19	2.517	1.457	0.551	0.885
PCL20	2.818	1.456	0.588	0.884

Appendix Table A8. Yazidi cohort HSCL-25 Item Statistics (n = 358)

Item	M	SD	Item-total correlation	α if Deleted
HSCL1	2.363	1.165	0.504	0.902
HSCL2	2.670	1.134	0.575	0.900
HSCL3	2.307	1.153	0.452	0.903
HSCL4	3.022	1.042	0.585	0.900
HSCL5	2.444	1.133	0.534	0.901
HSCL6	2.257	1.153	0.442	0.903
HSCL7	2.975	1.139	0.539	0.901

HSCL8	3.106	1.105	0.596	0.900
HSCL9	2.419	1.175	0.634	0.899
HSCL10	3.148	1.144	0.699	0.897
HSCL11	3.237	0.974	0.653	0.899
HSCL12	2.559	1.184	0.555	0.900
HSCL13	2.992	1.156	0.450	0.903
HSCL14	1.737	1.017	0.362	0.904
HSCL15	2.430	1.110	0.454	0.903
HSCL16	3.098	1.047	0.546	0.901
HSCL17	3.162	1.043	0.565	0.900
HSCL18	3.031	1.049	0.661	0.898
HSCL19	2.623	1.143	0.539	0.901
HSCL20	1.335	0.748	0.106	0.907
HSCL21	2.673	1.119	0.555	0.901
HSCL22	3.249	0.951	0.572	0.900
HSCL23	3.034	1.061	0.588	0.900
HSCL24	3.073	1.100	0.569	0.900
HSCL25	2.142	1.207	0.438	0.903

Appendix Table A9. Yazidi cohort PSS-14 Item Statistics (n = 358)

Item	M	SD	Item-total correlation	α if Deleted
PSS1	2.936	1.241	0.555	0.791
PSS2	2.972	1.204	0.636	0.786
PSS3	3.176	1.087	0.566	0.791
PSS4	2.059	1.305	0.580	0.786
PSS5	2.078	1.190	0.545	0.789
PSS6	2.031	1.246	0.490	0.792
PSS7	1.567	1.351	0.236	0.812
PSS8	2.223	1.230	0.414	0.797
PSS9	2.087	1.305	0.413	0.799
PSS10	2.385	1.331	0.499	0.791
PSS11	2.925	1.175	0.423	0.800
PSS12	3.201	1.125	0.532	0.790
PSS13	2.126	1.249	0.486	0.792
PSS14	2.902	1.263	0.527	0.792

Appendix Table A10. Complete Standardized Coefficients from Final Block 3 Models (chemical gas survivors)

Predictor	PHQ13K	PCL-5	HSCL-25	PSS-14
Gender	0.351	0.256	0.244	0.026
Income	-0.342	-0.242	-0.243	-0.122

Education	-0.206	-0.169	-0.196	-0.197
Occupation	0.027	0.069	0.008	-0.060
Living loaction	0.082	0.082	0.049	-0.006
Social status	-0.025	-0.042	-0.005	0.017
Number of children	-0.011	-0.034	-0.047	-0.119
Social support	0.041	-0.019	0.011	-0.023
Psychotropics	0.169	0.235	0.274	0.287
Cumulative trauma	0.208	0.239	0.235	0.175
Chronic disease	0.073	0.022	-0.011	0.006
Prior diagnosis	0.036	0.029	0.079	0.011

Appendix Table A11 Complete Standardized Coefficients from Final Block 3 Models (Yazidi survivors)

Predictor	PCL-5	HSCL-25	PHQ-14	PSS-14	HSCL-Status
Gender	0.128	0.202	0.205	-0.080	0.202
Age	0.104	0.081	0.222	0.162	0.081
Education	-0.234	-0.230	-0.188	-0.079	-0.230
Occupation	0.034	0.015	0.062	-0.158	0.015
Living location	0.155	0.131	0.065	0.038	0.131
Social status	0.015	0.020	0.048	0.033	0.020

Family member with mental illness	0.101	0.046	0.089	-0.100	0.046
Social support	0.100	0.075	0.077	0.057	0.075
Psychotropics	0.074	0.071	0.110	-0.019	0.071
Cumulative trauma	0.365	0.360	0.187	0.110	0.360
Chronic disease	-0.018	0.033	0.027	-0.053	0.033
Prior diagnosis	-0.185	-0.149	-0.152	-0.007	-0.149

Appendix Table A12. Full Standardized Betas, 95% CIs, and Diagnostics Across All Blocks

Predictor	PCL-5 β [95% CI]	HSCL-25 β [95% CI]	PSS-14 β [95% CI]	PHQ13K β [95% CI]
Block 1: Demographics				
Age	0.017 [-0.071, 0.110]	0.013 [-0.003, 0.004]	-0.024 [-0.074, 0.044]	0.061 [-0.012, 0.069]
Gender	0.163 [3.39, 8.32]	0.197 [0.19, 0.38]	-0.002 [-1.64, 1.56]	0.301 [3.38, 5.59]
Education	-0.189 [-4.26, -2.10]	-0.207 [-0.18, -0.10]	-0.133 [-1.96, -0.56]	-0.225 [-2.05, -1.09]
Occupation	0.004 [-0.66, 0.74]	-0.028 [-0.04, 0.02]	-0.115 [-1.13, -0.23]	0.030 [-0.18, 0.44]
Residential location	0.457 [4.99, 7.38]	0.416 [0.18, 0.28]	0.178 [0.58, 2.13]	0.120 [0.14, 1.21]
Block 2: Psychosocial				

Social status	-0.037 [-2.73, 0.79]	-0.020 [-0.09, 0.05]	-0.017 [-1.40, 0.89]	0.011 [-0.67, 0.90]
Social support	-0.017 [-2.48, 1.47]	0.003 [-0.07, 0.08]	-0.007 [-1.40, 1.17]	0.051 [-0.25, 1.52]
Psychotropics	0.204 [7.14, 13.54]	0.223 [0.33, 0.58]	0.206 [3.78, 7.94]	0.210 [2.99, 5.86]
Block 3: Trauma/Health				
Family disorder	-0.004 [-2.56, 2.20]	0.025 [-0.05, 0.14]	0.011 [-1.29, 1.80]	0.008 [-0.93, 1.20]
Chronic disease	0.051 [-0.44, 4.01]	0.020 [-0.06, 0.12]	-0.001 [-1.46, 1.43]	0.118 [0.73, 2.71]
Cumulative trauma	0.265 [0.93, 1.49]	0.257 [0.04, 0.06]	0.112 [0.11, 0.47]	0.207 [0.27, 0.52]
Prior diagnosis	-0.009 [-0.50, 0.39]	0.048 [-0.01, 0.03]	0.111 [0.11, 0.69]	-0.026 [-0.27, 0.13]
Model R²	0.402	0.443	0.203	0.308

Appendix Table A13. The table shows predictor $\times$ cohort interactions with β , SE, t-values, and p-values.

Outcome	Predictor	β Interaction	SE	t-value	p-value	Interpretation Stronger Effect /
PCL-5	Psychotropics	-0.384	0.183	-2.10	0.036	Effect smaller in Yazidi (Chemical stronger 0.58 vs 0.19)
PCL-5	Social Support	+0.242	0.106	2.28	0.023	Protective effect stronger in Yazidi (0.20 vs -0.04)
PCL-5	Diagnosis	-0.058	0.024	-2.46	0.014	Effect weaker in Yazidi (-0.04 vs 0.01)

PCL-5	Family Mental Illness	+0.303	0.120	2.52	0.012	Stronger effect in Yazidi (0.18 vs -0.12)
HSCL-25	Psychotropics	-0.482	0.131	-3.68	<0.001	Effect smaller in Yazidi (Chemical stronger: 0.62 vs 0.13)
PSS-14	Education	-0.084	0.036	-2.37	0.018	Protective effect stronger in Yazidi (-0.11 vs -0.03)
PSS-14	Gender	+0.270	0.083	3.25	0.001	Stronger female effect in Yazidi (0.43 vs 0.16)

Appendix B

“A study on long-term trauma-related mental health outcomes among Kurdish survivors of chemical attacks”

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Abstract

Background: The Kurdistan region experienced a series of devastating events, such as the 1988 chemical attacks and the 2014 Yazidi genocide, which have had substantial impacts on the psychological and physical health of survivors.

Objectives: This study evaluates the long-term mental health of individuals who were exposed to chemical gas, with a focus on the prevalence and severity of psychological symptoms and their associations with sociodemographic, clinical, and trauma-related factors.

Methods: A total of 534 participants were recruited (300 women and 234 men). Data collection was completed in 7 months, from March to September 2023. All participants completed validated psychological assessments, including the PTSD Checklist for DSM-5 (PCL-5), Patient Health Questionnaire-15 (PHQ-15), and Hopkins Symptom Checklist-25 (HSCL-25). Multivariate General Linear Modeling (GLM) was performed, adjusting for trauma exposure to simultaneously assess the effects of demographic and clinical variables on multiple symptom domains.

Results: Most of the participants exhibited clinically significant symptoms, with 78.8% meeting the post-traumatic stress disorder (PTSD) threshold and 46.3% exhibiting comorbid symptoms across multiple domains. The GLM demonstrated that gender, trauma exposure, education level, and clinical factors were significantly associated with symptom severity across PTSD, somatic, and anxiety/depression symptoms ($p < 0.001$). The model explained up to 47% of variance in symptom outcomes. After adjusting for covariates, women showed higher symptom severity than men. Greater trauma exposure and lower education independently predicted increased symptoms.

Conclusion: These findings highlight serious psychological and somatic effects among survivors and underscore the urgent need for targeted mental health interventions for those affected by chemical attacks, with particular attention to individuals with lower levels of education and socioeconomic status, while incorporating gender-sensitive approaches to address differential vulnerabilities.

1 Introduction

Kurdistan is the name given to a large region spanning parts of Syria, Turkey, Iran, and Iraq, which is home to the Kurdish people. The Kurds represent the fourth-largest population, following Arabs, Turks, and Persians, in the Middle East; their population is estimated to range from 32 to 34 million people (1–6).

The Kurdistan Region in Iraq has witnessed several large-scale atrocities, including the Anfal operations in 1988, during which approximately 182,000 Kurdish people were killed and buried in mass graves. During this time, millions of civilians were displaced, and tens of thousands were transported to concentration camps. Thousands disappeared, and many villages were completely destroyed (7, 8). The seizure of Kurdish property in Iraq and mass killings have been prevalent since what is now called the Kurdish genocide of 1963, characterized by the burning of Kurdish villages, chemical warfare, ground attacks, and massive aerial bombardment (9).

In 1988, the city of Halabja suffered one of the most significant chemical attacks in the history of Kurdish genocide (10). The Iraqi regime used nerve agents, cyanide, and mustard gas. Officially, approximately 5,000 people from Halabja are estimated to have died in the attack, and thousands continue to suffer from its long-term consequences. The Ministry of Martyrs and Anfal Affairs in the Kurdistan Regional Government (KRG) reported that approximately 6,000 victims have survived the attack, many of whom still live with ongoing health issues (11).

Research on related psychological and physiological influences of exposure to chemical gas has remained limited over the years. Nevertheless, some studies are now beginning to show the far-reaching consequences of this atrocity on its victims. Survivors report high rates of post-traumatic stress disorder (PTSD), sleep problems, depression, social isolation, itching, and respiratory complications (11, 12).

Further research from the Kurdish context highlights the persistent impact of mass violence. For example, 87% of children who survived the Kurdish Anfal have been reported to have PTSD. The estimated prevalence rates of depression and PTSD in adults were 49% and 86.2%, respectively. Similarly, findings are reported among Iraqi Kurdish torture survivors, Syrian Kurdish refugees, and Yazidi genocide survivors, who often experience numerous overlapping conditions including PTSD, depression, and anxiety (13–16).

When compared with worldwide estimates, PTSD impacts approximately 3.9% of the general population during their lifetime, with prevalence increasing to approximately

48% in trauma-exposed populations (17, 18). These findings underscore the extraordinary mental health consequences of conflict-related trauma. Meta-analytic evidence further shows that over one-quarter of adult war survivors suffer from PTSD, and trauma exposure is also associated with depression, anxiety, and somatic symptoms like headaches, fatigue, and respiratory problems. Additionally, longitudinal studies among survivors of chemical weapon exposure show persistent psychiatric symptoms and physical comorbidities decades after exposure, highlighting the chronicity and complexity of trauma effects among survivors (19–21). Collectively, studies have demonstrated that exposure to severe trauma, such as chemical attacks, renders survivors vulnerable to long-lasting mental health disorders. These psychiatric conditions continue many years after conflict ends, particularly in low- and middle-income countries with limited access to healthcare. This chronicity underscores the important need to assess mental health outcomes long after the acute consequences of trauma, even decades following chemical warfare exposure (14, 22–25). Additionally, the psychological outcomes for chemical attack survivors cannot be limited to a single event. Rather, the chemical attack comprised multiple, interconnected traumatic events, such as physical injury, witnessing death, deprivation, and severe loss, which together contribute significantly to long-term mental health consequences (26). This complexity is consistent with an ecological model of trauma, which underscores the combined impact of direct exposure to violence and ongoing environmental stressors in war-affected populations. Therefore, evaluating cumulative trauma through composite measures results in a more precise and nuanced understanding of PTSD symptomatology in such contexts (27, 28).

Most mental health studies currently available in this context focus on the psychological impacts of mass killing and utilize relatively simple or univariate statistical methods. While many have assessed mental health consequences among Kurdish survivors of genocide and chemical attacks, important gaps remain. Limited attention has been given to the comprehensive assessment of both somatic and psychological symptomatology, despite evidence that physical health consequences significantly impact survivors' overall health. Additionally, many studies have been limited by small sample sizes and lack of culturally adapted psychometric validation of measurement instruments (11, 14, 29, 30). In contrast, the present study integrates multiple psychological scales alongside sociodemographic and clinical factors to provide a more rigorous and multidimensional understanding of survivors' mental health. The early findings of this study highlight the urgent need for additional research and more robust measures to determine the full impact of such atrocities as genocide and chemical attacks. Further study should not only emphasize on the psychological impacts but also incorporate physiological factors to better understand the long-term consequences and mental health impacts of large-scale, traumatic, and life-threatening events.

This study has attempted to fill the identified gap by developing a multi-method assessment approach and applying it to a large sample of survivors of chemical

attacks. A comprehensive assessment of mental health status has been conducted, which reviews issues such as depression, anxiety, somatic symptoms, and post-traumatic stress. This research then contributes to establishing a comprehensive framework of the long-term psychological and psychiatric consequences of such traumatic events on the survivors.

In this first report, our aim is to comprehensively characterize the long-term psychological impact among Kurdish survivors of chemical weapons exposure by examining the prevalence, severity, and comorbidity of PTSD, depression/anxiety, somatic symptoms, and perceived stress. Additionally, the study seeks to investigate demographic, clinical, and trauma-related factors influencing symptom severity, including exploration of potential moderating effects. Prior to these analyses, the psychometric properties of the assessment instruments were evaluated to ensure that all instruments are reliable and appropriate to measure psychological symptoms of the study population.

2 Materials and methods

2.1 Ethical approval

This study received ethical approval from the ethics committee of the University of Duhok on 3 October 2021 (Approval Number: 141). In addition, we received permission from the Halabja governorate in April 2022 (Reference Number: 10/158), including the General Directorates of Security and Police. We were given permission to contact survivors at the Halabja Hospital for treatment of chemical victims. All survivors contacted (see below) were fully informed about the research and were voluntarily recruited to the study. If they decided not to participate, they did not face any negative consequences. All participants were assured of total confidentiality, and each participant consented in written form before the interviewing process began. The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2013.

2.2 Study setting, period, and design

The primary data collection took place in Halabja city (n = 373), Sulemani city (n = 54), the Khormal district (n = 44), the Serwan district (n = 41), and the Tawella district (n = 22). It extended over 7 months, from March 2023 to September 2023. The design was a cross-sectional observational study involving Kurdish chemical gas survivors attacks. The majority of participants were recruited from Halabja city, as this was the site of the 1988 chemical attack and the location of the largest population of survivors. Many survivors had relocated to other cities and districts due to displacement; therefore, recruitment efforts were extended to these areas to capture the dispersed survivor population. The sample size distribution reflects the practical realities of survivor location and accessibility rather than strict proportional allocation, as detailed population data for survivors across all areas were unavailable.

2.2.1 Data collection procedure

A total of 534 samples were collected, with an additional 145 subjects who refused to be interviewed or could not be contacted. Inclusion criteria required that participants (1) have confirmed exposure to chemical gas, (2) had given informed consent to participate, and (3) were physically and mentally capable of completing the assessments. The exclusion criteria included the inability to provide informed consent and severe illness precluding participation. Participants were primarily recruited as Kurdish survivors of the chemical gas attacks based on official identification by the Kurdistan Regional Government's Ministry of Martyrs and Anfal Affairs. Many survivors are officially registered as injured by chemical gas exposure and receive monthly financial compensation, which served as one pathway for recruitment. Considering that not all survivors are compensated, additional participants were identified through local NGOs working with survivors, especially those without official compensation. To further expand the sample size, snowball sampling was employed, in which initial participants referred other eligible survivors to the study. For those recruited outside official registries, victimization status for participants recruited outside official registries was assessed during structured interviews using standardized trauma exposure questionnaires. Initially, project details were explained to potential participants via phone, which was followed by in-person visits to their homes upon agreement to participate. Participants were briefed extensively on the project's objectives, procedures, and potential risks. Each assessment required approximately 60 to 90 min to complete all questionnaires and interviews.

2.2.2 Sample size determination and power analysis

The sample size was calculated using Cochran's formula for proportions (31).

$$n_0 = Z^2 \times p \times (1-p) / e^2$$

in which n_0 is the required minimum sample size, $Z = 1.96$ is the Z-score corresponding to a 95% confidence level, $p = 0.5$ is the estimated proportion of the attribute in the population (chosen as 0.5 to maximize variability), and $e = 0.05$ is the target margin of error (5%). Considering these values, the minimum sample size needed was 384. In order to ensure adequate power and representativeness of the study findings, the sample size was increased to 534 in order to account for potentially non-response and refusals, which were estimated to be 28% based on recruitment challenges. Non-response rates in similar vulnerable populations reported to range from 20% to 40% (32, 33).

Furthermore, this sample provides sufficient statistical power for multivariate analyses using a General Linear Modeling (GLM) including 12 predictors. Post-hoc calculations using Cohen's $f^2 = 0.15$ and alpha (α) = 0.05 yielded an estimated power of >0.99, confirming that the study is well powered to identify effects on psychiatric symptomatology.

2.3 Data collection instruments

2.3.1 PTSD checklist for DSM-5

PTSD checklist for DSM-5 (PCL-5) is a 20-item self-report scale assessing DSM-5 PTSD symptoms over the past month. Items are rated on a five-point Likert scale (0 = not at all to 4 = extremely). Total symptom severity scores are calculated by summing item responses, with a cutoff score ≥ 23 indicating probable PTSD (34). The PCL-5 has been validated for use in Kurdish trauma-affected populations and is conventionally designed to assess symptom severity related to a single, identified traumatic event. However, given the participants' exposure to multiple overlapping traumatic experiences during the chemical attacks, including injury, deprivation, loss of family members, respiratory distress, displacement, and witnessing death, it was necessary to adapt this approach. Participants were instructed to respond to the PCL-5 items with respect to their cumulative trauma experience throughout the chemical attack period. This adaptation is consistent with the ecological model of trauma, which recognizes that mental health outcomes arise from both direct violence exposure and ongoing contextual stressors. Existing psychometric and epidemiological evidence validates this composite trauma framework as an appropriate method for assessing PTSD symptoms in post-conflict populations (27, 28, 35).

