The Relationship Between Childhood Trauma and The Risk of Developing Psychosis Experiences: Epidemiological, Clinical, Psychodynamic, Neuropsychological, and Biological Perspectives

Mourad Ali Eissa
Professor of Special Education
KIE University
profmouradali@gmail.com

Abstract

Exposure to childhood trauma is common in patients with early psychosis and those with clinical high risk (CHR) for psychosis, with up to 80% of patients exposed to some traumatic experience and is associated with increased symptomatology. Researchers are now investigating the various processes by which childhood adversities may lead to symptoms of psychosis later in life. These include attachment, dissociation, dysfunctional cognitive processes, psychodynamic defenses, problematic coping responses, impaired access to social support, behavioral sensitization and revictimization. In this article, I discuss the results of epidemiological, clinical, psychodynamic, neuropsychological, and biological studies addressing the association between childhood trauma and psychosis.

Keywords. childhood trauma, the risk of developing psychosis experiences, epidemiological, clinical, psychodynamic, neuropsychological, and biological perspectives.

Introduction

The word “trauma” comes from the Greek and means “to damage, to harm;” it also contains a double reference to a wound with a laceration (Perrotta,2019). Childhood trauma is defined as a frightening, dangerous, or violent event that poses a threat to a child’s life or bodily integrity which can capture a range of severe adverse experiences, such as physical, sexual and emotional abuse, neglect, parental death, and bullying, affecting about one-third of the general population (Misiak et al.,2017). In early developmental years, adverse childhood experiences (ACEs) comprise exposure to long-term environmental stressors such as childhood maltreatment, domestic violence, living in a household with ongoing substance abuse, and interpersonal loss. Interpersonal loss entails experiencing parental death, divorce, or mental illness early in life before age 17(Inyang et al.,2022).

Exposure to childhood trauma is common in patients with early psychosis and those with clinical high risk (CHR) for psychosis, with up to 80% of patients having exposed to some traumatic experience, and is associated with increased symptomatology (Michel, et al.,2022). Hence, exposure to trauma may trigger a spectrum of negative reactions, such as dissociation, positive psychotic symptoms, and post-traumatic

stress disorder (PTSD) symptoms; preliminary evidence suggests that cognitive biases in the perception of threat link this complex network of relationships (Liu et al.,2022).

Psychosis is a common feature to many psychiatric, neuropsychiatric, neurologic, neurodevelopmental, and medical conditions. It is the hallmark feature of schizophrenia spectrum and other psychotic disorders, a co-occurring aspect to many mood and substance use disorders, as well as a challenging symptom to many neurologic and medical conditions. Psychosis can result in elevated levels of distress for patients and loved ones, which is why it has become a primary target of treatment for medical professionals (Calabrese & Al Khalili,2022).

The incidence of a first-time psychosis is approximately 50 in 100000 people, while the incidence of schizophrenia is about 15 in 100000 people (McGrath et al.,2004). The peak age of onset for males is teens to mid-twenties, while for females, the onset tends to be teens to late-20's. Earlier onset correlates with poorer outcomes, although early intervention correlates with better results. Psychosis is extremely uncommon in children (Calabrese & Al Khalili,2022). A higher prevalence of childhood trauma has been observed in first-episode psychosis patients compared to healthy ones. This

leads to the suggestion that psychosis may result from trauma experienced in childhood (Thomas et al.,2022).

Prevalence of psychosis-associated trauma symptoms among individuals with full psychotic disorders varies from 11–67% (Mayo et al.,2017). They may be associated with factors such as trauma history prior to inpatient hospitalization (e.g., physical, or sexual abuse) and other psychological factors (e.g., negative event appraisals, poor coping skills) (Mayo et al.,2017).

Cross-sectional studies (e.g., Bebbington et al.,2004; Gracie et al.,2007) have demonstrated that negative perceptions of self, anxiety, and depression partially mediated associations between trauma (not always limited to childhood) and psychotic symptoms. These studies suggest strong relationships between negative personal evaluations and low self-esteem, negative effects, and the characteristics of positive symptoms.

A systematic review found that most people who use mental health services never asked about traumatic experiences such as childhood abuse and neglect, and that people diagnosed with psychotic disorders asked even less than other service users (Campodonico et al.,2022).

