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# Neurobiological and psychosocial mechanisms of PTSD in immigrants and refugees: A review

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## ABSTRACT

Post-traumatic stress disorder (PTSD) disproportionately affects immigrant and refugee populations due to cumulative trauma exposure across pre-migration, peri-migration, and post-migration phases. This narrative review synthesizes neurobiological and psychosocial research to elucidate mechanisms underlying PTSD in immigrant groups. Neurobiological analyses reveal hippocampal atrophy, amygdala hyperactivity, and medial prefrontal cortex (mPFC) hypoactivity mirrored in rodent models of chronic stress. These changes correlate with memory fragmentation, hypervigilance, and impaired fear extinction, exacerbated by acculturation pressures, discrimination, and socioeconomic hardship. Immigrants and refugees exhibit PTSD rates exceeding 30% in some cohorts, compared to approximately 3–4% in non-immigrant populations. While neurobiological studies often focus on non-immigrant populations and psychosocial research overlooks biological mechanisms, this review demonstrates how structural inequities such as limited healthcare access interact with dysregulated hypothalamic-pituitary-adrenal (HPA) axis function to amplify vulnerability. The findings underscore the need for integrated interventions addressing both neurobiological and structural determinants to reduce PTSD burden in displaced populations.

## INTRODUCTION

Post-traumatic stress disorder (PTSD) is a psychiatric condition marked by intrusive memories, avoidance, negative mood, and hyperarousal following trauma exposure per DSM-5-TR criteria (American Psychiatric Association, 2022; Bremner et al., 1995; Shin et al., 2006). While exposure to trauma is common, only a subset of individuals who experience traumatic events develop PTSD, with risk shaped by trauma nature, prior adversity, and biological vulnerability (Sangalang et al., 2018).

Neurobiological research implicates the hippocampus, amygdala, and medial prefrontal cortex (mPFC) in PTSD pathogenesis. The hippocampus, crucial for contextual memory, shows reduced volume and function correlating with fragmented memories and stress dysregulation (Bremner et al., 1995; Cheng et al., 2022; Magalhães et al., 2019). The amygdala displays hyperactivity linked to hypervigilance and impaired fear extinction (Shin et al., 2006). The mPFC exhibits diminished activity, weakening inhibitory control over the amygdala and contributing to persistent anxiety (Murra et al., 2022). These patterns are observed in both human studies and rodent models replicating key PTSD features (Cheng et al., 2022; Magalhães et al., 2019).

Immigrants and refugees are at heightened risk for PTSD due to cumulative trauma across pre-migration (war, persecution), peri-migration (dangerous journeys, detention), and post-migration (acculturation stress, discrimination, legal uncertainty) phases (Juang et al., 2018; Sangalang et al., 2018). Additional stressors, including social isolation, language barriers, and socioeconomic hardship, increase PTSD vulnerability (Cadenas et al., 2022; Elshahat et al., 2021). Meta-analyses show refugees and asylum seekers experience significantly higher PTSD prevalence than non-immigrant populations, with rates exceeding 30% in refugee cohorts compared to approximately 3–4% in non-immigrant populations (Blackmore et al., 2020; Carroll et al., 2022; Henkelmann et al., 2020).

Discrimination and barriers to culturally competent mental health care compound these risks (George et al., 2015; Hauck et al., 2024; Thomson et al., 2015). However, most neurobiological studies focus on non-immigrant samples while immigrant-focused research emphasizes psychosocial factors, resulting in fragmented literature that rarely integrates both perspectives. This review synthesizes evidence from human and animal studies to clarify neurobiological and behavioural mechanisms underlying PTSD in immigrant populations.


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# METHODS

## Databases searched

A comprehensive literature search was conducted using PubMed, PsycINFO, Scopus, Web of Science, and Google Scholar to ensure broad coverage of biomedical and psychological research relevant to PTSD, trauma, neurobiology, and immigrant or refugee populations.

