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Is Childhood Bullying a Distinct and Causal Risk Factor for Psychotic Experiences? A Systematic Review of Longitudinal Studies

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Abstract. Childhood bullying is a prevalent adversity consistently linked with psychotic experiences in longitudinal studies, yet its causal status and distinct mechanisms remain to be rigorously investigated. This review synthesises longitudinal evidence to determine if bullying is a distinct and causal risk factor. Analysing prospective cohorts, twin designs, and causal models uncovers a clear hierarchy of evidence. Crucially, discordant monozygotic twin studies corroborate a causal environmental effect, with bullied twins exhibiting significantly elevated rates of paranoid ideation and cognitive disorganisation, independent of genetic confounders. Furthermore, bullying constitutes a distinct risk factor, often outweighing that of other adversities, with cumulative "double dose" exposure (e.g., from both peers and siblings) drastically elevating the odds of PEs. Mechanistically, this pathway is partially mediated (≈approximately one-third) by cognitive-affective factors, specifically, an external locus of control and low self-esteem, while a significant direct effect points to additional biological pathways, including hypothalamic-pituitary-adrenal (HPA) axis dysregulation and dopamine sensitisation. Synthesis of this evidence firmly establishes childhood bullying as a potent causal and environmental risk factor for psychosis, which operates via a complex network of interdependent psychological and biological pathways.

Keywords: Bullying victimisation, Psychotic Experiences, Causal inference, Twin studies

1. Introduction

Epidemiological data from a large UK cohort indicate that the majority of adolescents (58.7%) are victimised by their peers or siblings, with 15.6% enduring severe, frequent victimisation that confers substantial risk of harm [1]. Bullying frequently co-occurs with other forms of violence and adversity, as demonstrated by the finding that almost half (45.7%) of victimised adolescents experience multiple forms of severe victimisation [1]. Psychotic experiences (PEs), broadly defined as encompassing symptoms of paranoia, cognitive disorganisation, and sensory hallucinations, are relatively prevalent in adolescence and serve as a key risk factor for the subsequent onset of psychotic disorders. Notably, childhood bullying represents a pressing societal concern demonstrated to affect mental health adversely. One longitudinal twin study revealed that the most rigorous twin difference estimates (monozygotic) were consistent with a causal contribution of

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bullying exposure at age 11 to concurrent anxiety, depression, hyperactivity and impulsivity, inattention, and conduct problems [2]. The increasing co-prevalence of these two phenomena has prompted researchers to move beyond broad investigations of childhood trauma to explore the association between childhood bullying and psychotic experiences. While numerous cross-sectional and longitudinal studies have documented an association, it remains unclear how struggles from bullying work through neurobiological processes to translate into psychotic experiences—in short, the mechanisms. To establish causality, a synthesis of the mechanistic findings from these studies is essential. Accordingly, this review seeks to systematically evaluate the hypothesis and methodically assess the contention that childhood bullying is a causal risk factor for PES. This objective is achieved. It accomplishes this by examining and synthesizing findings from a range of integrating findings from an array of longitudinal and genetically sensitive cohort studies. Elucidating this causal relationship is essential, as school-based anti-bullying programs and early mental health support for bullying victims are effective strategies for preventing subsequent mental support for victims of bullying and could be validated as effective prevention of later mental illness. Ultimately, it benefits public health, child safety, and clinical practice.

2. Association

A substantial body of longitudinal evidence has consistently demonstrated that childhood bullying victimisation is a salient predictor of subsequent psychotic experiences. This association is particularly alarming given the prevalence of bullying. This association has been clearly established in seminal cohort studies. One critical piece of evidence from a longitudinal twin design study demonstrated that children who had experienced various forms of trauma, including bullying, exhibited an increased likelihood of developing psychotic symptoms in adolescence, with a clear dose-response relationship indicating that elevated risk correlates directly with the severity of victimisation [3]. Crucially, this study employed a genetically sensitive design utilising a twin cohort to account for shared environmental factors such as family socioeconomic status, parental education, and social class. Furthermore, it explicitly disentangled the environmental effect of bullying from genetic confounders by constructing a continuum of genetic risk based on each twin's zygosity and the co-twin's symptom status, and statistically adjusting for this genetic vulnerability in the analysis. This finding was corroborated in a prospective study that reported bullying exposure predicted new psychotic experiences at both the 3-month and 12-month follow-ups, after adjusting for baseline psychotic symptoms [4].

