Journal of Psychiatric Research 197 (2026) 275–288

Contents lists available at ScienceDirect

Journal of Psychiatric Research

journal homepage: www.elsevier.com/locate/jpsychires

The risk of schizophrenia or psychosis following traumatic brain injury: A systematic review and meta-analysis

Ting-Yi Chu [a,*], Chung-Che Wu [b,c,d,e], Zi-Xiang Xu [f], Yung-Hsiao Chiang [b,c,d,e], Shih-Chang Hsueh [g,h], Ju-Chi Ou [b,**], Kai-Yun Chen [d,i,j,***]

[a] Department of Medicine, College of Medicine, Taipei Medical University, Taipei, Taiwan
[b] Department of Surgery, School of Medicine, College of Medicine, Taipei Medical University, Taipei, Taiwan
[c] Department of Neurosurgery, Taipei Medical University Hospital, Taipei, Taiwan
[d] Research Center for Neuroscience, Taipei Medical University, Taipei, Taiwan
[e] Taipei Neuroscience Institute, Taipei Medical University, Taipei, Taiwan
[f] Family Medicine Division, Department of Community Medicine, Landseed International Hospital, Taoyuan, Taiwan
[g] Department of Pediatrics, Columbia University Irving Medical Center, New York, NY, USA
[h] Vagelos College of Physicians and Surgeons, Columbia University, New York, NY, USA
[i] PhD Program in Medical Neuroscience, Taipei Medical University, Taipei, Taiwan
[j] International Master Program in Medical Neuroscience, College of Medical Science and Technology, Taipei Medical University, Taipei, Taiwan

ARTICLE INFO

Keywords:
Neurotrauma
Psychiatric outcomes
Mental health disorders
Neuropsychiatric complications
Cognitive impairment
Long-term follow-up
Delayed-onset psychosis

ABSTRACT

Background: Traumatic brain injury (TBI) has been linked to an elevated risk of developing schizophrenia or psychosis, yet the extent of this risk and the factors that may modify it remain unclear. This meta-analysis aimed to quantify the overall risk and examine the influence of demographic and clinical variables.

Methods: A systematic review and meta-analysis were conducted following PRISMA 2020 guidelines. Databases including PubMed, EMBASE, and Cochrane Library were searched for studies reporting the incidence of schizophrenia or psychosis following TBI. Pooled odds ratios (ORs) and hazard ratios (HRs) were calculated using random-effects models. Subgroup analyses examined the effects of sex, age, TBI severity, and follow-up duration.

Results: Eleven studies met inclusion criteria. The pooled OR was 3.07 (95% CI: 1.95–4.84) and the pooled HR was 3.55 (95% CI: 2.17–5.80), confirming a significant association between TBI and psychosis. Subgroup analyses revealed elevated risks across sex and age groups, with particularly high risk among adults and individuals with moderate-to-severe TBI. Longer follow-up durations were associated with greater risk, suggesting a delayed onset pattern.

Conclusions: TBI significantly increases the risk of schizophrenia or psychosis, especially in adults and those with severe injuries. These findings underscore the need for long-term psychiatric monitoring in individuals with a history of TBI.

1. Introduction

Traumatic brain injury (TBI) has long been recognized as a significant public health issue, contributing to considerable neurological and psychiatric morbidity worldwide (Capizzi et al., 2020). Beyond its acute consequences, TBI is closely linked with various long-term mental health outcomes, such as depression, post-traumatic stress disorder (PTSD), and mood disorders, which profoundly affect patients' quality of life and functioning (Topolovec-Vranic et al., 2017; Xiong et al., 2019).

While the neuropsychiatric sequelae of TBI have received growing attention, psychosis—particularly schizophrenia spectrum disorders—remains a relatively understudied yet clinically significant outcome. Understanding the potential link between TBI and the onset of psychotic disorders is critical not only for early identification and intervention, but also for informing neurobiological models of psychosis.

Psychosis is characterized by an impaired perception of reality and

* Corresponding author.
** Corresponding author.
*** Corresponding author.
E-mail addresses: b101108027@tmu.edu.tw (T.-Y. Chu), juchi@tmu.edu.tw (J.-C. Ou), kychen08@tmu.edu.tw (K.-Y. Chen).

https://doi.org/10.1016/j.jpsychires.2026.03.005
Received 11 April 2025; Received in revised form 2 March 2026; Accepted 3 March 2026
Available online 6 March 2026

formal thought disorder and includes hallucinations, delusions, and cognitive impairment as core symptoms (American Psychiatric Association, 2013; Arciniegas, 2015). Schizophrenia, a chronic and severe mental disorder, is categorized under the broader spectrum of psychosis (Van Os and Kapur, 2009). The lifetime prevalence of psychosis is 9.57 per 1000 individuals (Moreno-Küstner et al., 2018). Although classified as a low-prevalence disease, psychosis is the prominent cause of years lived with disability in the global population (Vigo et al., 2016). According to a mental health report by the World Health Organization (2022), schizophrenia alone accounted for disability in 24 million individuals globally, and considerably influenced patients' academic and occupational performance. It also affects their quality of life and increases their social and economic burden (Vos et al., 2017). Its impact extends beyond neuropsychiatry, contributing to increased comorbidity and mortality across multiple systems, including type 2 diabetes mellitus (T2DM), chronic obstructive pulmonary disease (COPD), pneumonia, cardiovascular diseases, and Parkinsonism (Schoepf et al., 2014). Among these outcomes, psychosis has received relatively less attention despite its significant impact.

1.1. Potential association between TBI and schizophrenia or psychosis

In the acute phase, TBI induces diffuse axonal injury (DAI) and demyelination within white matter tracts due to direct impact, rotational acceleration, and shearing forces. These immediate insults disrupt cerebral cellular functions and compromise neuronal integrity, setting the stage for downstream neurodegenerative processes (Bramlett and Dietrich, 2015; Capizzi et al., 2020).

In the subacute to medium-term phase, progressive brain damage—particularly in the temporal and parietal lobes—has been associated with deficits in cognition, emotional processing, and semantic comprehension (Duarte et al., 2023; Rossell et al., 2010; Sachdev et al., 2001). These impairments may be further exacerbated by cortical thinning in prefrontal regions, which contributes to executive dysfunction and behavioral disinhibition (Hellstrøm et al., 2017). The cumulative impact of these alterations may increase vulnerability to the onset of schizophrenia or other psychotic disorders.

In the chronic phase, elevated Tau protein accumulation has been positively correlated with the emergence of late-onset neuropsychiatric symptoms post-TBI (Takahata et al., 2019). Structural brain changes, such as hippocampal atrophy, may further disrupt dopaminergic pathways—a core mechanism implicated in schizophrenia pathophysiology (Bray et al., 2021). These long-term neurobiological consequences may ultimately contribute to progressive cognitive decline and elevate the risk for chronic psychosis. These findings may provide a pathophysiological framework for understanding TBI as a potential risk factor for psychotic disorders.

1.2. Rationale

Although previous meta-analyses have identified a significant association between TBI and subsequent psychotic disorders, including schizophrenia, the precise relationship remains inconclusive due to methodological limitations and inconsistent findings across studies. A 2011 meta-analysis reported an increased risk of schizophrenia following TBI (odds ratio [OR] = 1.65; 95% CI = 1.17–2.32), but highlighted significant heterogeneity in the included studies (Molloy et al., 2011). A more recent meta-analysis also found a pooled OR of 1.80 (95% CI = 1.11–2.95) for psychosis outcomes, yet failed to establish a clear dose-response relationship between TBI severity and psychosis onset (Yau et al., 2024). In addition, sex differences in post-TBI outcomes remain underexplored: males appear to have a higher risk for schizophrenia, whereas females are more susceptible to epilepsy and suicidality (Cancelliere et al., 2016; Nielsen et al., 2002). These gaps underscore the need for an updated and methodologically rigorous meta-analysis focusing on the risk of schizophrenia or psychosis after TBI, stratified by key clinical characteristics. This study builds on existing literature by conducting a more stratified and rigorously assessed analysis.

1.3. Research objectives

This study aimed to conduct a systematic review and meta-analysis to evaluate the risk of schizophrenia or psychosis among individuals with a history of TBI compared to those without TBI exposure. Additionally, we investigated potential moderators of this association, including sex, age, TBI severity, and follow-up duration.

2. Methods

2.1. Study design

This systematic review and meta-analysis was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines to ensure methodological rigor in identification, screening, data extraction, and synthesis (Page et al., 2021).

2.2. Data sources and search strategy

We systematically searched PubMed, EMBASE, and the Cochrane Library databases on August 12, 2024, using the following Boolean logic: "(((((((((TBI) OR (traumatic brain injury)) OR (brain injury)) OR (head injury)) OR (cerebral trauma)) OR (craniocerebral injury)) OR (concussion)) OR (skull fracture))) AND ((((psychosis) OR (schizophrenia)) OR (psychotic)) OR (delusion))."