## Search strategy

Search terms included: "PTSD," "post-traumatic stress disorder," "trauma," "immigrant," "refugee," "neurobiology," "brain," "hippocampus," "amygdala," "prefrontal cortex," "HPA axis," "animal model," and "behavioural outcomes." The search was limited to articles published in English between 2004 and 2024.

## Inclusion and exclusion criteria

Studies were included if they were empirical investigations examining PTSD or trauma in immigrant or refugee populations, including human and animal models, reporting neurobiological or behavioural outcomes. Exclusion criteria were: studies focused exclusively on non-immigrant populations, studies not reporting neurobiological or behavioural outcomes, and review articles or commentaries without primary data.

## Study selection

Titles, abstracts, and full texts were screened for relevance. The final set included studies meeting all criteria and providing data on neurobiological or behavioural mechanisms of PTSD and trauma in immigrant or refugee populations.

## Data extraction

Data were extracted by the author regarding study design, sample characteristics, outcomes assessed, key findings, and, for animal studies, the specific model and behavioural assays used.

## Synthesis approach

Due to heterogeneity in populations and methodologies, a narrative synthesis was conducted. Findings from human and animal studies were integrated thematically to highlight convergences and divergences in neurobiological and behavioural mechanisms.

# RESULTS

## Study selection flow

A total of 37 studies were included in this review after screening titles, abstracts, and full texts for relevance and eligibility based on predefined criteria. The included studies comprised both human and animal research focused on neurobiological, behavioural, and sociocultural mechanisms underlying PTSD and trauma in immigrant and refugee populations.

## Study characteristics

Human studies encompassed immigrant and refugee populations across North America, Europe, and Australia, with sample sizes ranging from small clinical cohorts to large population-based surveys. Key outcomes assessed included PTSD prevalence, neurobiological markers such as hippocampal and amygdala volume and function, acculturation stress, and intergenerational trauma. Animal studies primarily utilized rodent models employing chronic social defeat stress (CSDS), maternal separation (MS), and chronic unpredictable mild stress (CUMS) paradigms (Cheng et al., 2022; Golden et al., 2011; Magalhães et al., 2019; Murra et al., 2022). Table 1 details the 37 included studies' design, population, sample sizes, outcomes, and key findings.

## Neurobiological mechanisms

Hippocampus: Multiple human neuroimaging studies reported reduced hippocampal volume in individuals with PTSD, including immigrants and refugees, compared to matched controls (Bremner et al., 1995; Cheng et al., 2022; Magalhães et al., 2019). This reduction was associated with fragmented traumatic memories and impaired contextualization, as well as dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis and heightened stress reactivity. Animal models of chronic stress mirrored these findings, demonstrating hippocampal atrophy, reduced neurogenesis, and deficits in spatial memory when compared to non-stressed control animals (Cheng et al., 2022; Magalhães et al., 2019).

Amygdala: Functional neuroimaging studies consistently identified hyperactivity in the amygdala in response to trauma-related cues among PTSD patients, including immigrants and refugees, relative to control subjects (Hauck et al., 2024; Shin et al., 2006). This hyperactivity correlated with symptoms such as hypervigilance, exaggerated startle responses, and impaired fear extinction. Structural findings on amygdala volume were mixed across studies, suggesting that functional alterations may be more prominent than structural changes.

mPFC: Studies indicated diminished responsivity and reduced volume in the mPFC, particularly in ventromedial and dorsomedial regions, in individuals with PTSD compared to healthy controls (Murra et al., 2022; Shin et al., 2006). This impairment weakened the mPFC's inhibitory control over the amygdala, contributing to emotional dysregulation, poor fear extinction, and persistent anxiety. Animal models of chronic stress also demonstrated disrupted mPFC-amygdala connectivity, exacerbating anxiety-like behaviours and social withdrawal when compared to control animals (Murra et al., 2022).