Furthermore, a dose-response relationship was further observed, where severity of bullying was positively correlated with psychosis risk—notably, cessation of bullying was associated with a marked reduction in PE incidence (OR=0.28). However, the relationship is also bidirectional, in that PEs predicted later bullying victimisation, ultimately perpetuating a complex vicious cycle [4]. A longitudinal study tracking children from elementary school through late adolescence revealed that bullying's impacts lasted for over a decade, extending these findings over a longer developmental span [5]. Notably, this study demonstrated that both bullies and victims exhibited the highest risk, and that chronic and persistent victimisation exerted the most pronounced effect, nearly doubling the odds of psychotic episodes relative to non-victims [5]. This dose-response relationship finds a plausible biological substrate in the stress-induced neuropsychological cascade triggered by chronic victimisation. Bullying is a severe psychosocial stressor that dysregulates the hypothalamic-pituitary-adrenal (HPA) axis and induces limbic hyperactivity, especially in the amygdala. This leads to structural and functional changes in prefrontal and hippocampal circuits critical for threat processing and emotion regulation. Additionally, these neurological effects are amplified by gene-

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environment interactions, in which individual neurobiological sensitivity to bullying-related stress is shaped by genetic predispositions (e.g., in serotonin and dopamine pathway genes). This confluence of chronic social stress, neurocircuitry disruption, and genetic predisposition offers a logical explanation for why longer and more severe bullying exposure elevates the risk of psychotic outcomes [6].

Critically, peer victimisation exerts a potent independent effect. Evidence from a direct comparison of childhood adversities between two cohorts discovered that being bullied by peers posed a larger risk for adult mental health issues than being mistreated by adult figures [7]. This highlights peer bullying as a distinct risk factor for psychopathology, separate from other traumatic experiences. This finding is supported by robust quantitative results: the study's adjusted analysis revealed that children who were bullied (but not maltreated) exhibited significantly higher odds of developing general adult mental health morbidity than those who were maltreated (but not bullied), with an odds ratio (OR) of 1.6 in the ALSPAC cohort and 3.8 in the GSMS cohort. In one cohort, the risk for adult anxiety among bullied children was nearly five times greater (OR=4.9) than for maltreated children [7]. This confirms bullying as a distinct and potent risk factor for psychopathology, one that can confer a greater risk than other severe forms of childhood trauma.

3. Causality: evidence from genetically sensitive designs

Though the longitudinal findings noted above have already indicated a clear association between childhood bullying and later psychotic experiences, it remains unclear whether the link is causal or whether extrinsic factors (e.g., environmental stressors) or pre-existing vulnerabilities predispose to psychotic experiences. In addressing this, studies have adopted increasingly rigorous methodologies to account for potential confounding factors, most specifically genetic influences.

Twin design research provides the most robust methodological framework for testing causality because genetic and environmental confounds can be systematically accounted for. A foundational study in this area employed a longitudinal twin cohort design to demonstrate that children who were bullied were significantly more likely to develop psychotic symptoms in adolescence. Notably, even when 2,232 pairs of monozygotic (identical) twins, who share 100% of their DNA, were examined, this association remained strong, indicating that the experience of bullying itself exerts an environmentally mediated causal effect independent of genetic factors or shared environmental confounders. The study further corroborated a dose-response association, which strengthened the case for a causal pathway by demonstrating that greater symptom incidence correlates with more frequent and severe victimisation [3]. The distinction between this finding and those from standard cohort studies resides in the level of causal inference. While prospective studies establish temporal sequence, they cannot rule out genetic confounding. The superior rigour of the twin design is exemplified by the discordant monozygotic twin comparison: it isolates the specific environmental effect of bullying from all shared genetic and shared familial environmental factors, providing the clearest possible evidence of an environmentally mediated causal pathway.

Building on this, a more recent and comprehensive population-based twin study yielded nuanced longitudinal insights [2]. Employing a twin-differences analytical framework with over 11,108 twins, this research validated a direct causal role of bullying in specific psychotic experiences. Their analysis demonstrated that the bullied twin in monozygotic pairs exhibited significantly elevated levels of paranoid ideation and cognitive disorganisation, even after controlling for all shared genetic and environmental factors. This effect was robust at concurrent assessment and 2-year follow-up points, thus corroborating a clear temporal and potentially causal pathway [2].