In addition to the database search, we performed a manual search of reference lists from included studies, identifying 11 additional relevant articles for full-text review and eligibility assessment, as shown in Fig. 1, which presents the PRISMA flow diagram (Chen et al., 2011; Fann et al., 2004; Helgeland and Torgersen, 2005; Ledoux et al., 2022; Lopez et al., 2022; Malaspina et al., 2001; Massagli et al., 2004; Orlovska et al., 2014; Sachdev et al., 2001; Timonen et al., 2002; Trivedi et al., 2024).

2.3. Eligible criteria

Inclusion Criteria. Studies were included if they met all of the following criteria.

a) Study design: randomized controlled trials, retrospective, prospective, longitudinal, and cross-sectional cohort studies, or case–control studies.
b) Population: individuals of any age or sex diagnosed with TBI.
c) Outcome: studies that compared the incidence or prevalence of schizophrenia, psychosis, or related disorders between TBI and a non-TBI control group, allowing the estimation of relative risk (RR), OR, or hazard ratio (HR). Studies that reported psychosis outcomes in TBI populations without a comparison group were included for qualitative synthesis only.
d) Publication: peer-reviewed articles published in English between January 1, 1945 and August 12, 2024.

Exclusion Criteria. Studies were excluded if they met any of the following conditions.

a) Non-human studies.
b) Reviews, editorials, book chapters, or conference abstracts without original data.
c) Non-peer-reviewed publications.
d) Studies that focused solely on therapeutic interventions without reporting outcome incidence.

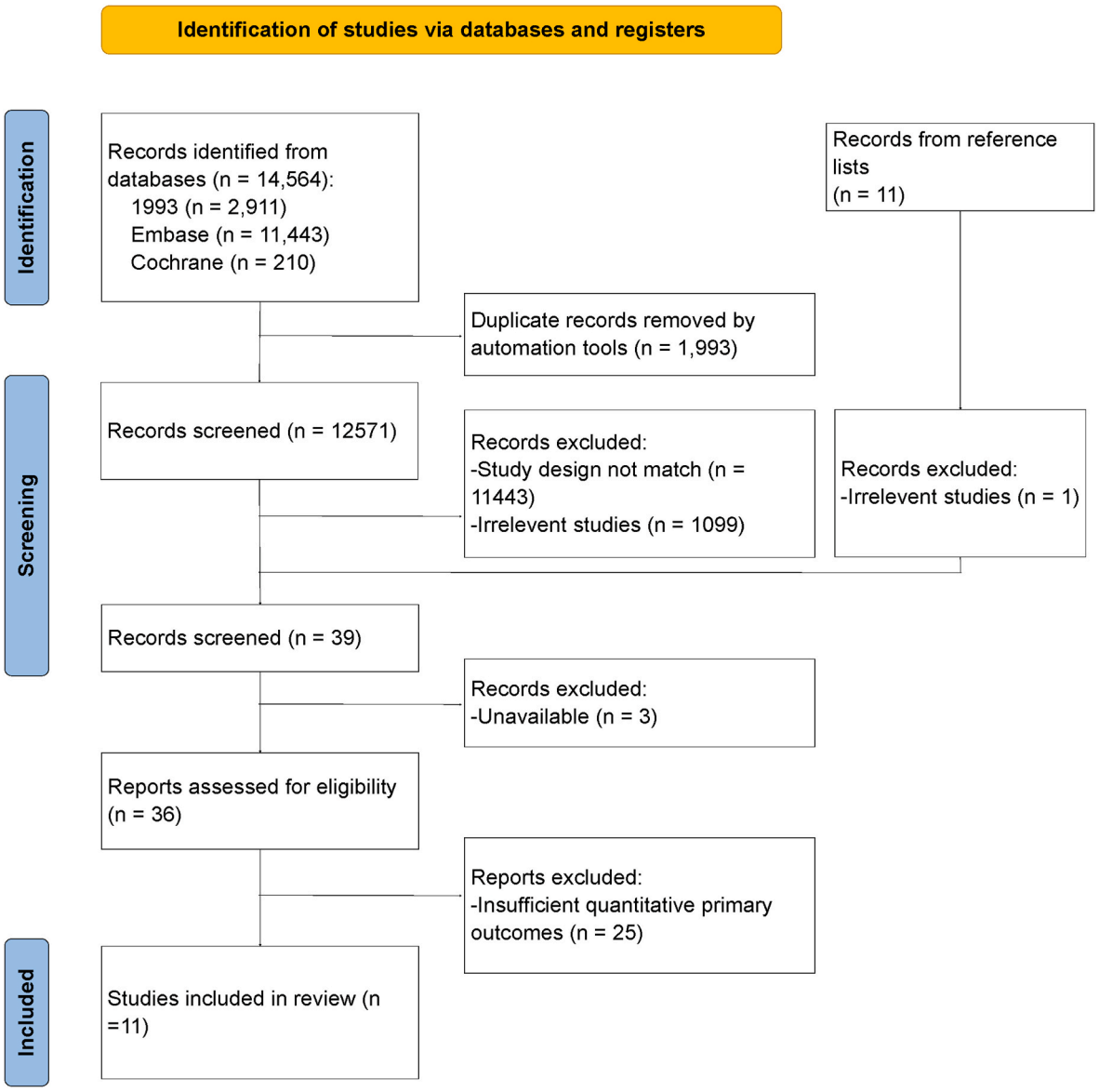


Fig. 1. PRISMA flow diagram for study selection.

e) Studies lacking sufficient quantitative outcome data (e.g., number of psychosis cases, total cohort size, or incidence/prevalence rates).
f) Studies without a non-TBI comparison group, or without sufficient statistical data to calculate risk estimates.

2.4. Study selection

Titles and abstracts were screened for relevance, duplicates were removed using EndNote, and full-text reviews were conducted based on inclusion and exclusion criteria.

2.5. Data extraction

One reviewer extracted data using a standardized Excel sheet, which included general study characteristics (first author, publication year, country, and study design), participant information (sample size, age, sex), and outcome measures (number of psychosis or schizophrenia cases, total cohort size, odds ratio, hazard ratio, and follow-up duration).

Additional data were collected regarding the diagnostic criteria or sources used to define psychosis and TBI, as well as the classification of TBI severity (mild, moderate, or severe), with concussion treated as a form of mild TBI (Kushner, 1998). A second reviewer cross-checked a random subset of the extracted data to ensure consistency and minimize bias.

2.6. Quality assessment

Two reviewers independently evaluated study quality using the Newcastle-Ottawa Scale (NOS) for cohort and case-control studies. The NOS assesses selection, comparability, and outcome/exposure domains, considering factors such as the representativeness of study groups, ascertainment of exposure and outcome, and adequacy of follow-up. The maximum possible score was adapted based on study design (see Results for details), with a total score ranging from 0 to 9, where higher scores indicate lower risk of bias.

To assess consistency between raters, a random subset of studies was independently re-evaluated. Inter-rater reliability was calculated using the Intraclass Correlation Coefficient (ICC) and Light's Kappa.

2.7. Data synthesis

We conducted both quantitative and qualitative syntheses. For the

meta-analysis, we included studies with a non-TBI comparison group that provided sufficient statistical data to calculate pooled effect estimates (e.g., OR, RR, or HR). Studies that reported psychosis outcomes in TBI populations but lacked a comparison group were included in the qualitative synthesis only, and were narratively summarized to provide clinical context.

2.8. Statistical analysis and heterogeneity assessment

All statistical analyses were conducted using R version 4.4.1. The primary meta-analysis estimated the pooled association between TBI and the subsequent development of schizophrenia or psychosis. Data included the number of cases, total sample sizes in the TBI and non-TBI groups, and reported effect measures (i.e., OR and HR).

Random-effects models were applied to account for between-study variability. Where stratified estimates were available (e.g., mild vs. moderate-to-severe TBI), they were treated as independent data points to preserve subgroup granularity. Heterogeneity was assessed using the Chi-square tests (χ^2), τ^2, and I^2, statistics to evaluate the magnitude and significance of between-study variance.

Subgroup analyses were conducted for studies reporting stratified data, with pooled estimates compared across sex, age groups, TBI severity, and follow-up duration. Consistent with most included studies, TBI severity was categorized as mild versus moderate-to-severe, since moderate and severe cases were often grouped together. Differences between subgroups were tested using the Chi-square test, with statistical significance defined as $p < .05$. Publication bias was assessed using funnel plots, Egger's test, Trim-and-Fill analysis, and leave-one-out sensitivity testing.

3. Results

3.1. Study selection

Of the 14,564 records initially identified from database searches, 1993 duplicates were removed. After screening the titles and abstracts of the remaining 12,571 records, 12,542 were excluded due to irrelevant topics or incompatible designs. An additional 11 records were identified through manual reference list screening, among which one was excluded for irrelevance. This resulted in 39 full-text articles assessed for eligibility. Of these, 3 were excluded due to unavailability of the full text, and 25 were excluded due to insufficient outcome data. Specifically, 8 studies primarily assessed TBI history among individuals with established psychiatric diagnoses rather than examining psychosis outcomes following TBI (AbdelMalik et al., 2003; Cheng et al., 2024; Deighton et al., 2016; Harrison et al., 2006; Malaspina et al., 2001; Nielsen et al., 2002; Sachdev et al., 2001; Wilcox and Nasrallah, 1987).