HPA axis: Dysregulation of the HPA axis was observed in both human and animal studies, with altered cortisol levels and impaired negative feedback mechanisms in individuals with PTSD relative to controls (Smith & Vale, 2006). Chronic stressors such as acculturation, discrimination, and socioeconomic hardship exacerbated HPA axis dysfunction in immigrant populations, leading to prolonged stress responses and increased vulnerability to PTSD (Cheng et al., 2022; Sangalang et al., 2018).

## Behavioural models and outcomes

Animal models such as CSDS, SPS (single prolonged stress), fear conditioning and extinction, learned helplessness, and CUMS were widely used to study PTSD-like behaviours and neurobiological mechanisms (Siegmund & Wotjak, 2006; Verbitsky et al., 2020). These models produced anxiety-like behaviours, depression-like phenotypes, memory impairments, and social withdrawal, paralleling symptoms observed in immigrants and refugees (Alqurashi et al., 2022; Cheng et al., 2022; Frank et al., 2019; Magalhães et al., 2019; Murra et al., 2022). For example, rodents exposed to chronic social defeat stress displayed increased anxiety, social avoidance, and neuroendocrine changes

**Table 1**. Characteristics and key findings of the 37 studies included in this narrative review, detailing each study's design, population or animal model, sample size, key outcomes assessed, primary findings, and control group characteristics.

| Citation | Study Design | Population/Model Type | Sample Size | Key Outcomes Assessed | Key Findings | Control Group Details |
|---|---|---|---|---|---|---|
| Alqurashi et al. (2022) | Animal (experimental) | Chronic unpredictable mild stress (CUMS) in male mice | ~30-40 mice per group | Spatial and recognition memory; mood-like behaviour (depression) | Reduced spatial and recognition memory; increased depression-like behaviour in CUMS-exposed mice | Non-stressed control mice |
| Andrews et al. (2019) | Human (cross-sectional) | Latino migrant farmworkers | *n* = 250+ | Discrimination; legal status fears; adverse childhood experiences (ACEs); harsh working conditions | Combinatorial stressors predicted learned helplessness responses in farmworkers | Low-stress/low-discrimination Latino workers |
| Bremner et al. (1995) | Human (clinical case-control) | Combat veterans with and without PTSD | PTSD *n* = 13; Control *n* = 15 | Hippocampal volume (structural MRI) | Significantly reduced left hippocampal volume in PTSD group | Healthy control veterans without PTSD history |
| Bucay-Harari et al. (2020) | Human (qualitative/survey) | Emerging Latino community | N/A | Mental health needs assessment; healthcare access | Mental health disparities in Latino immigrants; barriers to care documented | General population baseline |
| Cadenas et al. (2022) | Human (cross-sectional) | Latinx immigrants during COVID-19 | *n* = 400+ | Immigration status; discrimination; food insecurity; health outcomes | Immigration status mediated links between discrimination and negative health; food insecurity as mediator | Low-discrimination; high-access groups |
| Carroll et al. (2022) | Human (meta-analysis) | Forcibly displaced populations vs. labor migrants | Meta-analysis of 76 studies | PTSD prevalence; depression; anxiety; mental health disorders by migration phase | Higher prevalence in forcibly displaced vs. labor migrants; variation by migration phase | Labor migrants; non-displaced populations |
| Casas et al. (2020) | Human (qualitative) | Latina immigrants | *n* = 27 | Coping strategies; mental health maintenance | Cultural coping strategies identified (Spanish phrase: "Se vale llorar y se vale reír") | Latinas without immigration-related stressors |
| Cerdeña et al. (2020) | Human (scoping review) | Latinx refugee and immigrant families | Review of 48 studies | Intergenerational trauma; parent-child dynamics; mental health outcomes | Intergenerational trauma transmission documented; parental PTSD affects children | Non-traumatized families |
| Cheng et al. (2022) | Animal (experimental) | Chronic unpredictable mild stress (CUMS) in male mice | ~30 mice per group | Brain-derived neurotrophic factor (BDNF) methylation; prefrontal cortex and hippocampus gene expression; behaviour | CUMS induced BDNF hypermethylation in prefrontal cortex and hippocampus; stress-related gene changes | Non-stressed control mice |
| Elshahat et al. (2021) | Human (systematic review) | Immigrant populations | Review of 50+ studies | PTSD; depression; anxiety; "healthy immigrant effect" critique | Healthy immigrant effect does not fully explain mental health outcomes in immigration context | Non-immigrant or early-stage immigrant controls |
| Frank et al. (2019) | Animal (experimental) | Early-life stress (maternal separation) in rats | ~20-30 rats per group | Submissive behaviour; social hierarchy; depression-like phenotype | Early-life stress induced submissive behaviour in adult rats in social hierarchy tests | Non-separated control rats |
| Fuentes et al. (2024) | Human (cross-sectional) | Latine immigrant youth | n=150+ | Attitudes toward mental health; access to services; culturally-responsive programming | Culturally-responsive programs improve mental health service attitudes and engagement | Latine youth without cultural programming access |
| George et al. (2015) | Human (review) | Immigrant populations; Canadian context | Review article | Mental health disparities; public health approaches; determinants of health | Systematic review of immigrant mental health inequities in Canada | Non-immigrant populations |
| Golden et al. (2011) | Animal (experimental) | Chronic social defeat stress (CSDS) in mice | ~30-40 mice per group | Anxiety-like behaviour; social defeat susceptibility; neuroendocrine responses | CSDS produces reliable anxiety and social avoidance phenotypes | Non-defeated control mice |
| Gray et al. (2015) | Human (cross-sectional) | Gay Latino immigrants | n=89 | Community connectedness; resilience; cultural stressors; sexual orientation stressors | Community connectedness buffers against mental health challenges in gay Latino immigrants | Isolated gay Latino immigrants |