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Other prospective cohorts have elucidated the pervasiveness and specificity of bullying as a causative risk factor, even though twin studies, such as the one previously referred to, offer the strictest control for genetic and shared environmental confounds. These results were further validated by another prospective cohort study, which demonstrated that the risk of psychotic illness at the age of 18 was equally pronounced in the home environment, extending beyond peer bullying. Leveraging data from the ALSPAC birth cohort (children born 1991–1992), this study prospectively assessed sibling bullying at age 12 and psychotic disorder via a semi-structured clinical interview (PLIKSi) at age 18. Even after adjusting for a wide range of confounders, such as maternal depression, domestic violence, and childhood maltreatment, their study discovered that frequent sibling victimisation during middle childhood was associated with a nearly threefold increase in the odds of a psychotic disorder (OR 2.74). Notably, they found a "double dose" effect: children who experienced victimisation from both peers and siblings exhibited a more than fourfold elevated risk (OR 4.57), suggesting that the cumulative experience of victimisation across multiple ecological contexts significantly elevates psychotic risk. This additive effect, which persists after accounting for the child's behavioural issues and pre-existing family adversity, strongly implies that bullying itself—as opposed to pervasive adverse familial contexts—exerts a distinct and substantial causal contribution to the etiology of psychotic disorders [8].

4. Methodological rigour

Methodological approaches that adequately address key threats to validity, such as genetic confounding, reverse causality, and unspecific mediation, are necessary to establish a distinct causal risk factor from observational data. A clear hierarchy of evidence emerges from the literature, and the degree of confidence in the association between childhood bullying and psychotic experiences is directly contingent upon the rigour of the research designs used to examine it. The fundamental design for determining temporal sequence is embodied by prospective cohort studies. These studies provide critical preliminary evidence of an association and can demonstrate a dose-response relationship by evaluating bullying exposure prior to the onset of psychotic symptoms and adjusting for a variety of measured covariates, including familial adversity and socioeconomic status. Furthermore, to address potential genetic confounding, these studies may employ analytical strategies including stratified analysis, which involves examining associations within subgroups defined by high versus low genetic risk or multivariate regression models incorporating polygenic risk scores or family history of psychosis.

Genetically sensitive designs yield a higher tier of evidence, especially when it comes to investigations of monozygotic twin pairs that are discordant for bullying victimisation. This framework constitutes a powerful natural experiment, as it accounts for all shared genetic and environmental components. Strong evidence for a direct environmental causal effect is provided by a higher incidence of psychiatric sequelae in the bullied twin relative to their genetically identical co-twin, thus effectively ruling out genetic predisposition as the sole driver of the association.

Directed Acyclic Graphs (DAGs) and other causal modelling techniques are used in the most methodologically sophisticated approaches [9]. As a foundational tool for causal inference, a DAG (Directed Acyclic Graph) is a visual model that depicts the hypothesised directional relationships between variables, with no cyclical pathways. These models hypothesise how the causal route functions in addition to determining whether a link exists. DAGs, in contrast to traditional regression, simultaneously analyse complex interdependencies among multiple variables without imposing a predefined causal order, allowing the data to distinguish direct effects from downstream consequences and infer the most plausible causal structure [9].

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Progressing from correlation to causation, the shift from cohort studies to twin designs represents a pivotal step in accounting for confounding factors. For example, twin-discordant studies can establish that a dose-response relationship is likely caused by environmental variables, even though cohort studies can also discover this link. Building on this, complex causal models may then elucidate precise pathways—for instance, determining whether bullying directly affects psychotic symptoms or only acts through mediating factors like sadness or anxiety. This enables a more comprehensive understanding of the mechanistic pathways, as shown in Figure 1 [9].