2.3.2 Hopkins Symptom Checklist-25

Hopkins Symptom Checklist-25 (HSCL-25) is a 25-item scale measuring symptoms of depression and anxiety on a four-point Likert scale (not at all to extremely). The first 10 items measure anxiety symptoms, and the remaining 15 items assess depression symptoms. Mean scores are used, with a recommended cutoff of 1.75 for clinically significant symptoms validated in prior studies with chemical attack survivors (36).

2.3.3 Modified Patient Health Questionnaire-15 (13 items)

PHQ-15 consists of 15 items assessing somatic symptoms; items are rated on a three-point scale (0 = not bothered at all, 1 = bothered a little, and 2 = bothered a lot). Severity categories are defined by summed scores of ≥ 5 mild, ≥ 10 moderate, and ≥ 15 severe (37). Two items in PHQ15 were removed. Item PHQ4 ("menstrual cramps or other problem with your period") was removed as this item is specific to women and thus resulted in 49.4% missing data. According to the Item Response Theory (IRT) analysis, which was performed using a Graded Response Model (GRM) in R (38), PHQ4 showed low discrimination ($\alpha_1 = 0.653$), low factor loading (0.358), and low corrected item-total correlation (0.285), indicating that it poorly differentiates between respondents and contributes little to the overall scale. Similarly, item PHQ11 ("pain and problems during sexual intercourse") was eliminated because 34.8% of the data were missing. It also demonstrated low discrimination ($\alpha_1 = 0.659$), low factor loading (0.361), and a low corrected item-total correlation (0.274), suggesting limited

relevance to the measured construct. Additionally, a previous study suggested that item 4 and item 11 should be removed because these items reduce the overall sensitivity of the scale (39). To maintain comparability with the standard PHQ-15 scoring and cutoff thresholds (≥ 5 , ≥ 10 , and ≥ 15 for mild, moderate, and severe symptoms), we prorated total scores. In particular, the sum of the 13 items was multiplied by 15 (original item count) and divided by 13 (number of items retained), producing a prorated total score aligned with the original 15-item scale range (0–30). This prorated score was then used for all analyses and cutoff interpretations, preserving valid comparison with established severity categories.

2.3.4 Perceived Stress Scale-14

Perceived Stress Scale-14 (PSS-14) is a 14-item scale measuring subjective stress levels on a five-point Likert scale from (0 = never to 4 = very often) over the past month. Total scores are calculated by summing item responses, with higher scores reflecting greater perceived stress (40).

2.3.5 War and Adversity Exposure Checklist-26

War and Adversity Exposure Checklist-26 (WAE-26) combined with 11 supplementary context-specific questions related to chemical attack events for measuring traumatic events. Each item is dichotomously scored (yes/no). The total trauma exposure score was calculated by summing all endorsed items, including the 11 supplementary questions (34).

Scale thresholds were determined through consultation with individual experts and organizations experienced in working with trauma survivors in the Kurdistan region, as well as through review of the existing literature (30, 34, 36, 41, 42).

Demographic information, including age, gender, education, marital status, occupation, and income, as well as chronic disease status, family history of mental illness, prior psychiatric diagnoses, and current psychotropic medication were collected through structured interviews. Chronic disease was defined as the presence of diagnosed, long-term medical conditions requiring continuous management, such as cardiovascular disease and diabetes. Family history of mental illness referred to any first-degree relatives with a diagnosed psychiatric disorder. Information on participants' prior psychiatric diagnoses and current psychotropic medication use was obtained to determine whether participants had previously visited a psychiatrist; received formal diagnoses such as depression, PTSD, and anxiety; or were currently taking psychotropic medications. Although formal psychiatric diagnosis based on standardized criteria such as the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) and the International Classification of Diseases, 11th Revision (ICD-11) is the clinical gold standard, clinical history obtained via self-report or clinical interviews provides important pragmatic context in epidemiological research where structured clinical interviews may not always be feasible. Medication use was coded as a binary variable (yes/no) in analyses due to small numbers in

specific medication categories. These clinical variables were included as covariates in all relevant analyses to control for their potential confounding effects on symptom severity.

All scales were translated into Kurdish/Sorani by two independent, qualified translators who were proficient in both English and Kurdish. The Kurdish versions were back-translated into English by new translators to verify the quality of the back translation, i.e., to ensure linguistic equivalence and conceptual similarity. Discrepancies between the back-translated versions and the original English questionnaires were dealt with in the revisions.

2.4 Data quality control

Data quality control included examination of frequency distributions for categorical variables and descriptive statistics (mean, median, standard deviation, skewness, and kurtosis) for continuous and ordinal variables, including individual items of the key psychometric scales (HSCL-25, PCL-5, PHQ-15, and PSS-14) to assess response distributions and identify anomalies. Continuous demographic and clinical variables were similarly inspected. We identified and removed duplicate cases using unique participant identifiers and verified data variable types and coding consistency. Internal consistency reliability of the scales was assessed with Cronbach's alpha (α) and Kuder–Richardson Formula 20 (KR-20) as appropriate.

2.5 Statistical analysis

Both R version 4.4.2 and IBM SPSS Statistics version 29 were utilized for statistical analysis. Descriptive statistics, Cronbach's α , and KR-20 were performed to measure the internal consistency. IRT was used specifically for the PHQ-15 to assess individual item performance and guide the removal of poorly performing items. The GRM was employed because it is appropriate for analyzing Likert-scale items with ordered response categories, allowing estimation of item discrimination and difficulty parameters. Pearson correlation was conducted to assess relationships among psychological variables. Multivariate analyses were to evaluate the effects of demographic and clinical variables on mental health outcomes. The GLM was run in R using the `manova()` function from the base stats package, with Pillai's Trace as the test statistic due to its robustness against assumption violations such as heterogeneity of covariance matrices, which was assessed using Box's M test [`boxM()` function from the `heplots` package]. Homogeneity of variances for individual outcomes was evaluated using Levene's test [`leveneTest()` from the `car` package]. To ensure adequate statistical power for identifying medium effect sizes in models with 12 predictors, a post-hoc power analysis was conducted using the `pwr` package.

3 Results

3.1 Scales reliability

Data quality checks revealed no significant outliers or anomalies in item responses across all scales and variables. All translated and self-developed measures showed good to excellent internal consistency. The PCL-5 had $\alpha = 0.885$, the HSCL-25 scale reached $\alpha = 0.936$, the PHQ-13 had $\alpha = 0.868$, the PSS-14 had $\alpha = 0.875$, and the WAEC-26, when combined with the 11 additional supplementary questions, also demonstrated good internal consistency (KR-20 = 0.77), assessed using KR-20 since the scale was dichotomous with (yes/no) response options.

3.2 Sample characteristics and demographic variables

A total of 534 chemical gas survivors exposure participated in the study. Sample characteristics are summarized in Table 1. The sample was 56.2% female, with a mean age of 53.6 years. Most participants had elementary education 44% and low income. The majority were married and homemakers or government employees, with a high rate of chronic disease.

Table 1. Demographic and Clinical Characteristics of the Sample (N=534)

Demographic Variables	Frequency n (%) or Mean (SD)
Sample	534 (100%)
Female	300 (56.2%)
Age, Mean (SD)	53.6 (11.16)
Income in US\$, Mean (SD)	419.26 (269.49)
Level of Education	
No Formal Education	122 (22.8%)
Elementary School	235 (44.0%)
Secondary School	77 (14.4%)
Institution or University	100(18.7%)
Work Status	
Unemployed	24 (4.5%)
Homeworker	228 (42.7%)
Governmental employee	158 (29.6%)
Self-Employee	66 (12.4%)
Unable to Work	7 (1.3%)
Retired	51 (9.6%)
Marital Status	
Single	48 (9.0%)
Married	444 (83.1%)
Divorced	13 (2.4%)
Widow / Widower	29 (5.4%)
Number of Children. Mean (SD)	4.4 (2.6)
Chronic disease	
Having Chronic Disease	367 (68.7%)

3.2.1 Symptom severity and clinical prevalence

The mean (M) score and standard deviation (SD) for the PCL-5 were 36.14 and 16.05, respectively; PHQ-13: M = 15.72, SD = 7.33; HSCL25: M = 2.07, SD = 0.66; and PSS14: M = 24.30, SD = 11.14. Combined traumatic events (37 items) (M = 14.33, SD = 4.66) ranged from 2 to 30 traumatic events. The most frequently reported extreme symptoms for the PHQ-13 were “pain in your arms, legs, or joints” (74.7%), “feeling tired or having low energy” (66.1%), and “back pain” (64.6%). For the PCL-5, common symptoms were “repeated, disturbing, and unwanted memories” (38%), “feeling very upset when something reminded you of the stressful experience” (23.8%), and “trouble falling or staying asleep” (30.5%). Regarding HSCL-25, “feeling tense or keyed up” (26.4%) and “loss of sexual interest or pleasure” (26.8%) were the most frequently reported. The most frequently reported events were witnessing bombing or destruction (91.8%), experiencing life-threatening natural disasters (90.6%), exposure to armed combat (90.3%), and being severely deprived of food and water (83.9%). Less common events included seeing dead bodies and life-threatening illness or injury (Figure 1). The distribution of PCL-5 symptom severity categories is illustrated in Figure 2.

Figure 1. Common traumatic life events experienced by Kurdish survivors of chemical attacks.

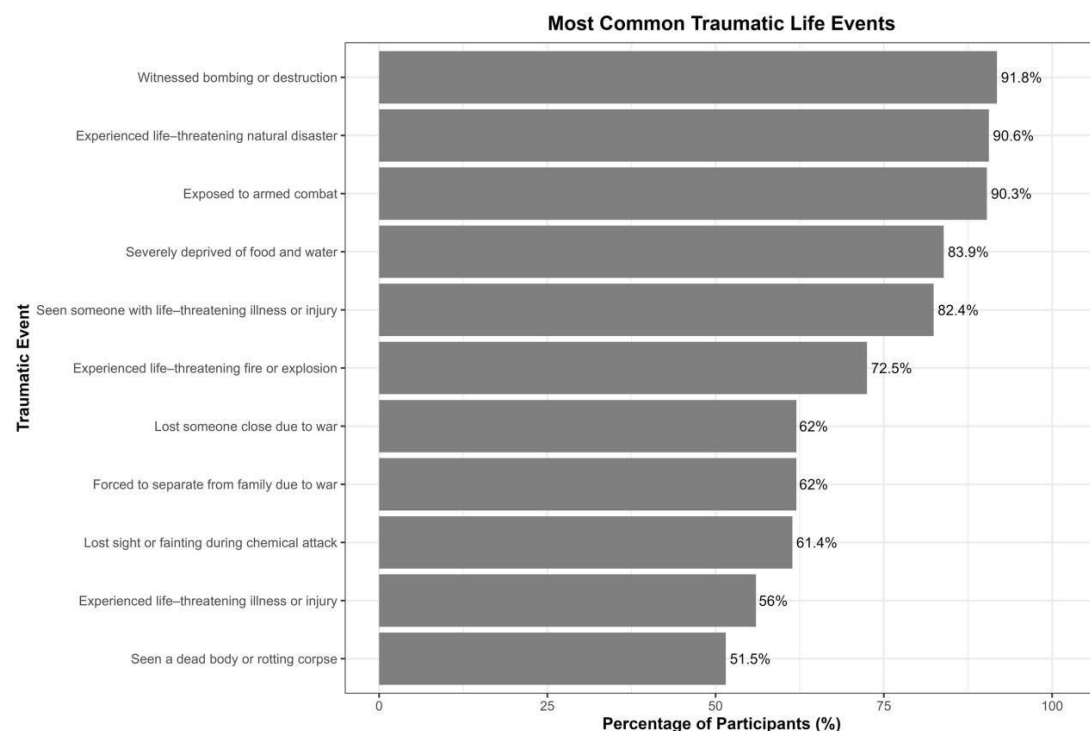
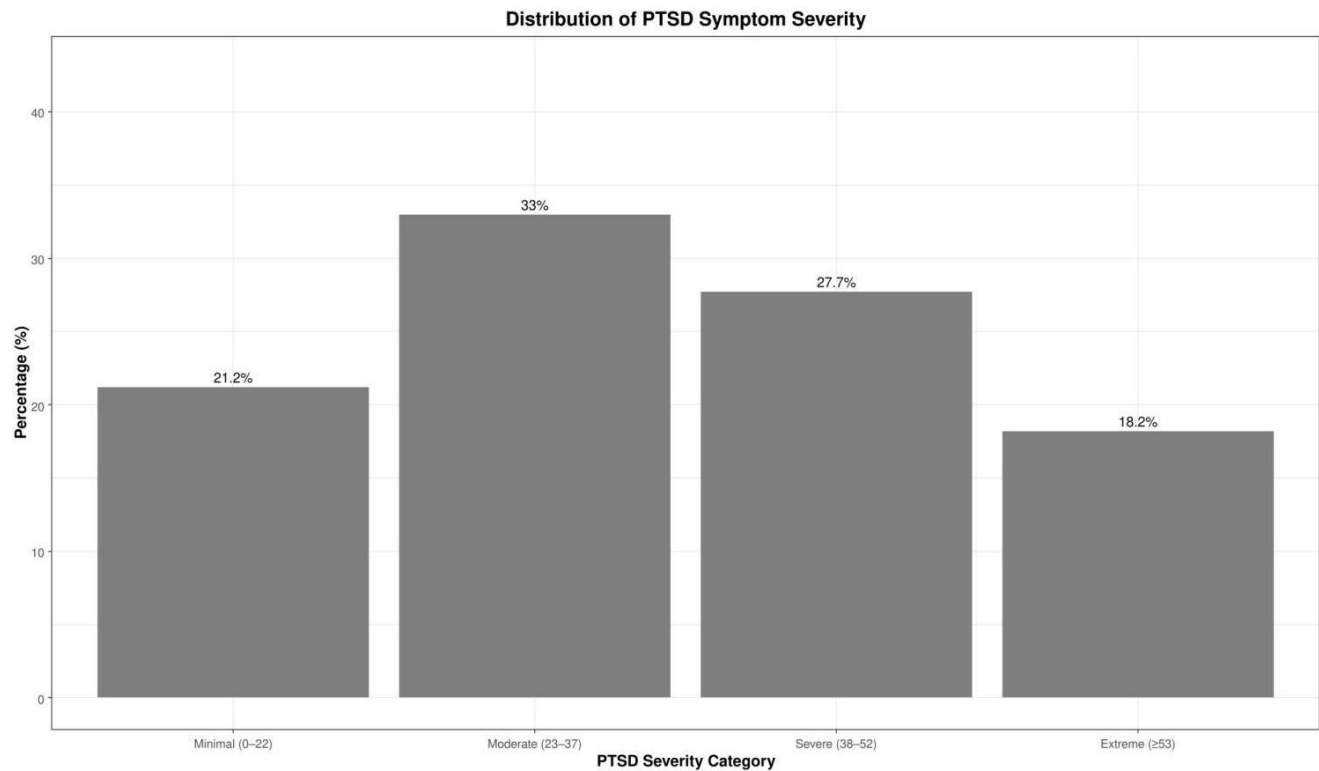


Figure 2. Distribution of PTSD symptom severity categories based on PCL-5 total scores. Categories were defined for descriptive purposes only. Bars represent the percentage of participants in each category; values on top of the bars show these percentages.



Related to clinically relevant cutoff scores, we found for PCL-5 that 78.8% of participants scored ≥ 23 on the PCL-5. For the HSCL-25, 65.2% had scored ≥ 1.75 . For PHQ13, 10.1% of participants had minimal symptoms (score < 5), 12.9% had mild symptoms (≥ 5), 21.2% had moderate symptoms (≥ 10), and 55.8% had severe somatic symptoms (≥ 15).

3.2.2 Symptom comorbidities

Comorbidities were common; 46.3% of participants met cutoff criteria for all three symptom domains (PTSD, somatic symptoms, and depression/anxiety); 65.2% of participants had PTSD and depression/anxiety symptoms, 51.5% had PTSD and somatic symptoms, while 47.8% had depression/anxiety and somatic symptom comorbidities.

3.2.3 Psychotropic medication use and diagnostic profiles

Of the total participants, 16.8% reported current use of psychotropic medications. The other psychotropics had the highest proportion (6.4%), followed by fluoxetine (3.1%), sertraline (2.6%), alprazolam (2.2%), and amitriptyline (1.5%) (Figure 3). Approximately 26.0% reported no prior psychological diagnosis, while 8.8% reported comorbid PTSD and depression, with smaller proportions reporting PTSD alone (1.9%), depression (3.7%), anxiety (1.8%), and obsessive–compulsive disorder (OCD) (0.8%), while the majority (57%) did not visit a healthcare provider (Figure 4). The comorbid group consists of participants meeting criteria for both PTSD and depression, distinct from those with only one disorder.

Figure 3. Psychotropic medication usage by type among study participants. Bar chart displays the proportion of participants prescribed each major psychotropic medication category.