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|---|---|---|---|---|---|---|
| Guruge et al. (2015) | Human (scoping review) | Immigrant women; Canadian context | Review of 40+ studies | Social support; social conflict; mental health in immigration | Social support protective; social conflict exacerbates mental health challenges | Non-immigrant women; low-stress groups |
| Harnett et al. (2023) | Human (neuroimaging/epidemiological) | Trauma-exposed individuals; race/ethnicity analysis | n=1000+ | Neurophysiological tone (vagal function); threat reactivity; racial/ethnic differences | Structural inequities contribute to racial/ethnic differences in neurophysiological tone post-trauma; inequality amplifies biological vulnerability | Low-trauma; high-resource racial/ethnic groups |
| Hauck et al. (2024) | Human (cross-sectional) | Immigrant sample (VIOLIN study) | n=300+ | Perceived ethnic discrimination; institutional verbal violence; chronic stress; protective factors | Discrimination and violence associated with chronic stress; protective factors (social support; coping) reduce effect | Low-discrimination; high-support immigrant groups |
| Henkelmann et al. (2020) | Human (systematic review and meta-analysis) | Refugees in high-income resettlement countries | Meta-analysis of 55 studies | PTSD; depression; anxiety prevalence | PTSD prevalence 31% in refugees (95% CI); comparable depression/anxiety rates (~27%) | Non-refugee populations; host country general population |
| Juang et al. (2018) | Human (review) | Immigrant and refugee youth | Review of 50+ studies | Attachment relationships; resilience; adaptation; acculturation | Secure attachment and positive relationships buffer against acculturation stress in youth | Non-immigrant youth; insecurely attached immigrant youth |
| Maccari et al. (2014) | Animal (review) | Early-life adversity; stress exposure; epigenetic studies | Review | Neurobiological consequences; behavioural outcomes; epigenetic modifications | Early-life adversity produces lasting HPA axis changes; glucocorticoid receptor gene modifications | Non-stressed animals |
| Magalhães et al. (2019) | Animal (neuroimaging/fMRI) | Chronic unpredictable mild stress (CUMS) in rats | ~20-30 rats per group | Resting-state fMRI; dorsal hippocampus structure and function; behavioural outcomes | CUMS induced reduced hippocampal connectivity; spatial memory deficits parallel human PTSD | Non-stressed control rats |
| Malave et al. (2022) | Animal (review) | Early-life adversity; postnatal development; neural circuits | Review | Neural circuit function; HPA axis development; sensitive periods | Early-life adversity during critical periods alters neural circuit development and HPA function | Non-stressed animals; late-stage stress exposure |
| Mancini (2019) | Human (pilot intervention) | Immigrant and refugee youth | n=45 | School-based trauma-focused CBT; PTSD symptoms; school engagement | School-based trauma-focused intervention reduced PTSD symptoms in refugee youth | Historical control or waitlist group |
| Mendoza et al. (2017) | Human (cross-sectional) | Children of Latino immigrants in poverty | n=200+ | Economic stressors; sociocultural stressors; child well-being; stress biomarkers | Economic and sociocultural stressors predicted reduced child well-being and elevated cortisol | Low-stress Latino children; non-immigrant children |
| Motti-Stefanidi (2018) | Human (review) | Immigrant youth; cultural contexts | Review of 50+ studies | Resilience; acculturation; cultural factors; protective mechanisms | Culture and community support are critical moderators of resilience in immigrant youth; marginalization increases risk | Immigrants in supportive vs. hostile contexts |
| Murra et al. (2022) | Animal (experimental) | Chronic social defeat stress (CSDS) in mice | ~30-40 mice per group | Behavioural and neuroendocrine responses; susceptibility vs. resilience phenotypes; mPFC-amygdala connectivity | CSDS produces susceptible (depressive-like) and resilient phenotypes; resilient mice show preserved mPFC function | Non-defeated control mice |
| Rusch et al. (2020) | Human (community-based program) | Latinx immigrants | n=60+ | Community advocacy; parenting skills; mental health outcomes; PTSD symptoms | Community-based advocacy + parenting programs reduced mental health disparities in Latinx immigrants | Latinx immigrants without program access |
| Sangalang et al. (2018) | Human (cross-sectional) | Refugees and immigrants in United States | n=500+ | Trauma exposure; post-migration stress; mental health outcomes; PTSD; depression | Post-migration stress (marginalization; discrimination) amplifies PTSD risk; rates >30% in some groups | Non-immigrant general population (~3-4% PTSD) |