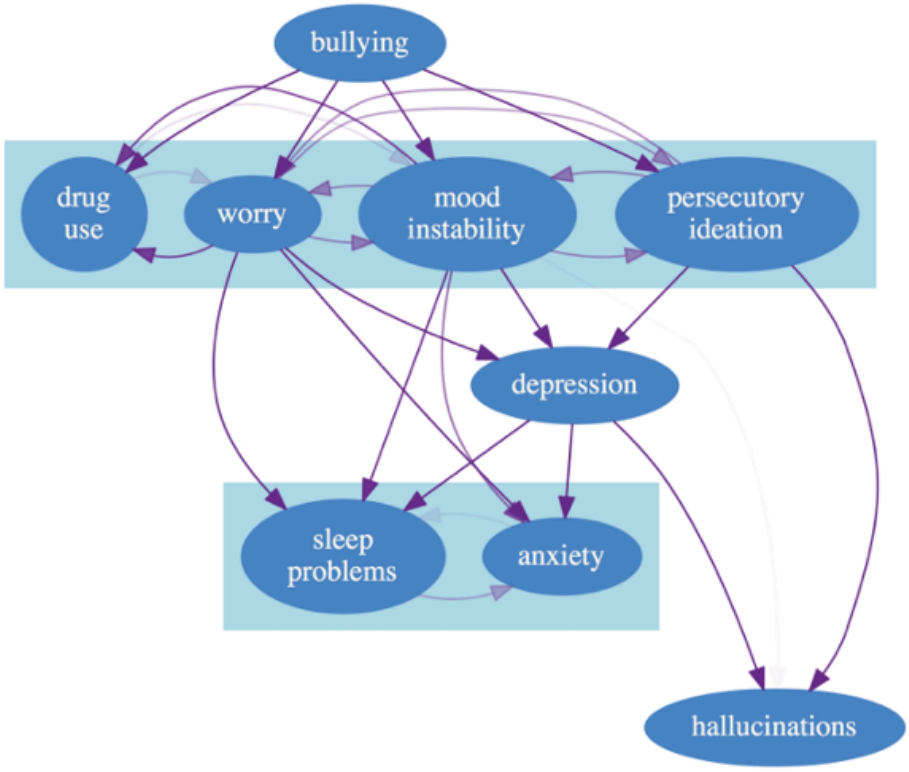

Figure 1. Directed acrylic graph of relationships between variables linked to bullying (adapted from [9])

5. Mechanics and complexity

Delving deeper than mere associations and causality, several prospective studies have advanced to investigating the psychological mechanisms linking bullying to psychotic experiences. One cohort study that tested a theoretical model where childhood bullying influences psychotic symptoms through cognitive mediators found that the association was approximately one-third mediated by cognitive mediating factors (with bullying's total effect on psychotic symptoms being highly significant, e.g., OR = 1.51, 95% CI 1.27–1.80 for definite symptoms), with an external locus of control and low self-esteem as particularly important pathways (see conceptual model, Figure 2) [10]. The study measured the extent to which these mediators explained bullying's effect relative to the direct effect of bullying. The pathway was also partially mediated by classic affective symptoms —anxiety and depression; however, these factors exerted only a secondary role relative to cognitive mediators in the context of bullying victimisation [10].

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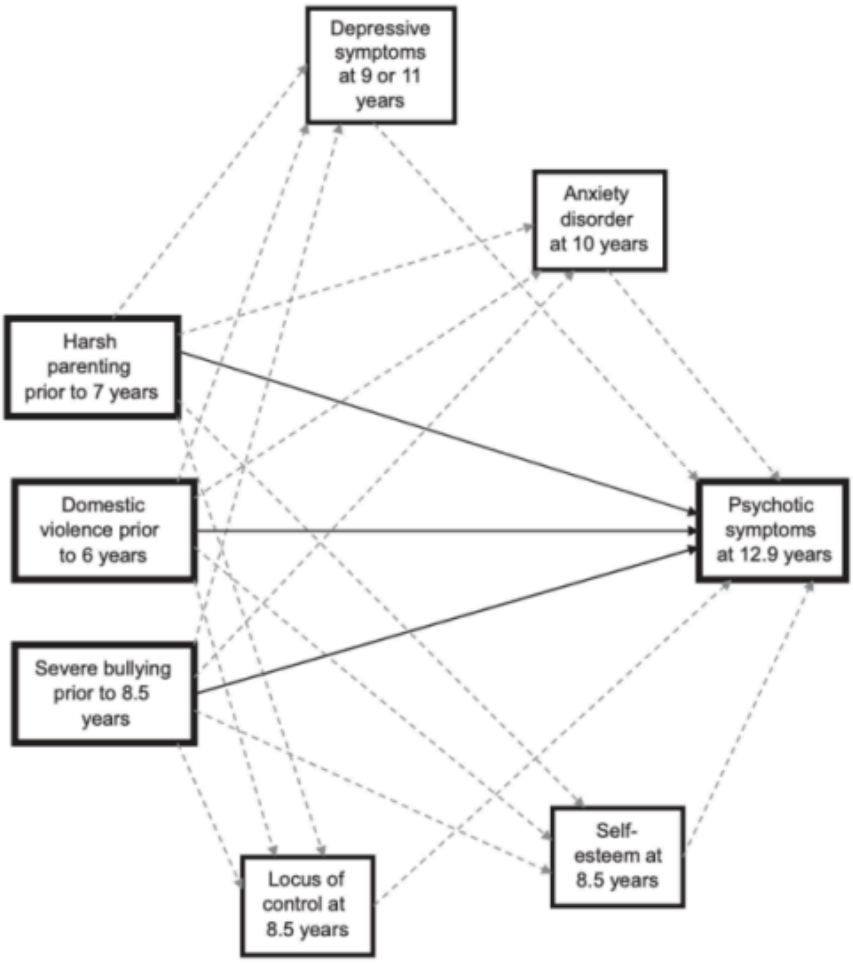

