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Research Article

Keywords:

Posted Date: November 16th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-11031509/v1

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Additional Declarations: No competing interests reported.

Human-Induced Trauma and Intimate Partner Violence: Longitudinal Associations with Mental Health and Functioning in A Six-wave Cohort of Women.

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Abstract

Background: Traumatic experiences differ in their causes, interpersonal meaning, and implications for recovery. Human-induced trauma (HIT) may be especially harmful because intentional or socially mediated harm can involve emotionally salient and unique experiences that include betrayal, coercion, injustice, and undermining of interpersonal trust. Intimate partner violence (IPV) is a salient form of HIT but is not always captured in conventional traumatic events indices.

Aims: This study examined longitudinally whether HIT, defined to include IPV, was more strongly associated than non-human-induced traumatic events with post-traumatic stress disorder (PTSD), major depressive episode (MDE), panic disorder, disability, and impaired quality of life. It also examined cumulative trauma burden and explored whether the apparent excess associated with HIT was attributable to IPV or to other human-induced exposures.

Methods: The analytic sample from the WATCH cohort study comprised 1,317 women from Sydney and Melbourne, contributing approximately 4,200 person-waves. Population-averaged generalized estimating equation models with robust standard errors were adjusted for age, site, refugee background, post-school education, marital status, and study wave. HIT comprised seven human-induced checklist events identified from within the World Mental Health Survey Traumatic Events Checklist and non-HIT comprised six items from the same list. IPV assessed with the WHO violence against women instrument. Exposure counts were standardized for direct comparison.

Results: 47.2% of participants had experienced at least one HIT exposure, 26.8% at least one non-HIT exposure, and 32.3% reported no traumatic event The two classes overlapped substantially: 17.8% of participants reported both, representing 37.8% of all women with any

HIT exposure and 66.6% of all women with any non-HIT exposure. Compared with women reporting no trauma, women exposed to both HIT and non-HIT had the highest morbidity across every outcome. Cumulative trauma burden showed a strong dose-response pattern: each additional trauma type increased the adjusted odds of PTSD 1.77-fold, MDE 1.39-fold, and panic disorder 1.45-fold, while increasing WHODAS disability by 0.90 points and AQoL impairment by 0.95 points. In lagged exposure models, HIT had larger point estimates than non-HIT across all five outcomes and was nominally stronger for MDE, panic disorder, and WHODAS disability, although multiplicity-adjusted q values were .079. Decomposition of HIT showed that IPV was strongly associated with every outcome, whereas the remaining checklist HIT and non-HIT counts were statistically indistinguishable from one another.

Conclusions: Greater cumulative trauma exposure was consistently associated with higher psychiatric morbidity and functional impairment, with women exposed to both human-induced trauma (HIT) and non-HIT showing the poorest outcomes. IPV-inclusive HIT demonstrated stronger associations than non-HIT across all outcomes, with the largest differences observed for major depressive episode, panic disorder, and disability. Decomposition analyses indicated that these excess associations were largely attributable to intimate partner violence (IPV); once IPV was considered separately, checklist-defined HIT and non-HIT showed similar associations with outcomes. Functional impairment associated with HIT remained evident after adjustment for concurrent depression, PTSD, and panic disorder, suggesting effects extending beyond diagnostic symptoms. PTSD was associated with both HIT and non-HIT, with no clear difference between trauma classes.

Background

Exposure to traumatic events is a major determinant of psychiatric morbidity, disability, and reduced quality of life.(1–4) The developmental stage at which trauma occurs may influence the type and severity of subsequent psychological difficulties, as well as accumulation of traumatic experiences across the life course. A substantial body of research suggests a dose-response pattern, however cumulative exposure is unlikely to be adequately captured by event counts alone. Different combinations of traumatic experiences may have distinct or interacting effects, such that the pattern, timing and nature of exposure can be as important as the total number of events experienced.(5) Longitudinal research also suggests that repeated and cumulative trauma may contribute to enduring vulnerability, with earlier exposure potentially shaping responses to later adversity and influencing longer-term trajectories of mental health. Our work has found that traumatic experiences differ substantially in their causes, interpersonal meaning, and implications for psychological recovery.(3,6–10) A natural disaster, serious accident, or life-threatening illness may generate intense fear, helplessness, and in some cases enduring post-traumatic symptoms, but these events generally do not involve another person deliberately inflicting harm.(8,11) Human-induced trauma (HIT), by contrast, encompasses traumatic experiences arising from intentional or socially mediated human actions, including interpersonal violence, persecution, captivity, armed conflict, forced displacement, and other forms of deliberate harm.(1,8,10,11) The conceptual premise of the current study is that the interpersonal and intentional nature of HIT may add psychologically important dimensions to the effects of threat itself, potentially

making the mental health impacts more serious or more prolonged. This is because human-inflicted harm can involve betrayal, coercion, moral transgression, injustice, and the violation of basic assumptions about safety and trust.(3,10,12–14). Recovery from trauma depends partly on restoring a sense of safety, predictability, interpersonal trust, and confidence in the social environment. Experiences in which another person deliberately causes harm may compromise these resources fundamentally and directly, and may therefore contribute not only to post-traumatic disorder related fear and arousal, as well as more complex manifestations of post-traumatic stress disorder (PTSD), depression, anxiety, interpersonal insecurity, diminished functioning, and poorer quality of life.(6,7) The term human-induced trauma is used herein for the class of events described in earlier taxonomies as human-caused or human-made,(1,9) and is retained in preference to interpersonal trauma because the class also includes collectively directed exposures such as armed conflict, persecution, and forced displacement, which are intentional but not interpersonal in the dyadic sense. No new construct is intended; the term is used for precision about the boundary of the class rather than to propose a novel category.