Ultimately, 11 studies were included in the systematic review, of which 8 were eligible for quantitative synthesis (Chen et al., 2011; Chin and Zeber, 2020; Fann et al., 2004; Izzy et al., 2021, 2022; Miller et al., 2015; Russell et al., 2023; Trivedi et al., 2024), and 3 studies without non-TBI comparison groups were narratively summarized in the qualitative synthesis (Nuhu and Yusuf, 2012; Orlovska et al., 2014; Whelan-Goodinson et al., 2009). Fig. 1 shows the PRISMA flow diagram. Supplementary Table 1 presents the PRISMA checklist.

3.2. Study characteristics

A total of 11 studies were included in the systematic review, encompassing diverse study designs (7 retrospective cohorts, 3 prospective cohorts, and 1 cross-sectional study) and various geographical locations, including the United States, Taiwan, Nigeria, Denmark, Canada, and Australia (see Table 1). Sample sizes varied substantially, ranging from 75 to over 113,000 participants. Two studies involved relatively small samples (Nuhu and Yusuf, 2012; Whelan-Goodinson et al., 2009), and three lacked a non-TBI control group, precluding their inclusion in the meta-analysis (Nuhu and Yusuf, 2012; Orlovska et al., 2014; Whelan-Goodinson et al., 2009).

Additionally, three studies contributed adjusted effect estimates (OR HR) and were thus included in the meta-analysis; however, they did not report the number of psychosis cases in the TBI or comparison groups (Chin and Zeber, 2020; Izzy et al., 2022; Miller et al., 2015). This lack of raw event data constrained further secondary analyses, such as leave-one-out procedures or direct comparisons of event rates.

Most studies relied on ICD diagnostic codes (ICD-9, ICD-10, or both) to identify psychotic outcomes. Some studies supplemented diagnostic classification with DSM criteria or structured clinical interviews.

TBI exposure was typically defined based on medical records, though the criteria for severity classification varied across studies. Concussion was considered a form of mild TBI, consistent with established classification systems (Kushner, 1998), and was operationalized as such in several studies. Other studies adopted broader definitions, utilizing criteria such as the Glasgow Coma Scale or CDC guidelines (Chen et al., 2011; Izzy et al., 2021, 2022; Miller et al., 2015).

Overall, the included studies demonstrated considerable variability in design, diagnostic criteria, TBI severity definitions, and reporting completeness. The characteristics of the included studies are summarized in Table 1.

3.3. Quality assessment

The maximum possible score on the Newcastle–Ottawa Scale (NOS) was adapted according to each study's design. Among the 11 included studies, three lacked a non-TBI control group (Nuhu and Yusuf, 2012; Orlovska et al., 2014; Whelan-Goodinson et al., 2009), rendering the items "selection of non-exposed cohort" and "comparability" not applicable. One study was cross-sectional in design, and thus "sufficient length of follow-up" and "adequacy of follow-up" could not be assessed (Trivedi et al., 2024).

Additionally, two studies focused on military cohorts, which may limit generalizability due to their distinct demographic and environmental characteristics (Chin and Zeber, 2020; Miller et al., 2015). One study reported a notably unbalanced sex distribution between groups, with the TBI group consisting of 1522 males (73.1%) and the non-TBI control group of 5198 males (49.9%) (Russell et al., 2023).

According to NOS scoring criteria, several studies did not receive a point in the "adequacy of follow-up" domain due to a lack of reporting on attrition rates or follow-up completeness (Chin and Zeber, 2020; Izzy et al., 2021, 2022; Nuhu and Yusuf, 2012; Orlovska et al., 2014; Russell et al., 2023; Whelan-Goodinson et al., 2009). Inter-rater reliability was high, with ICC = 0.986 (95% CI: 0.953–0.996, $p < .001$) and Kappa = 0.885 ($p < .001$), indicating strong agreement between raters. Details of the risk of bias assessment are provided in Supplementary Table 2.

3.4. Main analysis

Among the 11 included studies, seven reported OR (Chen et al., 2011; Chin and Zeber, 2020; Fann et al., 2004; Izzy et al., 2021, 2022; Russell et al., 2023; Trivedi et al., 2024), and five reported HR (Izzy et al., 2021, 2022; Miller et al., 2015; Russell et al., 2023; Trivedi et al., 2024) to quantify the association between traumatic brain injury (TBI) and the subsequent development of schizophrenia or psychosis. Given their distinct statistical properties, OR capture the overall likelihood of occurrence, whereas HR reflect the relative rate of onset over time. To ensure interpretative clarity and avoid estimation bias, we conducted separate meta-analyses for each effect measure.

Three studies that lacked a non-TBI comparison group were excluded from the quantitative synthesis but were narratively summarized in the qualitative analysis to retain their contextual insights (Nuhu and Yusuf, 2012; Orlovska et al., 2014; Whelan-Goodinson et al., 2009).

Fig. 2a shows the results from seven studies reporting OR. The pooled OR was 3.07 (95% CI: 1.95–4.84), indicating a significant increase in the

Table 1
Study characteristics.

First author, year	Study design	Country	Sample size	TBI group n/N_a	Non-TBI group n/N_b	Age (*M*/*Md*/Range)	Sex (M/F)	Outcomes	Psychiatric Diagnostic Criteria or Sources	TBI Diagnosis	TBI Severity Criteria
Chen et al. (2011)	Prospective cohort	Taiwan	20970	30/3495	82/17475	31.3 (18-50) [*M*]	13722/7248	Schizophrenia	ICD-9-CM	NR	GCS
Chin and Zeber (2020)	Retrospective cohort	United States	4980	NR/1574	NR/3406	25.5 [*M*]	4890/90	Schizophrenia/Psychosis	ICD-9-CM	NR	ISS
Fann et al. (2004)	Prospective cohort	United States	3756	35/696	60/2196	15-95	1840/1916	Psychosis	ICD-9-CM	ICD-9-CM	Loss of consciousness, Traumatic intracranial lesion
Izzy et al. (2022)	Prospective cohort	United States	13053	NR/8702	NR/4351	46 (>18) [*M*]	7188/5865	Schizophrenia/Psychosis	ICD-9, ICD-10	ICD-9	CDC
Izzy et al. (2021)	Retrospective cohort	United States	18393	309/9205 b	41/9188	31 (>18) [*Md*]	10427/7966	Schizophrenia/Psychosis	ICD-9, ICD-10	ICD-9/10	CDC
Miller et al. (2015)	Retrospective cohort	United States	49798	14/5065	NR/44733	NR (predominantly young adults)	37832/11966	Schizophrenia/Psychosis	ICD-9-CM, DSM-IV-TR	ICD-9/10	CDC
Nuhu and Yusuf (2012)	Retrospective cohort	Nigeria	75	21/75	NR	32.2 [*M*]	68/7	Psychosis	ICD-10, DSM-IV	NR	NR
Orlovska et al. (2014)	Retrospective cohort	Denmark	113906	1304/113906	NR	<33	NR	Schizophrenia	ICD-8, ICD-10	ICD-8, ICD-10	NR
Russell et al. (2023)	Retrospective cohort	Canada	12492	134/2082	528/10410	14.25 (10-18) [*M*]	6720/5772	Psychosis	ICD-9, ICD-10	ICD-9, ICD-10	Concussion = Mild TBI
Trivedi et al. (2024)	Cross-sectional	United States	52374	2854/26187	1230/26187	52.2 (>12) [*M*]	36505/15869	Psychotic disorder	ICD-10	ICD-10	NR
Whelan-Goodinson et al. (2009)	Retrospective cohort	Australia	100	3/100	NR	37 (19-74) [*M*]	71/29	Psychotic disorder	DSM-IV, SCID-I	NR	NR

Note. NR, not reported; GCS, Glasgow Coma Scale; ISS, Injury Severity Score; CDC, Centers for Disease Control and Prevention; SCID-I, Structured Clinical Interview for DSM Axis I Disorder.
[a]TBI group n/N = (number of participants with schizophrenia, psychosis, or psychotic disorder in the TBI exposure group)/(total number of participants in the TBI group).
[b]Non-TBI group n/N = (number of participants with schizophrenia, psychosis, or psychotic disorder in the non-TBI control group)/(total number of participants in the non-TBI group).

