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REVIEW

Bridging autism and psychosis: why childhood harm matters

Riccardo BORTOLETTO [1] *, Claudia SCIPIONI [1], Ruud VAN WINKEL [2], Marco COLIZZI [1, 3]

[1]Unit of Psychiatry and Eating Disorders, Department of Medicine (DMED), University of Udine, Udine, Italy; [2]University Psychiatric Centre KU Leuven, KU Leuven, Belgium; [3]Department of Psychosis Studies, Institute of Psychiatry, Psychology and Neuroscience, King's College London, London, UK

*Corresponding author. Unit of Psychiatry and Eating Disorders, Department of Medicine (DMED), University of Udine, 33100, Udine, Italy. Email: bortoletto.riccardo@spes.uniud.it

ABSTRACT

Autism and psychosis share genetic and neurobiological underpinnings, have overlapping clinical features, and often co-occur. Here we examine the impact of intention-to-harm adverse childhood experiences (ACEs) on the autism-psychosis pathway. Preclinical and genomic studies indicate that neurodevelopmental and genetic vulnerabilities interact with social adversity to heighten risk of psychosis. Observational findings link bullying and abuse to stronger associations between autistic traits (ATs) and psychotic experiences (PEs), though confirmatory research is warranted. Transdiagnostic mental health services should systematically screen for harmful environmental stressors and implement resilience-building strategies for autistic or high-ATs individuals.

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Increasing evidence indicates that autism spectrum disorder (ASD) and psychosis-spectrum conditions frequently co-occur, share transdiagnostic features, and overlap genetic and neurobiological underpinnings. Understanding how common environmental risk factors shape their interplay is therefore of utmost importance.[1-6] In previous research, we proposed a developmental continuity between both conditions, possibly mediated by the endocannabinoid signaling,[7] including heightened liability to cannabis-induced psychosis in autistic people (AP).[8] Building on this, it is worth interrogating whether ASD and autistic traits (ATs) may also represent a neurodevelopmental ground for increased vulnerability to other well-recognized environmental risk factors for psychosis, such as the exposure to adverse childhood experiences (ACEs) and childhood trauma.[9-12] Remarkably, AP face more ACEs than non-autistic people, including parental separation, bullying, domestic and neighborhood violence, psychiatric illness in the household, and financial difficulties.[13-17] The impact of these factors on AP's wellbeing and clinical risk cannot be overlooked, particularly given their lower perceived coping resources and increased

subjective stress.[18-22] Such considerations closely align with a sociodevelopmental-cognitive model of schizophrenia, which posits that neurodevelopmental hazards, social disadvantage, and cognitive processes interact to inform psychosis risk.[23] According to this framework, early neurodevelopmental risk (*i.e.*, genetic liability, perinatal factors) leads to presynaptic dopamine dysregulation, which is then amplified by the exposure to sociodevelopmental adversity. This fosters aberrant salience attribution in the context of biased cognitive schemas, eventually reinforcing psychotic interpretations.[23] Special attention should be paid to ACEs involving clear interpersonal intent to harm (*e.g.*, bullying, physical/sexual/emotional abuse, community/domestic violence), as these have shown a stronger association with later psychosis risk, possibly due to their impact on the perception of social threat and dysregulated emotional and physiological responses to stress.[24-26] Difficulties in social competence among AP, including reduced capacity to identify, interpret, and respond to others' intentions, may amplify the processing and impact of these ACEs, therefore leading to even more detrimental outcomes.[27, 28]

Our scope is to provide a perspective on how intention-to-harm ACEs may contribute to the autism-psychosis continuum, integrating findings from preclinical and clinical evidence. Given the exploratory aims of this reappraisal, a single major medical research database (*i.e.*, Pubmed/MEDLINE) was screened for paper retrieval, which may have resulted in the omission of relevant studies indexed elsewhere. To maximize sensitivity and inclusion, we used broad-meaning search terms encompassing autism, psychosis, and intention-to-harm ACEs.

Neurodevelopmental impact of intention-to-harm: insights from preclinical models and human genetics