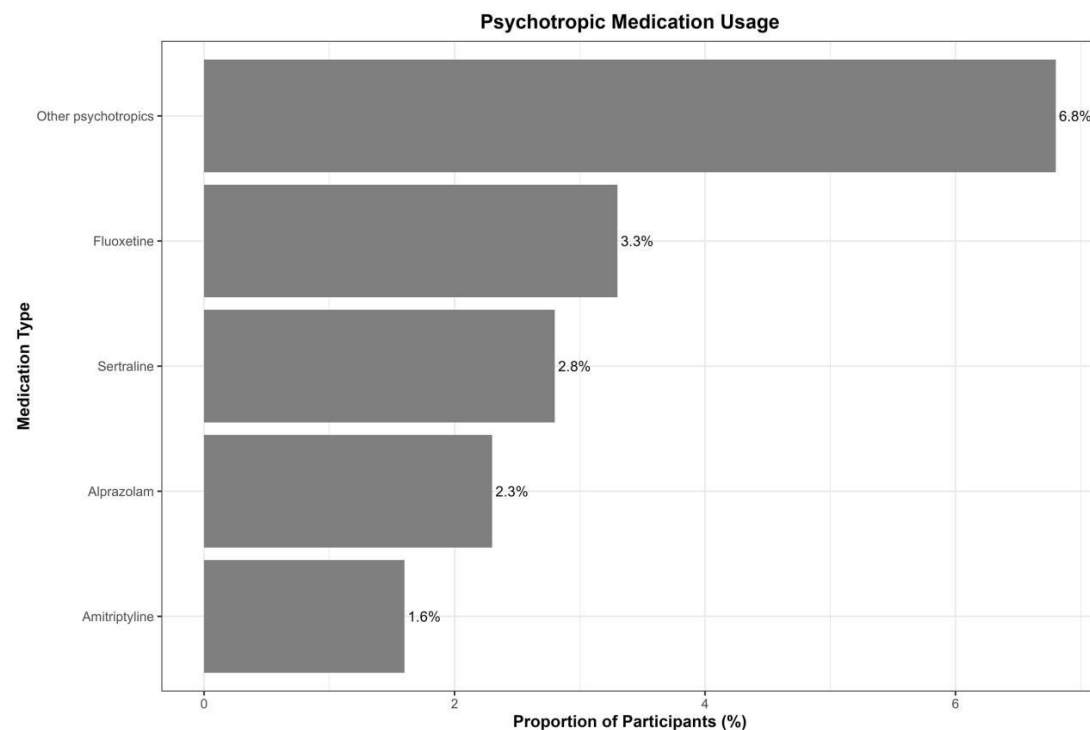
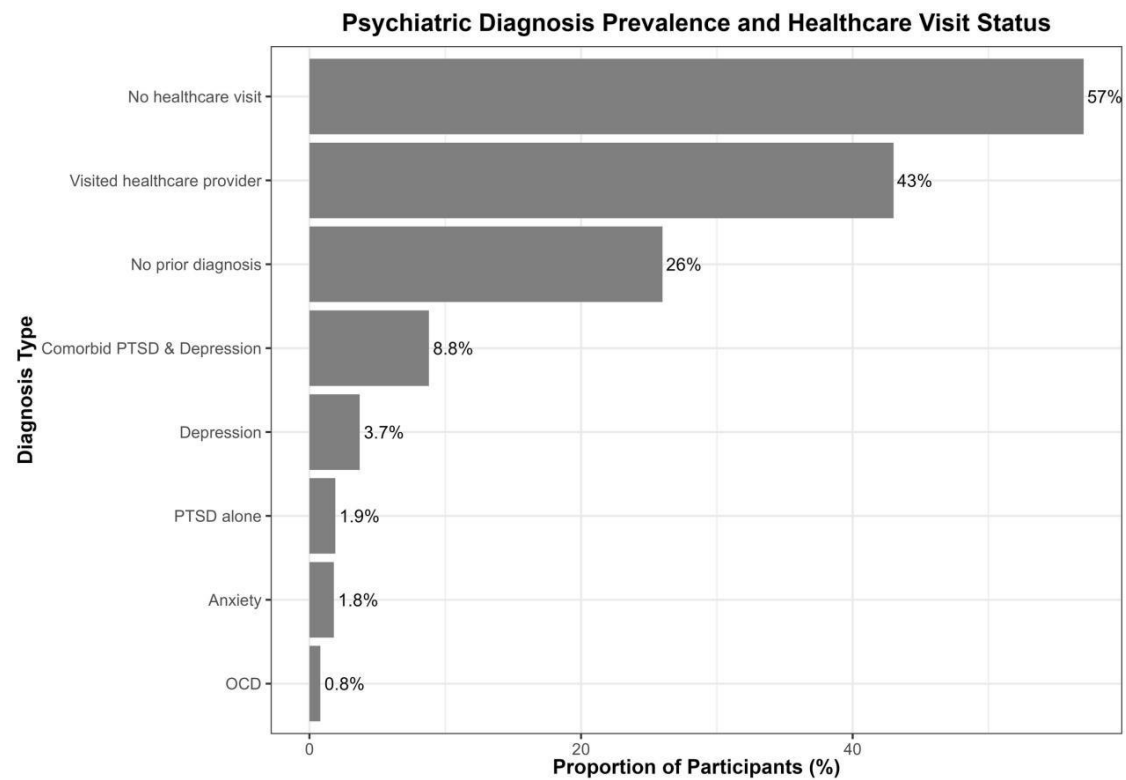


Figure 4. Prevalence of prior psychiatric diagnoses and healthcare professional visit status among participants.



3.3 Correlations

To explore the relationships among psychological symptoms, Pearson correlation analyses were performed. These revealed a significant positive correlation among all scales, $p < 0.001$. PCL-5 shows a strong positive correlation with HSCL-25 ($r = 0.80$) and somatic symptoms (PHQ-13, $r = 0.69$) and a moderate correlation with PSS-14 ($r = 0.58$), but a weaker positive correlation with the trauma checklist ($r = 0.25$). All correlations remained significant after Bonferroni correction ($p < 0.0033$) (Table 2).

Table 2. Shows Pearson correlations among psychological symptom scales, presenting significant positive associations between the PCL-5, HSCL-25, PHQ-13, PSS-14, and the Trauma Event Checklist, all significant at $p < .001$.

Variable	PHQ-13	PCL-5	HSCL-25	PSS-14	Trauma Event Checklist-37
PHQ-13	1	0.696**	0.742**	0.506**	0.196**
PCL-5	0.696**	1	0.805**	0.583**	0.258**

HSCL-25	0.742**	0.805**	1	0.719**	0.235**
PSS-14	0.506**	0.583**	0.719**	1	0.158**
Trauma Event Checklist-37	0.196**	0.258**	0.235**	0.158**	1

Note: **Indicates significance at $p < .001$ (two-tailed).

3.4 Multivariate general linear model analysis

GLM was performed to assess the effects of demographic, clinical, and treatment-related predictors on three related mental health outcomes: somatic symptoms (PHQ-13), PTSD symptoms (PCL-5), and anxiety/depression symptoms (HSCL-25). Assumption checks revealed a significant heterogeneity of covariance matrices across groups [Box's M, $\chi^2(42) = 65.84$, $p = 0.011$]. Since Box's M is sensitive to sample size and can detect minor differences, Pillai's Trace was used for multivariate testing due to its robustness against violation of covariance homogeneity assumptions. Additionally, Levene's tests for homogeneity of variance for each outcome were nonsignificant; PHQ-13 ($p = 0.236$), PCL-5 ($p = 0.936$), and HSCL-25 ($p = 0.114$), supporting the assumption of equal variances for the univariate analyses.

3.4.1 Explained variance of the model

The multivariate model explained significant variance in symptom severity across the three outcomes, with adjusted R^2 values of approximately 0.47 for somatic symptoms, 0.35 for PTSD symptoms, and 0.43 for anxiety/depression symptoms.

3.4.2 Multivariate effects

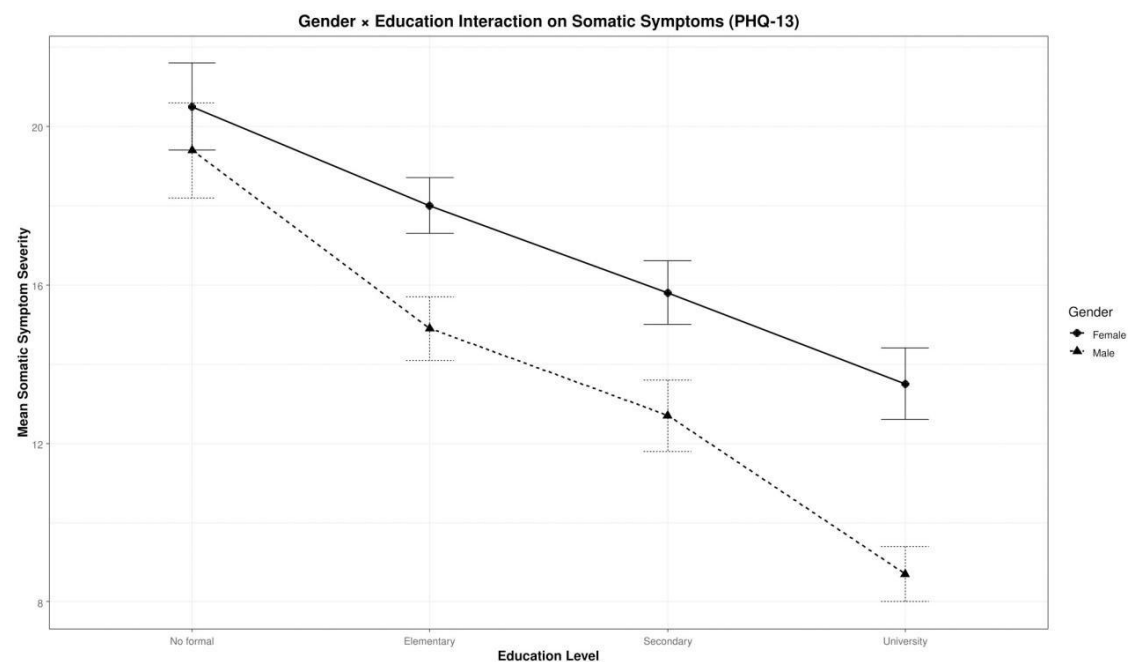
Significant main effects were observed for gender [Pillai's Trace = 0.187, $F(3,504) = 38.66$, $p < 0.001$], education [Pillai's Trace = 0.187, $F(9,1518) = 11.21$, $p < 0.001$], trauma exposure [Pillai's Trace = 0.184, $F(3,504) = 37.90$, $p < 0.001$], family history of mental disorder [Pillai's Trace = 0.038, $F(3,504) = 6.69$, $p < 0.001$], living location [Pillai's Trace = 0.049, $F(6,1010) = 4.25$, $p < 0.001$], chronic disease [Pillai's Trace = 0.023, $F(3,504) = 3.99$, $p = 0.008$], income [Pillai's Trace = 0.205, $F(3,504) = 43.41$, $p < 0.001$], occupation [Pillai's Trace = 0.056, $F(15,1518) = 1.92$, $p = 0.018$], psychotropic medication use [Pillai's Trace = 0.120, $F(3,504) = 22.87$, $p < 0.001$], and prior diagnosis [Pillai's Trace = 0.086, $F(18,1518) = 2.48$, $p < 0.001$]. Age, social status, and social support were not significant multivariately. The gender $\times$ education interaction was not significant [Pillai's Trace = 0.028, $F(9,1518) = 1.59$, $p = 0.11$] (see Supplementary Table S1).

3.4.3 Univariate effects and interaction details

Gender had significant main effects on all outcomes, somatic symptoms [$F(1,506) = 112.64$, $p < 0.001$, Partial eta squared effect size (partial η^2) = 0.18; coefficient $\beta = 0.037$], PTSD symptoms [$F(1,506) = 30.53$, $p < 0.001$, partial $\eta^2 = 0.06$; $\beta = 1.847$], and depression/anxiety symptoms [$F(1,506) = 54.95$, $p < 0.001$, partial $\eta^2 = 0.10$; $\beta = 0.034$]; symptom severity was higher among women.

Education showed significant main effects on all symptom domains, with the strongest effect on somatic symptoms [$F(3,506) = 30.91$, $p < 0.001$, partial $\eta^2 = 0.15$; $\beta = -5.17$ to -8.38 across levels]. Higher education levels were associated with lower symptom severity. The gender $\times$ education interaction was significant for somatic symptoms [$F(3,506) = 4.55$, $p = 0.004$, partial $\eta^2 = 0.03$; $\beta = 2.84$ to 5.99], revealing that higher education is a protective factor against somatic symptom severity in both genders, with a marked reduction observed among female participants (Figure 5).

Figure 5. Interaction between gender and education on somatic symptoms (PHQ-13). Estimated marginal means of PHQ-13 somatic symptoms are plotted by education level for female participants (solid line) and male participants (dashed line).



Income was significantly associated with symptom severity across all outcomes: somatic symptoms [$F(1,506) = 125.75$, $p < 0.001$, partial $\eta^2 = 0.46$; $\beta = -0.009$], PTSD [$F(1,506) = 51.46$, $p < 0.001$, partial $\eta^2 = 0.34$; $\beta = -0.012$], and depression/anxiety symptoms [$F(1,506) = 68.15$, $p < 0.001$, partial $\eta^2 = 0.57$; $\beta = -0.001$], with higher income associated with fewer symptoms.

Trauma exposure was strongly associated with increased symptoms across somatic [$F(1,506) = 84.58$, $p < 0.001$, partial $\eta^2 = 0.30$; $\beta = 0.34$], PTSD [$F(1,506) =$

80.43, $p < 0.001$, partial $\eta^2 = 0.38$; $\beta = 0.85$], and depression/anxiety symptoms [$F(1,506) = 83.82$, $p < 0.001$, partial $\eta^2 = 0.34$; $\beta = 0.031$].

Family history of mental disorder significantly predicted depression/anxiety symptoms [$F(1,506) = 10.98$, $p < 0.001$, partial $\eta^2 = 0.05$; $\beta = 0.089$]. Living location was associated with somatic [$F(2,506) = 8.82$, $p < 0.001$, partial $\eta^2 = 0.03$; $\beta = 2.54$] and depression/anxiety symptoms [$F(2,506) = 9.44$, $p < 0.001$, partial $\eta^2 = 0.04$; $\beta = 0.21$], with urban residents reporting higher symptom severity compared to those living in peripheral areas or in villages. Chronic disease showed a modest association with somatic symptoms [$F(1,506) = 9.57$, $p = 0.002$, partial $\eta^2 = 0.03$; $\beta = 1.07$].

Occupation had a significant effect only on depression/anxiety symptoms across occupational groups [$F(5,506) = 3.30$, $p = 0.006$, partial $\eta^2 = 0.03$], but showed no significant effect on PTSD and somatic symptoms.

Psychotropic medication use was associated with worse symptom severity across all outcomes, somatic symptoms [$F(1,506) = 25.74$, $p < 0.001$, partial $\eta^2 = 0.05$, $\beta = 2.31$], PTSD [$F(1,506) = 42.89$, $p < 0.001$, partial $\eta^2 = 0.08$, $\beta = 6.93$], and depression/anxiety symptoms [$F(1,506) = 67.43$, $p < 0.001$, partial $\eta^2 = 0.12$, $\beta = 0.30$].

Prior diagnosis was a significant predictor of PTSD [$F(6,506) = 4.62$, $p < 0.001$, partial $\eta^2 = 0.05$, $\beta = 10.11$] and depression/anxiety symptoms [$F(6,506) = 6.26$, $p < 0.001$, partial $\eta^2 = 0.05$, $\beta = 0.53$], but not somatic symptoms ($p = 0.17$).

Interactions between education $\times$ trauma exposure and income $\times$ trauma exposure were not significant, indicating that the effects of trauma exposure on symptoms do not differ across education or income levels in this sample (see Supplementary Table S2 for details).

4 Discussion

To our knowledge, this is the first study conducted on the long-term psychological outcomes of a chemical weapons attack on a large community. We studied survivors of a chemical attack that occurred more than three decades ago to assess their current wellbeing and psychopathology. These findings fill a critical gap in the literature on prolonged trauma effects.

Most survivors met criteria for probable PTSD (78.8%) using a cutoff score of ≥ 23 , which is lower than thresholds typically used in other populations (≥ 31 – 33) (43), possibly contributing to the high prevalence observed. Additionally, a significant proportion suffered from depression/anxiety and somatic symptoms.

These results show that extreme and chronic stress exposure has lasting negative effects on mental health, documenting the collective trauma experienced by populations exposed to genocidal attacks. The high rate of depression/anxiety and

somatic symptoms reflects the psychopathological burden beyond PTSD and the need for effective interventions that target comorbidity of psychopathology.

These results are in line with previous findings that revealed a high prevalence of PTSD and depression among Anfal survivors and a high prevalence of PTSD among survivors of the Yazidi genocide. They also align with earlier research showing that almost half of participants exceeded cutoff scores for PTSD and depression among chemical attack survivors (16, 40, 44).

Our sample also experienced multiple subsequent traumatic events during the chemical attack, with cumulative exposures ranging from 2 to 30 events. Consistent with the ecological model of trauma, prior studies indicate that cumulative exposure to various types of trauma and ongoing environmental stressors is associated with elevated prevalence and severity of PTSD and depression. In this study, we measured cumulative trauma exposure using a validated composite trauma scale. This comprehensive approach probably explains the heightened prevalence and severity of psychological symptoms identified in our sample. These results underscore the complex challenges survivors experience from multiple overlapping stressors, highlighting the need for multi-level mental health interventions (27, 28, 34, 45).

Based on these results, our GLM analysis offers a more nuanced view of how trauma exposure and sociodemographic and clinical factors interact to influence mental health consequences. The GLM analysis shows that psychological symptoms in this population are driven by many interconnected factors, with trauma exposure emerging as a particularly strong predictor across all symptom domains. Consistent gender differences were revealed, with women exhibiting greater vulnerability. Conversely, higher education and income appear as protective factors, particularly regarding somatic symptoms in women, as shown by the interaction between gender and education. Additionally, family psychiatric history, urban residence, and chronic disease were also shown to influence symptom variability, highlighting the intricate interaction of sociodemographic and clinical factors in shaping mental health outcomes. Altogether, our findings underscore the importance of integrated intervention strategies that address trauma, educational inequalities, gender-specific vulnerabilities, socioeconomic support, and broader social factors, in order to reduce long-term psychological distress and promote sustainable improvements in mental health across the Kurdistan region.

Notably, the association between elevated symptoms and psychotropic medication use observed probably indicates practical clinical circumstances where individuals with more severe or treatment-resistant PTSD and depression are predominantly prescribed medication. Meta-analyses reveal modest efficacy of selective serotonin reuptake inhibitors (SSRIs) and related medications, with many individuals showing partial or even no response. Persistent symptoms may arise from complex neurobiological modifications and pharmacological response variability, indicating the limitations of medication alone. These results highlight the importance of tailored treatment

strategies that integrate pharmacological, psychotherapeutic, and psychosocial interventions to effectively address the multifaceted needs of trauma survivors (46, 47).

Furthermore, while many participants met cutoff criteria for PTSD, somatic symptoms, and depression/anxiety, fewer reported formal diagnoses or receiving treatment. Specifically, over half had never visited a healthcare provider for mental health concerns, indicating a gap between symptom presence and clinical diagnosis.

Our findings are in line with previous studies that have found that the prevalence of PTSD, depression/anxiety, and somatic symptoms is far more common among women (16, 45–50), especially those who have little or no education (51). In the Kurdish cultural context, women often have limited education and are expected to marry and live as housewives raising children. Lacking employment and experiencing marital conflicts are significant vulnerability factors for developing low self-esteem. This, combined with additional stressful life events, can increase the risk of depression development (52, 53). Importantly, all scales showed good to excellent reliability and strong internal consistency. These findings are consistent with previous studies on these scales in other countries and other samples (37, 54–57). The strong correlations between PCL-5, PHQ-13, and HSCL-25 measures in our study ($r = 0.69$ to 0.80) are consistent with earlier research, confirming the expected overlap and convergent validity of these measures in trauma populations (58).

Although our psychometric measures were robust, the real challenge extends beyond measurement to the lack of adequate services. Despite evidence of a high prevalence of psychological symptoms, people living in the Kurdistan region have very limited or no access to health services in general, let alone psychiatric care, or psychotherapy. The high prevalence of psychopathology and its long-term, chronic effects highlight the urgent need for developing specific treatments and establishing services to support this large group of survivors. Effective treatment strategies are available but have, unfortunately, not yet been implemented in Kurdistan. However, these treatments need to be adapted to the specific local, cultural, and religious needs.

This work uniquely contributes to the literature by employing a multivariate analytical methodology to simultaneously model multiple risk factors, their interaction, and their distinct and combined contributions towards symptom severity. This adds robustness to evidence supporting customized, multidisciplinary intervention approaches encompassing educational support and socio-economically oriented empowerment, especially among susceptible subgroups.

4.1 Limitations and future research

In addition to the valuable insights contributed by this study, there are some limitations. First, our sample may be affected by the survivor bias, as a large part of exposed people have died during the elapsed time since the chemical attack. Second,

many survivors from Halabja city left the area and now live outside its territory, complicating the sample recruitment process.

Lastly, there is a lack of formal data on psychotherapeutic interventions, further limiting our ability to discern the impact of mental health support on the long-term outcomes. Many participants did not receive structured psychotherapy from trained psychotherapists; instead, they received irregular support and counseling from some organizations that had insufficient resources to address their mental health conditions and may have impacted their recovery.

Future research must investigate, in greater depth, the interaction of psychophysiological, psychological, and socio-demographic (environmental) factors in shaping trauma responses. Future studies should address the limitations related to the generalizability and applicability of our findings.