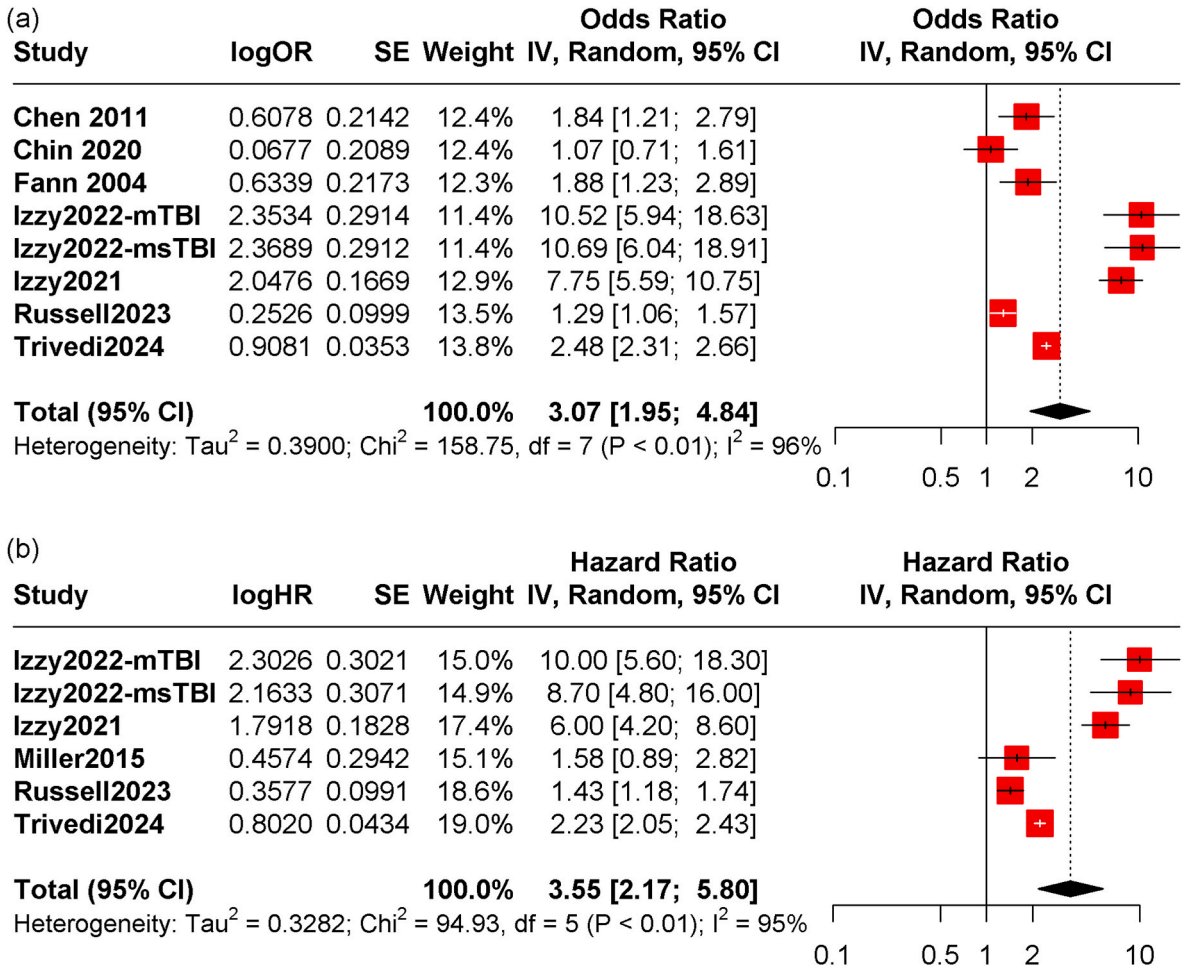


Fig. 2. Main meta-analysis results for (a) odds ratio (OR) and (b) hazard ratio (HR).

odds of developing schizophrenia or psychosis following TBI. While most studies demonstrated consistent effects, Chin and Zeber reported a non-significant OR of 1.07 (95% CI: 0.71–1.61), potentially due to methodological or sample-related differences (Chin and Zeber, 2020).

Fig. 2b presents the pooled HR derived from five studies. The meta-analysis yielded a significant association, with a pooled HR of 3.55 (95% CI: 2.17–5.80). Although one study reported a non-significant effect (HR = 1.58; 95% CI: 0.89–2.82) (Miller et al., 2015), most studies showed elevated and statistically robust HR.

While heterogeneity was substantial in both OR and HR models (OR: $\tau^2 = 0.3900$, $\chi^2 = 158.75$, df = 7, $I^2 = 96\%$, $p < .01$; HR: $\tau^2 = 0.3282$, $\chi^2 = 94.93$, df = 5, $I^2 = 95\%$, $p < .01$), this is not unexpected given the variability in study populations, effect measures, and outcome definitions. Subgroup and sensitivity analyses were conducted to explore these sources of variability.

3.5. Subgroup analyses

Subgroup analyses were conducted to examine whether the association between TBI and subsequent psychosis or schizophrenia varied by sex, age, TBI severity, follow-up duration.

Sex-based Subgroups. As shown in Fig. 3a (OR) and Fig. 4a (HR), both males and females with TBI demonstrated significantly elevated risks of developing schizophrenia or psychosis.

Pooled OR was 2.27 (95% CI: 2.10–2.45) for males and 2.65 (95% CI: 1.36–5.15) for females. Heterogeneity was negligible in the male subgroup ($\tau^2 = 0$, $\chi^2 = 0.51$, df = 1, $I^2 = 0\%$, $p = .48$), but moderate among females ($\tau^2 = 0.1595$, $\chi^2 = 2.41$, df = 1, $I^2 = 58\%$, $p = .12$).

Pooled HR was 1.48 (95% CI: 1.15–1.90) for males and 1.37 (95% CI: 1.01–1.86) for females. Overall heterogeneity for HR-based analyses was negligible ($\tau^2 = 0$, $\chi^2 = 0.15$, df = 1, $I^2 = 0\%$, $p = .70$), but interpretation is limited since both estimates were derived from the same study (Russell et al., 2023).

Notably, tests for subgroup differences were non-significant in both OR and HR analyses (OR: $\chi^2 = 0.20$, df = 1, $p = .65$; HR: $\chi^2 = 0.15$, df = 1, $p = .70$), suggesting that the association between TBI and psychosis does not differ significantly by sex.

Age-based Subgroups. As shown in Fig. 3b (OR) and Fig. 4b (HR), the association between TBI and subsequent psychosis or schizophrenia was examined separately for individuals above and below 18 years of age.

Pooled OR were 2.99 (95% CI: 1.75–5.10) for adults and 1.25 (95% CI: 1.03–1.53) for minors. Pooled HR were 3.14 (95% CI: 0.85–11.61) for adults and 1.43 (95% CI: 1.18–1.74) for minors. These results suggest a stronger association between TBI and psychosis or schizophrenia in adults. However, the HR estimate for adults did not reach statistical significance, as its confidence interval crossed 1.

Within-group heterogeneity was substantial among adults for both OR ($\tau^2 = 0.4081$, $\chi^2 = 90.99$, df = 5, $I^2 = 95\%$, $p < .01$) and HR ($\tau^2 = 0.8302$, $\chi^2 = 14.84$, df = 1, $I^2 = 93\%$, $p < .01$). In contrast, heterogeneity was low among minors for OR ($\tau^2 = 0.0012$, $\chi^2 = 1.01$, df = 1, $I^2 = 1\%$, $p = .31$).

Between-group differences were statistically significant for OR ($\chi^2 = 8.89$, df = 1, $p < .01$), but not for HR ($\chi^2 = 1.36$, df = 1, $p = .24$), suggesting that age may modify the OR-based association but not the HR-based one.