ISSN: 3009-612X الترقيم الدولي الموحد للطباعة
E. ISSN: 3009-6146 الترقيم الدولي الموحد الالكتروني

A recent meta-analysis found that people who suffered childhood adversity were significantly more likely to develop psychosis than those who had not (Varese et al.,2012). The meta-analysis also found a dose-response relationship in nine of the ten studies that assessed it. For example, a UK survey of 8580 individuals found that those subjected to two types of adversity (e.g., sexual abuse and bullying) were five-times more likely diagnosed with a psychotic disorder compared with 30-times more likely for three adversities (Shevlin et al. ,2008). A prospective study found (after controlling multiple factors including a family history of psychosis) that adults abused as children were 9.3-times more likely to have 'pathology-level psychosis.' The odds ratios for 'mild' and 'severe' abuse were 2.0 and 48.4, respectively (Janssen et al.,2004).

In recent years, some models focused on the significant role of stressful life events that may influence critical windows of brain development, triggering the onset of psychosis, as well as act during psychosis worsening long-term out-comes (Misiak et al.,2017).

Researchers are now investigating the various processes by which childhood adversities may lead to symptoms of psychosis later in life. These include attachment, dissociation,

dysfunctional cognitive processes, psychodynamic defenses, problematic coping responses, impaired access to social support, behavioral sensitization and revictimization (Bebbington,2009; Morrison ,2009; Read, Fink, Rudegeair & Whitfield, 2008). In this article, I will discuss the results of epidemiological, clinical, neuropsychological, and biological studies addressing the association between childhood trauma and psychosis.

Epidemiological findings:

Some models have proposed to explain the association between childhood traumatic events and psychotic symptoms, including the presence of dysfunctional cognitive patterns, affective dysregulation, insecure attachment style and dissociative mechanisms. Individuals with a personal history of childhood trauma have a threefold increased risk of developing Schizophrenia and tackling child abuse could result in a reduction of approximately 33% of cases of psychosis (Pugliese et al.,2020).

In a meta-analysis of 18 case-control studies (including 2048 patients with psychosis and 1856 non-psychiatric controls), 10 prospective studies (including 41,803 participants), and 8 population-based cross-sectional studies (35,546 participants), Varese et al. (2012) found that adverse

experiences in childhood significantly increased the risk to develop psychosis and schizophrenia. The group showed a significant association between childhood adversity, including trauma, and psychosis: the odds ratio was between 2.72 and 2.99, indicating a strong association between childhood adversity and psychosis, including schizophrenia. Epidemiological studies show that exposure to early stress in the form of abuse and neglect in childhood increases the risk to later develop schizophrenia (Bonoldi et al., 2013). In schizophrenia patients, the most frequent subtype of trauma was emotional neglect, but rates of physical abuse and physical neglect were also significantly increased (Larsson et al., 2013). Childhood abuse and neglect are known to have a negative influence on cognition in patients with schizophrenia and bipolar disorder (Shannon et al., 2011).

Patients with Schizophrenia who experienced abuse and neglect in childhood show more clinical severity than those who have undergone other types of traumatic experiences (Pugliese et al.,2020). Affected individuals with a history of physical abuse, sexual abuse and neglect also manifest prominent cognitive deficits, a reduced speed of processing of information and alterations in working memory (Rossi et al.,2016). Repeated childhood traumas, in fact, cause structural

and functional abnormalities on cerebral functioning (Pugliese et al.,2020).

Positive psychotic symptoms (i.e. hallucinations, delusions, thought insertion, thought control, and telepathic powers) form part of the key clinical presentation of schizophrenia spectrum disorders; however, they can also occur in mood disorders (e.g. major depressive disorder with psychotic features), as well as in non-clinical populations (Liu et al.,2022). Studies suggest that childhood trauma has a significant negative impact on a child's physical and psychological health throughout adolescence and even the entire life course and may be responsible for mental disorders (Wei et al.,2022).