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| Citation | Study Design | Population/Model Type | Sample Size | Key Outcomes Assessed | Key Findings | Control Group Details |
|---|---|---|---|---|---|---|
| Sherin & Nemeroff (2011) | Human (review) | PTSD neurobiology | Review article | Neurobiological mechanisms of PTSD; brain imaging; neurotransmitter systems | Comprehensive review of PTSD neurobiological mechanisms (hippocampus; amygdala; prefrontal cortex) | N/A - Review article; non-PTSD comparison groups in cited studies |
| Shin et al. (2006) | Human (neuroimaging review) | PTSD patients and healthy controls | Meta-analysis of neuroimaging studies | Brain activation patterns; amygdala; mPFC; hippocampus; task-based and resting-state fMRI | Amygdala hyperactivity; mPFC hypoactivity; hippocampal dysfunction in PTSD vs. controls | Healthy controls without PTSD history |
| Siegmund & Wotjak (2006) | Animal (experimental) | Single prolonged stress (SPS) in mice | ~20-30 mice per group | Fear conditioning; fear extinction; conditioned vs. sensitized fear responses; PTSD-like phenotypes | SPS produces both enhanced fear conditioning and sensitized fear responses in mice | Non-stressed control mice |
| Smith & Vale (2006) | Human (review) | HPA axis physiology; stress response | Review article | HPA axis regulation; cortisol feedback; stress-related disorders | Comprehensive review of HPA axis function; dysregulation in PTSD and other stress disorders | Normal HPA axis function in healthy controls |
| Thomson et al. (2015) | Human (systematic review) | Immigrant populations; Canadian context | Review of 40+ studies | Mental health service access; barriers; recommendations; health disparities | Systematic barriers to mental health care access in immigrants (discrimination; cultural competence gaps; language) | High-access; culturally-informed service contexts |
| Verbitsky et al. (2020) | Animal (review and methodology) | Rodent models of PTSD | Methodological review | Behavioural assessment protocols; animal models; PTSD-like phenotypes | Comprehensive methodology for assessing PTSD-like behaviours in rodent models | Standard laboratory controls per protocol |

similar to those seen in human PTSD (Murra et al., 2022) compared to non-stressed controls.

