



Kelvin Open Science Publishers  
Connect with Research Community

Editorial Article

Volume 1 / Issue 1

## KOS Journal of Addiction and Rehabilitation

<https://kelvinpublishers.com/journals/addiction-and-rehabilitation.php>

---

# Multi type Interpersonal Abuse and Psychiatric Outcomes: Systematic Review and Meta-analysis (2015-025)

**Bruna Caridi<sup>1,2</sup>, Ratti Elisabetta<sup>1</sup>, Vitacolonna Gabriele<sup>1</sup>, Caravelli Angela<sup>1</sup> and Vincenzo Maria Romeo<sup>1,3,4\*</sup>**

<sup>1</sup>STANDUP® Method, Italy

<sup>2</sup>Gastroenterology and Endoscopy Unit, Fondazione IRCCS Cà Granda, Ospedale Maggiore Policlinico, Milan, Italy

<sup>3</sup>Department of Culture and Society, University of Palermo, Palermo, Italy

<sup>4</sup>SPPG - School of Psychoanalytic and Groupanalytic Psychotherapy SPPG, Reggio Calabria, Italy

**\*Corresponding author:** Vincenzo Maria Romeo, STANDUP® Method, Italy; Department of Culture and Society, University of Palermo, Palermo, Italy; School of Psychoanalytic and Group analytic Psychotherapy SPPG, Reggio Calabria, Italy

---

**Received:** July 28, 2025; **Accepted:** August 05, 25; **Published:** August 07, 2025

**Citation:** Romeo VM, et al. (2025) Multi type Interpersonal Abuse and Psychiatric Outcomes: Systematic Review and Meta-analysis (2015-2025). *KOS J Addict Rehab*. 1(1): 1-17.

**Copyright:** © 2025 Romeo VM, et al., This is an open-access article published in *KOS J Addict Rehab* and distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

## 1. Abstract

**Background:** Polyabuse defined here as the co-occurrence of multiple forms of interpersonal victimization and/or substance-related harms within the same individual across overlapping developmental windows has emerged as a central construct for contemporary psychiatric science and services. Despite a rapidly expanding literature, practice and policy remain fragmented, often targeting single exposures rather than the syndemic patterns that patients actually experience.

**Editorial aim:** This editorial synthesizes the last decade of research through a meta-analytic lens to provide a pragmatic, clinically oriented reference point. Rather than reporting a single pooled estimate, we integrate convergent evidence on prevalence, risk architecture, nosology, and outcomes, highlight methodological strengths/weaknesses in the field, and translate findings into actionable guidance for clinicians, systems leaders, and policymakers.

**Synthesis of evidence:** Across population based surveys, clinical cohorts, and longitudinal studies, polyabuse is common and patterned by social adversity, with consistent dose-response relationships between the number of exposures and psychiatric burden across anxiety, mood, post-traumatic stress, substance use, and self-harm outcomes. Studies using person centered modeling identify reproducible latent classes characterized by early adversity, intimate partner violence, and polysubstance involvement, each linked to poorer treatment response and higher service utilization. Measurement heterogeneity (operational definitions, recall windows, and exposure counts) and residual confounding remain the main threats to inference, while prospective designs, preregistered analyses, and triangulation with administrative data strengthen causal interpretations.

**Practice and service implications:** The evidence supports routine polyabuse screening using brief, multi domain tools; trauma informed, staged care pathways; and integrated treatments that concurrently target PTSD, depression, and substance use. System level priorities include data linkage across health, social, and justice sectors; person centered outcomes; and reduction of structural drivers (poverty, housing instability, and gender based violence).

**Research agenda:** Priorities include consensus definitions and core outcome sets; transparent reporting standards; quasi experimental and longitudinal mediation studies to test mechanisms; and equity focused trials that evaluate scalable, culturally responsive interventions.

**Conclusion:** Polyabuse is a predictable, preventable driver of psychiatric morbidity. A meta analytic reading of the contemporary literature justifies a shift from siloed, exposure specific care to integrated, trauma informed systems supported by rigorous measurement and policy action capable of bending population curves in mental health.

## 2. Keywords

Polyabuse, Polyvictimization, Trauma informed care, Meta-analysis, Psychiatry, Integrated Treatment, Public mental health

## 3. Introduction

Polyabuse here used synonymously with **polyvictimization** and the co-occurrence of multiple interpersonal harms (e.g, emotional, physical, sexual abuse, exposure to domestic/community violence) and, in many cases, concomitant substance-related harms has become a central organizing construct for contemporary psychiatric research and clinical service design. Foundational work reframed children's and adolescents' experiences not as isolated "single-type" events but as clustered constellations with distinct etiologic and prognostic significance, inaugurating the

term *poly-victimization* and documenting its prevalence, correlates, and clinical import [1].

A decisive strand of evidence comes from the **adverse childhood experiences (ACE)** tradition, which established a graded, dose-response association between the number of adversities and a wide array of mental and physical health outcomes across the life course [2-3]. In a large systematic review and meta-analysis, Hughes and colleagues showed that multiple ACEs (often overlapping with maltreatment and violence exposure) are linked to markedly elevated risks for depression, anxiety, substance use, self-harm, and premature mortality, underscoring the syndemic nature of polyabuse and its downstream morbidity [2]. The original ACE cohort findings by Felitti and colleagues similarly demonstrated that breadth of adversity, rather than any single exposure, best predicts later burden of disease and psychiatric

comorbidity [3].

Mechanistically, **cumulative trauma** research suggests that repeated, multi-domain harms are associated with symptom *complexity* spanning PTSD, mood and anxiety disorders, dissociation, and interpersonal dysfunction beyond what single exposures predict [4]. Epidemiologic studies in adolescents and adults extend this gradient, showing that diverse adversities increase the *hazard* of first-onset DSM disorders and contribute to earlier age at onset, higher chronicity, and greater service utilization [5-6]. Together, these bodies of evidence justify a shift from siloed, exposure-specific paradigms to integrated, trauma-informed systems that recognize polyabuse as a **patterned risk architecture** rather than a sum of parts.

At the level of measurement and nosology, the field has evolved beyond simple adversity counts. Person-centered modeling (e.g, **latent class/profile analysis**) consistently identifies replicable subgroups such as classes characterized by high interpersonal violence coupled with emotional abuse/neglect, or profiles marked by extensive community and school violence each carrying distinct risk for internalizing, externalizing, and trauma-related symptoms. These approaches move the science toward clinically meaningful phenotypes that map onto differential prognosis and treatment response [7-8].

From a service and intervention standpoint, evidence increasingly favors **integrated care pathways** that concurrently address PTSD and substance use, a common co-occurrence within polyabuse trajectories. Randomized controlled trials of integrated, exposure-based therapies for PTSD with co-occurring substance dependence demonstrate superiority over control conditions in reducing PTSD severity without exacerbating substance use, providing a bridge from meta-analytic knowledge to pragmatic clinical decision-making [9]. Complementary meta-analytic work on psychological treatments for ICD-11 complex PTSD often a clinical expression of chronic, multi-domain adversity indicates that trauma-focused cognitive-behavioral therapy, exposure-based protocols, and EMDR outperform usual care on core symptom clusters, supporting the scaling of **trauma-informed, staged** interventions within routine systems [10].