Psychotic-like experiences (PLEs) resemble the positive symptoms of psychosis and are common in adolescents (Kelleher et al.,2012). While Psychotic-like experiences have considered as the mildest manifestation of psychosis tendencies, evidence exists that they are also associated with a wide range of mental health problems (Kalman et al.,2019). For example, Psychotic-like experiences, including unusual beliefs, perceptual abnormalities, and persecutory ideation during childhood, are positively associated with psychotic disorders, depression, and anxiety in adulthood (Fisher et

al.,2013). A number of studies have found that childhood trauma is associated with an increased risk of psychotic disorders (Lu et al.,2020). As suggested by previous research, specific types of traumas, such as witnessing violence (Mojtabai,2006) or emotional abuse (Alemany et al.,2011), may be strongly associated with PLEs.

Early and recent adversities have been associated with each other without an additive effect at the highest level of exposure to recent stressors. These findings might suggest that early childhood adversities may lead to psychosis development either by increasing exposure to later adversity or by making individuals more sensitive to later adversity if it is severe (Lataster et al.,2012).

Recent stressful life events also increase the risk of psychosis, and their effect is amplified by previous exposure to early adversity (Mansueto and Faravelli, 2017; Cullen et al., 2020). The Childhood Adversity and Psychosis (CAPsy) study (Morgan et al., 2020), a population-based case-control study of first-episode psychosis which included assessment of the frequency and severity of exposures, and adjusted for potential confounders of family history of psychosis and parental social class, found strong evidence that all forms of CA were associated with a two- to four-fold increased odds of psychotic

disorder. Exposure to multiple adversities further increased the odds in a linear fashion, and more severe forms of adversity involving threat, hostility and violence were most strongly associated.

Trauma exposure may lead to symptoms of PTSD – namely, re-experiencing and memory intrusions – that may appraise as anomalous experiences (e.g., hearing insults or phrases of past abusers appraised as an external voice in the present moment) (Morrison et al., 2003). Systematic reviews have indicated that PTSD symptoms mediate the relationship between trauma and psychosis (Alameda et al., 2020; Sideli et al., 2020). Systematic reviews suggest that these difficulties correlate with psychotic symptom severity, distress, and content (Hartley et al., 2013) and decrease quality of life (Nevarez-Flores et al., 2019), making affective problems key targets in psychological interventions for psychosis. PTSD is associated with a greater risk of anxiety and depression (Spinhoven et al., 2014), and cPTSD even more so (Karatzias et al., 2019).

Wei et al. (2022) aimed to investigate the epidemiology of childhood trauma and its relationship with insomnia and PLEs in Chinese Zhuang adolescents, focusing on the role of a specific type of trauma and accumulation. A questionnaire of

Childhood Trauma Questionnaire-Short Form (CTQ-SF), Athens Insomnia Scale (AIS), and Chinese Version Community assessment psychic experiences-8 (CCAPE- 8) were all completed by 1,493 Chinese Zhuang adolescents. Chi-square and multivariate logistic regression analyses examined the association between childhood trauma and insomnia/PLEs. The incidences of emotional abuse (EA), physical abuse (PA), sexual abuse (SA), emotional neglect (EN), and physical neglect (PN) occurred at rates of 5.63, 5.02, 6.56, 23.98, and 33.15%, respectively. EA, SA, EN, and PN were all positively related to insomnia (OR: 1.314–7.720, all $p < 0.05$). EA and SA were positively associated with PLEs (OR: 2.131–3.202, all $p < 0.001$). Adolescents who had experienced three or more types of traumas were more likely to have insomnia (OR = 6.961, $p < 0.001$) and PLEs (OR = 3.558, $p < 0.001$).

Clinical Findings:

Childhood trauma and psychopathological symptoms of psychosis.

Childhood traumatic events are risk factors for psychopathology. Studies have indicated that, childhood trauma (particularly childhood sexual abuse) may result in even higher rates of psychosis or psychotic symptoms when it, occurs together with cannabis use (Harley et al.,2010). Cross-

sectional studies have demonstrated that negative perceptions of self, anxiety, and depression partially mediate associations between trauma (not always limited to childhood) and psychotic symptoms (Gracie et al.,2007). They suggest strong relationships between negative personal evaluations and low self-esteem, negative effects, and the characteristics of positive symptoms. Lardinois et al. (2011) found a significant, interaction between daily life stress and childhood trauma on both negative affect, and intensity of symptoms in patients with psychosis, suggesting that, a history of childhood trauma is associated with increased sensitivity to stress.