5 Conclusion

This study provides vital evidence on the long-term psychological consequences of chemical weapons exposure, demonstrating the high incidence of PTSD, depression/anxiety, and somatic symptoms among survivors. Our findings show trauma exposure as a strong factor, with sociodemographic factors such as gender, education, and income significantly influencing symptom severity and resilience. Psychometric robustness across multiple validated scales supports the reliability of these insights.

The limited access to mental health services in the Kurdistan region, despite the need, highlights an urgent gap in clinical care and public health infrastructure. These results highlight the urgency for culturally sensitive, multidimensional intervention strategies addressing trauma and its complex socio-environmental correlates.

By combining detailed symptom profiling with multivariate analysis, this study enhances understanding of the long-term psychological effects of trauma and establishes a basis for developing targeted, evidence-based clinical and strategic interventions. Future research should expand on these findings by integrating biological or genetic data to explain trauma mechanisms and enhance intervention approaches.

Statements

Data availability statement

The raw data supporting the conclusions of this article will be made available by the authors, without undue reservation.

Ethics statement

The studies involving humans were approved by Research Center, University of Duhok. The studies were conducted in accordance with the local legislation and

institutional requirements. The participants provided their written informed consent to participate in this study. Written informed consent was obtained from the individual(s) for the publication of any potentially identifiable images or data included in this article.

Author contributions

IM: Investigation, Writing – review & editing, Writing – original draft, Data curation, Conceptualization, Resources, Formal Analysis, Methodology, Visualization, Project administration. HS: Data curation, Writing – original draft, Methodology. BH: Data curation, Methodology, Writing – review & editing. JK: Writing – review & editing, Methodology. HN: Writing – original draft, Data curation. SA: Writing – review & editing, Methodology. AQ: Writing – review & editing, Data curation. SP: Writing – review & editing. MR: Writing – review & editing. TS: Resources, Writing – review & editing, Supervision, Methodology. UH: Resources, Supervision, Methodology, Writing – review & editing. MH: Conceptualization, Methodology, Supervision, Funding acquisition, Resources, Writing – review & editing.

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Conflict of interest

The author(s) declared that this work was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Supplementary material

The Supplementary Material for this article can be found online at: <https://www.frontiersin.org/articles/10.3389/fpsy.2025.1693072/full#supplementary-material>

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Appendix C

“Reliability and validity of the Kurdish version of the patient health questionnaire- 15 in a trauma- affected population”

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Abstract

Background: Somatic symptoms are common among trauma-exposed populations and are often associated with persistent distress and functional impairment, highlighting the need for culturally appropriate assessment tools.

Objective: This study aimed to translate and culturally adapt the Patient Health Questionnaire-15 (PHQ-15), a screening measure of somatic symptom burden, into Central Kurdish and assess its psychometric properties among chemical gas survivors exposure.

Methods: A total of 534 survivors were recruited through community and registry sources. After translation and cultural adaptation, item 4 and item 11 were removed due to cultural and gender-specific considerations and limited psychometric performance, resulting in a 13-item version. Factor structure was examined using confirmatory factor analysis (CFA) and exploratory structural equation modeling (ESEM). Measurement invariance across gender and age groups was evaluated using multi-group CFA. Internal consistency was assessed using McDonald’s omega. An exploratory network analysis (EBICglasso) examined conditional associations among symptoms.

Results: Both a unidimensional CFA model (CFI = .988, RMSEA = .054) and a three-factor ESEM model (CFI = .992, RMSEA = .046) showed good model fit, with the ESEM structure providing a more differentiated representation of pain/fatigue, gastrointestinal, and cardiopulmonary domains. Measurement invariance testing supported full scalar invariance across gender, while partial metric invariance was observed across age groups. Internal consistency was acceptable ($\omega = .70-.83$). Network estimates demonstrated acceptable stability, with correlation stability coefficients of 0.59 for edge strength and 0.52 for bridge strength, and should be interpreted as exploratory.

Conclusion: The culturally adapted 13-item Kurdish PHQ demonstrates acceptable reliability and initial evidence of construct validity in trauma-exposed survivors of chemical attacks. The instrument may be used as a screening tool for somatic symptom burden in this context, while further validation in broader Kurdish populations is recommended.

Key words: Trauma, PHQ-15, Kurdistan, Translation, Psychometrics

Introduction

Somatic symptoms are common among the general population. Most people experience mild, temporary bodily sensations that are considered normal [1]. In certain cases, however, these symptoms become chronic and distressing, leading to marked functional impairment and psychological distress. Somatic symptom disorder (SSD) as defined in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision (DSM-5-TR) is a clinically significant condition defined by chronic physical symptoms associated with excessive thoughts, emotions, or behaviors related to these symptoms [2]. Affected individuals often report negative thoughts, increased negative emotions, and help-seeking behaviors [3]. Persistent somatic symptoms are also strongly associated with psychological distress, particularly depressive and anxiety symptoms. Trauma exposure also increases vulnerability to both somatic and psychological symptoms[3].

Survivors of traumatic events frequently report chronic pain, fatigue, and sleep disturbances alongside post-traumatic stress symptoms. A previous study of Kurdish chemical attack survivors has demonstrated lasting physical and psychological consequences, including impaired quality of life, increased bodily pain, and reduced energy compared with non-exposed populations. These combined effects highlight the need of culturally appropriate screening tools for trauma-affected populations[4-7].

Somatic symptoms create significant challenges not only for affected individuals but also for healthcare systems. They frequently lead to repeated medical consultations and unnecessary diagnostic procedures, contributing to personal suffering and economic cost [8,9]. Therefore, it is essential to validate instruments such as the PHQ-15 across cultural and linguistic contexts, which may improve the assessment of somatic symptoms and inform clinical decision-making [8,9].

The PHQ-15 is a widely used screening tool that measures somatic symptom burden and helps identify individuals who may require further evaluation rather than serving as a diagnostic instrument for SSD [9]. It assesses the frequency and severity of fifteen common physical symptoms. Factor-analytic studies consistently demonstrate a multidimensional structure, typically reflecting symptom domains related to pain, gastrointestinal disturbance, cardiopulmonary problems, and fatigue [10-13]. The instrument has been validated in diverse populations and settings, with strong evidence supporting its psychometric soundness [9].

Following cultural adaptation, the PHQ-15 was modified to a 13-item version to address cultural and gender-specific considerations among trauma-exposed survivors of chemical attacks. To examine the complex structure of somatic symptoms, we employed exploratory structural equation modeling (ESEM) [14,15], which allows theoretically plausible cross-loadings, and network analysis, which conceptualizes symptoms as dynamically interconnected components of a broader system. This approach complements factor analyses by identifying potential ‘bridge’ symptoms

that link different symptom domains, which may be important targets for clinical intervention or further research, especially in trauma-exposed populations where symptom interactions are complex [16]. These approaches provide insight into symptom patterns and relationships, complementing traditional latent factor analyses, without implying causal mechanisms.

By providing a culturally adapted and psychometrically evaluated 13-item Kurdish PHQ in trauma-exposed survivors of chemical attacks using ESEM and multi-group CFA, this study offers the first validation of the instrument in this population and presents evidence on its factor structure and symptom relationships.

The aims of the study are to validate the psychometric properties of the Kurdish PHQ-13 in the trauma-exposed survivors of chemical attacks in the Kurdistan region, including 1) examine the factor structure of the Kurdish PHQ-13 using both (CFA) and (ESEM), 2) evaluate measurement invariance across gender and age groups to assess the consistency and validity of the scale, 3) investigate associations between socio-demographic, clinical, and psychological factors and Kurdish PHQ-13 scores and 4) explore symptom interrelations using network analysis and identify bridge symptoms linking different somatic domains.

Method and Instruments

The validation of the Kurdish PHQ-13 was part of a larger study investigating factors influencing the development of depression, anxiety, and post-traumatic stress disorder among survivors of the Yazidi genocide and chemical attack. The study received ethical approval (see Ethics Approval and Consent to Participate section).

Data collected from multiple locations in the Kurdistan Region, Halabja (n = 373), Sulemani (n = 54), the Khormal (n = 44), the Serwan (n = 41), and the Tawella (n = 22) over seven months, from March to September 2023, resulting in a total of 534 participants from 679 contacted individuals, corresponding to a response rate of approximately 79%. Written informed consent was obtained from all enrolled participants. Additionally, 145 potential participants did not participate, 37 explicitly refused, while others were unreachable or deceased.

Participants were Kurdish chemical gas survivors attacks, recruited through three pathways. The largest group (n = 353, 66%) was identified via official registries maintained by the Kurdistan Regional Government's Ministry of Martyrs and Anfal Affairs, these registries document survivors eligible for financial compensation following formal evaluations. A second group (n = 102, 19%) comprised individuals with documented exposure and formal medical assessment who were not yet registered due to administrative delays. The remaining participants (n = 79, 15%) were recruited through snowball sampling, eligibility confirmed using structured trauma exposure scale (see Supplementary Table S1).

Kurdish PHQ-13 total scores were compared across the three recruitment pathways using one-way ANOVA to examine potential differences in symptom severity. No significant differences were observed, suggesting that recruitment source is unlikely to have substantially influenced reported symptom severity.

Inclusion criteria required confirmed exposure to chemical gas, informed consent to participate, and physical and mental capacity to complete assessments. Exclusion criteria included inability to consent or severe illness preventing participation.

Participants were informed in detail regarding the project's objectives, procedures, and potential risks. Each assessment took 60-90 minutes. Clinical trial number: not

applicable.

Data Collection Instruments

PTSD Checklist for DSM-5

The PTSD Checklist for DSM-5 (PCL-5) consists of 20 self-reported items assessing PTSD symptoms experienced over the past month, rated on a 5-point scale from 0 (not at all) to 4 (extremely). Total scores are calculated by summing items, with a cutoff score of ≥ 23 indicating probable PTSD [17].

Hopkins Symptom Checklist (HSCL25)

The 25-item HSCL-25 uses a 4-point Likert scale (not at all to extremely) to measure anxiety and depression symptoms. The first 10 items measure anxiety symptoms, and the remaining 15 items assess depression symptoms. Mean scores are employed, and a cutoff of 1.75 is suggested for clinically significant symptoms that have been confirmed in earlier research involving survivors of chemical attacks [18].

The Patient Health Questionnaire-15 (PHQ-15)

The PHQ-15 assesses the extent to which individuals have been bothered by common physical symptoms such as pain, gastrointestinal complaints, fatigue, cardiopulmonary symptoms, and sleep difficulties during the last 4 weeks. It includes 15 items, each rated on a 3-point scale (0 = “not bothered at all,” 1 = “bothered a little,” 2 = “bothered a lot”) [19].

Perceived Stress Scale-14 (PSS14)

PSS-14 has 14 items measure stress levels over the previous month using a 5-point frequency scale that ranges 0 (never) to 4 (very often). Higher summed scores indicate greater perceived stress [20].

War and Adversity Exposure Checklist-26 (WAEC-26)

The WAEC-26 measures traumatic event exposure through 26 dichotomous (yes/no) items, with total exposure scored by summing endorsed items.

The PCL-5 and WAEC-26 have been validated in Kurdish trauma survivor, showing strong psychometric properties, including strong internal consistency [17]. Other measures, such as the HSCL-25 and PSS-14, have not undergone formal validation studies in the region but have been widely used in previous research and intervention settings with traumatized Kurdish populations, including chemical gas survivors attacks[21]. This extensive use provides practical support for their applicability in these contexts. The Kurdish PHQ-15 and all other instruments utilized in this study demonstrated good to excellent internal consistency with alpha coefficients (α) for PCL-5 ($\alpha = .885$), HSCL-25 ($\alpha = .936$), PHQ-15 ($\alpha = .868$), PSS-14 ($\alpha = .875$), and WAEC-26 ($\alpha = .736$).

Additionally, in our study chronic disease was defined as the presence of diagnosed, long-term medical conditions requiring ongoing care, such as cardiopulmonary disease and diabetes.

Translation of PHQ-15

The Patient Health Questionnaire-15 (PHQ-15) was translated into Kurdish using a forward-backward translation approach following Beaton et al. guidelines[22]. Two bilingual translators, both native Kurdish speakers, independently translated the original English version into Kurdish. One translator had a background in clinical psychology to ensure conceptual accuracy, while the other had no medical training in order to capture more natural, everyday language. The two forward translations were compared and discussed within the research team, and a single reconciled version was agreed upon.

This version was then back-translated into English by an independent bilingual translator who had not been involved in the initial translation process. The back-translated version was compared with the original PHQ-15 to identify discrepancies and to ensure that the meaning of the items had been preserved. Minor wording adjustments were made where necessary to maintain semantic and conceptual equivalence.

The pre-final Kurdish version was reviewed by three clinical psychologists with experience in trauma assessment and cross-cultural research. They assessed the items for clarity and cultural appropriateness. The measure was subsequently pilot tested with 18 trauma-exposed chemical gas survivors attacks. Participants were asked whether any items were unclear or culturally inappropriate and were invited to paraphrase selected items to confirm their understanding. No major modifications were required following this stage.

The other measures used in the study (PTSD Checklist for DSM-5 (PCL-5), the Hopkins Symptom Checklist-25 (HSCL-25), and the Perceived Stress Scale-14 (PSS-14)) were translated and back-translated using the same procedure.

Statistical Analysis

All analyses were conducted using R (version 4.4.2) and SPSS (version 29), with R applied for structural equation modeling and network analyses, and SPSS used for descriptive statistics, independent-samples t-tests, and multiple regression.

Items were screened for potential exclusion prior to model estimation based on predefined data-quality and applicability criteria. These criteria included (1) item-level missingness greater than 30%, given concerns about the stability and interpretability of parameter estimates under substantial nonresponse[23-25,14], and (2) lack of applicability across participants (e.g., gender-specific items). Factor loadings were not used as primary exclusion criteria, instead, they were examined subsequently to evaluate the psychometric implications of the initial screening decisions.

PHQ-11 (“pain or problems during sexual intercourse”) demonstrated 34% missingness, exceeding the predefined threshold. Accordingly, the item was excluded from the primary ESEM analyses. The remaining PHQ items contained complete data. Because retaining PHQ-11 without imputation would have resulted in approximately one-third of the sample being excluded due to listwise deletion, multiple imputation using predictive mean matching (five imputed datasets, ten iterations) was conducted solely for sensitivity analyses in which PHQ-11 was temporarily reinstated. In these models, PHQ-11 showed low standardized loadings on the pain/fatigue factor ($\lambda = 0.30$, $R^2 = 0.09$), both before and after imputation, indicating a limited contribution to the latent construct.

PHQ-4 (“ menstrual cramps or problems with periods ”) is inherently gender-specific and therefore cannot be included in a unified cross-gender ESEM model.

Among female participants, the item also demonstrated low loadings on the pain/fatigue factor ($\lambda = 0.32$, $R^2 = 0.10$). The item was therefore excluded from factor and invariance analyses. Because measurement invariance requires shared indicators across groups, gender comparisons were performed using only items applicable to both males and females. Sensitivity analyses including both PHQ-4 and PHQ-11 in the three-factor ESEM solution yielded slightly poorer model fit (CFI = 0.898, RMSEA = 0.069) compared with the model excluding these items (CFI = 0.992, RMSEA = 0.046) (see Supplementary Table S2).

All subsequent analyses were conducted using a 13-item version, representing a culturally adapted derivative of the original PHQ-15. This adaptation acknowledges that the removal of gender-specific or culturally sensitive items reduces strict comparability with the original scale, while maintaining measurement validity and interpretability within this trauma-exposed Kurdish sample.

Structural Validity and Invariance Testing

Structural validity was assessed using ESEM, which allows theoretically plausible cross-loadings between items. A single-factor confirmatory factor analysis (CFA) was first estimated using diagonally weighted least squares (DWLS) on polychoric correlations for the 13 ordinal items [14,26]. A three-factor ESEM solution was then fitted with geomin rotation, appropriate for ordinal indicators. Model fit was assessed using $CFI \geq 0.95$, $RMSEA \leq 0.06$, and $SRMR \leq 0.08$ [27].

Separate ESEM models were fitted for male and female participants to examine gender differences in the factor structure. To facilitate cross-group comparison, the female factor solution was aligned to the male reference using target rotation. Configural similarity across genders was assessed using factor congruence coefficients (ϕ), and metric similarity was examined by examining the mean absolute differences in factor loadings. To test measurement invariance and support latent mean comparisons, multi-group CFA (MG-CFA) was run across genders using the Weighted Least Squares Means and Variance (WLSMV) estimator, evaluating configural, metric, and scalar invariance.

For age-related analyses, participants were categorized into three age groups based on meaningful age ranges: Young (37–44 years, $n = 125$, $M = 40.1$, $SD = 3.1$), Middle (45–59 years, $n = 262$, $M = 51.9$, $SD = 3.8$), and Older (≥ 60 years, $n = 147$, $M = 68.4$, $SD = 6.3$). Factor scores obtained from the final ESEM model were used for group comparisons. Group differences were summarized using unstandardized mean differences (ΔM) and Cohen's d effect sizes [28].

Latent variable reliability was estimated using McDonald's omega (ω). Convergent validity was evaluated by correlating ESEM factor scores with the PCL-5, HSCL-25, and PSS-14 scales.

Network Analysis

Network analysis was performed on the 13 retained items to examine associations among somatic symptoms. Polychoric correlations were used to accommodate the ordinal nature of the items. A regularized partial correlation network was estimated

using the EBICglasso procedure with a tuning parameter of $\gamma = 0.5$ to reduce spurious edges. Bridge strength centrality was calculated to identify symptoms connecting the predefined communities (pain/fatigue, gastrointestinal, cardiopulmonary) [29].

Network robustness was evaluated via nonparametric case-dropping bootstrap procedures with 1,000 resamples using the bootnet package. Edge-weight accuracy was examined through 95% bootstrapped confidence intervals, and centrality stability was assessed using the correlation stability (CS) coefficient. CS values above 0.50 were considered sufficient to support interpretation of centrality metrics [30]. All network analyses were interpreted cautiously, emphasizing their exploratory and hypothesis-generating nature without implying causality.

Descriptive statistics (means, standard deviations, and ranges) were calculated for the adapted Kurdish PHQ total scores and demographic variables. To examine potential selection bias across recruitment pathways, PHQ-13 total scores were compared across the three recruitment sources using one-way ANOVA with Welch correction for unequal variances and group sizes.

Results

Descriptive statistics and independent t-test

534 participants included, with 56.2% were female, with a mean age of 53.6 years (standard deviation SD = 11.2). and an average income of \$419 (SD = 269). Most were married (83%) and had some formal education, ranging from elementary school (44%) to university level (19%). Employment included housewives (43%), government employees (30%), self-employed (12%), retired (10%), and unemployed (5%), 69% reported at least one chronic disease.

The mean total score of PHQ-13 was $M = 13.62$ ($SD = 6.36$), with a significant difference between males and females ($t(532) = -8.18$, $p < .001$, and a large effect size Cohen's $d = 0.71$), females ($M = 15.51$, $SD = 5.67$), and males ($M = 11.22$, $SD = 6.40$).

Associations with the Adapted Kurdish PHQ-13

A multiple linear regression was run to assess the associations between Kurdish PHQ-13 scores and key demographic, clinical, and psychological variables. The fully adjusted model accounted for 62.9% of the variance in PHQ-13 total scores ($R^2 = .629$, $F(7, 526) = 127.49$, $p < .001$).

Depressive/anxiety symptoms, showed the strongest association with PHQ-13 scores (HSCL-25: $\beta = .44$, $p < .001$), followed by PTSD symptoms (PCL-5: $\beta = .25$, $p < .001$). Female gender was associated with higher PHQ-13 scores ($\beta = .16$, $p < .001$), higher education was associated with lower scores ($\beta = -.11$, $p < .001$). Chronic disease ($\beta = .08$, $p = .004$) and trauma exposure ($\beta = .08$, $p = .011$) were also positively associated with somatic symptom burden.