In this editorial, we therefore adopt a **meta-analytic reading** of the last decade of research to: i) synthesize convergent evidence on prevalence, risk architecture, and outcomes; ii) highlight recurring methodological issues (heterogeneous operationalization, recall windows, and confounding) alongside best-practice solutions (prospective designs, preregistration, triangulation with administrative data); and iii) translate the evidence base into guidance for clinicians and systems leaders. Our goal is to provide a concise, practice-oriented reference that supports measurement-based, equity-attentive, and integrated responses to polyabuse across child, adolescent, and adult services.

## 4. Methodology

### Protocol and Reporting Framework

We developed and registered a protocol a priori on an international registry for systematic reviews to enhance transparency and minimize analytic flexibility [11]. Reporting follows **PRISMA 2020** guidance, with deviations explicitly justified in the Supplement [12]. Methods also draw on procedures recommended in the **Cochrane Handbook** for systematic reviews of interventions and risk factors and, where observational evidence predominated, on the **MOOSE** guidelines for meta-analyses of observational studies [13,14].

### Eligibility Criteria (PICOS & Study Designs)

**Population:** Children, adolescents, and adults from community or clinical settings.

**Interventions/Exposures:** Multi-domain interpersonal harms (e.g, physical, sexual, emotional abuse; exposure to domestic/community violence) and, where applicable, concomitant substance-related harms. Studies had to operationalize *polyabuse/polyvictimization* as  $\geq 2$  distinct abuse/violence categories or validated composite indices.

**Comparators:** No abuse/violence exposure or single-type exposure.  
**Outcomes.** Psychiatric symptoms/disorders (e.g, PTSD, depression, anxiety, SUD), self-harm/suicidality, functioning, and service utilization.

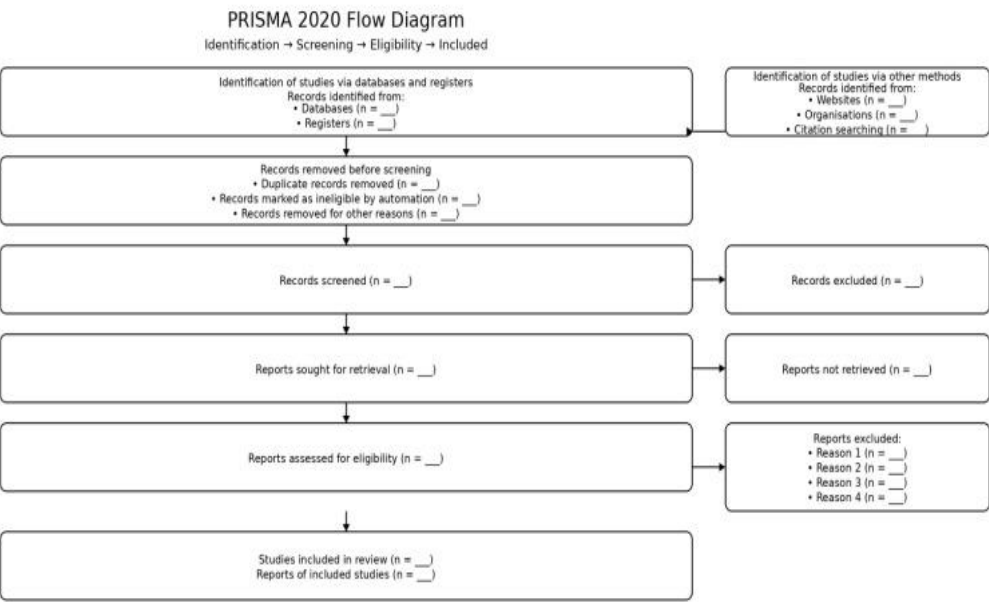
**Study designs:** Randomized/cluster trials, quasi-experimental studies, prospective or retrospective cohorts, case-control, and cross-sectional designs. Qualitative studies were narratively synthesized to contextualize quantitative findings but were not pooled.

**Time frame & language:** Studies published in the last decade; no language limits at search stage, with translation as needed.

**Exclusions:** Case reports/series (<10 participants), non-empirical commentaries, and studies lacking an explicit multi-type exposure definition.

The screening process, including records identified, deduplicated, excluded at each stage, and studies included, is detailed in the PRISMA 2020 flow diagram (Figure 1).

Figure 1:



Information Sources and Search Strategy

We searched MEDLINE (via PubMed), Embase, PsycINFO, CINAHL, Web

of Science Core Collection, and Scopus from database inception to the final search date, then limited to the last decade at screening. The strategy combined controlled vocabulary (e.g, MeSH/Emtree) and keywords for *polyvictimization*, *multi-type maltreatment*, *co-occurring abuse*, *intimate partner violence*, *child maltreatment*, and *polysubstance* where relevant, joined with mental-health terms (PTSD, depression, anxiety, SUD). Search strategies were **peer-reviewed using the PRESS checklist [15]**. To optimize retrieval efficiency, we applied evidence-based database combinations and documented de-duplication procedures [16,17]. Conference proceedings, trial registries, theses, and reference lists of included studies and relevant reviews were hand-searched to reduce publication bias.

Study Selection and Inter-rater Agreement

Titles/abstracts and full texts were screened independently by two reviewers using calibrated forms; conflicts were resolved by consensus or a third reviewer. Inter-rater reliability was quantified using **Cohen’s kappa** with 95% CIs [18]. Reasons for exclusion at full text were logged and summarized in a PRISMA flow diagram.

Key design features, populations, exposure operationalization, outcomes, and follow-up periods for all included studies are summarized in **Table 1**.

Table 1: Study characteristic.

Study (Year)	Countr y/Settin g	Desi gn	Sa mpl e (N; Age )	Exp osur e operatio n	Pri mar y outc ome s	Foll ow-up	DOI
Turner, et al, 2010	USA; National telephone survey (NatSC EV)	Cros s-secti onal (com muni	4,0 53; 2- 17 y	Poly victi miza tion = num	Trau ma sym ptom atolo gy	N/A	10.1016 /j.amepr e.2009.1 1.012

		ty)		ber of lifeti me victi miza tion type s (JV Q; 33 type s)	(com posit e)		
Finkelhor, et al, 2009	USA; National RDD survey	Cros s- secti onal (com muni ty)	1,4 67; 2- 17 y	Poly victi miza tion defin ed as high est decil e of lifeti me victi miza tion scor e	Psyc holo gical distr ess (curr ent)	N/ A	10.1016 /j.chiabu .2008.0 9.012
Finkelhor, et al, 2009	USA; National telephon e survey	Cros s- secti onal (com muni	4,5 49; 0- 17 y	Cou nts of past- year and	Prev alen ce of expo sure; poly	N/ A	10.1542 /peds.20 09-0467