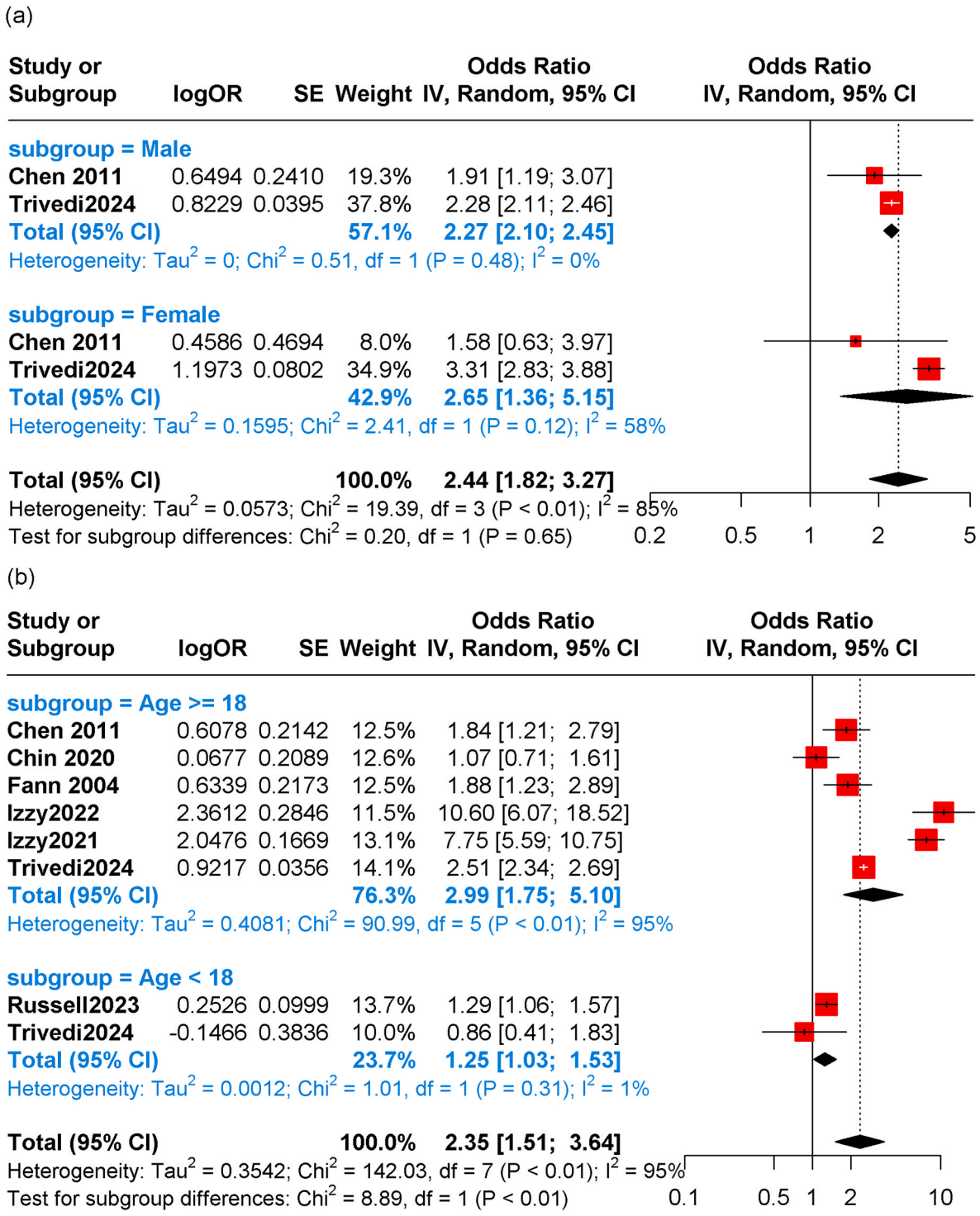


Fig. 3. Subgroup analyses of OR: (a) Sex, (b) Age (before/after 18), (c) TBI severity, (d) Follow-up time.

Overall heterogeneity remained high across age-stratified studies (OR: $\tau^2 = 0.3542$, $\chi^2 = 142.03$, df = 7, $I^2 = 95\%$, $p < .01$; HR: $\tau^2 = 0.7230$, $\chi^2 = 48.10$, df = 2, $I^2 = 96\%$, $p < .01$), indicating the presence of residual variability not fully explained by age stratification.

TBI Severity Subgroups. As shown in Fig. 3c (OR) and Fig. 4c (HR), individuals with both mild and moderate-to-severe TBI exhibited significantly elevated risks of developing schizophrenia or psychosis.

For moderate-to-severe TBI, the pooled OR was 5.42 (95% CI: 2.26–13.05), and the HR was 8.35 (95% CI: 5.42–12.87). For mild TBI, the pooled OR was 3.59 (95% CI: 1.76–7.32), and the HR was 4.45 (95% CI: 2.01–9.84).

These results indicated that TBI severity may influence the strength of association, with more severe injuries corresponding to higher risk estimates. However, all pooled effects remained statistically significant, suggesting that even mild TBI carries a meaningful risk.

Within-group heterogeneity was substantial for both mild TBI (OR: $\tau^2 = 1.0029$, $\chi^2 = 184.6$, df = 7, $I^2 = 96\%$, $p < .01$; HR: $\tau^2 = 0.9248$, $\chi^2 = 118.28$, df = 5, $I^2 = 96\%$, $p < .01$) and moderate-to-severe TBI in the OR

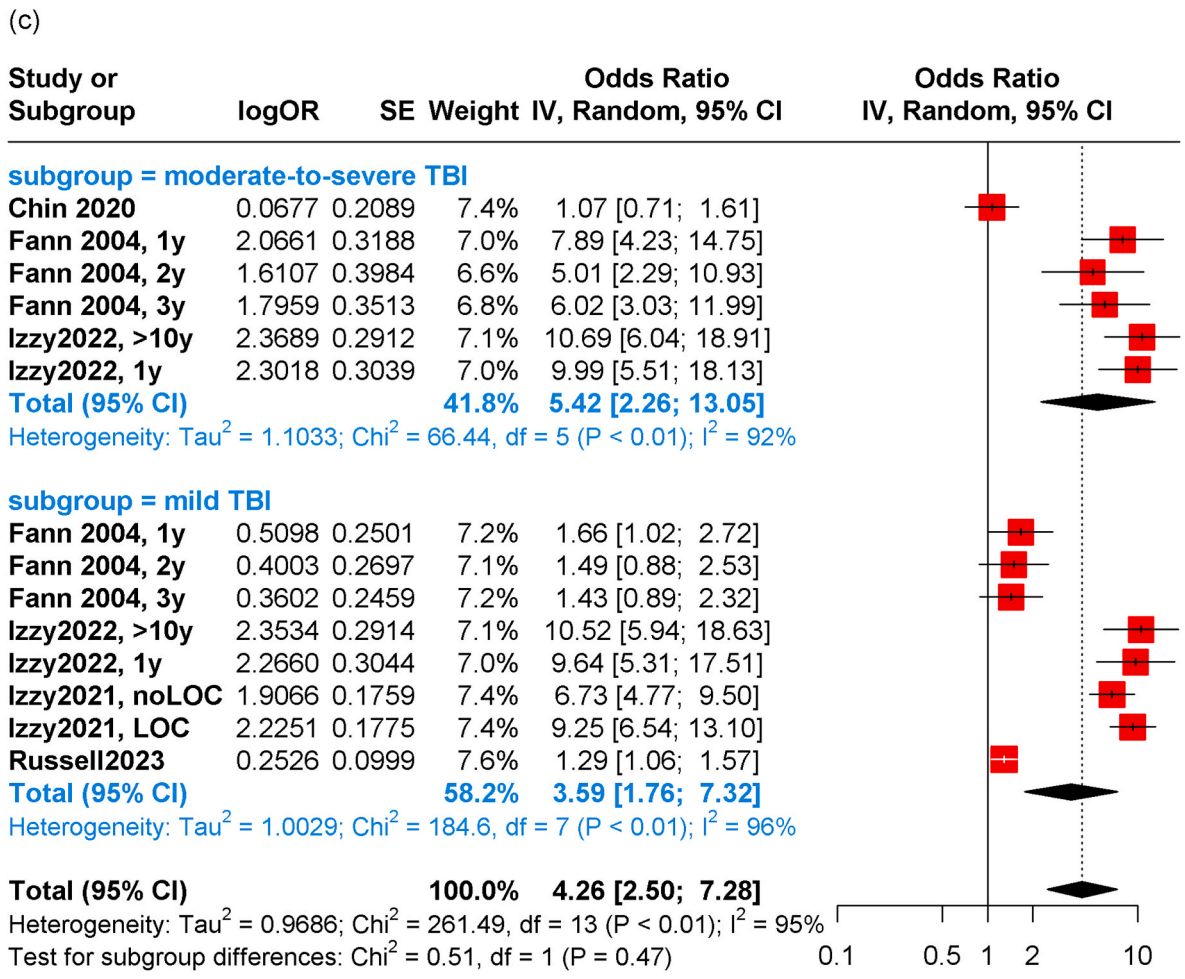


Fig. 3. (*continued*).

analysis ($\tau^2 = 1.1033$, $\chi^2 = 66.44$, df = 5, $I^2 = 92\%$, $p < .01$), but was negligible for the HR-based estimates ($\tau^2 = 0$, $\chi^2 = 0.04$, df = 1, $I^2 = 0\%$, $p = .85$).

Between-subgroup differences were not statistically significant (OR: $\chi^2 = 0.51$, df = 1, $p = .47$; HR: $\chi^2 = 1.86$, df = 1, $p = .17$), suggesting no conclusive evidence that the strength of association differed by TBI severity, although the magnitude of pooled estimates varied. Overall heterogeneity was considerable for both OR and HR models (OR: $\tau^2 = 0.9686$, $\chi^2 = 261.49$, df = 13, $I^2 = 95\%$, $p < .01$; HR: $\tau^2 = 0.9088$, $\chi^2 = 141.88$, df = 7, $I^2 = 95\%$, $p < .01$), indicating substantial between-study variability despite subgroup stratification.

Follow-up Duration Subgroups. As shown in Fig. 3d (OR) and Fig. 4d (HR), the association between TBI and subsequent psychosis or schizophrenia was examined across different follow-up durations.

For OR, the pooled estimate was 3.29 (95% CI: 1.78–6.08) for the 1–5 years group and 6.25 (95% CI: 2.71–14.40) for the >5 years group. For HR, the estimates were 1.58 (95% CI: 0.89–2.82) within 1 year, 5.31 (95% CI: 1.79–15.76) for 1–5 years, and 7.43 (95% CI: 5.39–10.25) for >5 years post-TBI.

These results suggested a time-dependent pattern: the risk of developing schizophrenia or psychosis appeared to increase with longer follow-up duration.