Variance inflation factors (VIFs) ranged from 1.06 to 2.99, indicating no evidence of problematic multicollinearity. These results indicate associations between

demographic, clinical, and psychological variables and PHQ-13 symptom levels within this trauma-exposed sample, and should not be interpreted as evidence of causal relationships.

Kurdish PHQ-13 Factor Structure and Model Fit

A single-factor confirmatory factor analysis (CFA) of the PHQ showed adequate fit (CFI = .988, TLI = .986, RMSEA = .054, SRMR = .064), with all item loadings significant ($\lambda = 0.602-0.809$) and high reliability ($\omega = .894$).

However, comparison with the three-factor ESEM model revealed significantly better fit ($\Delta\chi^2 = 33.27$, $df = 3$, $p < .001$), supporting a multidimensional structure.

Overall, the model demonstrated good fit to the data, $\chi^2(62) = 132.09$, CFI = .992, TLI = .990, RMSEA = .046, 90% CI [.035, .057], and SRMR = .058.

All items loaded significantly on their respective latent factors ($p < .001$). Standardized factor loadings ranged from .59 to .85, reflecting moderate to strong associations between observed indicators and their underlying constructs. Items loading on Factor 1 (pain/fatigue) (PHQ2, PHQ3, PHQ5, PHQ14) showed standardized loadings between .73 and .85. For Factor 2 (gastrointestinal)(PHQ1, PHQ12, PHQ13, PHQ15), standardized loadings ranged from .59 to .73. Items loading on Factor 3 (cardiopulmonary) (PHQ6-PHQ10) demonstrated standardized loadings between .62 and .80.

Item-level explained variance (R^2), obtained from the standardized solution, ranged from .349 to .721. The highest proportion of explained variance was observed for PHQ14 ($R^2 = .721$), while PHQ12 and PHQ13 showed lower explained variance ($R^2 = .360$ and .349, respectively) (see Table 1).

Omega reliability estimates (ω) showed adequate to good internal consistency for all three factors. Reliability was highest for Factor 3 ($\omega = .83$), followed by Factor 1 ($\omega = .77$) and Factor 2 ($\omega = .70$).

Table 1: ESEM Standardized Factor Loadings and Item-Level Explained Variance (R^2)

Factor	Item	Std. Loading	R^2
Pain/fatigue	PHQ2	0.732	0.536
	PHQ3	0.762	0.580
	PHQ5	0.746	0.556
	PHQ14	0.849	0.721
Gastrointestinal	PHQ1	0.670	0.449
	PHQ12	0.600	0.360

	PHQ13	0.591	0.349
	PHQ15	0.732	0.536
Cardiopulmonary	PHQ6	0.783	0.613
	PHQ7	0.773	0.598
	PHQ8	0.623	0.388
	PHQ9	0.769	0.591
	PHQ10	0.804	0.646

Gender-Specific ESEM and Cross-Gender Comparisons

Exploratory structural equation modeling showed excellent fit in both gender. For males ($n = 234$), $\chi^2(62) = 75.89$, $p = .11$, CFI = .996, TLI = .996, RMSEA = .031 [90% CI = .000, .053], SRMR = .069. For females ($n = 300$), $\chi^2(62) = 87.74$, $p = .02$, CFI = .992, TLI = .990, RMSEA = .037 [90% CI = .016, .054], SRMR = .070.

Standardized factor loadings ranged from .550-.875 for males and .618-.756 for females, with all items loading significantly ($p < .001$). McDonald's omega coefficients indicated good to strong internal consistency across factors, males: pain/fatigue ($\omega = .84$), gastrointestinal ($\omega = .71$), cardiopulmonary ($\omega = .88$), and females: pain/fatigue ($\omega = .82$), gastrointestinal ($\omega = .70$), cardiopulmonary (.84).

To allow comparisons between genders, we applied a target rotation alignment, using the male solution as the reference to rotate the female factors. The factor structure was very similar for males and females. The pain/fatigue factor matched extremely well across groups ($\phi = 0.95$), exceeding the standard 0.90 threshold. Cardiopulmonary also showed strong similarity ($\phi = 0.97$), while the gastrointestinal factor was somewhat lower ($\phi = 0.72$), indicating that the main structure is comparable, but there are minor differences for some factors (see Table 2). Overall, these results suggest that the Kurdish PHQ-13 factors are comparable between genders.

Table 2. Factor congruence coefficients (ϕ) between male and female ESEM factors

Female Factor	Male (pain/fatigue)	Male (cardiopulmonary)	Male (gastrointestinal)
Pain/Fatigue	0.95	0.21	0.50
Cardiopulmonary	0.97	0.30	0.22
Gastrointestinal	0.37	0.72	0.15

Measurement Invariance

Gender Invariance

Multi-group confirmatory factor analysis (MG-CFA) was conducted to evaluate measurement invariance of the three-factor PHQ model (pain/fatigue, gastrointestinal, cardiopulmonary) across males ($n = 234$) and females ($n = 300$).

The configural model, imposing no equality restrictions between gender, demonstrated acceptable fit ($CFI = 0.975$, $TLI = 0.971$, $RMSEA = 0.051$, $SRMR = 0.074$), supporting the same three-factor structure across males and females. Metric invariance, assessed by constraining factor loadings, resulted in minimal changes in fit ($\Delta CFI = 0.004$, $\Delta RMSEA = -0.006$), suggesting that the relationships between items and latent factors are largely comparable. Scalar invariance showed acceptable fit ($\Delta\chi^2(10) = 9.23$, $p = 0.51$; $\Delta CFI = -0.008$, $\Delta RMSEA = 0.006$), supporting full scalar invariance across gender. Latent factor means can therefore be directly compared (Supplementary Table S3).

Following invariance assessment, latent factor means were compared across gender. Females endorsed higher somatic symptom levels on all factors, with the largest estimated latent difference between male and female for pain/fatigue ($\Delta = 1.73$, Cohen's $d = 0.78$), followed by cardiopulmonary ($\Delta = 1.08$, $d = 0.48$) and gastrointestinal symptoms ($\Delta = 1.06$, $d = 0.45$) (see Table 3). These differences should be interpreted cautiously, as unobserved confounders such as co-occurring depression, anxiety, or chronic medical conditions may influence observed patterns.

Exploratory Age Invariance

To examine age differences, participants were divided into three groups (Young: $n = 125$; Middle: $n = 262$; Older: $n = 147$). Multi-group CFA showed acceptable configural fit ($CFI = 0.965$, $TLI = 0.956$, $RMSEA = 0.048$, $SRMR = 0.062$), indicating that the same three-factor structure was present across age groups. When factor loadings were constrained to be equal, model fit worsened significantly [$\Delta\chi^2(20) = 34.70$, $p = 0.022$; $\Delta CFI = -0.022$, $\Delta RMSEA = +0.012$], suggesting that full metric invariance was not supported. Examination of the loadings indicated that most were similar across groups, consistent with partial metric invariance.

Estimated latent mean differences were small ($d = 0.04$ - 0.17) and were not adjusted for depression or PTSD. Therefore, comparisons of latent factor scores across age groups should be considered exploratory. Differences were small: pain/fatigue ($\Delta = 0.10$, $d = 0.04$), cardiopulmonary ($\Delta = 0.39$, $d = 0.17$), and gastrointestinal ($\Delta = 0.08$, $d = 0.04$). These findings should be interpreted cautiously given the cross-sectional design and the possibility of unmeasured confounding factors.

Table 3. Latent factor scores ($M \pm SD$) for somatic symptom factors across females and males. Δ represents the mean difference (Female – Male); Cohen's d indicates effect size; *** $p < .001$.

Factor	Female M (SD)	Male M (SD)	Δ	Cohen's d
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Pain/Fatigue	6.61 (1.71)	4.88 (2.43)	1.73***	0.78
Cardiopulmonary	3.76 (2.16)	2.68 (2.22)	1.08***	0.48
Gastrointestinal	4.05 (2.28)	2.98 (2.30)	1.06***	0.45

Convergent Validity

The PHQ latent factors demonstrated moderate to strong positive correlations with PTSD symptom severity (PCL-5), anxiety and depression symptoms (HSCL-25), and perceived stress (PSS-14), indicating substantial convergence with established measures of psychological distress (see Table 4). Correlation patterns were consistent across male and female participants, although associations with perceived stress were modestly stronger among males. Because all external measures were self-reported and administered within the same assessment context, these associations may partly reflect shared method variance and general distress rather than construct-specific overlap. Accordingly, the present findings support convergent validity with distress-related constructs but do not constitute evidence for discriminant validity or somatic symptom specificity.

Table 4. Correlations Between PHQ Latent Factors and External Validity Measures (Total Sample)

Variable	Factor 1	Factor 2	Factor 3
PCL5	.67	.71	.70
HSCL25	.73	.74	.76
PSS14	.51	.50	.52

Network Estimation

A network analysis was performed to examine associations among symptoms. The resulting network exhibited moderate sparsity, with 36.1% of possible edges retained. Symptom clusters were broadly consistent with the ESEM domains (pain/fatigue, gastrointestinal, cardiopulmonary) (Fig. 1), although the network does not assume latent factors and should be interpreted as representing conditional dependencies among observed items.

Bridge strength centrality was calculated to identify symptoms connecting different clusters. PHQ15 (“trouble sleeping”), PHQ14 (“feeling tired or having low energy”), and PHQ8 (“fainting spells”) showed the highest bridge strength values, suggesting a relatively greater role in linking domains (see Table 5). Expected influence centrality indicated that several cardiopulmonary items were highly connected within their community. However, centrality metrics reflect statistical interrelations and do not

imply causality or temporal influence.

To evaluate robustness, case-dropping bootstrap procedures (1,000 resamples) were conducted. Edge-weight accuracy showed narrower 95% confidence intervals for stronger edges and greater variability for weaker edges (Supplementary Fig. S1). The correlation stability (CS) coefficients were 0.59 for edge strength and 0.52 for bridge strength, meeting the preferred threshold of 0.50. These findings show adequate stability of centrality estimates. Results are interpreted as exploratory and hypothesis-generating.

Fig. 1. symptom network estimated using EBICglasso, showing three symptom communities (cardiopulmonary (blue), gastrointestinal (orange), and pain/fatigue (green)). Nodes represent individual symptoms, and edges reflect regularized partial correlations, with thicker edges indicating stronger associations.

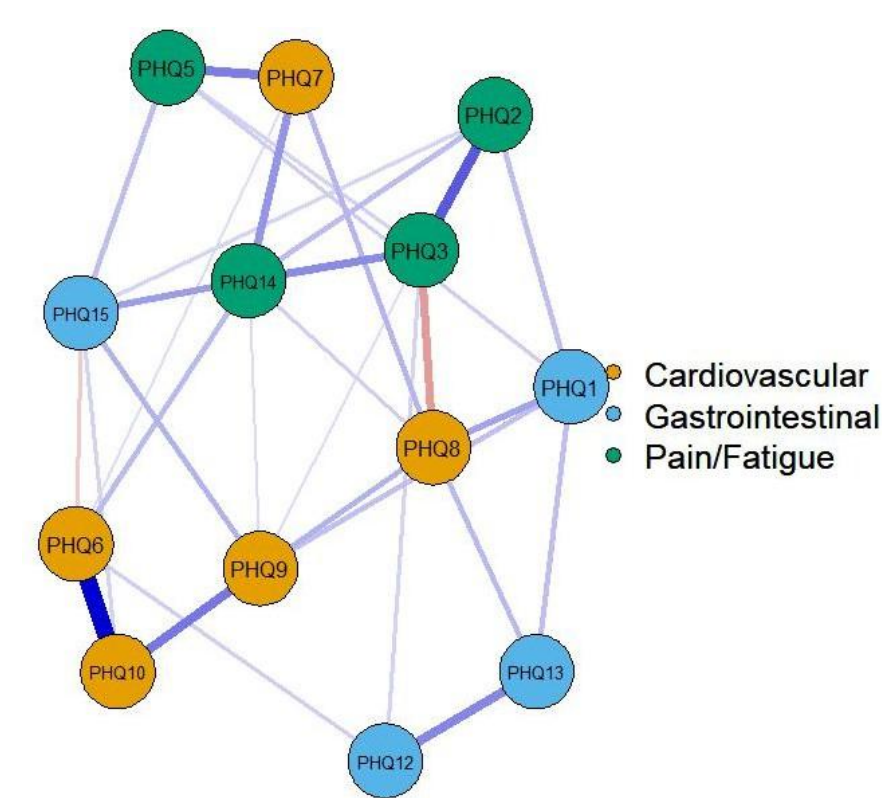


Table 5. Bridge Strength Centrality in Kurdish PHQ-13 Symptom Network (N = 534)

Rank	Item	Bridge Strength	Community
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1	PHQ15	0.849	Gastrointestinal
2	PHQ14	0.798	Pain/fatigue
3	PHQ8	0.696	Cardiopulmonary
4	PHQ1	0.601	Gastrointestinal
5	PHQ5	0.549	Pain/fatigue
6	PHQ7	0.527	Cardiopulmonary
7	PHQ9	0.478	Cardiopulmonary
8	PHQ3	0.408	Pain/fatigue
9	PHQ6	0.381	Cardiopulmonary
10	PHQ2	0.237	Pain/fatigue
11	PHQ12	0.207	Gastrointestinal
12	PHQ13	0.156	Gastrointestinal
13	PHQ10	0.097	Cardiopulmonary

Discussion

The present study assessed the structure and validity of the Kurdish PHQ-13, a culturally adapted 13-item version of the original PHQ-15, among survivors of chemical attacks. The findings suggest adequate reliability and structural validity in this trauma-exposed sample.

The three- factor structure identified in this sample consistent to previous PHQ- 15 studies that describe pain/fatigue, gastrointestinal, and cardiopulmonary dimensions alongside a general somatic symptom factor. This consistency suggests that key dimensions of somatic symptom reporting may show relative stability across contexts, including trauma-exposed populations. Reliability estimates for the total scale and subscales were in the adequate to good range, consistent with prior validations and supporting the use of the Kurdish PHQ-13, as a brief index of somatic symptom

burden in Kurdish survivors of chemical attacks[11, 31-34]. At the same time, the somewhat lower reliability of the gastrointestinal factor relative to the other factors indicates that domain-specific interpretations should be made with caution, and that further refinement of items capturing culturally salient gastrointestinal idioms of distress may be warranted.

The exclusion of PHQ-11 and PHQ-4 was informed by predefined psychometric criteria and sensitivity analyses. PHQ-11 showed substantial missing data and low factor loadings even after multiple imputation, while PHQ-4 showed similarly weak loadings among female participants and was not applicable to male participants. Retaining these items reduced overall model fit and added minimal information to the latent structure. These findings align with previous systematic reviews indicating that sexual- and gender-specific items in the PHQ-15 often show low item-total correlations and limited contribution to the general somatic symptom factor, and that their removal can improve structural coherence and reliability across diverse populations [16,31,23-26]. While this supports the use of the Kurdish PHQ-13, it does reduce strict comparability with the original PHQ-15, particularly with respect to severity interpretation.

PHQ-4 was administered as part of the complete 15-item version to allow evaluation of the adapted instrument in line with the original PHQ structure prior to item screening. However, menstrual-related symptoms represent a biologically specific experience that is not applicable to male participants and therefore differs conceptually from the broader construct of general somatic distress examined in this study. Including such an item within a unified cross-gender factor model would introduce structural imbalance and complicate interpretation of measurement invariance findings. The primary analyses therefore focused on indicators that were conceptually and structurally relevant to both genders. This decision should not be interpreted as diminishing the clinical importance of menstrual-related symptoms in Kurdish women. Rather, it reflects the methodological challenge of modeling gender-specific symptoms within a shared latent framework. Future validation studies may benefit from investigating reproductive items separately or through gender-stratified analyses to preserve both cultural sensitivity and psychometric clarity.

Women in this sample reported substantially higher somatic symptom levels than men, in line with evidence that women tend to endorse more frequent and intense PHQ-15 symptoms in both community and clinical settings. These differences may indicate a combination of biological factors, exposure to chronic disease and caregiving roles, and greater readiness to report bodily distress, although the present design cannot distinguish between these mechanisms[14,35-38].

Multi-group CFA analyses showed full scalar invariance across gender, indicating that latent factor means can be compared. Comparisons should focus on the broader symptom domains rather than individual items, particularly given the possible influence of unmeasured factors, while age comparisons demonstrated only partial metric invariance. Accordingly, observed age differences should be interpreted as exploratory, and further validation across developmental stages is warranted.

It is important to note that the depression and anxiety items in the Hopkins Symptom

Checklist-25 are predominantly focused on cognitive and emotional symptoms, with relatively little somatic content, consistent with prior research indicating limited overlap between these scales in terms of somatic items [39, 40]. The strong correlations observed between PHQ factors and PTSD, depression/anxiety, and perceived stress are in line with previous work showing that higher PHQ-15 scores are closely associated with general psychological distress, functional impairment, and health service use [19]. However, because all measures were self-reported in a single session and some auxiliary instruments have not been extensively validated in Kurdish clinical settings, these associations may partly reflect shared method variance and broader construct overlap. In addition, the absence of objective medical data or measures of unrelated constructs (e.g., cognitive ability, personality traits) limits formal tests of discriminant validity. Overall, these findings support convergent validity with distress-related constructs but do not establish specificity for DSM-5 somatic symptom disorder or for medically unexplained symptoms.

Network analyses complemented the latent variable findings by showing how individual symptoms are directly related to each other, after accounting for all other symptoms. Trouble sleeping, selected gastrointestinal symptoms, and cardiopulmonary items showed higher bridge strength, suggesting a central role in connecting symptom clusters. Edge weights and centrality indices were examined and demonstrated acceptable stability, supporting cautious interpretation. Importantly, these networks represent statistical associations rather than causal or temporal pathways, and findings should be regarded as exploratory and hypothesis-generating. Overall, these findings indicate that the Kurdish PHQ-13 demonstrates acceptable psychometric properties as a culturally sensitive measure of somatic symptom burden in trauma-exposed Kurdish chemical gas survivors attacks. Because two items were removed, severity classifications should be interpreted as adapted thresholds rather than directly equivalent to the original PHQ-15 cutoffs.

Limitations

This study has several limitations. First, the sample consisted solely of survivors of chemical attacks, which may limit the generalizability of the findings to other Kurdish populations. The symptom patterns identified likely reflect the specific experiences and historical context of this highly trauma-exposed group.

Second, random sampling was not feasible due to ethical and logistical constraints. Participants were recruited through registries, non-governmental organizations, and peer networks, which may have introduced selection bias and potentially led to an overrepresentation of individuals with greater health concerns or stronger engagement with support services. Although no major differences in symptom scores were observed across recruitment sources, unmeasured biases may still have influenced symptom estimates.

Third, the PHQ-11 and PHQ-4 were excluded from certain analyses. The PHQ-4, which assesses menstrual pain, was omitted from cross-gender comparisons to ensure

measurement equivalence between male and female participants. While methodologically necessary, this may have resulted in reduced coverage of female-specific symptomatology.

Fourth, despite careful translation, expert review, and pilot testing of the PHQ-15, some culturally specific expressions of somatic distress may not have been fully captured. In addition, other measures used in the study, including the HSCL-25 and PSS-14, have not undergone formal validation in Kurdish populations. Therefore, findings related to convergent validity should be interpreted with caution.