Certain populations appear to have higher risk for HITs, which may also be more strongly correlated with mental disorders. For example, people who experienced sexual and physical abuse and neglect during childhood,(15) military veterans,(5) people subject to ethnic, religious or racial persecution and civilians in war zones.(16,17)

Intimate partner violence (IPV) is a salient form of human-induced trauma.(18–20). Physical, sexual, and severe emotional violence perpetrated by an intimate partner is inherently interpersonal and intentional and may be characterised by repeated and long term patterns of exposure, coercive control involving pervasive sense of threat and fear, humiliation, degradation, betrayal, and restricted opportunities for escape.(8) Conceptually, IPV therefore belongs within the HIT construct rather than constituting a separate or ancillary traumatic exposure. The WATCH cohort is an Australian community based longitudinal mental health and IPV study of 1335 women (half who arrived from conflict affected countries) and their children. It was established in 2016-2016 and participants have been interviewed at six timepoints, approximately 12-18 month intervals (21–23). Mental disorder symptoms associated with IPV are very high in women including in women born in Australia, and higher amongst women who arrived from conflict-affected countries.(21,22) Children born to women who have experienced IPV have substantive differences in development compared with children whose mothers are not IPV survivors.(20) Throughout this paper, IPV refers to lifetime exposure with the current or most recent partner, as assessed by the WHO instrument, and not to 12-month prevalence. The traumatic events checklist also records lifetime exposure, but without restriction as to perpetrator or relationship. The two measures therefore overlap in timeframes (lifetime-to-date), but differ in scope: the IPV measure is tied to a single relationship and does not capture violence by earlier partners.

Women comprise approximately half of the population and experience disproportionately high rates of certain forms of trauma and mental disorders, underscoring the importance of research that specifically examines women’s experiences, risk factors, and mental health

outcomes. Australia's substantial migrant population includes many people who have arrived from conflict-affected regions; with 32.0% of the Australian population born overseas in 2025 (Australian Bureau of Statistics, 2026), this demographic context underscores the importance of research specifically examining the mental health and trauma-related experiences of people from refugee and conflict-affected backgrounds. The WATCH cohort, comprising half the sample of women who arrived from a conflict affected country, also included substantial exposure to potentially traumatic events, measured by the World Mental Health Survey.(21,24) In the WATCH cohort, war-zone exposure was reported by 28.0% of participants and having to flee home due to being a refugee by 21.8%. Other directly targeted human-induced events included being mugged or threatened with a weapon (7.4%), childhood beating by a caregiver (4.6%), and kidnapping or captivity (1.4%). (21,22) These high rates of exposure to human induced traumatic events amongst women make it important to distinguish and study the broad effects of cumulative trauma from any additional effect associated with human causation or direct interpersonal violence.

From a clinical and public health perspective PTSD is the hallmark psychiatric consequence of trauma, frequently occurring alongside depression and anxiety disorders.(25) A growing consensus is that functioning outcomes related to trauma may be more clinically meaningful to patients than symptom reduction.(6,26,27) Although trauma-related symptoms and functioning are correlated, they are distinct constructs, and symptom reduction alone does not necessarily restore an individual's functioning. For example, following human-induced trauma some people with PTSD or depression can function well, and others without mental disorder can exhibit poor functioning.

A further consideration is that women exposed to HIT frequently also experience other traumatic events (non-HIT) including accidents, serious illness, disasters and bereavement. Accumulated adversity may contribute to psychiatric morbidity through repeated stress, disruption of coping resources, and reduced economic opportunities to aid recovery.(6,7,14) Longitudinal follow-up therefore provides an opportunity to examine whether trauma histories predict subsequent psychiatric and functional outcomes and whether any differences between trauma classes persist across assessment waves.

Aims

The primary aim was to examine whether HIT was more strongly associated than non-human-induced trauma with PTSD, mood disorder, panic disorder, disability, and quality of life impairment among women participating in the WATCH Study.

A second aim was to characterize cumulative trauma burden by comparing women reporting no trauma, unclassified events only, non-HIT only, HIT only, and both classes, and by estimating the effect of each additional trauma type. A third aim was to clarify what carried any excess association of HIT by entering IPV separately alongside checklist HIT and non-HIT exposure counts. The previously proposed distinction between personally targeted and contextual HIT was retained as a supplementary analysis rather than a primary explanatory framework because, when IPV was removed from the targeted category, targeted checklist

events were no more strongly associated with outcomes than non-HIT events. Three hypotheses were thus specified as follows. Accordingly, we distinguish herein between those tested as confirmatory analyses and those developed following the primary results. The first two were confirmatory: that human-induced trauma would show stronger associations than non-human-induced trauma with each of the five outcomes, and that outcomes would worsen with the number of trauma types reported, with women exposed to both classes showing the greatest morbidity. The third hypothesis, that any excess associated with HIT would be attributable in substantial part to IPV rather than distributed evenly across human-induced events, is exploratory. The decision to include IPV within the HIT class, the decomposition of the HIT class, and the targeted-versus-contextual partition were all developed after an initial comparison using the checklist items alone returned a null result, and all three are reported here as exploratory rather than confirmatory analyses.

Methods

Study sample and design

The analysis used data from 1,317 women participating in the WATCH Study in Sydney and Melbourne, including 1,003 participants in Sydney and 314 in Melbourne. The cohort contributed approximately 4,200 person-waves across follow-up. WATCH is an Australian community-based longitudinal study of women and their children, recruited from public antenatal clinics in Sydney and Melbourne from 2015 to 2016, and designed to include approximately equal numbers of women from refugee or conflict-affected backgrounds and women born in Australia.(17,18) Participants have been interviewed at six timepoints at intervals of roughly 12 to 18 months, with assessment of intimate partner violence, mental disorder, disability and quality of life at each wave. The trauma events checklist was administered at waves 1, 2, 4, 5 and 6, and was not included at wave 3; the present analysis used the baseline report only and treated lifetime trauma exposure as a fixed baseline characteristic, so events first reported at subsequent waves were not incorporated. The cohort profile and measures are described in full elsewhere. (17)