A sociodevelopmental-cognitive paradigm is consistent with animal models examining how the social environment may shape autism- and schizophrenia-like phenotypes from a biobehavioral standpoint. Interestingly, several studies employing early-insult animal models of neurodevelopmental disorders (NDDs), either genetically induced (*e.g.*, DLG associated protein 2 [Dlgap2] mutations, Phosphatase and tensin homolog [Pten] mutations),[29, 30] or chemically induced during gestation (*e.g.*, methylazoxymethanol acetate [MAM], valproate acid [VPA]),[31, 32] or socially induced through postnatal stressors (*e.g.*, maternal separation),[33-35] have investigated the effects of the resident-intruder paradigm, conceptualized as a proxy for intention-to-harm stress during late adolescence to early adulthood. Across these studies, the resident-intruder test has been shown to exacerbate frustrative behaviors, aggression, and irritability in neurodevelopmentally sensitized animals. Indeed, such behavioral responses are best understood within a broader neurodevelopmental vulnerability framework, characterized by disrupted gene expression (*e.g.*, CLDN5, CLDN12, MMP9), dysregulated neural activity, heightened neuroinflammatory states, and structural abnormalities in brain regions critical to NDDs pathophysiology (*e.g.*, prefrontal cortex, hypothalamus, orbitofrontal cortex, hippocampus).[29, 31, 33-35] Conversely, "double-hit" neurodevelopmental models specifically assessing whether resident-intruder stress induces additional disruptions in brain or peripheral systems (*e.g.*, immune, endocrine, gastrointestinal) relevant to autism and schizophrenia are underrepresented in literature — or may have been missed by our broad-mapping search — despite well-established evidence that aggressive social confrontation elicits marked structural and functional changes in mesolimbic dopaminergic pathways[36, 37] and serotoninergic signaling in the prefrontal cortex.[38] Moreover, evidence from different intention-to-harm paradigms including chronic social defeat or predator scent exposure as second hit in animal models of NDDs seem to be less described.

Complementing preclinical evidence, findings from human genetics support a model in which ASD and psychotic disorders appear closely intertwined with each other and with early intention-to-harm social exposures. First, polygenic risk scores (PRSs) for ASD and both non-affective (*i.e.*, schizophrenia) and affective (*i.e.*, bipolar disorder) psychosis have each been independently associated with increased expo-

sure to ACEs involving clear interpersonal intent to harm, including physical and emotional abuse and assault. These associations indicate that no single PRS fully explains the link between genetic liability and childhood abuse, supporting a converging vulnerability model where shared genetic risk across different diagnoses fuels susceptibility to harmful early-life events.[39] Second, some genetic variants implicated in ASD and schizophrenia interact with both intentional and non-intentional types of prenatal and postnatal stress to influence subcortical brain development in childhood. Specifically, loci within genes involved in synaptic development, neuronal plasticity, and stress-response signaling (*e.g.*, PRSS12, NDST3, SYNGAP1, CUTA) have been shown to moderate the effects of early life stress on the volume of caudate nucleus and nucleus accumbens — brain regions associated with emotional regulation and reward processing.[40] Finally, the polygenic architecture of childhood maltreatment presents substantial overlap with genetic risk for both ASD and schizophrenia. Intriguingly, AP exposed to childhood maltreatment showed a trend-level increase (P=0.08) in schizophrenia polygenic risk compared to non-exposed peers, suggesting that intention-to-harm ACEs may accentuate genetic liability for psychosis within ASD and reinforce a gene-environment interplay model.[41] Together, evidence from both preclinical and genomic studies supports a model in which neurodevelopmental vulnerability — whether induced genetically or via early-life insult — interacts with interpersonal adversity to shape psychopathological risk along the autism-psychosis continuum.

From bench to bedside: effects of bullying and abuse on the autism-psychosis continuum

In recent years, a growing body of observational studies questioned the impact of bullying/peer victimization (PV) on the pathway between ATs/ASD and PEs[42-44] or psychotic symptoms,[45] in both the general population[42-44] and clinical settings.[45] A large-scale study using longitudinal data from the Tokyo Teen Cohort found that bullying/PV significantly mediated the ATs-PEs association in general population adolescents aged 10 to 14, even after controlling for confounders.[44] Consistently, school-related bullying/PV was associated with an increased risk of inpatient admission for psychotic symptoms in autistic adolescents, suggesting that such psychosocial stressor may serve as acute trigger for severe psychiatric manifestations in neurodevelopmentally vulnerable individuals.[45] Conversely, evidence from both the Avon Longitudinal Study of Parents and Children (ALSPAC) and from the Belgian population-based multiple-birth cohort TwinssCan indicated that, although ATs[42, 43] and bullying/PV[43] in childhood were associated with later PEs, bullying/PV neither explained[42] nor moderated[43] the relationship between ATs and PEs. Nonetheless, findings from the same Belgian cohort pointed to an amplified ATs-PEs association in the presence of emotional, physical, and sexual abuse, indicating a cumulative interaction between neurodevelopmental factors and harmful environmental exposures.[43] Additionally, a further recent study from the ALSPAC cohort found that early ATs and social communication difficulties predicted distressing or frequent PEs in young adulthood, with a cumulative burden of childhood trauma, comprising primarily yet not exclusively intention-to-harm stressors (*i.e.*, domestic violence, abuse, neglect, bullying/PV), mediating up to 36% of this association.[46] Similar traumatic exposures (*i.e.*, sexual/physical/emotional abuse, physical neglect, emotional neglect) did not confound the association between schizotypy (conceptualized as a latent personality organization predisposing to psychosis) and ATs in an adult student sample, potentially supporting a trait-level overlap between the two dimensions.[47]

Overall, despite limited, compelling evidence suggests a developmental trajectory linking autism and psychosis, particularly when shaped by exposure to bullying/PV[44, 45] and various forms of abuse[43, 46] (Table I).[42-47]