Within-group heterogeneity was high for OR across both follow-up periods (1–5 y: $\tau^2 = 0.4372$, $\chi^2 = 58.69$, df = 4, $I^2 = 93\%$, $p < .01$; >5 y: $\tau^2 = 0.6665$, $\chi^2 = 40.42$, df = 3, $I^2 = 93\%$, $p < .01$), and also for HR in the 1–5 y group ($\tau^2 = 0.8572$, $\chi^2 = 35.08$, df = 2, $I^2 = 94\%$, $p < .01$), while heterogeneity in the >5 y group was relatively low ($\tau^2 = 0.0188$, $\chi^2 = 2.56$, df = 2, $I^2 = 22\%$, $p = .28$).

Overall heterogeneity remained high in both models (OR: $\tau^2 = 0.5355$, $\chi^2 = 151.42$, df = 8, $I^2 = 95\%$, $p < .01$; HR: $\tau^2 = 0.6183$, $\chi^2 = 100.08$, df = 6, $I^2 = 94\%$, $p < .01$), suggesting substantial between-study variability even after stratification.

The test for subgroup differences was significant in HR ($\chi^2 = 21.11$, df = 2, $p < .01$), but not in OR ($\chi^2 = 1.47$, df = 1, $p = .23$), indicating that follow-up duration may influence the association in some models but not consistently across all.

3.6. Sensitivity analysis

Sensitivity analyses excluding studies with potential methodological concerns yielded consistent results (OR = 2.68, 95% CI: 1.28–5.58; HR = 3.27, 95% CI: 1.28–8.38), supporting the robustness of the primary findings. Detailed sensitivity analyses are provided in Supplementary Figs. 1–9.

3.7. Publication bias assessment and robustness check

We conducted a comprehensive assessment of publication bias and robustness for both the odds ratio (OR) and hazard ratio (HR) meta-analyses.

For the OR analysis, although the funnel plot suggested slight asymmetry, Egger's test indicated no significant publication bias ($t = 0.50$, df = 6, $p = .6342$). The Trim-and-Fill method imputed one potentially missing study, yielding an adjusted pooled estimate (OR = 2.57, 95% CI: 1.63–4.04) that remained consistent with the original result. Leave-one-out sensitivity analysis showed that the pooled OR

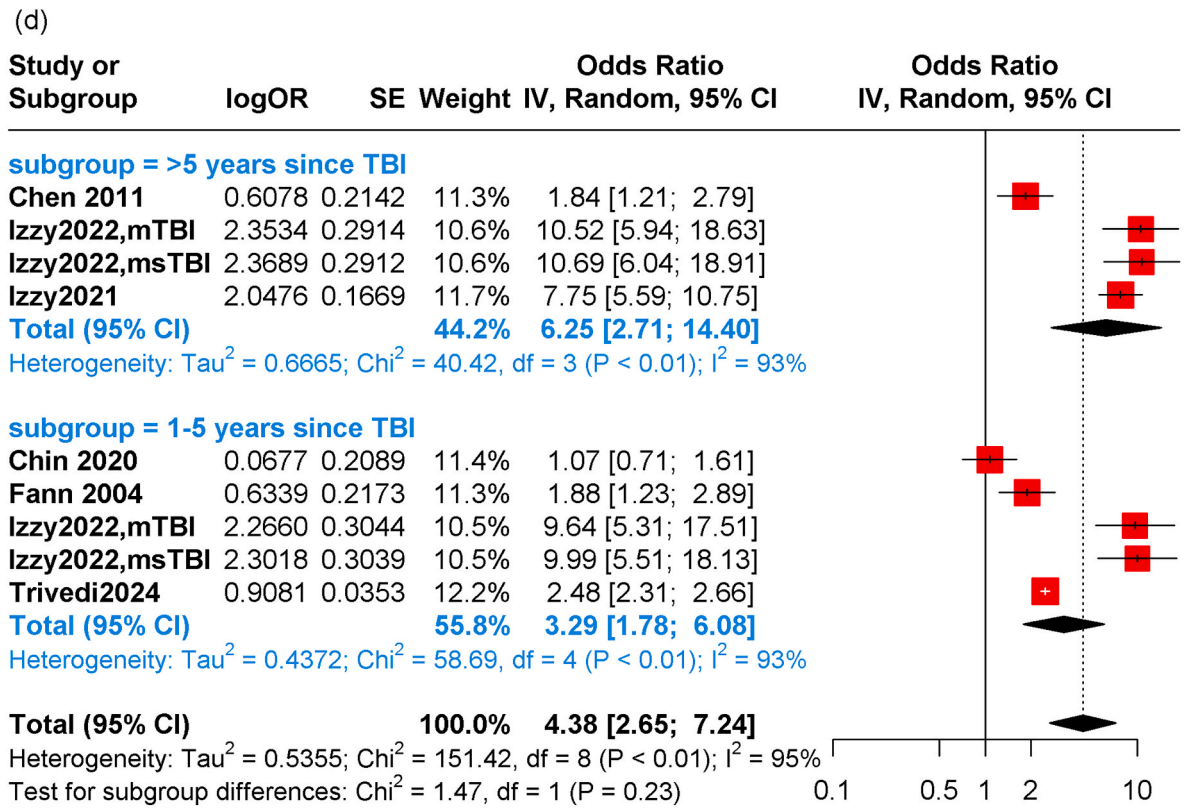


Fig. 3. (*continued*).

ranged from 2.61 to 3.56, suggesting no single study unduly influenced the overall association.

For the HR analysis, Egger's test also showed no evidence of significant asymmetry ($t = 1.26$, df = 4, $p = .2774$), and Trim-and-Fill analysis imputed two potentially missing studies. The adjusted pooled HR (2.26, 95% CI: 1.39–3.69) was comparable to the unadjusted estimate, reinforcing the robustness of the findings. Leave-one-out analysis revealed a pooled HR range of 2.94 to 4.42, again confirming that no single study dominated the overall result.

Overall, Trim-and-Fill adjustments yielded slightly attenuated pooled estimates for both OR (3.07 → 2.57) and HR (3.55 → 2.26), suggesting potential publication bias may have led to overestimation. Nevertheless, the adjusted estimates remained statistically significant and directionally consistent, supporting the robustness of the observed associations. Visual inspection of publication bias, including funnel and trim-and-fill plots, is presented in Supplementary Figs. 10-11 and 13-14. Detailed publication bias assessments are provided in Supplementary Tables 3–4, Supplementary Figs. 12 and 15.

4. Discussion

This meta-analysis found a significant association between TBI and the subsequent development of schizophrenia or psychosis, in line with previous meta-analyses (Molloy et al., 2011; Yau et al., 2024). By incorporating both OR and HR, this study provided a more comprehensive perspective on both cumulative risk and time-to-event patterns.

The pooled effect sizes in the present study (OR = 3.07; HR = 3.55) were notably larger than those reported by Molloy et al. (2011) (OR = 1.65) and Yau et al. (2024) (OR = 1.59). Several factors may account for this discrepancy. The present review specifically focused on studies that examined the incidence of psychosis following TBI, and excluded studies that primarily assessed the prevalence of prior TBI among individuals with established psychiatric diagnoses, as such studies address a different research question and may introduce reverse-causality concerns. We excluded 8 such studies (AbdelMalik et al., 2003; Cheng et al., 2024; Deighton et al., 2016; Harrison et al., 2006; Malaspina et al., 2001; Nielsen et al., 2002; Sachdev et al., 2001; Wilcox and Nasrallah, 1987) to maintain a consistent exposure-outcome framework. Additionally, differences in inclusion criteria, publication dates, and the incorporation of both OR- and HR-based analyses may have contributed to the observed differences. Despite these methodological variations, all three meta-analyses consistently support an elevated risk of psychosis following TBI.

4.1. Sex-based findings

Subgroup analyses indicated that both males and females with TBI had significantly increased risks of developing schizophrenia or psychosis. These findings were consistent across both OR- and HR-based models, and no significant subgroup difference by sex was detected. While prior reviews suggested male-specific vulnerability (Cancelliere et al., 2016), the present findings support a sex-independent risk pattern.

Clinically, these findings suggest that psychiatric surveillance following TBI should not be limited to male patients. Although prior literature has emphasized male vulnerability to both TBI and psychosis, the present results indicate that female TBI survivors warrant equal attention in long-term monitoring protocols.

4.2. Age groups

The observed stronger association in adults compared with minors may partially reflect the low baseline incidence of psychosis in children and adolescents. Nevertheless, the increased risk in the minor subgroup (OR = 1.25, 95% CI: 1.03–1.53) suggests that TBI may elevate risk even in populations with lower base rates. Between-group heterogeneity was statistically significant for OR-based analyses ($p < .01$), suggesting that age may modify the strength of the association. These findings support the need for age-tailored surveillance strategies after TBI.