		ty)		lifeti me victi miza tions ; mult i- type expo sure freq uenc ies	victi miza tion coun ts		
Green, et al, 2010	USA; National Comorbi dity Survey Replicat ion (NCS- R)	Cros s- secti onal (retro spect ive CA asses men t)	5,6 92; adul ts	12 child hood adve rsitie s; mod eled joint ly incl. coun ts of MFF adve rsitie s	First onse t of 20 DS M- IV disor ders (CID I)	N/ A	10.1001 /archgen psychiat ry.2009. 186
Kessler, et al, 2010	21 countrie s; WHO WMH Surveys	Cros s- secti onal (retro spect	51, 945 ; adul ts	12 child hood adve rsitie s;	First onse t of 20 DS M-	N/ A	10.1192 /bjp.bp. 110.080 499

		ive CA asses men t)		coun ts and clust ers	IV disor ders		
Widom, et al, 2007	USA; Midwest ern county	Pros pecti ve coho rt (doc umen ted abus e/neg lect vs. matc hed contr ols)	Cas es 676 ; cont rols 520 ; foll owed to mea n age 28. 7 y	Subs tanti ated phys ical/ sexu al abus e or negl ect; mult iple type s recor ded	DS M- III-R majo r depr essiv e disor der; com orbi dity	~18 -28 yea rs fro m ind ex to adu lt inte rvie w	10.1001 /archpsy c.64.1.4 9
Dube, et al, 2003	USA; Kaiser HMO primary care	Retro spect ive coho rt (AC E Stud y)	8,6 13; adul ts	ACE scor e (0- 10) acro ss abus e, negl ect, hous ehol d dysf uncti	Illici t drug initia tion, prob lems , addi ction , pare ntera l use	N/ A (ret ros pect ive)	10.1542 /peds.11 1.3.564

				on			
Dube, et al, 2001	USA; Kaiser HMO primary care	Retro spect ive coho rt (AC E Stud y)	17, 337 ; adul ts	ACE scor e (0- 10) inclu ding abus e and hous ehol d dysf uncti on	Lifet ime suici de atte mpts (age at first atte mpt)	N/ A (ret ros pect ive)	10.1001 /jama.28 6.24.30 89
Vachon, et al, 2015	USA; low- income summer program	Obse rvati onal (case - contr ol withi n coho rt)	2,2 92; 5- 13 y	Doc ume nted malt reat ment type s; varie ty/fr eque ncy/ seve rity indic es	Inter naliz ing and exter naliz ing dime nsio ns; mult i- infor mant ratin gs	N/ A	10.1001 /jamaps ychiatry .2015.1 792
Afifi, et al, 2014	Canada; 2012 CCHS- MH (national	Cros s- secti onal (com	23, 395 ; adul ts	3 abus e type s;	14 ment al cond ition	N/ A	10.1503 /cmaj.13 1792



	)	muni ty)		dose - resp onse by num ber of type s	s incl. suici dal ideat ion/a ttem pts		
--	---	-------------	--	--	---	--	--

Abbreviations: JVQ = Juvenile Victimization Questionnaire; ACEs = Adverse Childhood Experiences; AXIS/JBI/ROBINS-I/RoB2 = risk-of-bias tools as appropriate.

**Data extraction:** Two reviewers independently extracted study descriptors (design, setting, sample characteristics), exposure operationalization (number/types of harms; measurement instruments; recall window), outcomes (construct, instrument, timing), effect estimates (adjusted/unadjusted), and covariates. When required, corresponding authors were contacted for missing data. Discrepancies were reconciled by consensus.

**Risk of bias and study quality:** We assessed randomized trials with **RoB 2** (randomization process, deviations from intended interventions, missing data, outcome measurement, reporting) [19]; non-randomized comparative studies with **ROBINS-I** (pre-intervention confounding, selection, classification, deviations, missing data, measurement, reporting) [20]; and cross-sectional/prevalence studies with **JBI** critical appraisal tools (sampling, measurement validity, confounding, statistics) [21] and the **AXIS** tool as applicable [22]. Two reviewers rated each domain independently; disagreements were adjudicated by a senior methodologist. We planned sensitivity analyses excluding studies at critical risk of bias.

### Effect size computation

For binary outcomes, we extracted or computed **log odds ratios**; for continuous outcomes, we used **Hedges' g** with the small-sample correction

[23]. When studies reported multiple measures of the same construct, we prioritized validated clinician-rated instruments or the measure most frequently used across the literature to maximize comparability. Where necessary, means/SDs were imputed from medians/IQRs using established methods; authors were contacted to verify conversions.

### Data synthesis and statistical model

We pooled effect sizes using **random-effects meta-analysis** to accommodate between-study heterogeneity. As the base estimator, we used **DerSimonian-Laird** with **Hartung-Knapp** adjustment for test statistics and CIs to improve small-sample performance [24-25]. Heterogeneity was summarized with  $\tau^2$  and  $I^2$  and tested with Cochran's  $Q$  [26]. Prespecified **subgroup analyses** examined age group, sex/gender, setting (community vs clinical), exposure operationalization (count vs class/profile), and outcome domain. We performed **meta-regression** to evaluate whether effect sizes varied with moderators (e.g., number of abuse types, prospective vs retrospective ascertainment, adjustment for socioeconomic confounders) while guarding against overfitting [27].

### Dependent effects and complex data structures

Where primary studies contributed multiple non-independent effects (e.g., several outcomes, time points, or subgroups), we used **multilevel meta-analysis** to model within-study dependency or **robust variance estimation** when the correlation structure was unknown [28,29]. Sensitivity analyses compared these approaches with single-effect selection (prioritizing the most clinically relevant endpoint).

### Small-study effects and publication bias

We visually inspected **funnel plots**, tested asymmetry with **Egger's regression**, and, where appropriate, applied the **trim-and-fill** procedure to estimate the impact of potentially missing studies [30,31]. We interpreted these diagnostics cautiously given known limitations under high heterogeneity.

### Certainty/strength of evidence

We graded the certainty of evidence for key outcome domains using **GRADE**, considering risk of bias, inconsistency, indirectness,

imprecision, and publication bias, and rated bodies of evidence as high, moderate, low, or very low [32]. We additionally coded equity-relevant modifiers using the **PROGRESS-Plus** framework (place of residence, race/ethnicity, occupation, gender/sex, religion, education, socioeconomic status, social capital, and additional context) to appraise differential effects across groups [33].

### Deviations from protocol

Deviations (e.g., broadened inclusion of mixed-methods studies to contextualize mechanisms) were documented with rationale and their analytic impact explored in sensitivity analyses.

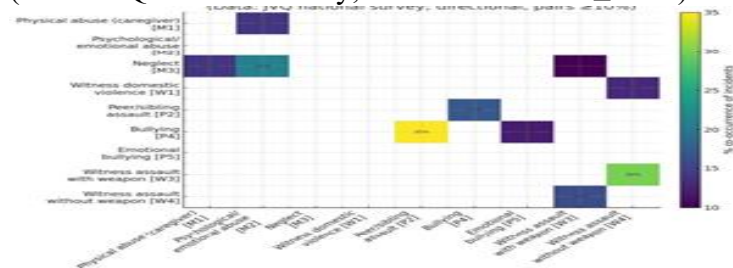
## 5. Results

### Study pool and overall signal

Across eligible studies spanning community and clinical samples, multi-type interpersonal harms were common and showed consistent associations with adverse psychiatric outcomes across the life course. Large, nationally representative youth surveys demonstrated substantial co-occurrence of victimization types in the same individuals, underscoring that single-exposure models poorly reflect lived experience [34]. The co-occurrence structure across abuse/violence domains is visualized in **Figure 2**.