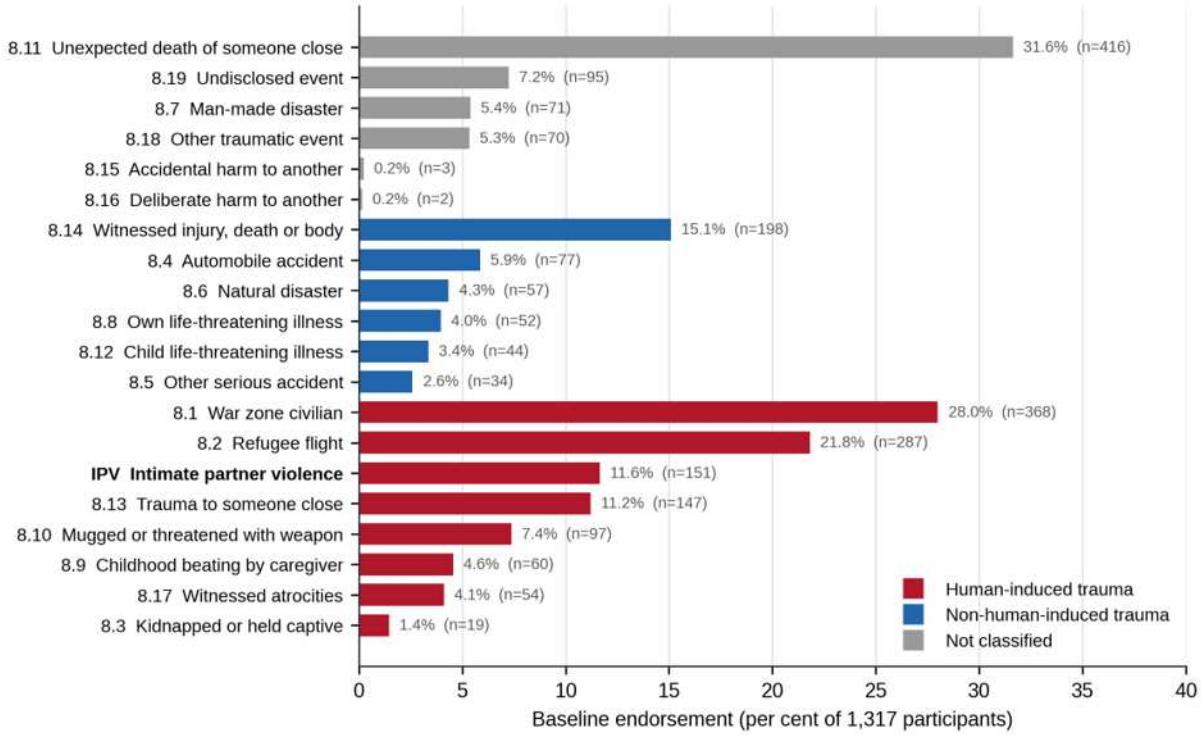
Trauma classification

HIT comprised seven human-induced items selected from the world trauma events checklist (refer Appendix A): war-zone exposure, refugee flight, kidnapping or captivity, childhood beating by a caregiver, mugging or weapon threat, trauma to someone close, and witnessing atrocities. IPV was also included in the main HIT definition and was identified using the WHO violence against women instrument, including any physical act, sexual coercion, or severe emotional abuse.

Non-HIT comprised six non-human-induced events: two accident items, natural disaster, the participant's own life-threatening illness, a child's life-threatening illness, and witnessing injury or death. Six checklist items remained unclassified due to ambiguity; unexpected death of someone close was the most common of these, endorsed by 31.6% of participants.

Under the revised classification, 621 women (47.2%) reported at least one HIT exposure once IPV was included, 353 (26.8%) reported at least one non-HIT exposure, and 425 (32.3%) reported no traumatic event of any kind. The mutually exclusive contrast groups comprised 151 women reporting unclassified events only, 118 reporting non-HIT only, 386 reporting HIT only, and 235 reporting both HIT and non-HIT.

Figure 1. Traumatic exposures at baseline. Intimate partner violence is classified as human-induced trauma and is measured with the WHO instrument.



In each wave of the WATCH study, we assessed intimate partner violence (IPV) using the World Health Organization (WHO) self-report measure, administered after privacy was confirmed and only to women who reported having married or lived with a partner.(21,28,29) For the purposes of the present trauma classification, IPV was defined more restrictively than in previous WATCH studies such that: an IPV exposure was coded when a woman endorsed any of eleven items of the WHO instrument: being humiliated in front of others, or threatened with harm to herself or to someone close to her (the two severe emotional-abuse items); being pushed, shaken or having something thrown at her, slapped or having her arm twisted, punched, kicked or dragged, strangled or burned, threatened with a weapon, or attacked with a weapon (the seven physical-violence items); or being forced to perform sexual acts she did not want (the sexual-violence item). The six controlling-behaviour items of the instrument, which inquire about jealousy, accusations of infidelity, restriction of contact with friends and with family, surveillance of her whereabouts, and control of money, were excluded. This threshold was adopted so that IPV entered the analysis as a discrete violent act comparable in kind to the events of the trauma events checklist. Earlier WATCH publications reported broader IPV constructs, including psychological IPV comprising controlling-behaviour items, and their prevalence estimates are therefore not directly comparable with the traumatic IPV indicator used herein. The indicator used in the present analysis corresponds closely to the

physical IPV category of those reports, which was 5.4% among Australian-born women and 6.6% among refugee-background women at baseline; the IPV estimates in those reports (25.8% and 44.4% respectively) additionally incorporated the severe psychological IPV category (20.5% and 37.8%), which is composed largely of the controlling-behaviour items excluded in the present classification. Applying that broader construct to the present data returned a baseline prevalence of 36.7% overall, 27.3% among Australian-born women and 45.4% among women born overseas, against the 6.8% yielded by the narrow definition. A sensitivity analysis using the broader construct is reported in Supplementary Table S1. For the longitudinal analyses, IPV identified at T1 was used for analyses of T2 to T6 outcomes, and IPV identified at either T1 or T2 was used for analyses of T3 to T6 outcomes.

Five outcomes were examined: PTSD, MDE and panic disorder using the MINI neuropsychiatric interview,(30) disability measured using the WHODAS-12,(31) and impaired quality of life measured using the AQoL.(32) The study protocol and methods are described elsewhere. (12,20,21)

Previous analyses of the WATCH cohort

The WATCH cohort has supported several analyses drawing on overlapping measures, and the present study differs from each in the definitions of exposure and outcome variables. Specifically, the cohort profile and the antenatal depression study that followed immediately documented IPV using the broader WHO construct, which included the controlling-behaviour items, and focused on cross-sectional analyses of prevalence estimates and correlates; whereas present analysis applied the narrow definition of IPV described above and examined the longitudinal patterns of the narrowly defined IPV, with baseline exposure predicting outcomes at five subsequent waves (see Table 1 in the Supplemental File).