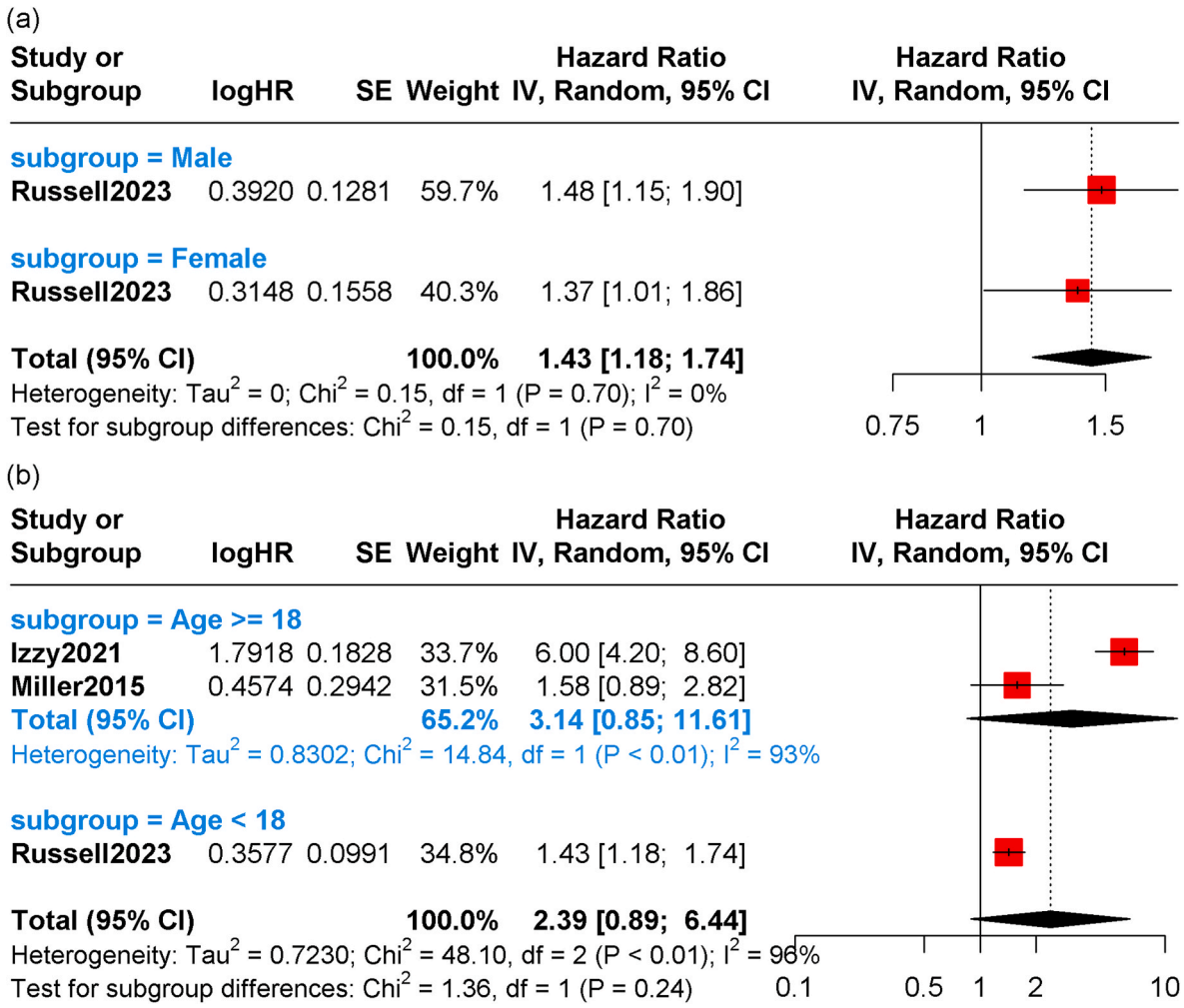


Fig. 4. Subgroup analyses of HR: (a) Sex, (b) Age (before/after 18), (c) TBI severity, (d) Follow-up time.

From a clinical standpoint, while adults with TBI may represent a higher-risk group warranting prioritized psychiatric follow-up, the significant elevation in risk among minors suggests that pediatric TBI patients should not be overlooked. Given that psychosis typically emerges in late adolescence or early adulthood, children and adolescents who sustain TBI may require extended monitoring into adulthood to capture delayed-onset cases. These findings support the development of age-specific follow-up guidelines for TBI populations.

4.3. Follow-up duration

Longer follow-up durations were associated with higher risk estimates. Although it is intuitive that more psychosis cases emerge over time, the HR models account for follow-up time and still showed higher effect sizes in longer periods (e.g., HR = 7.43 for >5 years vs. HR = 1.58 for <1 year). This pattern suggests not merely case accumulation, but an increasing hazard over time. However, overlapping confidence intervals and limited study numbers warrant caution in interpretation.

4.4. TBI severity

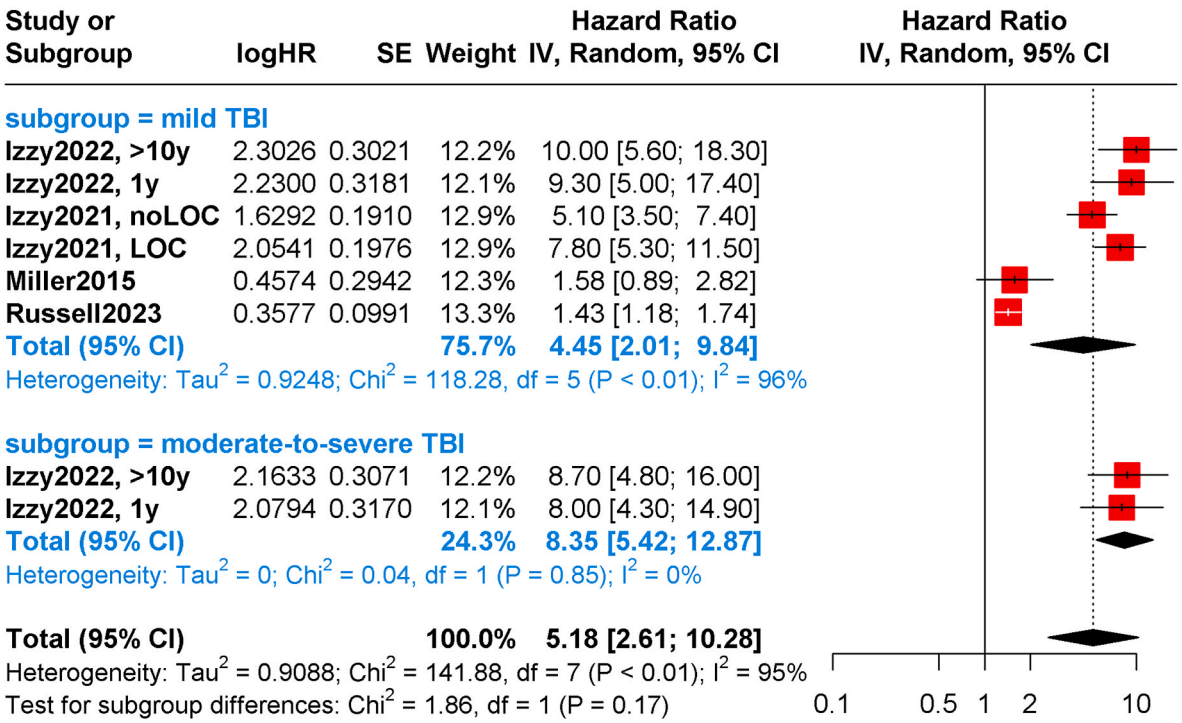
A dose-response relationship was observed across TBI severity levels. Moderate-to-severe TBI showed a higher risk (OR = 5.42; HR = 8.35) than mild TBI (OR = 3.59; HR = 4.45). While both levels conferred significant risk, the stronger association with more severe injuries is consistent with neurobiological models of cumulative damage. Discrepancies between OR and HR values may reflect differences in outcome definitions and follow-up timelines; HR may capture more immediate effects post-injury.

Notably, Yau et al. did not observe a significant dose-response relationship between TBI severity and psychosis risk. This discrepancy may reflect differences in how TBI severity was classified across included studies, as well as variation in the number of studies reporting severity-stratified data. The present analysis benefited from additional recent studies that provided more granular severity classifications, which may have enhanced the ability to detect a severity-dependent gradient.

4.5. Links to neurobiological mechanisms

The observed dose-response relationship and time-dependent increase in risk are consistent with the pathophysiological framework outlined in the Introduction. The finding that more severe TBI conferred greater risk aligns with models suggesting cumulative structural damage influences psychiatric vulnerability. Similarly, the increasing hazard over longer follow-up periods supports the role of progressive neurodegeneration—including Tau accumulation and hippocampal atrophy—in delayed-onset psychosis (Bray et al., 2021; Takahata et al., 2019). However, the present meta-analysis was limited to clinical outcome data and could not directly assess underlying neurobiological processes. Future studies integrating neuroimaging biomarkers with longitudinal psychiatric outcomes would help clarify these mechanistic pathways.