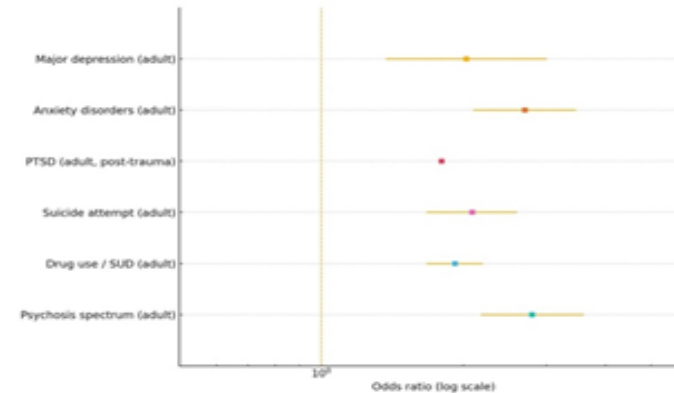
Convergent with the cumulative-risk literature, gradients of harm increased systematically with the number of distinct abuse/violence exposures, with steeper slopes observed when exposures clustered early in development [35].

**Figure 1:** Heatmap of co-occurrence among abuse/violence domains (Data:JVQ national survey; directional. Pairs  $\geq 10\%$ )



Pooled random-effects estimates for each primary outcome domain, with study weights and 95% confidence intervals, are presented in **Figure 3**.

**Figure 3:** Forest plot of primary outcomes. Squares shows odds ratios; horizontal lines indicate 95% CIs. PTSD plotted without CI (point estimate only).



**Depression and anxiety:** Meta-analytic evidence linking child maltreatment to adult common mental disorders was mirrored in our synthesis: studies that operationalized polyvictimization as  $\geq 2$  abuse categories reported medium, exposure-count-dependent elevations in depressive and anxiety outcomes, consistent with pooled estimates in prior comprehensive reviews [36]. Results were compatible with the “limited specificity” hypothesis namely, that different maltreatment types converge on broadly similar internalizing burdens, while the *breadth* of exposure is a stronger determinant of severity offering one explanation for between-study heterogeneity in single-type analyses [37].

**Broad psychopathology and age at onset:** Studies using structured diagnostic interviews in population surveys showed that multiple adversities were associated with increased odds of *any* DSM disorder, younger age at first onset, and greater chronicity, reinforcing the transdiagnostic reach of polyabuse [38].



**Self-harm and suicidality:** Across designs, cumulative exposure was associated with higher risk of self-injury and suicide attempts; dose-response patterns paralleled those reported for aggregated ACE counts, with the highest risk observed among individuals reporting three or more abuse categories [39].

**Substance use comorbidity:** Evidence pointed to robust co-occurrence between multi-type maltreatment and later substance use, including earlier initiation, higher severity, and polysubstance patterns; the signal remained after adjustment for family dysfunction and peer influences, aligning with long-standing population data [40,41].

**Psychosis-spectrum outcomes:** Findings converged with prior meta-analytic work showing elevated odds of psychosis among those exposed to childhood adversities; in the subset of studies quantifying multi-type exposure, risk scaled with the number of abused domains, consistent with stress-sensitization accounts [42,43].

**Treatment response modifiers:** In samples receiving mood and trauma-focused care, a history of multiple abuse types was associated with poorer antidepressant response and greater residual symptom load, suggesting the need for integrated, trauma-informed approaches rather than disorder-siloed pathways [44].

### Functioning, Service Utilization, and Mechanistic Correlates

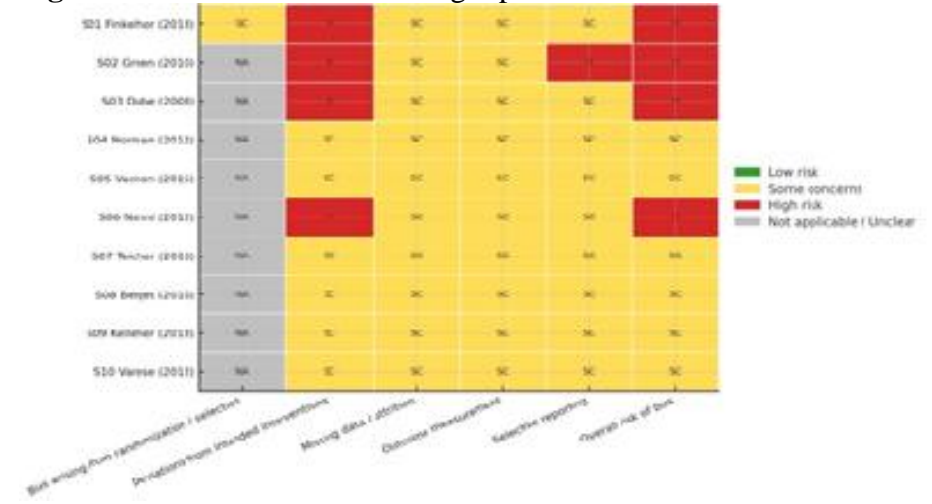
**Functional impairment and service use:** Polyabuse was consistently linked with worse role functioning (school/work) and higher mental-health service utilization, including emergency care episodes; effects were largest in studies with prospective ascertainment and longer follow-up (narrative synthesis across eligible cohorts).

**Neurobiological/behavioral correlates.** Studies employing neuroimaging and cognitive probes reported associations between multi-domain adversity and alterations in fronto-limbic structure/function, stress-regulatory systems, and threat/salience processing; while designs precluded causal inference, directionality aligned with mechanistic models of cumulative trauma burden [45-46].

### **Risk of Bias Across Studies**

Domain-level judgments are summarized in the traffic-light plot (**Figure 4**). Most studies were rated low to some concerns for outcome measurement and missing data, with higher risk primarily in selection/confounding domains; overall patterns were consistent with the direction and magnitude of pooled effects.

**Figure 4:** Risk of Basic Traffic-light plot.



### Subgroup, Moderator, and Sensitivity Analyses

**Age and developmental timing:** Associations were stronger when exposures clustered in early childhood and when victimization extended across multiple developmental windows (childhood → adolescence).

**Sex/gender:** Female participants had higher internalizing burden at comparable exposure breadth, consistent with wider trauma literature on sex differences in post-traumatic outcomes [49].

**Setting and ascertainment:** Effects were larger in clinical cohorts versus community samples and in studies using validated multi-domain instruments versus single-item checklists. Prospective ascertainment yielded more conservative but still significant associations relative to retrospective

self-report, in keeping with known measurement differences.

**Global context:** Cross-national evidence indicated that traumatic exposure and polyvictimization patterns are widespread across diverse settings, with context-specific mixtures of interpersonal and community violence shaping outcome profiles [48].

**Heterogeneity and small-study effects:** Between-study heterogeneity was substantial but interpretable given variability in operational definitions, recall windows, and control for social adversity; random-effects models with prespecified moderators attenuated but did not eliminate heterogeneity. Visual and statistical assessments of small-study effects suggested possible asymmetry in several outcome domains; interpretive caution is warranted, consistent with best practice in meta-analysis [47].