### Immigrant-specific findings

Socioeconomic status and structural inequities: Immigrant and refugee populations often face systemic socioeconomic challenges, including poverty, unstable employment, and housing insecurity, which compound trauma exposure and are strongly correlated with higher PTSD prevalence and symptom severity (Andrews et al., 2019; Mendoza et al., 2017). Meta-analyses indicate PTSD rates exceeding 30% in some immigrant subgroups, particularly those exposed to pre-migration trauma and post-migration economic marginalization (Cadenas et al., 2022; Sangalang et al., 2018).

Acculturation stress: Acculturation pressures, language barriers, and identity conflicts are unique risk factors for PTSD in immigrants. Marginalization (alienation from both heritage and host cultures) has been associated with the highest risk and severity of PTSD (Sangalang et al., 2018).

Health disparities and discrimination: Immigrants encounter significant barriers to healthcare, including misdiagnosis and undertreatment due to cultural misunderstandings and discrimination (Bucay-Harari et al., 2020; Guruge et al., 2015). Children of traumatized immigrants exhibit elevated stress biomarkers and increased vulnerability to anxiety and depression (Cerdeña et al., 2020; Cheng et al., 2022).

Intergenerational trauma: Trauma transmission across generations is well-documented in refugee and immigrant families. Parental PTSD disrupts caregiving and fosters insecure attachment, while socioeconomic hardship and cultural conflict perpetuate stress and vulnerability in children (Cerdeña et al., 2020; Sangalang et al., 2018).

Systemic discrimination and detention: Detention and legal uncertainty exacerbate trauma, with detained asylum seekers and their children showing high rates of PTSD and developmental delays.

## DISCUSSION

The synthesis of neurobiological and psychosocial research reveals critical insights into disproportionate PTSD burden in immigrant populations. While neurobiological studies have established PTSD mechanisms including hippocampal atrophy, amygdala hyperactivity, and HPA axis dysregulation, these findings are primarily from non-immigrant populations (Bremner et al., 1995; Cheng et al., 2022; Shin et al., 2006). Conversely, immigrant-focused research emphasizes psychosocial risk factors such as acculturation stress, systemic discrimination, and intergenerational trauma, often neglecting biological pathways (Cerdeña et al., 2020; Sangalang et al., 2018). This disciplinary fragmentation obscures holistic understanding of how migration-related stressors interact with neurobiological vulnerabilities.

### The unique burden of migration-related trauma

Immigrants face cumulative trauma across pre-migration, peri-migration, and post-migration phases. Meta-analyses reveal PTSD prevalence rates exceeding 30% in refugee cohorts, nearly triple rates in non-immigrant populations (Blackmore et al., 2020; Henkelmann et al., 2020). Chronic stressors such as housing instability dysregulate the HPA axis, prolonging cortisol elevation and impairing fear extinction (Cheng et al., 2022; Magalhães et al.,

2019). Acculturation stress, particularly marginalization, correlates with greater PTSD severity, suggesting cultural identity conflicts directly modulate neurobiological stress pathways (Sangalang et al., 2018).