Fifth, several items displayed partial metric invariance across age groups, suggesting that certain symptoms may be perceived differently at various stages of life, which may affect the comparability of scores across age categories.

Finally, all measures were self-reported and collected at a single time point. Given the cross-sectional design, causal inferences cannot be drawn. In addition, shared method variance and unobserved confounders such as medication use or healthcare access may have influenced the relationships. Nonetheless, these findings provide preliminary support for the reliability and potential cultural relevance of the Kurdish PHQ-13 within this trauma-exposed sample.

Conclusion

The present findings suggest that the Kurdish PHQ-13 shows acceptable reliability and promising cultural relevance among survivors of chemical attacks. A three-factor configuration consisting of pain/fatigue, gastrointestinal, and cardiopulmonary domains demonstrated acceptable model fit, with full scalar invariance across gender and partial metric invariance across age groups. The associations observed with measures of psychological distress indicate overlap with broader distress constructs.

Network analyses further highlighted associations among somatic symptoms, with several items functioning as potential bridges across domains. Taken together, these findings provide preliminary support for the use of the Kurdish PHQ-13 as a tool for assessing somatic symptom burden in this trauma-exposed population. However, as the instrument was adapted and evaluated in a highly specific population, its generalizability to non-traumatized groups remains uncertain.

Future research employing longitudinal designs and more diverse samples is needed to examine temporal dynamics, assess long-term measurement stability, and further evaluate its applicability across different populations.

Declarations

List of Abbreviations

PHQ-15: Patient Health Questionnaire-15

PTSD: Post-Traumatic Stress Disorder

PCL-5: PTSD Checklist for DSM-5

HSCL-25: Hopkins Symptom Checklist-25

PSS-14: Perceived Stress Scale-14

WAEC-26: War and Adversity Exposure Checklist-26

DSM-5-TR: Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition-
Text Revision

CFA: Confirmatory Factor Analysis

MG-CFA: Multi-Group Confirmatory Factor Analysis

ESEM: Exploratory Structural Equation Modeling **CFI:**

Comparative Fit Index

TLI: Tucker-Lewis Index

RMSEA: Root Mean Square Error of Approximation

SRMR: Standardized Root Mean Square Residual

DWLS: Diagonally Weighted Least Squares

WLSMV: Weighted Least Squares Mean and Variance Adjusted

χ^2 : Chi-square statistic

df: Degrees of freedom **p:**

p-value

M: Mean

SD: Standard Deviation

d (Cohen's d): Effect size

β : Standardized regression coefficient

R²: Coefficient of determination

ω (Omega): McDonald's Omega

ϕ (Phi): Tucker's coefficient of congruence

CI: Confidence Interval

$\Delta\chi^2$: Change in Chi-square

Δ CFI: Change in Comparative Fit Index

Δ M: Difference in Means

Ethics Approval and Consent to Participate

Ethical Approval: This study received ethical approval from the University of Duhok, ethics committee on October 3rd, 2021 (approval number: 141). In addition, permission was obtained from the Halabja governorate in April 2022 (reference number: 10/158), including the general directorates of security and police.

Consent Procedure: Written informed consent was obtained from all participants prior to data collection. Participation was completely voluntary, confidentiality was ensured, and participants were informed of their right to withdraw from the study at any time without consequences. For participants not able to read or write, the consent form was read aloud, and informed consent was obtained using a thumbprint in the presence of a witness.

Ethical Guidelines: The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2013.

Consent for publication

Not applicable.

Data Availability

The data that support the findings of this study are available from the corresponding author (Ibrahim Mohammed), upon reasonable request.

Competing interests

The authors declare that they have no competing interest.

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All authors read and approved the final manuscript.

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Appendix D

"Psychological and biological long-term consequences of trauma in the Yazidi population displaced from Shingal after the genocide"

Consecuencias psicológicas y biológicas a largo plazo del trauma en la población yazidí desplazada de Shingal tras el genocidio

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ABSTRACT

Background: About a decade after the 2014 ISIS genocide in Shingal, Iraq, the effects of mass trauma and displacement continue to be felt among the Yazidi population. In particular, the long-term interplay between psychological distress and biological stress regulation remains poorly understood.

Objective: The present study aimed to investigate the prevalence and severity of PTSD, depression, anxiety, somatic symptoms, and perceived stress among displaced Yazidi participants. It also aimed to explore how these psychological indicators relate to chronic physiological stress, as measured by hair cortisol and cortisone concentrations.

Method: A total of 264 Yazidi adults residing in internally displaced person (IDP) camps in Duhok, Iraq, underwent structured interviews by trained psychologists. Standardized Kurdish versions of the PCL-5, HSCL-25, PHQ-15, PSS-14, and War and Adversity Checklist-26 were administered. Hair cortisol and cortisone were analyzed by liquid chromatography tandem mass spectrometry (LC–MS). Data were evaluated using multiple regressions and latent profile analysis (LPA).

Result: Severe mental health burden was evident: 93.2% reached the cutoff for PTSD, 83.7% for anxiety, and 87.5% for depression (HSCL-25 subscales). LPA revealed three classes of PTSD symptoms – low, moderate, and high – along with four symptom classes reflecting various distress patterns. Compared to men, women exhibited significantly higher levels of

psychological distress and somatic symptoms ($p < .01$, $d = 0.60-0.70$). Hair cortisol related moderately to all symptom scales ($r = .17-.25$, $p < .01$), indicating cumulative HPA axis activation. Cross-class correlations suggest that PTSD, anxiety, and depression represent, at least partly, independent symptom dimensions.

Conclusions: Findings stress the long-lasting psychobiological effects of genocide-related trauma among Yazidi IDPs. This finding is further underlined by the integrated psychometric and biological data, which point to the urgent need for long-term, culturally sensitive, and gender-responsive interventions.

HIGHLIGHTS

Yazidi Shingal IDPs show very high levels of post-traumatic stress, depression, anxiety, and somatic symptoms. Hair cortisol shows modest but consistent associations with psychological distress, indicating cumulative trauma-related stress on body and mind. Women, older adults, and camp residents are especially highly burdened, underscoring the need for gender-sensitive and culturally specific mental health care.

Antecedentes: A casi una década del genocidio perpetrado por ISIS en 2014 en Shingal, Irak, los efectos del trauma masivo y el desplazamiento continúan afectando a la población yazidí. En particular, la interacción a largo plazo entre el malestar psicológico y la regulación biológica del estrés sigue siendo escasamente comprendida. Objetivo: El presente estudio tuvo como objetivo investigar la prevalencia y gravedad del trastorno de estrés postraumático (TEPT), depresión, ansiedad, síntomas somáticos y estrés percibido en participantes yazidíes desplazados. Asimismo, buscó explorar cómo estos indicadores psicológicos se relacionan con el estrés fisiológico crónico, medido a través de las concentraciones de cortisol y cortisona en cabello.

Método: Un total de 264 adultos yazidíes residentes en campamentos para personas desplazadas internamente (internally displaced persons, IDPs) en Duhok, Irak, fueron evaluados mediante entrevistas estructuradas realizadas por psicólogos entrenados. Se administraron versiones kurdas estandarizadas del PCL-5, HSCL-25, PHQ-15, PSS-14 y la War and Adversity Checklist-26. El cortisol y la cortisona en cabello se analizaron mediante cromatografía líquida acoplada a espectrometría de masas en tándem (LC-MS). Los datos se evaluaron mediante regresiones múltiples y análisis de perfiles latentes (LPA por sus siglas en inglés).

Resultados: Se evidenció una carga severa de patología en salud mental: el 93,2% alcanzó el punto de corte para TEPT, el 83,7% para ansiedad y el 87,5% para depresión (subescalas del HSCL-25). El análisis de perfiles latentes identificó tres clases de síntomas de TEPT – baja, moderada y alta –, así como cuatro clases sintomáticas que reflejan distintos patrones de malestar. En comparación con los hombres, las mujeres presentaron niveles significativamente más altos de malestar psicológico y síntomas somáticos ($p < 0,01$; $d = 0,60-0,70$). El cortisol en cabello se asoció de manera moderada con todas las escalas sintomáticas ($r = 0,17-0,25$, $p < 0,01$), lo que indica una activación acumulativa del eje hipotálamo-hipófisis-adrenal (HHA). Las correlaciones

entre clases sugieren que el TEPT, la ansiedad y la depresión representan, al menos parcialmente, dimensiones sintomáticas independientes.

Conclusiones: Los hallazgos subrayan los efectos psicobiológicos persistentes del trauma asociado al genocidio en la población yazidí desplazada internamente (IDPs). Esto se ve reforzado por la integración de datos psicométricos y biológicos, que evidencian la necesidad urgente de intervenciones a largo plazo, culturalmente sensibles y con enfoque de género.

KEYWORDS: Genocide, post-traumatic stress disorder, depression, hair cortisol, Yazidi population

PALABRAS CLAVE: Genocidio, trastorno de estrés postraumático, depresión, cortisol en cabello, población yazidí,

1. Introduction

The Yazidi population is an ethnoreligious group that speaks the Kurdish language and is native to the Kurdistan region of Iraq, with smaller Yazidi communities also residing in Syria, Turkey, Armenia, and Azerbaijan (Lobanov-Rostovsky & Kiss, Citation2022). In August 2014, ISIS attacked the Yazidis in Shingal, killing thousands of men and abducting approximately 7,000 women and children, who were then forced to convert, tortured, sexually enslaved, and subjected to inhumane treatment (Cetorelli & Ashraph, Citation2019).

The Yazidi population, displaced by ISIS attacks, was forced into internally displaced persons (IDP) camps around Duhok, which host more than 60% of all displacement camps in the Kurdistan Region (Duhok Governorate, Citation2016). IDPs are individuals who have fled their homes but, unlike refugees, have not crossed an internationally recognized border (Cantor et al., Citation2021). Shingal, located along the 133-kilometer-long Shingal anticline, has come to symbolize both the region's profound trauma and its cultural resilience (Sissakian & Al-Ansari, Citation2023).

Mental disorders are highly prevalent among war-affected, mass-displaced, and socioeconomically insecure populations. Recent research shows that mental health problems are widespread: 74.3% of respondents reported PTSD, 63.8% major depression, and 40.0% anxiety disorders (Manafe et al., Citation2024). Similarly, most Yazidi survivors of Shingal remain displaced and continue to experience long-term psychological symptoms such as PTSD, major depression, anxiety disorders, and somatic distress (Kizilhan, Citation2025).

These psychological consequences are closely linked to how the body responds to stress. Stress is a physiological and psychological reaction to perceived strain. Acute stress, or eustress, can be adaptive, whereas chronic stress, or distress, has been shown to be harmful (Salleh, Citation2008). The three phases of the stress response – alarm, resistance, and exhaustion – were first described by Hans Selye as the general adaptation syndrome, mediated

by the hypothalamic–pituitary–adrenal (HPA) axis (Selye, Citation1936). This axis regulates cortisol release, coordinating the body's adaptation to stress (Sharan & Vellapandian, Citation2024). Cortisol, a glucocorticoid hormone produced by the adrenal glands, plays a vital role in numerous physiological processes (Knezevic et al., Citation2023). Recent studies suggest that cortisol dysregulation negatively affects mental health and contributes to the emergence and severity of diverse mental disorders (Sic et al., Citation2024). Specifically, cortisone is the hormonally inactive metabolite of cortisol, generated primarily by the enzyme 11 β -hydroxysteroid dehydrogenase type 2 (11 β -HSD2) and converted back to active cortisol by 11 β -hydroxysteroid dehydrogenase type 1 (11 β -HSD1), thereby regulating local glucocorticoid availability within tissues (Dineen et al., Citation2023). In this context, cortisone is measured alongside cortisol as a complementary biological indicator, providing an aligned measure of HPA axis function and peripheral conversion mechanisms relevant to chronic stress regulation.

Stress and cortisol dysregulation are primary precursors of post-traumatic stress disorder (PTSD), a mental disorder that may develop following such traumatic events as war, rape, or forced confinement (American Psychiatric Association [APA], Citation2013; Kleber, Citation2019). Traumatic events are inherently stressful, and the intensity and duration of the stress experienced during and after the event significantly influence the risk of developing PTSD. PTSD is particularly common among vulnerable populations like the Yazidis, especially among women with a history of captivity or sexual violence (Kizilhan et al., Citation2020). Women are also two to three times more likely than men to develop PTSD, in part due to differences in trauma type, coping strategies, and neurobiological stress responses (Olf, Citation2017). A hallmark of PTSD is also high comorbidity, most commonly with depression, and the risk of PTSD is influenced by the type and number of traumatic experiences (Kizilhan et al., Citation2023). The disorder is characterized by mentally re-experiencing traumatic events, avoidance, hyperarousal, and cognitive or mood disturbances (Chen, Citation2023). The development of PTSD is shaped by several risk factors, including previous trauma, social support, sex, and psychiatric comorbidities such as depression and anxiety (National Institute of Mental Health, Citation2023). Cortisol dysregulation observed in different biomaterials (hair, blood, or saliva), has been correlated with PTSD severity and the number of traumatic events (Steudte et al., Citation2011). Despite being intensely studied across different populations and settings, it remains unclear whether cortisol dysregulation is still present after very long follow-up intervals, such as in the present study.

2. Objectives of the present study and hypotheses

The present study investigates the long-term psychological and biological consequences of the genocide experienced by the Yazidi population and their subsequent displacement in 2014. Nearly a decade later, limited evidence exists regarding how prolonged exposure to trauma, within the context of post conflict distress and displacement, continues to affect mental health and physiological stress markers. Therefore, the study aimed to assess the prevalence and severity of PTSD and its common comorbid conditions, particularly depression, anxiety, stress, and somatic symptoms, and to examine how these psychological symptoms relate to

sociodemographic characteristics such as sex, marital status, income, number of children, education, and trauma-related experiences. A further objective was to explore associations between psychological symptoms and biological markers of stress, specifically hair cortisol and cortisone, as indicators of potential hypothalamic–pituitary–adrenal (HPA) axis dysregulation in populations exposed to chronic trauma. In addition, the study sought to apply latent profile analysis (LPA) to the PTSD Checklist for DSM-5 (PCL-5) and the Hopkins Symptom Checklist-25 (HSCL-25) to identify distinct psychological symptom profiles and to characterize patterns of trauma-related and general psychological distress among internally displaced Yazidis, within a multidimensional biopsychosocial framework integrating psychological, biological, and social factors.

Based on prior research on trauma and stress regulation, it was hypothesized that ...

... Yazidis affected by the 2014 genocide show high levels of PTSD, depression, anxiety, somatic symptoms, and perceived stress.

1... greater PTSD severity is positively associated with depression, anxiety, physical symptoms, perceived stress, and trauma exposure.

2... psychological symptom severity depends on sex, marital status, income, education, and number of children.

3... higher hair cortisol and cortisone levels are linked to greater psychological distress, indicating HPA axis dysregulation.

4... women report higher levels of psychological symptoms than men.

3. Methods

3.1. Ethical approval and consent procedures

This study received ethical approval from the University of Duhok Ethics Committee on 3 October 2021 (Approval Number: 141). Additional approval was obtained from the Governorate of Duhok in June 2022 (Reference Number: 1024) following coordination with the General Directorates of Police and Security, as well as regional humanitarian and medical authorities. Before enrolment, participants were contacted by phone, informed about the study, and invited to participate. During home visits, the study's objectives, potential risks, and confidentiality procedures were explained in detail. All participants provided written informed consent and were assured of their right to withdraw from the study at any time without negative consequences. Ninety-five individuals declined participation for cultural reasons, particularly in relation to hair sampling.

3.2. Pilot study

A pilot study was conducted with 20 participants seven months before the actual study, to assess feasibility and cultural adaptation. Pilot participants were not included in the main study. The psychometric scales were translated into Kurdish (Kurmanji) by two independent translators and back-translated by two additional translators to establish linguistic equivalence. Discrepancies were resolved through discussion. Pre-testing of the questionnaires for cultural appropriateness was also performed with a bilingual validator. Because of low literacy levels among participants, all scales were administered as structured interviews by trained psychologists rather than as self-report measures. Five female and two male psychologists were trained for one week in psychometric assessment, interview protocol, hair sampling, and supportive intervention with bereaved participants. No wording modifications were required after the pilot, as all items were clearly understood and culturally suitable. In summary, the pilot study confirmed that the adapted instruments were feasible and appropriate.

3.3. Main study and sampling

The study targeted Yazidi individuals displaced from Shingal during the 2014 ISIS genocide living in internally displaced persons (IDP) camps in Duhok Governorate, Kurdistan Region. Participants were aged 18 years or older and had directly witnessed the attacks or experienced captivity, displacement, loss of relatives, or other war-related trauma. Inclusion criteria included being Yazidi, aged 18 or older, residing in Shingal in 2014 during the attacks, and having been displaced during the conflict. Exclusion criteria were having a severe cognitive impairment, being unable to provide consent, or lacking sufficient data for inclusion. Eligibility was verified by phone, confirming residence in Shingal during the attack and subsequent displacement. Of the 359 individuals screened, 264 (73.5%) met the inclusion criteria and provided informed consent for the interview and hair sample collection. Data were collected between May and September 2024 in Kurmanji, Iraq. Participants were recruited using convenience and snowball sampling. Distressed participants were referred to NGO-provided psychological services, and follow-up support was organized through camp administrators.

3.4. Data collection procedures

Standardized instruments were used to assess psychological symptoms and trauma exposure. As in the pilot study, due to low literacy levels among participants, structured interviews were conducted instead of self-administered questionnaires. As shown in Table 1, 43.6% of participants reported no formal education, 20.8% had only basic (primary) schooling, 22.7% had completed high school, and 11.7% had university education. Because a significant proportion of participants had no or only primary schooling, educational level was used as an indicator of limited literacy, and all questionnaires were therefore administered as interviewer-led measures rather than self-report forms. All assessments were carried out by trained psychologists in Kurdish (Kurmanji) following translation, back-translation, and cultural validation. Interviews lasted 60–90 min and included psychological questionnaires and hair sampling for cortisol analysis. During the first week, supervisors monitored interviews to ensure adherence to

protocols. Thereafter, psychologists worked independently under weekly supervision for consistency and quality control. Although standardized measures were used, formal clinician-administered diagnostic tools (e.g. CAPS-5) were not included.

Trauma and mental health symptoms were assessed with validated instruments:

PCL-5 (Post-Traumatic Stress Disorder Checklist for DSM-5 (20 items)). This instrument assesses PTSD symptoms in four domains – re-experiencing, avoidance, negative cognition/mood, and hyperarousal (Weathers et al., Citation2013). Items use a 0–4 scale, where higher scores reflect greater symptom severity.

War and Adversity Checklist-26: Using dichotomous items (0 = no, 1 = yes), this instrument assesses exposure to 26 war-related traumatic events (Mahmood et al., Citation2022).

HSCL-25 (Hopkins Symptom Checklist). This instrument measures anxiety (10 items) and depression (15 items), each item rated from 1 ('not at all') to 4 ('extremely') (Mahmood et al., Citation2019). For descriptive purposes (see Table 3, Section 4.3), mean subscale scores for anxiety and depression were computed and reported separately, with the clinical cutoff of ≥ 1.75 applied to each subscale. However, for all inferential analyses – including correlations (Section 4.4), sex comparisons (Section 4.6), regression models (Section 4.6), and latent profile analysis (Section 4.8) – the HSCL-25 total score was used. The total score has been validated as an overall index of psychological distress in conflict-affected populations (Ibrahim et al., Citation2018; Mahmood et al., Citation2019) and captures the combined burden of anxiety and depression symptoms.