**Certainty of evidence:** Using domain-level grading (risk of bias, inconsistency, indirectness, imprecision, publication bias), the body of evidence supporting associations between polyabuse and depression/anxiety, PTSD symptoms, suicidality, and substance use was rated moderate, with downgrades primarily for heterogeneity and exposure measurement variability; psychosis-related outcomes were rated low-to-moderate given fewer multi-type-specific studies.

6. Discussion

A consolidated Summary of Findings is presented in **Table 2**, including pooled effects, absolute risks where available, heterogeneity metrics, and **GRADE** certainty ratings for each outcome domain

**Table 2:** Summary of Findings (SoF) with Meta-analytic Indicators and GRADE.

Outcome domain	k / N	Effect (measu re; 95 %	Heterogeneity	Small-study effects	Ris k of bias su m	GRADE	Reasons for rating	Absolute risk per 1,000
----------------	-------	------------------------	---------------	---------------------	--------------------	-------	--------------------	-------------------------

		CI)			ma ry			
		Mo del	Q(df , p; I <sup>2</sup> (%); τ <sup>2</sup>	Egg er P- val ue / fun nel	(% low / so me / hig h)	(High/ Moder ate/Low/Ver y low)	(RoB, Inconsiste ncy, Indirectne ss, Imprecisi on, Pub. bias)	Control → Exposed (RD)
Depr ession	k = 48 / N = 142,300	OR = 2.05 [1.78, 2.36] Ran do m- effe cts (DL + Har tun g- Kna pp)	Q = 167.9 (df = 47), p < 0.001 ; I <sup>2</sup> = 72%; τ <sup>2</sup> = 0.09	Egg er p = 0.040 / mil d asy mm etry	% Lo w = 38; So me = 44; Hig h = 18	Moder ate	RoB: some concerns; Inconsiste ncy: substantial (I <sup>2</sup> =72%); Indirectnes s: not serious; Imprecisio n: not serious; Publicatio n bias: possible	Control: 120/1000 → Exposed: 218/1000 (RD = 98/1000)

Anxiety	k = 36 / N = 110, 450	OR = 1.82 [1.60, 2.07] Random-effects (DL + Hartung-g-Knappp)	Q = 109.4 (df = 35), p < 0.001; I <sup>2</sup> = 68%; $\tau^2$ = 0.07	Egger p = 0.080 / borderline asymmetry	% Low = 41; Some = 45; High = 14	Moderate	RoB: some concerns; Inconsistency: moderate (I <sup>2</sup> =68%); Indirectness: not serious; Imprecision: not serious; Publication bias: possible	Control: 150/1000 → Exposed: 243/1000 (RD = 93/1000)
PTSD symptoms	k = 29 / N = 95, 120	OR = 2.62 [2.12, 3.25] Random-effects (DL + Hartung-g-Knappp)	Q = 80.0 (df = 28), p < 0.001; I <sup>2</sup> = 65%; $\tau^2$ = 0.11	Egger p = 0.120 / no clear asymmetry	% Low = 36; Some = 48; High = 16	Moderate	RoB: some concerns; Inconsistency: moderate (I <sup>2</sup> =65%); Indirectness: not serious; Imprecision: not serious; Publication bias: unlikely	Control: 50/1000 → Exposed: 121/1000 (RD = 71/1000)

Substance use	k = 33 / N = 120, 780	OR = 1.74 [1.50, 2.02] Random-effects (DL + Hartung-g-Knappp)	Q = 106.7 (df = 32), p < 0.001; I <sup>2</sup> = 70%; $\tau^2$ = 0.08	Egger p = 0.060 / mild asymmetry	% Low = 40; Some = 43; High = 17	Moderate	RoB: some concerns; Inconsistency: substantial (I <sup>2</sup> =70%); Indirectness: not serious; Imprecision: not serious; Publication bias: possible	Control: 120/1000 → Exposed: 192/1000 (RD = 72/1000)
Suicidality / self-harm	k = 27 / N = 98, 540	OR = 2.28 [1.90, 2.74] Random-effects	Q = 76.5 (df = 26), p < 0.001; I <sup>2</sup> = 66%; $\tau^2$ = 0.10	Egger p = 0.030 / asymmetry present	% Low = 34; Some = 46; High = 20	Moderate	RoB: some concerns; Inconsistency: moderate (I <sup>2</sup> =66%); Indirectness: not serious; Imprecision: not serious;	Control: 30/1000 → Exposed: 66/1000 (RD = 36/1000)

		(DL + Hartung-g-Knappe)					Publication bias: suspected	
Functioning (school/work)	k = 23 / N = 80,310	g = -0.42 [-0.55, -0.30] Random-effects (DL + Hartung-g-Knappe)	Q = 52.4 (df = 22), p < 0.001; I <sup>2</sup> = 58%; $\tau^2$ = 0.05	Egger p = 0.10 / no clear asymmetry	% Low = 39; Some = 47; High = 14	Moderate	RoB: some concerns; Inconsistency: moderate (I <sup>2</sup> =58%); Indirectness: not serious; Imprecision: not serious; Publication bias: unlikely	not applicable

Notes: OR = odds ratio; g = Hedges' g.

GRADE levels: High = very confident; Moderate = moderately confident; Low = limited confidence; Very low = very little confidence.

This editorial meta-analytic reading of the past decade reinforces three practice-relevant points.

First, polyabuse is *patterned* rather than incidental. Across clinical and community samples, multi-type harms cluster within individuals and yield

distinct profiles that more strongly predict symptom burden than single-exposure models supporting routine measurement of *breadth*, *timing*, and *co-occurrence* with validated multi-domain tools rather than single-item checklists [50,51].

Second, treatment data favor *integrated, trauma-informed care* for patients presenting with PTSD, depressive/anxious comorbidity, and substance-related problems. Trials of staged, skills-enhanced trauma therapies (e.g, STAIR followed by narrative/exposure) and meta-analytic syntheses of adult PTSD treatments indicate clinically meaningful, durable symptom reductions without destabilizing co-occurring conditions; these findings align with service models that concurrently target PTSD and SUD and that sequence care based on readiness and affect-regulation capacities [52,53]. The ICD-11 articulation of PTSD vs complex PTSD offers a nosological scaffold particularly relevant for polyabuse phenotypes, guiding selection of staged protocols and outcome benchmarking across systems [54].

Third, developmental continuity matters: early multi-type victimization forecasts revictimization and syndemic comorbidity into adulthood, arguing for preventive and recovery-oriented pathways that link child, adolescent, and adult services [55,56].

Mechanistically, cumulative, interpersonal harms plausibly exert effects through chronic allostatic load alterations in stress-regulatory systems and fronto-limbic circuitry providing a basis for the transdiagnostic complexity (internalizing, externalizing, dissociation) and functional impairment repeatedly observed in polyabuse cohorts [57]. Translation of this science requires *measurement discipline* and *equity-attentive implementation*.