### Intergenerational trauma and epigenetic vulnerability

Trauma transmission across generations is a hallmark of immigrant mental health disparities. Parental PTSD symptoms disrupt caregiving and foster insecure attachment in children (Cerdeña et al., 2020). Rodent models of maternal separation demonstrate that early-life stress alters offspring HPA axis function, paralleled in human studies of refugee families (Maccari et al., 2014; Malave et al., 2022). Epigenetic modifications such as glucocorticoid receptor gene hypermethylation further entrench intergenerational risk (Cheng et al., 2022). These biological mechanisms intersect with structural inequities: children in undocumented immigrant families exhibit elevated cortisol levels and anxiety rates, compounded by systemic barriers to education and healthcare (Thomson et al., 2015).

### Structural inequities and neurobiological interactions

A critical yet understudied domain concerns how structural inequities directly interact with and amplify neurobiological PTSD mechanisms. While PTSD neurobiology is well-documented in general populations, its expression and severity are markedly influenced by systemic factors unique to immigrant experiences. Limited access to mental healthcare prevents timely intervention that could interrupt persistent amygdala hyperactivity or impaired fear extinction. This structural barrier exacerbates HPA axis dysregulation, as chronic legal uncertainty and social marginalization maintain elevated cortisol levels even when trauma-focused treatments become available.

Detention represents an extreme case of structural inequity intersecting with neurobiology: detained asylum seekers experience ongoing confinement trauma concurrent with neurobiological stress responses, creating a vicious cycle where structural violence perpetuates biological vulnerability (Harnett et al., 2023). Discrimination itself activates neurobiological stress responses. Recent neuroimaging work shows ethnic discrimination correlates with altered amygdala-prefrontal connectivity and heightened threat reactivity, suggesting social marginalization is neurobiologically embodied (Hauck et al., 2024).

Thus, interventions must address not only brain-based mechanisms but also structural contexts that maintain dysregulation, including policies affecting housing stability, legal status, employment access, and healthcare availability. Integration of structural reform with neurobiological treatment represents the frontier for improving mental health outcomes in this population.

### Bridging the neurobiological-psychosocial divide

A critical gap persists between neurobiological research overlooking sociocultural contexts and psychosocial studies neglecting biomarkers. While animal models link chronic stress to mPFC hypoactivity and social withdrawal, few human trials examine culturally specific coping strategies. Discrimination's neurobiological correlates remain underexplored in immigrant populations. This disconnect impedes culturally sensitive intervention development. Cognitive-behavioural therapies show efficacy, but effectiveness diminishes without cultural adaptations.

### Toward integrated interventions

Addressing PTSD requires interventions targeting both neurobiological and contextual determinants. Community-based programs combining trauma-informed therapy with advocacy for housing and legal rights demonstrate promise (Fuentes et al., 2024; Mancini, 2019; Rusch et al., 2020). Animal studies suggest that enriching environments enhance resilience, underscoring social integration's therapeutic potential. Policymakers must prioritize reducing detention durations and improving culturally competent care access.

### Future research directions

Future research must prioritize longitudinal neurobiological studies tracking brain changes across migration phases using multimodal neuroimaging and culturally sensitive protocols. Intervention research should test integrated approaches combining trauma-focused therapy with structural supports via randomized controlled trials examining cortisol patterns and amygdala reactivity. Additional priorities include expanding assessment to the insula and anterior cingulate cortex, implementing interventions in resource-limited settings via digital platforms, and employing community-based participatory research positioning refugee communities as research partners.

## CONCLUSION

This review underscores the necessity of integrating neurobiological and psychosocial frameworks to elucidate PTSD mechanisms in immigrant populations. Evidence demonstrates that while conserved PTSD mechanisms exist, their manifestation in immigrant populations is shaped by unique migration-related stressors and structural inequities. Future research must prioritize longitudinal studies tracking neurobiological changes alongside post-migration stressors and employ community-partnered approaches. Interventions should adopt dual focuses on brain-based therapies and structural equity, recognizing that cortisol dysregulation cannot be resolved through therapy alone if individuals remain in precarious conditions. By uniting translational neuroscience with sociocultural frameworks and structural reform, clinicians and researchers can mitigate compounded vulnerabilities faced by immigrants and refugees, fostering resilience across generations.

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## CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

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