Statistical analysis

Population-averaged generalized estimating equation models with robust standard errors were adjusted for age, site, refugee background, post-school education, marital status, and study wave. Exposure counts were standardized so that trauma classes containing different numbers of component events could be compared on a common scale. Analyses included exposure-group contrasts relative to women reporting no trauma, cumulative trauma-count models, simultaneous HIT and non-HIT models, disability models additionally adjusted for concurrent psychiatric disorder, and decomposition models entering IPV, checklist HIT, and non-HIT simultaneously. Sensitivity analyses were undertaken comparing the broader and narrow definitions of IPV employed in earlier WATCH studies (see Table 2 in the Supplemental File).

Two IPV ascertainment windows were considered in the comparison of HIT and non-HIT: baseline IPV predicting outcomes from T2 to T6, and IPV ascertained at T1 or T2 predicting outcomes from T3 to T6. The latter specification reduced the possibility that IPV measured at T2 and outcomes measured at the same wave contributed to temporal ambiguity. Equality

tests compared the HIT and non-HIT coefficients, and Benjamini-Hochberg adjustment was used across the five primary contrasts.

Results

Trauma exposure groups and outcomes relative to no trauma

Compared with the trauma-free reference group, non-HIT exposure alone was associated with poorer functioning, including a 1.47-point increase in AQoL impairment (95% CI 0.67 to 2.27) and a 0.81-point increase in WHODAS disability (95% CI 0.04 to 1.59), with non-significant elevations in psychiatric disorder.

HIT exposure at any point up to the baseline interview, that is lifetime exposure to date, and in the absence of any non-HIT exposure, was associated with higher odds of MDE (OR 1.85, 95% CI 1.23 to 2.80) and panic disorder (OR 3.00, 95% CI 1.38 to 6.50), together with functional impairment of similar magnitude to that seen with non-HIT alone. Women with experiences of to both trauma classes were the most affected on every outcome. Relative to women reporting no trauma, they had nearly six times the odds of PTSD (OR 5.94, 95% CI 2.12 to 16.67), more than three times the odds of MDE (OR 3.21, 95% CI 2.11 to 4.88), and over four times the odds of panic disorder (OR 4.17, 95% CI 1.87 to 9.31). They also had substantially greater disability and quality-of-life impairment, with WHODAS and AQoL increases of 2.47 and 3.03 points, respectively.

Women reporting only unclassified events, predominantly unexpected bereavement, were indistinguishable from the trauma-free group across all outcomes. For example, the OR for MDE was 0.81 (95% CI 0.46 to 1.44) and the WHODAS difference was 0.25 points (95% CI -0.33 to 0.84).

In a sensitivity analysis, human-induced trauma no longer exceeded non-human-induced trauma on any outcome: PTSD OR 1.45 versus 1.86, MDE OR 1.40 versus 1.29, panic disorder OR 1.26 versus 1.57, WHODAS b +0.92 versus +0.78, and AQoL b +0.93 versus +0.77, with equality p values of .27, .49, .39, .60 and .53 respectively.

Outcome	Unclassified only	Non-HIT only	HIT only	Both classes
PTSD (OR)	0.29 (0.03-2.39)	1.72 (0.48-6.17)	1.37 (0.42-4.46)	5.94 (2.12-16.67)
MDE (OR)	0.81 (0.46-1.44)	1.28 (0.72-2.28)	1.85 (1.23-2.80)	3.21 (2.11-4.88)
Panic disorder (OR)	1.57 (0.70-3.53)	1.53 (0.62-3.74)	3.00 (1.38-6.50)	4.17 (1.87-9.31)
WHODAS-12 (b)	+0.25 (-0.33, 0.84)	+0.81 (0.04, 1.59)	+0.87 (0.33, 1.41)	+2.47 (1.64, 3.30)
AQoL (b)	+0.24 (-0.33, 0.80)	+1.47 (0.67, 2.27)	+1.37 (0.82, 1.92)	+3.03 (2.27, 3.79)

Adjusted GEE estimates relative to 425 women reporting no traumatic event of any kind. HIT includes IPV ascertained at T1 or T2.