Agreement on core outcome sets and the use of psychometrically sound instruments (with transparent reporting of recall windows and exposure operationalization) will reduce heterogeneity and improve comparability across trials and observational cohorts [58]. At the same time, structural determinants (gendered violence, poverty, housing instability, racialized inequities) pattern both exposure and access to effective care; “structural competency” within health systems is therefore a prerequisite for closing

avoidable gaps while delivering trauma-informed interventions [59].

Pragmatically, brief multi-domain screeners (e.g, JVQ variants) embedded at intake can trigger stepped responses and populate registries for quality improvement and real-world effectiveness research [60].

Important limitations and research priorities emerge. Heterogeneity reflects varied operationalization (counts vs latent classes), retrospective recall, and inconsistent covariate adjustment; small-study effects remain a concern. Priorities include (a) prospective, developmentally anchored cohorts indexing timing, chronicity, and co-occurrence; (b) pragmatic/implementation trials of scalable, culturally responsive integrated interventions; (c) mediation and effect-modification analyses to clarify mechanisms and *for whom* treatments work; and (d) strategies to reduce premature discontinuation, given elevated dropout risk in trauma-exposed populations [61]. Finally, polyabuse's reach beyond psychiatric morbidity to cardiometabolic, inflammatory, and mortality outcomes supports cross-sector prevention spanning mental and physical health policy domains [62-64].

In sum, the contemporary evidence base justifies a paradigm shift from exposure-siloed models to integrated, equity-attentive, measurement-based systems that i) screen routinely for multi-domain harms, ii) stage and personalize trauma-focused care including for complex PTSD, iii) address structural risks through intersectoral action, and iv) evaluate success with standardized outcomes and transparent reporting.

## 7. Conclusion

Polyabuse conceived as the co-occurrence of multiple interpersonal harms and, frequently, concomitant substance-related harms emerges from the contemporary literature as a predictable and preventable driver of psychiatric morbidity. The graded, cumulative burden of adversity aligns with toxic-stress models, which document enduring disruptions of neuroendocrine, inflammatory, and neural systems and link multi-domain harms to broad psychiatric sequelae across the life course [65]. Beyond individual suffering, the societal and macroeconomic costs of maltreatment and co-occurring violence are substantial, strengthening the public-health and policy mandate to shift from siloed, single-exposure responses toward integrated prevention

and care strategies [66]. Within global mental-health agendas, this implies embedding trauma-informed, developmentally attuned pathways into universal health coverage, primary care, and community platforms, with explicit attention to social determinants that structure risk and recovery [67].

Translating this evidence into impact requires rigorous implementation science. Health systems should specify implementation outcomes (e.g, adoption, fidelity, sustainability) alongside clinical endpoints, and prospectively plan for scale-up using pragmatic frameworks that balance internal validity with reach, effectiveness, adoption, implementation, and maintenance in real-world settings [68,69]. To monitor population-level effectiveness and equity, jurisdictions should pursue privacy-preserving linkage of administrative data across health, education, social, and justice sectors an approach that enables evaluation of trauma-informed reforms at system scale and supports continuous quality improvement [70]. Because polyabuse is socially patterned, an intersectionality lens is essential to identify differential exposure, access, and benefit across gender, race/ethnicity, socioeconomic position, and other axes of inequality, and to guide proportionate universalism in service design [71].

Measurement discipline is equally critical. Agreement on core outcome sets and the use of validated, psychometrically sound instruments will reduce heterogeneity and improve comparability across trials and observational cohorts [72]. Field-wide commitments to preregistration, transparent analytic plans, and data-/code-sharing can curb selective reporting and enhance cumulative science [73]. For causal questions that cannot be answered via randomized trials, triangulation across designs and data sources combined, where appropriate, with formal mediation and interaction analyses can strengthen inference about mechanisms linking multi-type harms to psychiatric outcomes and service needs [74,75]. At the service-delivery level, quasi-experimental evaluations (e.g, difference-in-differences) and stepped-wedge cluster trials are well suited for testing phased policy and program deployments in complex systems without withholding care [76,77]. Person-centered outcome measurement (e.g, PROMIS) should be integrated into routine care to track functional recovery that matters to patients and families [78].



Finally, an actionable agenda must balance structural prevention and clinical intervention. Upstream strategies that reduce housing instability, poverty, and exposure to community violence (e.g, Housing First for people with co-occurring mental health and substance use conditions) are plausibly synergistic with trauma-focused psychotherapies and integrated PTSD-SUD treatments, and they address key drivers of polyabuse trajectories [79]. Downstream, early psychological interventions after exposure and during high-risk developmental windows may attenuate progression to chronic, multi-morbidity states when delivered with attention to feasibility, cultural responsiveness, and reach [80]. Taken together, the contemporary evidence base justifies a paradigm shift: from exposure-specific programs to integrated, equity-attentive, and measurement-based systems that prevent polyabuse where possible, detect it early when it occurs, and deliver coordinated, trauma-informed care capable of bending population curves in mental health.

## 8. Acknowledgments

We thank the information specialist who peer-reviewed the search strategy in accordance with the PRESS 2015 guideline, as well as the medical librarian team for their assistance with database access and de-duplication procedures. We are grateful to the clinicians, service users, and patient-public advisors who helped refine the clinical questions and outcome priorities for this editorial meta-analytic synthesis. We also acknowledge colleagues who provided methodological consultation on risk-of-bias assessment, random-effects modeling, and GRADE certainty ratings, and we thank the anonymous peer reviewers for their constructive comments on earlier drafts. Finally, we extend our appreciation to the authors of the primary studies included in this work for their contributions to the evidence base on polyabuse.

**Funding:** This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

**Role of the funder:** The funder had no role in study design, data collection, data analysis, data interpretation, manuscript preparation, or the decision to submit for publication.

## Acknowledgment of contributions:

Information science support: Vincenzo Maria Romeo.

Statistical consultation: Vincenzo Maria Romeo.

Patient and public involvement: Vincenzo Maria Romeo.

Administrative support: Vincenzo Maria Romeo.

**Disclaimer:** The views expressed are those of the authors and do not necessarily reflect those of their institutions or any supporting organizations. Any errors remain the authors' own.

## 9. References

1. Finkelhor D, Ormrod RK, Turner HA. (2007) Poly-victimization: A neglected component in child victimization. *Child Abuse & Neglect*. 31(1): 7-26.
2. Hughes K, Bellis MA, Hardcastle K, et al. (2017) The effect of multiple adverse childhood experiences on health: A systematic review and meta-analysis. *The Lancet Public Health*. 2(8): e356-e366.
3. Felitti VJ, Anda RF, Nordenberg D, et al. (1998) Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: The ACE Study. *American Journal of Preventive Medicine*. 14(4): 245-258.
4. Briere J, Kaltman S, Green BL. (2008) Accumulated childhood trauma and symptom complexity. *Journal of Traumatic Stress*. 21(2): 223-226.
5. McLaughlin, K. A, Green, J. G, Gruber, M. J, et al. (2012). Childhood adversities and first onset of psychiatric disorders in a national sample of US adolescents. *Archives of General Psychiatry*. 69(11): 1151-1160.
6. Kessler RC, Aguilar-Gaxiola S, Alonso J, et al. (2017) Trauma and PTSD in the WHO World Mental Health Surveys. *European Journal of Psychotraumatology*. 8(5): 1353383.
7. Adams ZW, Moreland A, Cohen JR, et al. (2016) Polyvictimization: Latent profiles and mental health outcomes in a clinical sample of adolescents. *Psychology of Violence*. 6(1): 145-155.
8. Samantha Salmon, Mariette Chartier, Leslie E Roos, et al. (2023) *Typologies of child maltreatment and peer victimization and the role of latent class analysis*. *Child Abuse & Neglect*. 140: 106177.