Why childhood harm matters: closing gaps and addressing psychosis risk along the autism spectrum

Net of some conflicting[42, 43, 47] and partly inconclusive findings — particularly when non-inten-

TABLE I.—*Summary of observational evidence on intention-to-harm childhood adversities in the autism-psychosis pathway.*[42-47]

Study	Sample size	Age	Gender	Summary of evidence	Model type
Bevan Jones 2012 (UK)[42]	8232	7-12 y	Female: 4043 Male: 4189	Bullying/PV does not mediate the ATs-PEs association	Developmental trajectory – negative
Gong 2017 (China)[47]	2757	Female: 18.74±1.14 y Male: 18.79±1.09 y	Female: 1785 Male: 684	CT [a] does not account for the STs-ATs association	Overlapping vulnerability – negative
Dardani 2022 (UK)[46]	3707	3-24 y	Not applicable	CT [b] partially mediates the ATs-PEs association	Developmental trajectory – positive
Stanyon 2022 (Japan)[44]	3171	10-14 y	Female: 1487 Male: 1684	Bullying/PV mediates the ATs-PEs association in adolescence; indirect effect statistically significant	Developmental trajectory – positive
Bortoletto 2023 (Italy)[45]	69	10-19 y	Female: 13 Male: 56	Bullying/PV at school/work increases risk of hospitalization for psychotic symptoms in ASD adolescents	Developmental trajectory – positive
Karaçam Dogan 2025 (Belgium)[43]	792	17.47±3.60 y	Female: 477 Male: 315	EA, PA, and SA moderate the ATs-PEs association Bullying/PV does not moderate the ATs-PEs association	Developmental trajectory – mixed

ASD: autism spectrum disorder; ATs: autistic traits; CT: childhood trauma; EA: emotional abuse; PA: physical abuse; PEs: psychotic experiences; PV: peer victimization; SA: sexual abuse; STs: schizotypal traits; y: years of age.
[a] Measured using the Childhood Trauma Questionnaire - Short Form (CTQ-SF), including SA, PA, EA, physical neglect, and emotional neglect; [b] measured using a 57-question tool, including domestic violence (regular acts of physical violence taking place in the home), PA, EA, emotional neglect, SA, and bullying/PV.

tion-to-harm adversities are included[46, 47] — the available evidence converges on the notion of a genetic, neurobiological, and clinical autism-psychosis continuum modulated by environmental risk, warranting the need for further robust investigation.

To our knowledge, no studies in AP or individuals with high ATs exposed to intention-to-harm ACEs have yet combined psychometric assessments with biological stress markers (*e.g.*, salivary/serum cortisol, functional measures of altered neural circuitry), an approach that would certainly provide a better understanding of how such stressors shape neurodevelopmental trajectories. Also, future research should clarify the psychological mechanisms linking ATs and PEs in the context of intention-to-harm ACEs. In individuals with high ATs, maladaptive emotion regulation (*e.g.*, rumination, suppression), impaired interpretation of social cues, and low social support may interact with ACEs exposure to facilitate biased cognitive schemas, aberrant salience attribution, threat anticipation, and feelings of social defeat, ultimately increasing vulnerability to paranoid ideation and PEs.[21, 23, 48-53] A deeper knowledge of such mechanisms would have relevant clinical implications for mental health professionals, as they would target possible preventive strategies to enhance coping in individuals with high ATs (*e.g.*, engagement coping, extraversion, optimal parenting), thereby mitigating the risk of comorbid psychopathology or psychosis.[54] Notably, while ATs such as social communication difficulties and restricted/repetitive behaviors exist on a continuum in the general population, a clinical diagnosis of ASD is often associated with greater cognitive-behavioral inflexibility, potentially leading to increased mental health challenges.[55] This highlights the importance of a truly transdiagnostic

approach in mental health services — spanning adolescence, transition to adulthood, and adult care — systematically addressing neurodevelopmental traits and NDDs, and actively screening for environmental risk factors, particularly intention-to-harm ACEs.[56, 57] In fact, exposure to such psychosocial stressors appear to be more strongly linked to externalizing psychiatric outcomes and the occurrence of positive psychotic symptoms via engagement of fear-learning circuits (*e.g.*, amygdala, medioventral prefrontal cortex), compared to deprivation-related ACEs (*e.g.*, neglect), which are more often associated with impaired cognitive control.[24-26] Integrating this risk-factor-driven perspective into assessment and intervention methods would allow clinicians to more precisely identify subgroups of AP or individuals with high ATs who may be at heightened risk for psychosis or PEs, and to implement targeted approaches aimed at building resilience and reducing risk.

Within this broader vulnerability framework, ASD can be understood not only as a distinct neurodevelopmental condition, but also as an at-risk mental trait for psychosis, warranting specific attention in prevention and early intervention efforts, especially in those exposed to harmful environmental stressors.

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Conflicts of interest
The authors certify that there is no conflict of interest with any financial organization regarding the material discussed in the manuscript.

Authors' contributions
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