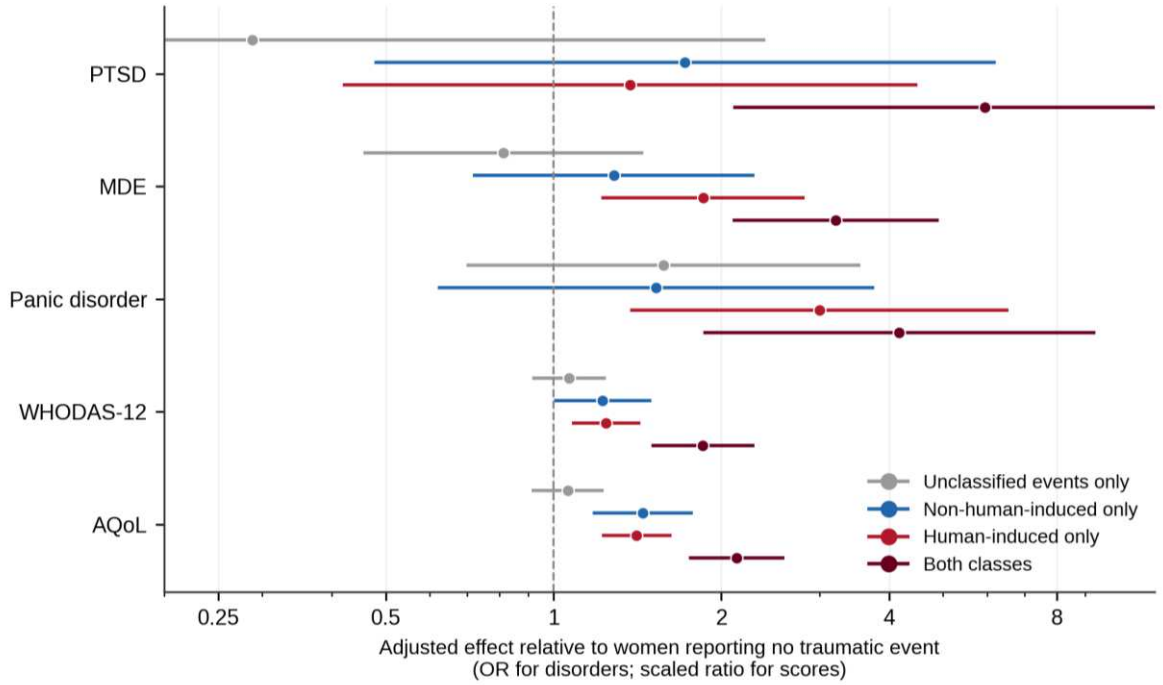
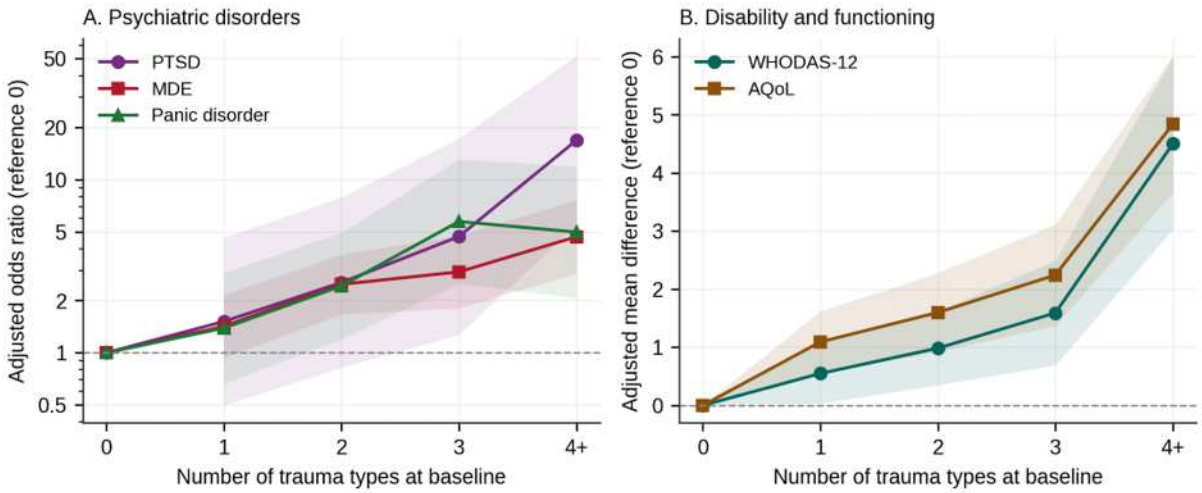


Figure 2. Exposure groups against a trauma-free reference

Cumulative trauma burden

The dose-response relationship was the most robust result in the analysis. Each additional trauma type increased the adjusted odds of PTSD 1.77-fold (95% CI 1.50 to 2.09), MDE 1.39-fold (95% CI 1.28 to 1.51), and panic disorder 1.45-fold (95% CI 1.26 to 1.68). Each additional trauma type also increased WHODAS disability by 0.90 points and AQoL impairment by 0.95 points. Women reporting four or more trauma types had roughly seventeen times the odds of PTSD relative to women reporting no trauma.

Figure 3. Cumulative trauma burden, human-induced and non-human-induced combined



HIT versus non-HIT

When HIT and non-HIT counts were entered simultaneously and expressed per standard deviation, HIT including IPV had the larger point estimate for every outcome under both IPV ascertainment windows. In the lagged exposure T3-to-T6 specification, each standard deviation of HIT was associated with an OR of 1.92 for PTSD (95% CI 1.30 to 2.84), 1.59 for MDE (95% CI 1.32 to 1.92), and 1.97 for panic disorder (95% CI 1.41 to 2.75), together with increases of 1.23 points in WHODAS disability (95% CI 0.72 to 1.75) and 1.14 points in AQoL impairment (95% CI 0.72 to 1.57).

The corresponding non-HIT estimates were 1.52 for PTSD (95% CI 1.16 to 1.99), 1.17 for MDE (95% CI 1.00 to 1.37), 1.09 for panic disorder (95% CI 0.83 to 1.42), 0.57 points for WHODAS (95% CI 0.21 to 0.94), and 0.73 points for AQoL (95% CI 0.38 to 1.07). Equality tests were nominally significant for MDE (p=.032), panic disorder (p=.020), and WHODAS (p=.048), but not for PTSD (p=.403) or AQoL (p=.152). After Benjamini-Hochberg correction across the five contrasts, the q value for the three nominally significant comparisons was .079. The consistency of direction across outcomes and ascertainment windows is therefore more informative than any single p value.

In an additional analyses, we repeated these models with the broader IPV construct used in earlier WATCH studies and the estimates reported herein remain unchanged, with human-induced trauma retaining the larger coefficient across all five outcomes, but attenuated the contrast: OR 1.85 versus 1.58 for PTSD, 1.53 versus 1.20 for MDE, 1.86 versus 1.14 for panic disorder, b +1.13 versus +0.62 for WHODAS, and b +1.08 versus +0.76 for AQoL, with equality p values of .581, .093, .070, .111 and .270. No equality test reached conventional significance under the broader construct in the lagged exposure window.

Outcome	HIT per SD	Non-HIT per SD	p	HIT per SD	Non-HIT per SD	p
PTSD (OR)	1.89 (1.41-2.54)	1.46 (1.18-1.82)	0.226	1.92 (1.30-2.84)	1.52 (1.16-1.99)	0.403
MDE (OR)	1.49 (1.28-1.73)	1.18 (1.03-1.34)	0.040	1.59 (1.32-1.92)	1.17 (1.00-1.37)	0.032
Panic disorder (OR)	1.62 (1.19-2.21)	1.13 (0.89-1.44)	0.140	1.97 (1.41-2.75)	1.09 (0.83-1.42)	0.020
WHODAS-12 (b)	+1.09 (0.68, 1.51)	+0.51 (0.22, 0.80)	0.028	+1.23 (0.72, 1.75)	+0.57 (0.21, 0.94)	0.048
AQoL (b)	+1.05 (0.70, 1.39)	+0.64 (0.37, 0.92)	0.087	+1.14 (0.72, 1.57)	+0.73 (0.38, 1.07)	0.152

Left block: IPV ascertained at baseline, outcomes T2 to T6. Right block: IPV ascertained at T1 or T2, outcomes T3 to T6. p values test equality of the two coefficients.