Figure 2. Path diagram of associations between victimisation and psychotic symptoms (adapted from [10])

Notably, these psychological mediators account for only a fraction of the observed association. Approximately one-third of the total effect of bullying on psychotic outcomes was explained by the investigated mediators taken together, with a substantial direct effect remaining unaccounted for [5]. Aside from the HPA axis and dopamine systems mentioned, other unmeasured pathways include neuroinflammation, epigenetic changes such as FKBP5 gene methylation, the default mode network, and others. This indicates that while these cognitive and emotional adversities are substantial, the detrimental effects of bullying also operate via additional unmeasured pathways [10].

Integrative theoretical frameworks positing concurrent biological and complex cognitive processes help elucidate these unmeasured pathways. Beyond these well-characterised pathways, new research suggests additional biological mechanisms that may contribute to psychotic risk following victimisation, such as stress-induced epigenetic modifications (e.g., DNA methylation changes) and perturbations in the gut microbiome-brain axis. Nevertheless, further validation via integrated multi-omics approaches is required to corroborate these emerging pathways. In terms of biology, victimisation stress is associated with a dysregulated hypothalamic-pituitary-adrenal (HPA) axis, evidenced by blunted salivary cortisol responses—an established risk factor for psychosis [11]. The axis's negative feedback loop may be disrupted, possibly as a result of stress-induced epigenetic

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changes that alter the expression or sensitivity of key elements, such as glucocorticoid receptors [12]. Moreover, enhanced dopamine release, a neurotransmitter critically implicated in psychotic symptoms, may result from the subsequent heightened emotional reactivity. Genetic variables, such as certain FKBP5 haplotypes, which influence glucocorticoid receptor sensitivity, modulate this process by interacting with bullying exposure to increase psychotic risk. Simultaneously, more intricate cognitive processes beyond locus of control and self-esteem have been identified, such as the development of maladaptive schemas regarding the self and others, as well as increased interpersonal sensitivity, which has been shown to be a particular mediator of paranoid ideation [11].

6. Conclusion

In conclusion, converging evidence from prospective cohort studies robustly demonstrates that childhood trauma, including peer and sibling bullying, is a significant causal risk factor for the subsequent onset of psychotic symptoms and disorders later in life. The findings confirm a dose-response relationship, wherein greater or more chronic exposure to victimisation increases risk, and highlight potential psychological pathways such as heightened anxiety, depression, and paranoid ideation. Despite the methodological rigour of longitudinal and twin designs, several limitations in research still persist. Notably, several limitations constrain the interpretation of this evidence, including potential residual confounding, measurement biases inherent to self-reports, and inherent challenges of causal inference from observational longitudinal data. These constraints do not negate the findings; rather, they underscore that the relationship is likely part of a complex, transactional process. Therefore, to adequately account for genetic and environmental confounding, future research should prioritise multi-informant, longitudinal measurements from an early age; investigate specific mechanistic pathways and vulnerable subpopulations; and adopt innovative techniques. To transition from proving association to completely clarifying the causal pathways, this will be essential. Future research should also focus on investigating behavioural therapy, such as raising self-esteem and modifying attributional styles in the face of stress or problems. Another major direction worthy of deeper investigation is exploring why some victimised children do not experience clinical outcomes. This approach should prioritise the mechanisms of social support and psychological resilience's impact on different outcomes of childhood bullying.

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