PHQ-15 (Patient Health Questionnaire). This instrument assesses 15 common somatic symptoms, rated 0 ('not bothered at all') to 2 ('bothered a lot') (Kroenke et al., Citation2002).

PSS-14 (Perceived Stress Scale). The 14 items of the Perceived Stress Scale (PSS-14) were administered on a 5-point Likert scale ranging from 0 ('never') to 4 ('very often'), with positively worded items reverse-scored in accordance with established scoring conventions (Santiago et al., Citation2019). Item responses were summed to yield a global index of perceived stress. In addition to this continuous score, perceived stress was summarized descriptively using an ordinal three-level variable (low, moderate, and high stress), derived by recoding the total PSS-14 score for descriptive purposes, without applying any external cut-off values. Because no empirically validated diagnostic or severity cutoffs are available for the PSS-14, these categories were used solely to facilitate the presentation of prevalence estimates and should not be interpreted as reflecting clinical thresholds.

Cutoff scores were applied to classify probable clinical symptom levels. For the PCL-5, a total score of 33 or higher was used as the cutoff for probable PTSD, consistent with

recommendations by Weathers et al. (Citation2013). For the HSCL-25, a mean item score ≥ 1.75 indicated clinically relevant anxiety or depression, in line with prior studies in conflict-affected populations (Ibrahim et al., Citation2018; Mahmood et al., Citation2019). For the PHQ-15, scores ≥ 15 reflected severe somatic symptom burden (Kroenke et al., Citation2002). For the PSS-14, higher scores indicated greater perceived stress, but no formal diagnostic cutoff was applied.

All five scales showed very good reliability (internal consistency) in the present study (Cronbach's α : Adversity Checklist = .805; PCL-5 = .893; HSCL-25 = .913; PHQ-15 = .893; PSS-14 = .803). Validity was supported by previous cross-cultural studies (Fares et al., Citation2021; Ibrahim et al., Citation2018; Kroenke et al., Citation2002; Neldner et al., Citation2023; Rodríguez-Barragán et al., Citation2021; Santiago et al., Citation2020).

3.5. Hair sampling

Cortisol was selected as a biomarker of chronic stress, reflecting cumulative activation of the hypothalamic–pituitary–adrenal (HPA) axis. Unlike serum or saliva cortisol, which captures acute fluctuations, hair cortisol concentration (HCC) measures long-term stress exposure (Stalder & Kirschbaum, Citation2012). Following standardized procedures, 30–50 strands of hair were cut close to the scalp with sterilized scissors and stored for analysis (Wright et al., Citation2018). The proximal 2 cm segment was analyzed, reflecting cortisol accumulation over the preceding two months, based on an average hair growth rate of approximately 1 cm per month (Stalder et al., Citation2017). Cortisone was measured alongside cortisol as an index of peripheral conversion and an additional indicator of HPA axis activity. Hair cortisol and cortisone levels were quantified using the LC–APCI–MS/MS assay (Gao et al., Citation2013), based on 10 mg of prewashed, non-pulverized hair. The analytical method had quantification limits < 0.1 pg/mg and intra-/interassay coefficients of variation $< 10\%$.

3.6. Statistical analysis

All statistical analyses were conducted in R (r-project.org) and IBM SPSS Statistics (version 29.2.0). Correlations among PTSD, depression, anxiety, somatic symptoms, perceived stress, trauma exposure, and hair-based biomarkers (log-transformed cortisol and cortisone) were examined using Pearson or Spearman coefficients, as appropriate. Group differences by sex, age, education, marital status, occupation, and living location were tested with independent samples t-tests and one-way ANOVAs, with effect sizes reported as Cohen's d and eta-squared. Multiple linear regression analyses were used to assess the contributions of demographic, psychosocial, and biological variables to PTSD symptoms, depression symptoms, somatic symptoms, and perceived stress, and potential moderation effects of sex, age, and education were examined using hierarchical regression models. Latent profile analysis (LPA) was applied separately to PCL-5 and HSCL-25 scores to identify symptom classes; one to six class solutions were compared using log likelihood, AIC, BIC, sample size adjusted BIC, and entropy, and the best-

fitting models were retained. Finally, multinomial logistic regression models were estimated to examine demographic, psychosocial, and biological predictors of latent class membership, with the intermediate symptom class specified as the reference group. All tests were two-tailed with α set at .05, and false discovery rate corrections were applied to adjust for multiple comparisons.

4. Results

4.1. Description of study sample

The sample consisted of 264 participants, of whom 68.9% were female. Sociodemographic characteristics for the total sample are presented in Table 1. Education levels were generally low, with a high proportion of participants having no formal education. Students and unemployed individuals comprised the most common occupational groups. Most respondents were married (73.5%), while 25.0% were single and 1.5% divorced, residing primarily in camps or rural areas. A large proportion of participants reported dependence on social support, and approximately one-fifth indicated having a chronic illness. These sociodemographic variables provide essential contextual information for understanding the psychological outcomes assessed in this study. Table 2 presents the descriptive characteristics for the PTSD diagnostic groups according to sex, age, education level, marital status, and living location.

Table 1. Sociodemographic Characteristics of the Study Sample (N = 264).

Variable	Category	%
Sex		
	Female	68.9
	Male	31.1
Age group		
	18–29	53.0
	30–39	26.9
	40–49	11.4
	50–59	3.4

Variable	Category	%
	60+	5.3
Level of education		
	No formal education	43.6
	Basic (primary school)	20.8
	High school	22.7
	University	11.7
	No information	1.1
Occupation		
	Unemployed	37.1
	Housewife	13.6
	Student	33.0
	Civil servant	2.7
	Independent	12.9
	Unable to work	0.8
Living location		
	Inside city	0.8
	Around city	2.7

Variable	Category	%
	Village	37.9
	Camp	58.7
Marital status		
	Single	25.0
	Married	73.5
	Divorced	1.5
Number of children		
	No children	51.1
	1–2	9.1
	3–5	13.6
	6+	26.1
Mother alive		
	Yes	79.5
	No	20.5
Father alive		
	Yes	65.0
	No	35.0

Variable	Category	%
Number of siblings		
	None	0.8
	1–3	12.5
	4–6	33.7
	7–9	37.5
	10+	15.5
Social support		
	Yes	74.6
	No	25.4
Chronic disease		
	Yes	20.5
	No	79.5

Note. Values are percentages of the total sample (N = 264). Percentages may not sum to 100 due to rounding.

Table 2. Sociodemographic Characteristics by Probable PTSD Status (PCL-5 Cutoff).

Variable	Category	Below Cutoff (n=18)	Probable PTSD (n=246)
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Variable	Category	Below Cutoff (n=18)	Probable PTSD (n=246)
Age group	18-29	88.9%	50.4%
	30-39	11.1%	28.0%
	40-49	0.0%	12.2%
	50-59	0.0%	3.7%
	60+	0.0%	5.7%
Sex	Female	50.0%	70.3%
	Male	50.0%	29.7%
Education	No formal	16.7%	45.5%
	Basic/primary	11.1%	21.5%
	High school	33.3%	22.0%
	University	33.3%	10.2%
	Other	5.6%	0.8%
Occupation	Unemployed	16.7%	38.6%
	Housewife	33.3%	12.2%
	Student	0.0%	35.4%
	Civil servant	5.6%	2.4%
	Independent	44.4%	10.6%

Variable	Category	Below Cutoff (n=18)	Probable PTSD (n=246)
	Unable to work	0.0%	0.8%
Living location	Village	94.4%	33.7%
	Camp	5.6%	62.6%
Marital status	Married	66.7%	74.0%
	Single	33.3%	24.4%
	Divorced	0.0%	1.6%
Children	None	83.3%	48.8%
	1-2	5.6%	9.3%
	3-5	11.1%	13.8%
	6+	0.0%	28.0%
Mother alive	Yes	88.9%	78.9%
Father alive	Yes	88.9%	63.0%
Social support	Yes	66.7%	75.2%
Chronic disease	Yes	0.0%	22.0%

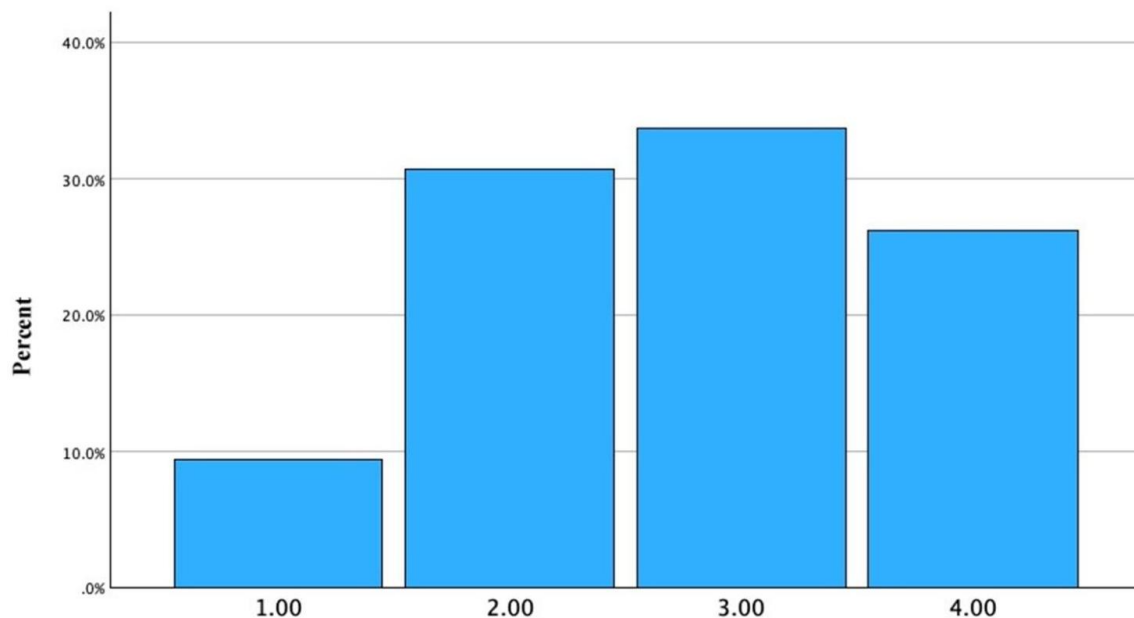
Note: Percentages within PTSD groups; small below-cutoff n=18 limits interpretation.

4.2. Traumatic event exposure

Based on the statistical analysis, participants were categorized into four groups according to the number of lifetime traumatic events: Group 1 (0–2 events), Group 2 (3–6 events), Group 3 (7–10 events), and Group 4 (11–36 events). A total of 9.5% of participants were classified in Group 1, reflecting low exposure, while 30.7% were in Group 2. Group 3 contained the largest proportion

of participants (33.7%), and 26.1% were in Group 4, representing very high trauma exposure (Figure 1).

Figure 1. Distribution of participants by number of lifetime traumatic events. Note. Values are percentages within each PTSD group. Probable PTSD was defined as a PCL-5 total score ≥ 33 . Percentages may not sum to 100 due to rounding. Percentages in the below-cutoff group should be interpreted cautiously because of the small sample size ($n = 18$).



4.3. Current symptomatology

Analysis of symptom severity among the 264 participants from the Shingal group revealed a considerable burden of psychological and physical health problems. The mean (SD) PCL-5 score was 49.67 (15.93), with 93.2% of participants exceeding the recommended cutoff for posttraumatic stress symptoms. For the HSCL-25 subscales, 83.7% scored above the clinical threshold for anxiety ($M = 2.66$, $SD = 0.74$), and 87.5% exceeded the cutoff for depression ($M = 2.64$, $SD = 0.62$).

For physical symptoms, the PHQ-15 yielded a mean (SD) score of 15.23 (7.82). Among participants, 49.2% reported severe symptoms, 17.8% moderate, 18.2% mild, and 14.8% minimal. The mean (SD) standardized severity rating was 3.02 (1.13) on a three-point Likert scale.

Regarding stress levels measured by the PSS-14, the mean (SD) score was 33.75 (9.36). High stress was reported by 34.5% of participants, moderate stress by 62.9%, and low stress by 2.7%. Additional information is presented in Table 3.

Measure	M (SD)	% Above Cutoff	Symptom Severity Distribution
PCL-5 (posttraumatic stress)	49.67 (15.93)	93.2	—
HSCL-25 Anxiety	2.66 (0.74)	83.7	—
HSCL-25 Depression	2.64 (0.62)	87.5	—
PHQ-15 (somatic symptoms)	15.23 (7.82)	—	49.2% severe, 17.8% moderate, 18.2% mild, 14.8% minimal
PSS-14 (perceived stress)	33.75 (9.36)	—	34.5% high, 62.9% moderate, 2.7% low

4.4. Correlation analysis

Only statistically significant correlations are presented here; the full correlation matrix, including nonsignificant associations, is provided in the online supplemental material (Supplementary File S1 Correlation Matrix). Correlation analyses examined the relationships among the HSCL-25, PCL-5, PSS-14, and PHQ-15 measures. The HSCL-25 was significantly correlated with the PCL-5 ($r = .66$, $p < .001$), PHQ-15 ($r = .51$, $p < .001$), and PSS-14 ($r = .24$, $p < .001$). The PCL-5 was also positively related to the PHQ-15 ($r = .39$, $p < .001$), and the PHQ-15 was correlated with the PSS-14 ($r = .14$, $p = .03$). Perceived stress scores (PSS-14) were associated with somatic symptom severity (PHQ-15; $r = .25$, $p < .001$). A statistically significant positive correlation was also found between PCL-5 severity and the War and Adversity Checklist-26 ($r = .22$, $p < .001$), indicating that participants with higher PCL-5 scores reported experiencing more traumatic events.

4.5. Sex comparison

Independent-samples *t*-tests indicated that males reported significantly higher trauma exposure on the War and Adversity Checklist-26 ($M = 8.74$, $SD = 4.58$) than females ($M = 7.52$, $SD = 4.19$), $t(262) = 2.13$, $p = .034$, $d = 0.28$. In contrast, females reported significantly greater PTSD symptom severity, with lower PCL-5 total scores observed in males ($M = 45.05$, $SD = 16.36$) than in females ($M = 51.75$, $SD = 15.33$), $t(262) = -3.22$, $p = .001$, $d = -0.43$. Further descriptive information on sex differences in trauma exposure and PTSD symptom severity is presented in Table 4.

Table 4. Sex Differences in Trauma Exposure and PTSD Symptom Severity.

Variable	Male (n = 82) M (SD)	Female (n = 182) M (SD)	t(262)	p	Cohen's d	95% CI for d
Trauma exposure (War and Adversity Checklist-26)	8.74 (4.58)	7.52 (4.19)	2.13	.034	0.28	[0.02, 0.54]
PTSD symptoms (PCL-5 total score)	45.05 (16.36)	51.75 (15.33)	-3.22	.001	-0.43	[-0.69, -0.16]

Note. Values are presented as M (SD). Higher scores indicate greater trauma exposure and more severe PTSD symptoms. Independent-samples t tests were conducted assuming equal variances. Effect sizes are reported as Cohen's d.

4.6. Factors contributing to the severity of psychological symptoms

4.6.1. Sex differences

Independent-samples t-tests showed that women reported significantly higher levels of psychological distress than men on all symptom scales. Women scored higher on depression (HSCL-25; $t(262) = 3.37$, $p < .001$, $d = 0.70$), anxiety (HSCL-25; $t(262) = 3.68$, $p < .001$, $d = 0.65$), perceived stress (PSS-14; $t(262) = 3.31$, $p < .001$, $d = 0.60$), and somatic symptoms (PHQ-15; $t(262) = 3.80$, $p < .001$, $d = 0.66$). These effect sizes indicate moderate to large sex differences in symptom severity, with women experiencing a greater burden of depression, anxiety, stress, and somatic complaints than men.

4.6.2. Age and education

Age significantly moderated symptom severity, particularly for somatic and PTSD symptoms. Older individuals (> 50 years) exhibited higher stress levels, as reflected in large effect sizes ($\eta^2 = .096-.141$) and FDR-corrected p-values significant for PHQ-15 and depression ($p = .001$). This finding suggests that older adults are vulnerable to age-related issues. Education demonstrated a strong protective effect: participants with higher education reported lower HSCL-25 ($\eta^2 = .182$), PCL-5 ($\eta^2 = .184$), and PHQ-15 ($\eta^2 = .223$) scores. These relationships remained significant in multivariate regression analyses: $\beta = -.21$, $p = .004$ for HSCL-25; $\beta = -.14$, $p = .043$ for PCL-5; and $\beta = -.17$, $p = .019$ for PHQ-15.

4.6.3. Living environment and occupation

The living environment also affected symptom severity. Participants residing in peri-urban or camp settings reported greater psychological distress than those in rural villages, with effect sizes

ranging from $\eta^2 = .072$ to $.131$. Occupational status was significantly associated with mental health outcomes, in particular with somatic symptoms: unemployed participants, students, and housewives reported higher symptom levels, with a large effect size for PHQ-15 ($\eta^2 = .328$). Differences in stress levels (PSS) by occupation were not statistically significant after FDR correction ($p = .339$), suggesting that occupation influences some domains of psychological distress more than others.

4.6.4. Family and other social factors

Family structure variables were also linked to symptom severity. The number of children was positively associated with psychological distress: participants with six or more children reported significantly higher PTSD, depression, stress, and somatic symptoms, with large effect sizes (up to $\eta^2 = .175$). In contrast, the number of siblings was weakly and non-significantly related to outcomes ($\eta^2 < .03$). Marital status showed mixed associations: married participants had slightly higher PCL-5 ($\eta^2 = .058$) and HSCL-25 ($\eta^2 = .041$) scores, whereas effects on stress ($\eta^2 = .015$) and somatic symptoms ($\eta^2 = .023$) were small and non-significant.

4.6.5. Health and social support

Chronic illness was significantly associated with higher scores across all psychological symptom scales: PTSD ($\eta^2 = .050$), HSCL-25 ($\eta^2 = .047$), PSS ($\eta^2 = .044$), and PHQ-15 ($\eta^2 = .048$), identifying it as a clinically relevant risk factor. Social support, though correlated with slightly lower mean symptom levels in bivariate analyses, did not reach statistical significance in multivariate models and showed small effect sizes ($\eta^2 < .05$). Further details are presented in Table 5.

Table 5: Factors Associated with Symptom Severity

Predictor	PCL-5 β	p	HSCL-25 β	p	PHQ-15 β	p	PSS-14 β	p
Age	.14	.104	.06	.472	.14	.104	.05	.574
Sex	.17	.005	.11	.078	.17	.005	-.07	.287
Education level	-.17	.019	-.21	.004	-.17	.019	-.01	.875
Occupation	.01	.851	-.06	.326	.01	.851	-.04	.509
Living location	.13	.033	.21	< .001	.13	.033	-.05	.467

Predictor	PCL-5 β	p	HSCL-25 β	p	PHQ-15 β	p	PSS-14 β	p
Marital status	.10	.163	-.04	.513	.10	.163	-.16	.037
Number of children	.06	.487	.05	.609	.06	.487	.24	.019
Number of siblings	.05	.370	-.04	.539	.05	.370	.04	.521
Social support	.03	.561	-.05	.384	.03	.561	-.08	.229
Past diagnosis	-.15	.039	-.09	.201	-.15	.039	.21	.008
Chronic illness	.04	.574	-.10	.123	.04	.574	.00	.994
Lost everything	-.11	.096	-.00	.968	-.11	.096	-.19	.007

Note. Values are standardized regression coefficients (β) from multiple linear regression analyses. PCL-5 = PTSD symptom severity; HSCL-25 = anxiety and depression symptoms; PHQ-15 = somatic symptoms; PSS-14 = perceived stress. Sex was coded with higher values indicating female sex. Boldface indicates statistically significant coefficients ($p < .05$).