Figure 4. Human-induced trauma including IPV is more potent than non-human-induced trauma. p values test equality of the two coefficients.

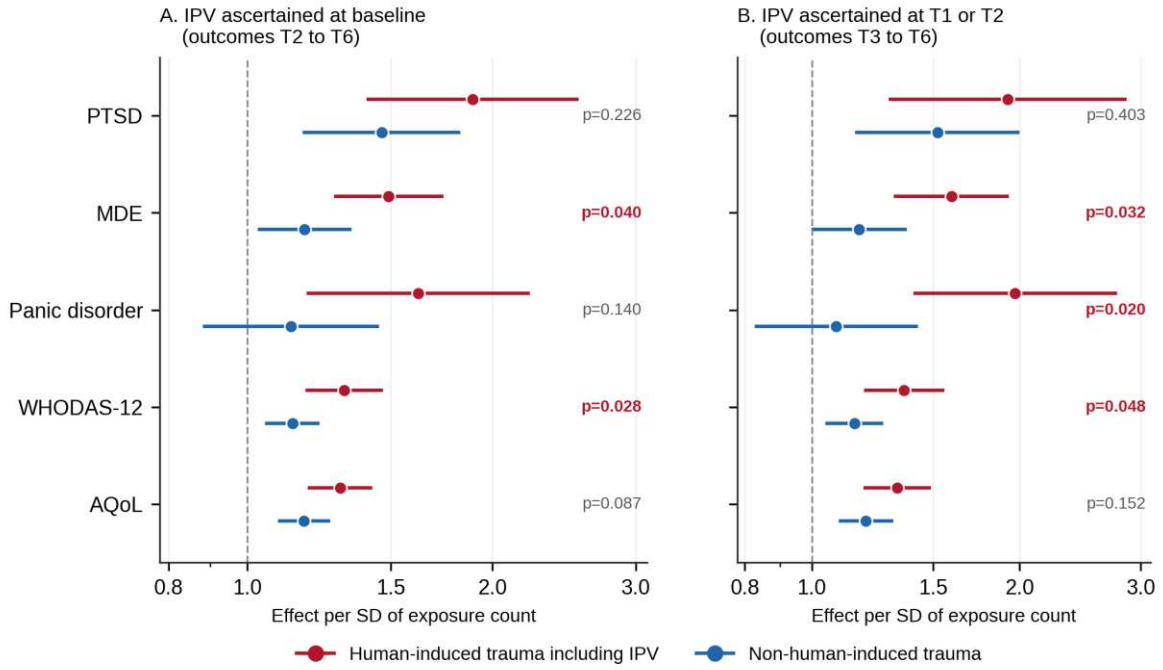


Figure 4. Human-induced versus non-human-induced trauma under two IPV ascertainment windows.

Functional impairment beyond psychiatric diagnosis

The functional consequences of HIT were not wholly explained by concurrent psychiatric disorder. After adjustment for concurrent MDE, PTSD, and panic disorder, 64% of the association between HIT and WHODAS disability remained: the coefficient fell from 1.18 to 0.76. For AQoL, 76% remained, with the coefficient declining from 1.16 to 0.88. These

findings indicate that a substantial share of functional impairment associated with HIT fell outside the three diagnosed disorders examined.

Decomposition of HIT: the role of IPV

The excess association of the broad HIT class was largely carried by IPV. When IPV was entered as a separate term alongside the checklist HIT count and the non-HIT count, IPV was strongly associated with every outcome: PTSD OR 3.17 (95% CI 1.46 to 6.88), MDE OR 3.48 (95% CI 2.48 to 4.89), panic disorder OR 4.59 (95% CI 2.56 to 8.25), WHODAS +2.64 points (95% CI 1.55 to 3.73), and AQoL +2.96 points (95% CI 2.02 to 3.89).

In the same models, checklist HIT and non-HIT remained independently predictive but were statistically indistinguishable from one another on all five outcomes, with equality p values ranging from .28 to .82. Thus, the finding is not that other human-induced events are harmless; rather, per unit of exposure, the remaining checklist HIT events were no more harmful than accidents, disasters, and other non-HIT events once IPV was modelled separately.

Outcome	IPV (any vs none)	Checklist HIT per SD	Non-HIT per SD	HIT vs non-HIT p
PTSD (OR)	3.17 (1.46-6.88)	1.70 (1.22-2.38)	1.52 (1.21-1.91)	0.63
MDE (OR)	3.48 (2.48-4.89)	1.29 (1.11-1.51)	1.20 (1.06-1.37)	0.55
Panic disorder (OR)	4.59 (2.56-8.25)	1.34 (0.92-1.94)	1.12 (0.87-1.44)	0.51
WHODAS-12 (b)	+2.64 (1.55, 3.73)	+0.80 (0.41, 1.19)	+0.53 (0.24, 0.82)	0.28
AQoL (b)	+2.96 (2.02, 3.89)	+0.72 (0.40, 1.04)	+0.67 (0.39, 0.94)	0.82

All three exposure terms entered simultaneously with covariates, T2 to T6.