(c)



(d)

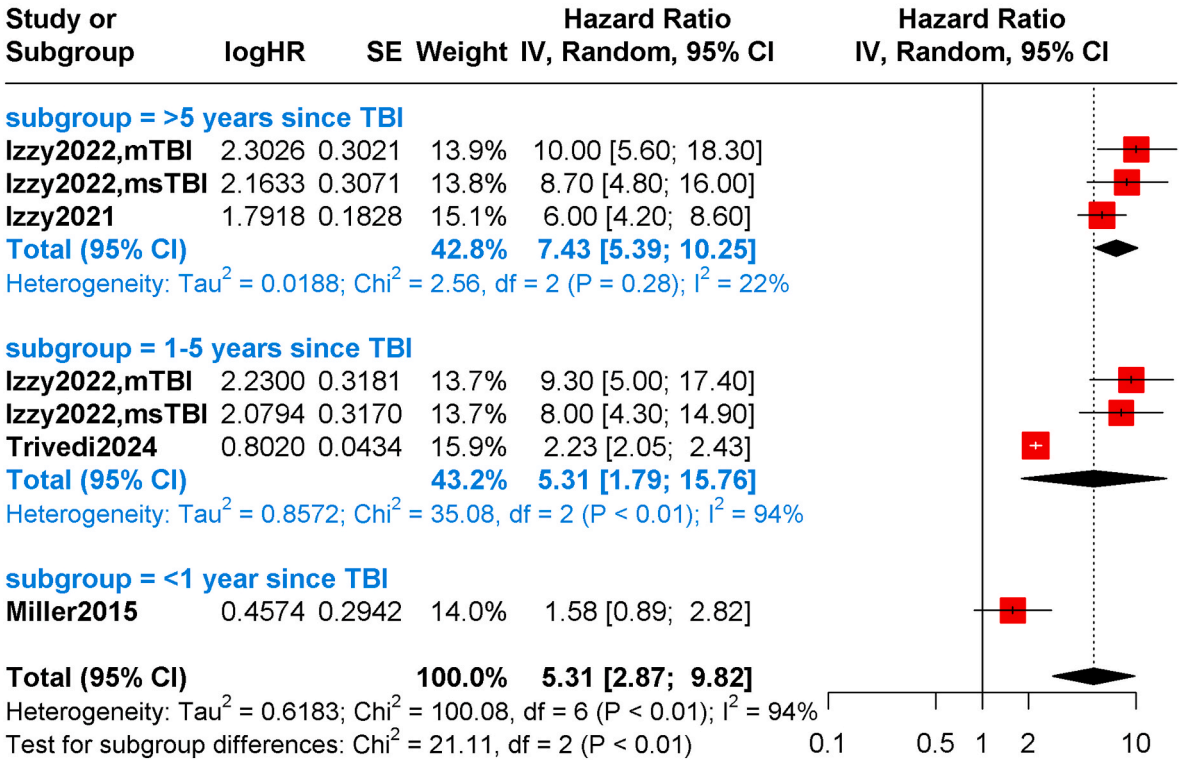


Fig. 4. (*continued*).

4.6. *Heterogeneity and publication bias*

The substantial heterogeneity observed across analyses ($I^2 > 95\%$) warrants careful interpretation. This level of heterogeneity suggests that the true effect size likely varies across study populations and contexts, and the pooled estimate should be viewed as an average tendency rather than a precise universal effect. Sources of heterogeneity may include differences in TBI ascertainment methods, psychosis diagnostic criteria, follow-up duration, population demographics, and healthcare settings. While subgroup analyses by age, sex, severity, and follow-up duration

were conducted to explore potential moderators, considerable residual heterogeneity remained. The use of random-effects models accommodates this variability by providing wider confidence intervals, but the clinical application of these findings should account for the likelihood that effects may differ across settings.

4.7. Reverse causality

An important consideration in interpreting these findings is the possibility of reverse causality. Prodromal symptoms of schizophrenia—such as impaired attention, impulsivity, cognitive decline, or social dysfunction—may increase the likelihood of sustaining a TBI, rather than TBI causing subsequent psychosis. This is particularly relevant for studies with shorter follow-up periods or those unable to ascertain the precise timing of TBI relative to the onset of psychiatric symptoms. Although several included studies attempted to address this by excluding individuals with pre-existing psychiatric diagnoses or by requiring a minimum interval between TBI and psychosis onset, residual confounding by prodromal states cannot be fully ruled out. Future prospective studies with detailed premorbid assessments are needed to disentangle the temporal sequence of TBI and psychosis.

4.8. Confounding

Relatedly, the degree of covariate adjustment varied considerably across included studies. While some studies controlled for potential confounders such as age, sex, substance use, and socioeconomic status, others provided only unadjusted estimates. Residual confounding from unmeasured factors—including genetic predisposition, family psychiatric history, and lifestyle factors—may have influenced the observed associations. The present meta-analysis was limited to synthesizing published effect sizes and could not perform individual-level adjustment for confounders. Readers should therefore interpret the pooled estimates as reflecting associations that may be partially attributable to confounding rather than direct causal effects.

4.9. Strengths and limitations

Key strengths of this meta-analysis include its dual use of OR and HR, its stratified subgroup analyses (by age, sex, severity, and follow-up), and its incorporation of multiple bias assessments.

However, this study has several limitations. First, although our search strategy covered major databases with high coverage of psychiatric literature, the exclusion of certain specialized databases, grey literature, or unpublished studies may have resulted in incomplete retrieval of relevant studies or introduced publication bias. Second, the cross-sectional design of Trivedi et al. (2024) limits causal inference, although sensitivity analyses excluding this study yielded consistent results. Third, including multiple subgroups from single studies may result in overlapping control groups (Izzy et al., 2022); this was addressed through sensitivity analyses for the primary analysis but retained in severity subgroup analysis by design. Fourth, TBI definitions varied across studies, with four studies not reporting diagnostic criteria, which may have contributed to between-study variability. Finally, substantial heterogeneity ($I^2 > 95\%$) was observed, likely reflecting variability in study designs, populations, and outcome assessments. Results should therefore be interpreted with caution.

5. Implications and future directions

Despite these limitations, the findings underscore the need for long-term psychiatric monitoring following TBI. The elevated risk across time and severity levels suggests that psychosis may emerge as a delayed consequence of neurotrauma. Clinically, these results support integrated care models that include psychiatric screening for individuals with a history of TBI. Future studies should employ standardized definitions of TBI and psychosis, report stratified results, and use longer follow-up intervals to better understand temporal risk trajectories.

6. Conclusion

This meta-analysis confirmed a significant association between traumatic brain injury and the subsequent development of schizophrenia or psychosis. The strength of this association varied by age, injury severity, and follow-up duration, highlighting the need for long-term monitoring and individualized psychiatric risk assessment in TBI survivors.

CRediT authorship contribution statement

Ting-Yi Chu: Writing – review & editing, Writing – original draft, Visualization, Methodology, Formal analysis, Data curation, Conceptualization. **Chung-Che Wu:** Supervision, Formal analysis. **Zi-Xiang Xu:** Investigation, Data curation. **Yung-Hsiao Chiang:** Methodology, Conceptualization. **Shih-Chang Hsueh:** Validation, Investigation. **Ju-Chi Ou:** Writing – review & editing, Supervision, Project administration. **Kai-Yun Chen:** Writing – review & editing, Supervision, Funding acquisition.

Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Availability of data and materials

All data generated or analyzed during this study are included in this published article and its supplementary material.

Protocol registration

This systematic review and meta-analysis was not prospectively registered in PROSPERO. However, a predefined protocol was developed and is available in Supplementary Text 1.

Use of AI and AI-assisted technologies

During the preparation of this work, the author used ChatGPT-4o (OpenAI) and Claude Opus 4.5 (Anthropic) to assist with language refinement, formatting clarity, and consistency checking. The author reviewed and edited all content as needed and takes full responsibility for the integrity and accuracy of the manuscript.

Funding

This research was supported by the National Science and Technology Council (NSTC) under grants NSTC 113-2811-B-038-032, 112-2811-B-038-016, 112-2314-B-038-108, 111-2314-B-038-134-MY3, 113-2314-B-038-017, and 113-2314-B-038-142. Additional support was provided by the University System of Taipei Joint Research Program (USTP-NTUT-TMU-110-01) and the Higher Education Sprout Project by the Ministry of Education (MOE) in Taiwan (DP2-TMU-113-N-01).

Declaration of competing interest

The authors declare that they have no competing interests.

Acknowledgements

We thank the University System of Taipei and the Ministry of Education in Taiwan for supporting this research. We also acknowledge the valuable feedback from peer reviewers during manuscript revision.

Glossary

CI	Confidence Interval
COPD	Chronic Obstructive Pulmonary Disease
DSM	Diagnostic and Statistical Manual of Mental Disorders
GCS	Glasgow Coma Scale
HR	Hazard Ratio
ICC	Intraclass Correlation Coefficient
ICD	International Classification of Diseases
I^2	I-squared Statistic
ISS	Injury Severity Score
NOS	Newcastle-Ottawa Scale
OR	Odds Ratio
PRISMA	Preferred Reporting Items for Systematic Reviews and Meta-Analyses
PTSD	Post-Traumatic Stress Disorder
RR	Relative Risk
SCID-I:	Structured Clinical Interview for DSM Axis I Disorder
TBI	Traumatic Brain Injury
T2DM	Type 2 Diabetes Mellitus

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jpsychires.2026.03.005.

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