4.7. Cortisol in hair samples

Cortisol showed statistically significant positive correlations with all psychological symptom scales included in this study. Higher levels of LF-cortisol were associated with higher scores on the PCL-5 ($r = .18$, $p = .004$), HSCL-25 ($r = .18$, $p = .004$), PSS-14 ($r = .18$, $p = .004$), and PHQ-15 ($r = .25$, $p < .001$). These findings align with the concept of cortisol as a biological marker of cumulative psychological distress, demonstrating consistent associations across multiple standardized measures. In contrast, LF-Cortisone displayed a weaker pattern of associations. It was significantly correlated only with the PHQ-15 ($r = .18$, $p = .01$) but not with the PCL-5, HSCL-25, or PSS-14 (all $p > .05$). Additional details are shown in Table 6.

Table 6: Cortisol Correlations

Variables	r	p	N
PCL-5 – LF-Cortisol	.18	.004	264
PCL-5 – LF-Cortisone	.10	.093	263

Variables	r	p	N
HSCL-25 – LF-Cortisol	.18	.004	264
HSCL-25 – LF-Cortisone	.08	.181	263
PSS-14 – LF-Cortisol	.18	.004	264
PSS-14 – LF-Cortisone	.10	.093	263
PHQ-15 – LF-Cortisol	.25	< .001	215
PHQ-15 – LF-Cortisone	.18	.008	214
LF-Cortisol – LF-Cortisone	.85	< .001	263

Note. r = Pearson correlation coefficient. LF = log-transformed values. PCL-5 = PTSD symptom severity; HSCL-25 = anxiety and depression symptoms; PHQ-15 = somatic symptoms; PSS-14 = perceived stress. Sample sizes vary due to missing data. Correlations significant at $p < .05$ are shown in bold (optional, depending on journal guidelines).

4.8. Latent profile analysis

Latent Profile Analysis (LPA) was conducted separately for PCL-5 subscale scores (intrusions, avoidance, negative alterations in cognition and mood, and arousal) and HSCL-25 total scores in 264 participants. This approach allowed the identification of latent symptom profiles representing clinically meaningful subgroups and clarified heterogeneity in trauma-related symptom expression beyond mean-level differences.

Models with one to six classes were estimated and compared using log-likelihood, AIC, BIC, sample-size-adjusted BIC (SABIC), and entropy. For the PCL-5, a three-class solution provided the best fit, with the lowest BIC (5758), good balance across indices, and acceptable entropy (0.81); models with more than three classes showed slightly lower AIC but poorer interpretability and highly unequal class sizes, so the three-class solution was retained.

For the PCL-5 model, class membership was distributed as follows: Low Symptom Group (Class 2; $n = 153$, 58.0%), Moderate Symptom Group (Class 1; $n = 59$, 22.3%), and High Symptom Group (Class 3; $n = 52$, 19.7%). Class 1 comprised participants with moderate PTSD symptom levels across all four domains, Class 2 (Low Symptom Group, potentially resilient) included

individuals with low distress and minimal trauma-related symptoms, and Class 3 consisted of participants with high PTSD, depression, and anxiety scores across all domains. Mean PTSD, depression, and anxiety scores differed significantly across the three classes (see Table 7), indicating that the profiles represent distinct constellations of symptoms rather than simple linear gradients of severity. Symptom severity was highest in the High Symptom Group, intermediate in the Moderate Symptom Group, and lowest in the Low Symptom Group.

Table 7: Latent Profile Model Fit Indices

Measure	Classes	Log Likelihood	AIC	BIC	SABIC	Entropy
PCL-5	1	−3058.0	6133	6161	6136	1.000
	2	−2859.0	5745	5791	5750	0.911
	3	−2829.0	5694	5758	5701	0.810
	4	−2823.0	5692	5774	5701	0.809
	5	−2809.0	5674	5774	5685	0.775
	6	−2794.0	5653	5771	5666	0.770
HSCL-25	1	−544.0	1096	1110	1097	1.000
	2	−416.0	847	872	850	0.882
	3	−392.0	803	839	807	0.805
	4	−380.0	786	832	791	0.769
	5	−377.0	786	843	793	0.783
	6	−376.0	791	859	799	0.720

Note. AIC = Akaike information criterion; BIC = Bayesian information criterion; SABIC = sample-size-adjusted Bayesian information criterion. Higher entropy values indicate better classification accuracy. Lower AIC, BIC, and SABIC values indicate better model fit. Final model selection was based on a combination of statistical fit indices and interpretability.

For the HSCL-25, a four-class model was selected as best fitting based on the lowest BIC (832), although entropy (0.77) was slightly lower than for some smaller-class models. The classes were: Low Distress Group (Class 2; 15.5%, $n \approx 41$), Moderate Distress Group (Class 1; 47.3%, $n \approx 125$), Intermediate Distress Group (Class 3; 17.4%, $n \approx 46$), and High Distress Group (Class 4; 19.8%, $n \approx 52$), which primarily differed in overall levels of anxiety and depression, with Class 4 showing the highest scores and Class 2 the lowest.

4.9. Multinomial logistic regression predicting class membership

Multinomial logistic regression models were used to examine predictors of latent class membership for both PCL-5 and HSCL-25 models, with the intermediate symptom class specified as the reference group. For the PCL-5 classes, higher education was associated with lower odds of belonging to the Low Symptom Group (Class 2) compared with the Moderate Symptom Group (Class 1; OR = 0.57, 95% CI [0.43, 0.65], $p < .001$). Greater trauma exposure increased the odds of membership in the Low Symptom Group (Class 2; OR = 1.12, 95% CI [1.03, 1.20], $p = .008$) but decreased the odds of belonging to the High Symptom Group (Class 3; OR = 0.86, 95% CI [0.75, 0.94], $p = .017$), suggesting that, relative to the intermediate group, some highly exposed individuals may show resilience or benefit from protective factors that buffer the impact of trauma, although these modest odds ratios warrant cautious interpretation. Cortisol, sex, occupation, and marital status did not predict PCL-5 class membership.

For the HSCL-25 classes, higher education reduced the likelihood of belonging to both the Moderate Distress Group (Class 2; OR = 0.66, 95% CI [0.50, 0.81], $p = .017$) and the High Distress Group (Class 4; OR = 0.51, 95% CI [0.40, 0.72], $p < .001$). Occupational status increased the odds of membership in the Intermediate Distress Group (Class 3; OR = 1.56, 95% CI [1.12, 2.03], $p = .011$), and female sex was associated with higher odds of belonging to the High Distress Group (Class 4; OR = 2.99, 95% CI [1.40, 4.50], $p = .017$). Trauma exposure was positively related to High Distress Group membership (OR = 1.18, 95% CI [1.05, 1.30], $p = .002$), whereas cortisol and marital status did not significantly predict HSCL-25 class membership. Fit indices for all latent profile models are summarized in Table 7.

4.10. Comparison of PCL-5 and HSCL-25 latent classes

Cross-tabulation of PCL-5 and HSCL-25 class memberships showed partial overlap but overall low concordance, indicating that PTSD-specific and general distress profiles capture related yet distinct dimensions of psychological burden in this sample. The joint distribution of participants across PCL-5 and HSCL-25 classes is presented in Table 8.

Table 8. Cross-Classification of PCL-5 and HSCL-25 Latent Classes.

PCL-5 Class	HSCL-25 Class 1	HSCL-25 Class 2	HSCL-25 Class 3	HSCL-25 Class 4
Class 1	22 (37.3%)	28 (47.5%)	5 (8.5%)	4 (6.8%)
Class 2	11 (7.2%)	54 (35.3%)	0 (0.0%)	88 (57.5%)
Class 3	14 (26.9%)	3 (5.8%)	35 (67.3%)	0 (0.0%)

Note. Values are n (%), with percentages calculated within PCL-5 classes (row percentages). Cohen's κ indicated poor agreement between the two classifications (average unweighted $\kappa = -0.02$; average weighted $\kappa = -0.23$), suggesting low concordance and supporting the interpretation that PTSD symptom profiles (PCL-5) and anxiety/depression profiles (HSCL-25) represent partly independent symptom dimensions.

5. Discussion

5.1. Overall symptom severity and chronic trauma effects

Nearly a decade after the 2014 ISIS genocide of the Yazidis in Shingal, the findings of this study reveal the chronic manifestation of trauma, reflected in extremely high PCL-5, HSCL-25, PHQ-15, and PSS-14 symptom severity scores. These results provide new evidence of post-genocide mental health outcomes and are further supported by biological data, with hair cortisol concentration serving as a marker of chronic stress. The exceptionally high prevalence of PTSD symptoms reported on the PCL-5 underscores that, although exposure to trauma was nearly universal, the intensity of symptoms indicates that psychological wounds remain raw and unhealed for many participants. Similarly, the elevated HSCL-25 and PHQ-15 scores suggest a strong interconnection between physical and psychological health. The concordance between psychological disorders and somatic symptoms in this sample is consistent with models of trauma-related psychopathology, which propose that chronic psychological distress may manifest through physical symptoms such as headaches, gastrointestinal disturbances, and fatigue (Hinton & Lewis-Fernández, Citation2011; McFarlane, Citation2010). These findings align with theoretical frameworks emphasizing hypothalamic–pituitary–adrenal (HPA) axis dysregulation and stress system adaptation among war-affected populations (Dhabhar, Citation2018; Yehuda & Lehrner, Citation2018).

5.2. Latent profile analysis findings

The LPA results highlight meaningful heterogeneity in trauma-related symptom patterns. The three PTSD classes and four anxiety/depression classes differed significantly in severity, with education and trauma exposure emerging as key predictors of class membership. However, the

low concordance between PCL-5 and HSCL-25 classifications suggests that PTSD and anxiety/depression represent partially distinct, though interrelated, symptom dimensions within this population.

5.3. Sex differences in symptom severity

One of the most important findings of this study is the substantially higher psychological distress observed among women. Across all psychological assessments – including PTSD, depression, somatic symptoms, and perceived stress – females scored consistently higher than males, and these differences remained statistically significant after corrections for multiple comparisons with moderate-to-large effect sizes. This pattern aligns with research in conflict-affected settings showing that women often bear a disproportionate psychological burden related to gender-based violence, caregiving responsibilities, and structural inequality (e.g. Otten et al., Citation2021; Taha & Sijbrandij, Citation2021; Tekin et al., Citation2016). Within Iraq, Taha and Sijbrandij (Citation2021) reported higher levels of depression, anxiety, and somatic symptoms among displaced Iraqi women than men, while Tekin et al. (Citation2016) found that female Yazidi refugees showed more severe PTSD and depression symptoms than male refugees, including flashbacks and hypervigilance.

The high proportion of female participants in this study likely reflects the demographic aftermath of the genocide. Many Yazidi men were killed during the ISIS attacks, resulting in a pronounced gender imbalance among surviving community members (Cetorelli et al., Citation2017). Cultural expectations of toughness or stoicism may therefore have discouraged remaining men from participating in the study or from disclosing their emotional suffering during interviews (Courtenay, Citation2000). Societal stigma surrounding mental illness can further inhibit help-seeking and open discussion of psychological difficulties among men (Branam et al., Citation2023). Consequently, women were not only overrepresented in the sample but also more likely to express emotional suffering during structured interviews, underscoring the need for sex- and gender-sensitive approaches that address both barriers to men's help-seeking and the distinct trauma narratives of male and female internally displaced Yazidis.

5.4. Cortisol

Hair cortisol levels showed positive correlations with PTSD, depression, stress, and somatic symptom severity. Although these associations had small effect sizes, they support the interpretation that higher cortisol concentrations reflect greater cumulative psychological distress and chronic activation of the HPA axis, even long after trauma. Similar associations between elevated hair cortisol and greater PTSD, depressive, and anxiety symptoms have been reported (Elnazer et al., Citation2021; Koumantarou Malisiova et al., Citation2021; Van den Heuvel et al., Citation2020), and a recent systematic review confirmed hair cortisol concentration as a reliable, non-invasive biomarker of long-term stress exposure (Schindler-Gmelch et al., Citation2024). Women with depression and anxiety also tend to have elevated cortisol levels

(Qin et al., Citation2023). While some studies have reported lower cortisol levels in chronic PTSD, suggesting increased glucocorticoid receptor sensitivity, others have observed elevated early levels; for example, Walsh et al. (Citation2013) found that higher cortisol measured shortly after trauma predicted greater subsequent PTSD severity, indicating stage-dependent variability in HPA axis function. Lawrence and Scofield (Citation2024) further highlighted that PTSD-related dysregulation of the HPA axis can alter immune functioning and increase risk for a wide range of physical health conditions.

In contrast to hair cortisol, hair cortisone showed limited associations with psychological outcomes in the present study. Aside from its association with somatic symptom severity, cortisone was not significantly related to PTSD symptoms, depression, anxiety, or perceived stress. This pattern suggests that, within this sample, cortisone may be less sensitive than cortisol to variations in psychological distress. Although cortisone was included as an additional indicator of HPA-axis-related activity, the absence of broader associations highlights potential differences in how these two glucocorticoids reflect stress-related processes. These findings underscore the need for further research to clarify the role of cortisone as a biomarker of chronic psychological stress, particularly in trauma-exposed populations.

5.5. Socioeconomic and demographic factors

Socioeconomic and demographic factors also contributed to the complexity of psychological symptom expression. Older participants reported higher symptom levels, likely reflecting cumulative exposure to displacement, bereavement, and chronic illness. Lower education, residence in camps or peri-urban settings, and unemployment were associated with poorer mental health, consistent with the compounded effects of poverty, insecurity, and restricted access to resources.

Education appeared to function as a protective factor, potentially through greater coping resources, access to information and services, and enhanced perceived agency. In contrast, social support showed a weaker buffering effect than expected, which may reflect fragmentation of social networks and the loss of key family members in displaced communities. Marital status had nuanced effects: for some women, caregiving responsibilities, financial strain, and widowhood may have increased psychological distress rather than alleviating it.

Somatic expressions of trauma were common, with nearly half of the participants reporting severe bodily symptoms. In contexts where mental illness carries stigma, psychological distress may be communicated preferentially through physical complaints that are more culturally acceptable, a pattern widely described as somatic expression of trauma-related suffering (Hinton & Lewis-Fernández, Citation2011), underscoring the importance of culturally sensitive clinical approaches that address both emotional and somatic manifestations of trauma.

5.6. Implications for intervention and policy

The findings show that the Yazidi population continues to bear the combined burden of past violence and ongoing adversity. There is a pressing need for long-term, culturally appropriate, and gender-sensitive mental health care that addresses psychological symptoms, physical health, social functioning, and biological stress regulation.

Interventions should include strategies to engage men in discussions about trauma and treatment, challenge norms that equate masculinity with emotional suppression, and provide safe spaces for both men and women to disclose suffering. The latent profile analysis demonstrates that individuals within the same traumatic context show distinct symptom patterns, indicating that stepped and tailored care is required rather than one-size-fits-all approaches. By integrating interview-based assessments with biological measures, this study offers a more comprehensive understanding of how genocide-related-trauma affects displaced Yazidis and provides actionable insights for clinicians, humanitarian actors, and policymakers seeking to mitigate long-term consequences of mass violence.

5.7. Limitations

The use of convenience sampling within IDP camps in Duhok may limit the generalizability of the findings to other Yazidi or trauma-exposed populations. The absence of a non-trauma-exposed control group also restricts comparison and limits causal inference. Cultural stigma regarding mental health remains a major barrier to help-seeking and open disclosure, which may have influenced participation and self-report data. Fear and mistrust related to biological data collection further affected participation: Nearly 95 potential participants declined to provide hair samples, primarily due to cultural or privacy concerns. Interviewers reported that several participants feared that their hair could be misused, reflecting local spiritual and religious beliefs that hair may be used for harmful purposes (e.g. superstition or spiritual control). Others, particularly male participants, opted out due to short hair. Because relatively few studies have been conducted in the Kurdistan Region, there is limited regional data for comparison. This scarcity of local research restricts contextualization of the findings and further underscores the need for additional studies to strengthen the regional evidence base and to refine culturally appropriate recruitment and data-collection strategies.

5.8. Future directions

Future research should incorporate clinician-administered assessments of PTSD to complement self-report data and enhance diagnostic accuracy. Longitudinal studies are also needed to track changes in hair cortisol and cortisone levels over time, providing a clearer understanding of chronic stress trajectories and recovery patterns. Future studies should also examine protective factors such as social support, coping flexibility, and community resilience to better capture mechanisms of adaptation among Yazidis and other traumatized populations. A higher number of male participants would allow to research possible sex differences in HPA-axis functioning and cortisol regulation. Including a control group of non-traumatized individuals would improve

causal interpretation by accounting for potentially confounding variables such as age, environmental factors, and medical comorbidities. Finally, research should investigate the effects of prior trauma exposure and intergenerational trauma to better understand the biological and psychosocial mechanisms underlying adaptation and resilience in post conflict communities.

6. Conclusion

This study provides strong evidence of the profound psychological and biological consequences of genocidal trauma. The elevated symptom scores demonstrate the enduring nature of trauma-related distress within this population and emphasize the urgent need for trauma-informed mental health interventions.

The modest yet statistically significant correlations between hair cortisol levels and psychiatric symptoms support the integration of biological stress markers with psychometric assessments, in line with prior work identifying hair cortisol concentration as an indicator of cumulative stress exposure (Schindler-Gmelch et al., Citation2024; Stalder & Kirschbaum, Citation2012). When combined with culturally sensitive methodologies, the inclusion of biological data contributes to a more comprehensive and contextually grounded framework for both clinical practice and research on trauma in war-affected populations.

Author contributions

Hataw Ahmed Sharif: Study design, organization of data collection, statistical analysis, and manuscript writing; Prof. Dr. Martin Hautzinger: Supervision, funding, study design, review of statistical analysis, and manuscript drafting; Prof. Dr. Thomas G. Schulze: Supervision, study design, and manuscript revision; Ibrahim Mohammed: Organization of data collection and study design; Dr. Urs Heilbronner: Supervision, Study design, and manuscript review; Prof. Dr. Dr. Jan Kizilhan: Study design and Manuscript review.

Transparency declaration

As the first author (Hataw Ahmed Sharif), I affirm that this manuscript is an honest, accurate, and transparent account of the study being reported; that no important aspects of the study have been omitted; and that any discrepancies from the study as planned and reported have been fully explained.

Analytic code

The analytic code used in this study is openly available in Zenodo (details to be provided after peer review).

Research materials

The research materials for this study are available in Zenodo (details to be provided after peer review).

AI transparency declaration

Language editing was facilitated using generative AI (Perplexity AI) to enhance manuscript clarity and readability in accordance with Taylor & Francis guidelines on responsible AI use. This support was limited to editorial refinement; all scientific content, methodological decisions, analyses, and conclusions remain the original work of the authors. All AI-assisted revisions were reviewed and formally approved by the authors.

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Disclosure statement

No potential conflict of interest was reported by the author(s).

Data availability statement

The dataset supporting the findings of this study is deposited in the Zenodo repository (DOI: to be added). Due to the sensitive nature of human subject data, access is restricted to protect participant confidentiality. Peer reviewers are granted access via a private link during the review process.

Supplemental Material

Supplemental data for this article can be accessed online at <https://doi.org/10.1080/20008066.2026.2627701>.

Additional information

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