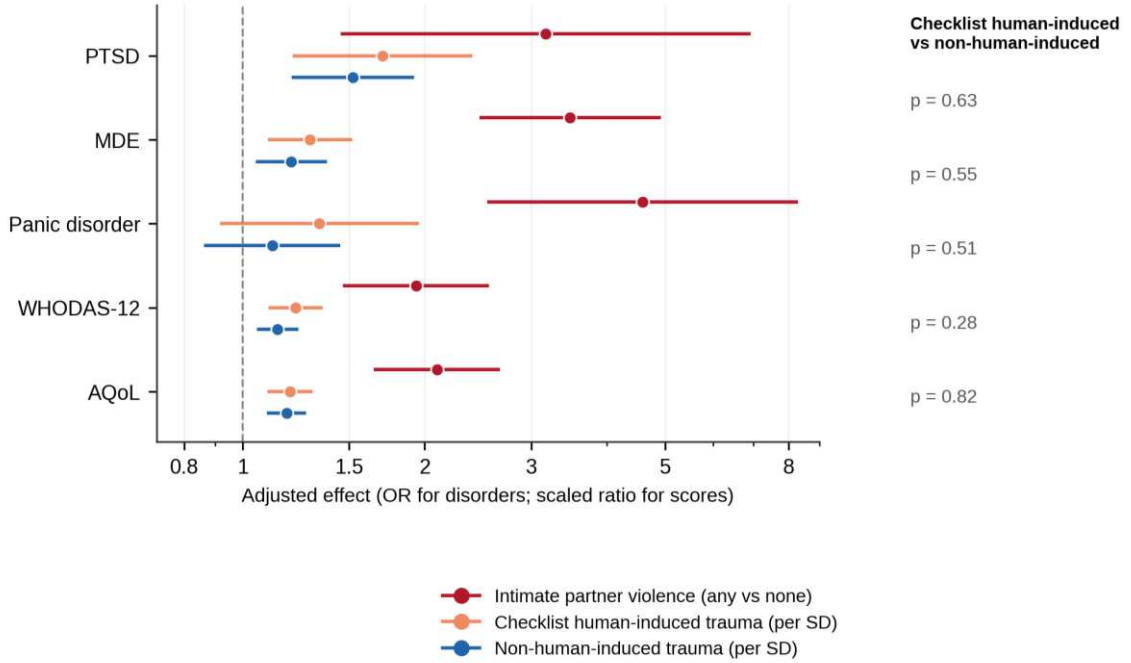


Figure 5. Decomposition of the human-induced class.

Supplementary targeted-versus-contextual analysis

For completeness, human-induced exposures were also partitioned into personally targeted events—kidnapping, childhood beating, mugging or weapon threat, with or without IPV—and collective or contextual events—war-zone exposure, refugee flight, witnessing atrocities, and trauma to someone close. When IPV was included in the targeted count, targeted exposure exceeded non-HIT for MDE, panic disorder, and WHODAS disability (p=.017, .017, and .028, respectively). However, when IPV was removed, no targeted-versus-non-HIT contrast approached significance on any outcome, with p values from .22 to .79; targeted-versus-contextual p values ranged from .13 to .90. The confidence intervals for MDE and the functioning measures made a large independent effect of directedness unlikely, although

power remained limited for PTSD and panic disorder because the targeted checklist events were rare.

Discussion

This study examined human-induced trauma using a revised definition that incorporates IPV as an integral component of HIT and uses women reporting no trauma as the principal reference group. Three findings stand out. First, cumulative trauma burden showed a strong and graded relationship with psychiatric disorder symptoms and functional impairment. Second, IPV-inclusive HIT had larger estimated associations than non-HIT across every outcome, with the clearest differences for MDE, panic disorder, and disability. Third, decomposition of the HIT class showed that the excess was largely attributable to IPV rather than to a general property shared by all human-induced checklist events.

The cumulative exposure finding is central to interpretation. Women exposed to both HIT and non-HIT had the poorest outcomes across the board, and each additional trauma type was associated with progressively greater psychiatric morbidity and functional impairment. This pattern suggests that classification by trauma type should not displace attention to total trauma burden. Women who experience interpersonal violence, conflict, persecution, or displacement may also experience accidents, illness, bereavement, and other adversities, and their mental health may reflect the accumulation of these exposures.

At the same time, the consistent direction of the HIT-versus-non-HIT comparisons indicates that trauma classification contains potentially meaningful information. Depression and panic disorder showed particularly marked differences in the IPV-inclusive models. Intentionally targeted interpersonal harm may affect domains that extend beyond the physical injury and psychological harm most often associated with fear and threat. These HIT specific forms of abuse may in addition impact self-worth, trust, agency, perceived control, and expectations of safety within relationships.(8,33) Repeated intentional harm within a close relationship may also shape attachment representations, and insecure attachment has been proposed as one pathway through which interpersonal trauma contributes to later depression, anxiety, and difficulty in relationships.(34) The present data cannot test this pathway, since attachment was not assessed, but it is consistent with the pattern of impairment observed and is a candidate mechanism for future work.

The functional findings are also important. HIT was associated with more disability than non-HIT, and much of the association with WHODAS and AQoL remained after adjustment for concurrent MDE, PTSD, and panic disorder. Trauma-related morbidity therefore cannot be fully characterized by the presence or absence of these diagnoses. Interpersonal violence may affect social, occupational, and everyday functioning through psychological distress, avoidance, impaired relationships, physical consequences of violence, continuing insecurity, or other pathways not captured by the diagnostic outcomes considered here.(4,35,36)

IPV emerged as the most distinctive component of HIT. Unlike many checklist events, IPV involves intentional interpersonal harm within a close relationship that is expected to be

characterised by trust, care and kindness, and may be recurrent, coercive, and difficult to escape.(7,8,18,19) It can combine threat with betrayal, relational proximity, and ongoing exposure. The finding that checklist HIT and non-HIT were statistically indistinguishable once IPV was entered separately indicates that the broader HIT effect should not be interpreted as evidence that all human-caused events are intrinsically more harmful than accidents or disasters. Rather, IPV appears to account for much of the observed human-induced excess in this cohort. Earlier iterations of the present analysis suggested that personally targeted HIT might be the key explanatory dimension, but the supplementary analyses showed that this pattern depended on IPV being included in the targeted category. Once IPV was removed, targeted checklist events were no more strongly associated with outcomes than non-HIT events. Directness of targeting may still be clinically meaningful, but the present data do not support it as an independent explanation for the HIT effect.