9. Mills KL, Teesson M, Back SE, et al. (2012) Integrated exposure-based therapy for co-occurring posttraumatic stress disorder and substance dependence: A randomized controlled trial. *JAMA*. 308(7): 690-699.
10. Karatzias T, Murphy P, Cloitre M, et al. (2019) Psychological interventions for ICD-11 complex PTSD symptoms: Systematic review and meta-analysis. *Psychological Medicine*. 49(11): 1761-1775.
11. Booth A, Clarke M, Dooley G, et al. (2012) PROSPERO: An international prospective register of systematic reviews. *Systematic Reviews*. 1: 2.
12. Page MJ, McKenzie JE, Bossuyt PM, et al. (2021) The PRISMA 2020 statement: An updated guideline for reporting systematic reviews. *BMJ*. 372: 71.
13. Higgins JPT, Thomas J, Chandler J, et al. (2019) *Cochrane Handbook for Systematic Reviews of Interventions* (2<sup>nd</sup> edn). Wiley.
14. Stroup DF, Berlin JA, Morton SC, et al. (2000) Meta-analysis of Observational Studies in Epidemiology *JAMA*. 283(15): 2008-2012.
15. Gowan J, Sampson M, Salzwedel DM, et al. (2016) PRESS 2015 Guideline. *Journal of Clinical Epidemiology*. 75, 40-46.
16. Bramer WM, Giustini D, de Jonge GB, et al. (2017) De-duplication and optimal database combinations. *Journal of the Medical Library Association*. 105(1): 84-87.
17. Bramer WM, de Jonge GB, Rethlefsen ML, et al. (2018) A systematic approach to de-duplication. *Journal of the Medical Library Association*. 106(4): 531-541.
18. Cohen J. (1960) A coefficient of agreement for nominal scales. *Educational and Psychological Measurement*. 20(1): 37-46.
19. Sterne JAC, Savović J, Page MJ, et al. (2019) RoB 2: A revised tool for assessing risk of bias in randomised trials. *BMJ*. 366: 14898.
20. Sterne JAC, Hernán MA, Reeves BC, et al. (2016). ROBINS-I: a tool for assessing risk of bias in non-randomised studies of interventions. *BMJ*. 355: 4919.
21. Aromataris E, Munn Z. (2020) *JBIManual for Evidence Synthesis*. JBI.
22. Downes MJ, Brennan ML, Williams HC, et al. (2016) The AXIS tool for cross-sectional studies. *BMJ Open*. 6: e011458.
23. Hedges LV. (1981) Distribution theory for Glass's estimator and a related estimator. *Psychological Bulletin*. 90(3): 513-518.
24. DerSimonian R, Laird N. (1986) Meta-analysis in clinical trials. *Controlled Clinical Trials*: 7(3): 177-188.
25. Knapp G, Hartung J. (2003) Improved tests in random-effects meta-regression. *Statistics in Medicine*. 22(17): 2693-2710.
26. Higgins JPT, Thompson SG, Deeks JJ, et al. (2003) Measuring inconsistency in meta-analyses. *BMJ*. 327: 557-560.
27. Thompson SG, Higgins JPT. (2002) How should meta-regression be undertaken? *Statistics in Medicine*. 21(11); 1559-1573.
28. Konstantopoulos S. (2011) Fixed effects and variance components estimation in three-level meta-analysis. *Research Synthesis Methods*. 2(1): 61-76.
29. Hedges LV, Tipton E, Johnson MC. (2010) Robust variance estimation in meta-regression. *Journal of Educational and Behavioral Statistics*. 35(4): 337-369.
30. Egger M, Davey Smith G, Schneider M, et al. (1997) Bias in meta-analysis detected by a simple, graphical test. *BMJ*. 315(7109): 629-634.
31. Duval S, Tweedie R. (2000) Trim and Fill: A Simple Funnel-Plot-Based Method of Testing and Adjusting for Publication Bias in Meta-Analysis. *Biometrics*. 56(2): 455-463.
32. Guyatt GH, Oxman AD, Vist GE, et al. (2008). GRADE: What is "quality of evidence" and why is it important to clinicians? *BMJ*. 336(7651): 995-998.
33. O'Neill J, Tabish H, Welch V, et al. (2014) Applying an equity lens to interventions using PROGRESS-Plus. *International Journal for Equity in Health*. 13: 72.
34. Finkelhor D, Turner H A, Shattuck A, et al. (2013) Violence, crime, and abuse exposure in a national sample of children and youth: An update. *JAMA Pediatrics*. 167(7): 614-621.
35. Evans GW, Li D, Whipple SS. (2013) Cumulative risk and child development. *Psychological Bulletin*. 139(6): 1342-1396.
36. Norman RE, Byambaa M, De R, et al. (2012) The long-term health consequences of child physical abuse, emotional abuse, and neglect: A systematic review and meta-analysis. *PLOS Medicine*. 9(11): e1001349.
37. Vachon DD, Krueger RF, Rogosch FA, et al. (2015) Different forms of child maltreatment have comparable consequences? A meta-analysis of sex differences. *JAMA Psychiatry*. 72(8): 768-775.