PTSD differed somewhat from the pattern observed for MDE, panic disorder, and disability. Both HIT and non-HIT were associated with PTSD, but the difference between them was not statistically significant. Severe threat may therefore be sufficient to produce PTSD risk regardless of whether the event originates from intentional human action. This interpretation remains provisional because current PTSD prevalence was low and estimates were imprecise.(6,25,33,37)

Strengths and limitations

Strengths of the study include the longitudinal design, repeated outcome assessment, the use of population-averaged GEE models, adjustment for key demographic and study covariates, standardized exposure counts, and the explicit incorporation of IPV into the HIT construct. The use of a trauma-free reference group also clarified the degree to which each exposure pattern differed from women reporting no traumatic event. Retention was satisfactory over an extended follow-up: of the 1,317 women assessed at baseline, 1,124 (85.3%) contributed outcome data at wave 2, 941 (71.5%) at wave 3, 842 (63.9%) at wave 4, 748 (56.8%) at wave 5, and 547 (41.5%) at wave 6, the last of which was not able to be completed, and therefore not reflective of retention. Attrition was addressed analytically by the use of population-averaged models, which retain all available person-waves.

Several limitations should be considered. Current PTSD prevalence was low, limiting precision for PTSD estimates. The IPV index employed herein included acts of emotional, physical and sexual violence but not controlling behaviours, and is therefore narrower than the construct used in earlier WATCH studies. The exposure is linked to the current or most recent partner, so violence by earlier partners is not captured. IPV reporting rose from 6.8% at T1 to 11.6% across T1 and T2, this could reflect additional experience of IPV that was not disclosed in the antenatal setting where recruitment occurred, or other measurement effects that cannot be distinguished here. Together, these factors might have accounted for under-ascertainment, so the associations reported here are likely to be conservative. Trauma exposures other than IPV were measured as lifetime exposures and treated as fixed, so

subsequent events were not incorporated. The rarer targeted checklist events also limited power in the supplementary analyses.

Multiplicity is another important consideration. Although HIT exceeded non-HIT at nominal significance levels for MDE, panic disorder, and WHODAS disability in the lagged exposure models, the Benjamini-Hochberg q value was .079 across these contrasts, and no single comparison exceeded a strict Bonferroni threshold. The coherent direction across all outcomes and both ascertainment windows warrants further investigation, but the results should not be described as definitive evidence that HIT is universally more harmful than non-HIT.

Implications

The findings have implications for trauma measurement, research, and clinical assessment. Conventional trauma indices may underrepresent one of the most consequential forms of intentional interpersonal harm if IPV victimisation is omitted or treated separately. Trauma assessments should therefore include physical, sexual, and severe emotional IPV alongside other human-induced events.

At the same time, a simple binary division between human- and non-human-caused events is insufficient on its own. The present results indicate that cumulative burden is highly consequential and that the apparent excess associated with HIT is largely driven by IPV. Future trauma taxonomies may therefore benefit from capturing multiple dimensions simultaneously, including cumulative exposure, intentionality, relationship to the perpetrator, coercion, repetition, and ongoing threat.

Clinically, comprehensive assessment should document the range of traumatic exposures a woman has experienced and should ask specifically about violence within intimate relationships. Such information may be relevant to psychiatric presentation, functional impairment, interpersonal difficulties, and continuing safety needs.

These considerations also have implications for treatment. Although established trauma-focused interventions such as Prolonged Exposure (PE) and Cognitive Processing Therapy (CPT) are effective first-line treatments for PTSD, treatment of women exposed to IPV may require additional attention to the distinctive interpersonal and relational consequences of such trauma, including disrupted trust, diminished agency and perceived control, altered expectations of safety, difficulties in relationships, and, for some women, ongoing exposure to coercion or threat. This suggests a role for interventions that complement trauma processing with a more explicit focus on restoring agency, affect regulation, interpersonal functioning and relational safety. Interpersonal and affect-regulation approaches are potentially relevant because they directly target emotion regulation and interpersonal difficulties, although the evidence base for these approaches in IPV populations remains less established than that for first-line trauma-focused therapies. IPV-specific interventions may also be particularly valuable especially if they incorporate PTSD treatment alongside

modules addressing ongoing contact with abusive former partners and risk of revictimization(38)

Conclusion

In the WATCH cohort, trauma exposure was strongly associated with psychiatric morbidity, disability, and impaired quality of life, with cumulative trauma burden showing the most robust dose-response relationship. Human-induced trauma defined to include IPV had larger estimated associations than non-human-induced trauma across all five outcomes, particularly for MDE, panic disorder, and disability, although multiplicity-adjusted evidence was not definitive. Decomposition of the HIT class showed that IPV was strongly associated with every outcome and accounted for much of the observed excess, while checklist HIT and non-HIT were statistically indistinguishable when modelled alongside IPV. These findings support conceptualizing IPV as a central form of human-induced trauma and emphasize the need to assess both cumulative trauma burden and intentional interpersonal violence when evaluating mental health and functioning.

Acknowledgements

We wish to thank the WATCH study participants and our research assistant team. JF receives support from the Australian Government through the Australian Research Council's Centre of Excellence Projects funding scheme (project CE230100004).

Ethics Approval and Consent to Participate

The WATCH study is approved by the Southwestern Sydney Local Health District Human Research Ethics Committee (HREC/15/POOL/28) and Monash Health Ethics Committees. Participants gave informed consent to participate in the study before taking part and were remunerated for their time.

This study is reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) reporting guideline for cohort studies.

Consent for Publication

All authors have read and verified the manuscript for publication.

Competing interests

The authors declare no competing interests.

Funding/Support: This research was funded by the National Health & Medical Research Council of Australia (GNT1164736). The funding body had no role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; preparation, review, or approval of the manuscript; and decision to submit the manuscript for publication

Author Contributions Statement

S.R. wrote the main manuscript; S.R, O.G, S.C, D.S, A.S, T.W, J.F, L,R, A.T contributed to the main manuscript; S.R, O.G, S.C, D.S, A.S, T.W, J.F, L,R, A.T edited the main manuscript; A.T, T.W, A.S conducted statistical analysis; A.T prepared figures and tables; S.R, J.F received funding; S.R supervised the study and fieldwork.

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