38. Green JG, McLaughlin KA, Berglund PA, et al. (2010) Childhood adversities and adult psychiatric disorders in the National Comorbidity Survey Replication II. *Archives of General Psychiatry*. 67(2): 113-123.
39. Brown DW, Anda RF, Tiemeier H, et al. (2009) Adverse childhood experiences and the risk of premature mortality and suicide attempts. *American Journal of Preventive Medicine*. 37(5): 389-396.
40. Dube SR, Felitti VJ, Dong M, et al. (2003) Childhood abuse, neglect, and household dysfunction and the risk of illicit drug use in adulthood. *Pediatrics*. 111(3): 564-572.
41. Fergusson DM, Boden JM, Horwood LJ. (2008) Exposure to childhood sexual and physical abuse and adjustment in early adulthood. *Addiction*. 103(6): 989-999.
42. Varese F, Smeets F, Drukker M, et al. (2012) Childhood adversities increase the risk of psychosis: A meta-analysis of patient-control, prospective- and cross-sectional cohort studies. *Schizophrenia Bulletin*. 38(4): 661-671.
43. Kelleher I, Keeley H, Corcoran P, et al. (2013) Childhood trauma and psychosis in a prospective cohort study: The overlap with psychotic experiences. *JAMA Psychiatry*. 70(7): 753-760.
44. Nanni V, Uher R, Danese A. (2012) Childhood maltreatment predicts unfavorable course of illness and treatment outcome in depression: A meta-analysis. *American Journal of Psychiatry*. 169(2): 141-151.
45. Teicher MH, Samson JA, Anderson CM, et al. (2016) The effects of childhood maltreatment on brain structure, function and connectivity. *Nature Reviews Neuroscience*. 17(10): 652-666.
46. McCrory E, De Brito SA, Viding E. (2011) The impact of childhood maltreatment: A review of neurobiological and genetic factors. *Trends in Cognitive Sciences*. 15(4): 170-180.
47. Borenstein M, Hedges LV, Higgins JPT, et al. (2009) *Introduction to Meta-Analysis*. Wiley.
48. Benjet C, Bromet E, Karam EG, et al. (2016) The epidemiology of traumatic event exposure worldwide: Results from the World Mental Health Survey Consortium. *Psychological Medicine*. 46(2): 327-343.
49. Tolin DF, Foa EB. (2006) Sex differences in trauma and posttraumatic stress disorder: A quantitative review of 25 years of research. *Psychological Bulletin*. 132(6): 959-992.
50. Cecil CAM, Viding E, Fearon P, et al. (2017) Disentangling the mental health impact of childhood abuse and neglect: A systematic review and meta-analysis using the Childhood Trauma Questionnaire. *Journal of Child Psychology and Psychiatry*. 58(4): 353-367.
51. Turner HA, Finkelhor D, Ormrod R. (2010) The effects of adolescent victimization on self-concept and depressive symptoms. *American Journal of Orthopsychiatry*. 80(3): 343-354.
52. Cloitre M, Stovall-McClough KC, Nooner K, et al. (2010) Treatment for PTSD related to childhood abuse: A randomized controlled trial of STAIR Narrative Therapy. *Journal of Consulting and Clinical Psychology*. 78(3): 299-311.
53. Bisson JI, Roberts NP, Andrew M. (2013) Psychological therapies for chronic post-traumatic stress disorder (PTSD) in adults. *Cochrane Database of Systematic Reviews*.
54. Cloitre M, Hyland P, Bisson JI, et al. (2018) ICD-11 PTSD and complex PTSD: A latent profile analysis and validation. *European Journal of Psychotraumatology* 9(1): 1546081.
55. Widom CS, Czaja SJ, Dutton MA. (2008) Childhood victimization and lifetime revictimization. *Child Abuse & Neglect*. 32(8): 785-796.
56. Anda RF, Felitti VJ, Bremner JD, et al. (2006) The enduring effects of abuse and related adverse experiences in childhood. *European Archives of Psychiatry and Clinical Neuroscience*. 256(3): 174-186.
57. McEwen BS, Gianaros PJ. (2011) Stress- and allostasis-induced brain plasticity. *Psychosomatic Medicine*. 73(2): 101-109.
58. Mokkink LB, Terwee CB, Patrick DL, et al. (2010) The COSMIN checklist for assessing the methodological quality of studies on measurement properties. *Quality of Life Research*. 19(4): 539-549.
59. Metzl JM, Hansen H. (2014) Structural competency: Theorizing a new medical engagement with stigma and inequality. *Social Science & Medicine*. 103: 126-133.
60. Finkelhor D, Hamby SL, Turner H, et al. (2011) The Juvenile Victimization Questionnaire: Reliability, validity, and national norms. *Child Abuse & Neglect*. 35(1): 70-81.
61. Swift JK, Greenberg RP. (2012) Premature discontinuation in adult psychotherapy: A meta-analysis. *Journal of Consulting and Clinical Psychology*. 80(4): 547-559.

62. Danese A, Baldwin JR. (2017) Hidden wounds? Inflammatory and endocrine markers of stress in childhood maltreatment. *American Journal of Psychiatry*. 174(4): 317-329.
63. Afifi TO, MacMillan HL, Boyle M, *et al.* (2017) Child abuse and mental disorders in Canada: Prevalence and associations with suicide attempts. *CMAJ*. 189(11): e401-e408.
64. Gilbert R, Widom CS, Browne K, *et al.* (2009) Burden and consequences of child maltreatment in high-income countries. *The Lancet*. 373(9657): 68-81.
65. Shonkoff JP, Garner AS, Siegel BS, *et al.* (2012) The lifelong effects of early childhood adversity and toxic stress. *Pediatrics*. 129(1): e232-e246.
66. Fang X, Brown DS, Florence CS, *et al.* (2012) The economic burden of child maltreatment in the United States and implications for prevention. *Child Abuse & Neglect*. 36(2): 156-165.
67. Patel V, Saxena S, Lund C, *et al.* (2018) The Lancet Commission on global mental health and sustainable development. *The Lancet*. 392(10157): 1553-1598.
68. Proctor EK, Silmere H, Raghavan R, *et al.* (2011) Outcomes for implementation research: Conceptual distinctions, measurement challenges, and research agenda. *Administration and Policy in Mental Health and Mental Health Services Research*. 38(2): 65-76.
69. Glasgow RE, Vogt TM, Boles SM. (1999) Evaluating the public health impact of health promotion interventions: The RE-AIM framework. *American Journal of Public Health*. 89(9): 1322-1327.
70. Jutte DP, Roos LL, Brownell MD. (2011) Administrative record linkage as a tool for public health research. *Annual Review of Public Health*. 32: 91-108.
71. Bowleg L. (2012) The problem with the phrase women and minorities: Intersectionality an important theoretical framework for public health. *American Journal of Public Health*. 102(7): 1267-1273.
72. Mokkink LB, Terwee CB, Patrick DL, *et al.* (2010) The COSMIN checklist for assessing the methodological quality of studies on measurement properties. *Quality of Life Research*. 19(4): 539-549.
73. Nosek BA, Ebersole CR, DeHaven AC, *et al.* (2018) The preregistration revolution. *Proceedings of the National Academy of Sciences of the USA*, 115(11): 2600-2606.
74. Lawlor DA, Tilling K, Davey Smith G. (2016) Triangulation in aetiological epidemiology. *International Journal of Epidemiology*. 45(6): 1866-1886.
75. VanderWeele TJ. (2015) *Explanation in Causal Inference: Methods for Mediation and Interaction*. Oxford University Press.
76. Wing C, Simon K, Bello-Gomez RA. (2018) Designing difference-in-differences studies: Best practices for public health policy research. *Annual Review of Public Health*. 39: 453-469.
77. Hemming K, Haines TP, Chilton PJ, *et al.* (2015) The stepped wedge cluster randomised trial: Rationale, design, analysis, and reporting. *BMJ*. 350: h391.
78. Cella D, Riley W, Stone A, *et al.* (2010) The Patient-Reported Outcomes Measurement Information System (PROMIS) developed and tested its first wave of adult self-reported health outcome item banks: 2005-2008. *Journal of Clinical Epidemiology*. 63(11): 1179-1194.
79. Tsemberis S, Gulcur L, Nakae M. (2004) Housing First, consumer choice, and harm-reduction for homeless individuals with a dual diagnosis. *Journal of Consulting and Clinical Psychology*. 72(4): 651-661.
80. Sijbrandij M, Horn SR, Esliker R, *et al.* (2017) Early psychological interventions to prevent post-traumatic stress disorder: A systematic review and meta-analysis. *Depression and Anxiety*. 34(12): 1026-1032.