Biological mechanisms such as reduced cortical thickness(Habets et al.,2011) and dysregulated Cortisol(Faravelli et al.,2010) following exposure to childhood trauma have also been recently investigated which may well facilitate the development of psychosis. Moreover, gene-environmental interactions are likely to play a role in the relationship between childhood trauma and psychosis. In a recent study, Alemany et al.(Alemany et al.,2011) found that the relationship between childhood abuse and psychosis was moderated by the BDNF-Va166Met polymorphism. In a sample of 533 students, met carriers reported more positive psychotic-like experiences when exposed to childhood abuse than did individuals carrying the Val/Val genotype.

Psychotic patients with a history of childhood trauma and/or PTSD have a more severe clinical profile compared with those without these experiences. They report more current or lifetime substance abuse, (Conus et al.,2010) higher levels of current depression and anxiety, (Lysaker & Salyers,2007) and more dissociative symptoms (Schâfer et al.,2011). Childhood sexual abuse has specifically linked to hallucinations and delusions (Mason et al.,2009) and the content of these positive symptoms may be related to patients' traumatic experiences. Psychotic patients with a history of childhood trauma tend to present with a variety of additional problems, like that of other populations with childhood trauma.

Victims of abuse report increased levels of suicidal ideation and more frequent suicide attempts. They have also reported to be less able to sustain intimacy, and to be more prone to emotional instability. A history of childhood abuse is associated with worse overall social functioning, lower remission rates, and poorer compliance with treatment (Schäfer & Fisher,2011).

Daalman et al. (2012) showing that sexual and emotional trauma during childhood serve as a rendering factor for subjects more vulnerable to experience auditory verbal

hallucinations in general − both psychotic and non-psychotic individuals.

Among population-based studies, childhood neglect has found to have an association with psychotic experiences (van Nierop et al. 2014) and with paranoia specifically (Sitko et al. 2014). Differing findings have emerged from clinical population studies. One Dutch study found that there was an association between childhood neglect and hallucinations among patients with a psychotic disorder (Daalman et al. 2012) while a study from New Zealand found no such association with either hallucinations or any other psychotic symptom type (Longden et al. 2015)

Childhood trauma and cognitive functions:

Cognitive functioning comprises numerous interrelated cognitive functions, including intelligence, language, perceptual/visuospatial functions, memory as well as attention and executive functions (EF) (Perfect et al., 2016). On the one hand, trauma may have a stronger or weaker impact on certain cognitive functions that are vulnerable to toxic stress because they rely on brain regions with varying periods of vulnerability to toxic stress. On the other hand, given that toxic stress is expected to detrimentally impact numerous brain regions that are collectively responsible for many cognitive functions,

trauma may have a pervasive impact on several aspects of cognitive functioning (Bick &Nelson, 2016).

The previous reviews mostly included retrospective or cross-sectional design studies; only one systematic review focused exclusively on longitudinal studies (Su et al., 2019). It indicated that childhood maltreatment was associated with poorer overall cognitive functioning. Aas et al. (2012), showing a reduction in cognitive functioning among patients with schizophrenia-spectrum and bipolar disorders, regarding working memory and executive functions.

Masson et al. (2015) systematically reviewed and conducted meta-analyses on the cognitive functioning of children, adolescents, and adults with histories of childhood maltreatment. They found that those with such histories had lower scores in all cognitive functions and further identified that participants assessed at a younger age (0–5 years old vs. adulthood) had poorer cognitive functioning. Because childhood maltreatment was necessarily closer in time for participants assessed between the ages of 0 and 5 years (vs. adulthood), this points to an effect of recency the impact of maltreatment on cognitive functions being stronger when trauma was more recent.

Kavanaugh et al. (2017) instead found convincing evidence for poorer intelligence and attention/EF in maltreated children, whereas findings were not as conclusive for language and memory. In contrast, other reviews did highlight poorer memory in maltreated or trauma-exposed children compared to controls (Irigaray et al., 2013; Perfect et al., 2016). Some reviews further suggest poorer perceptual/visuospatial functions in maltreated children in comparison to controls (Irigaray et al., 2013; Kavanaugh et al., 2017).

Matte-Landry et al. (2022) compare the cognitive outcomes of children with complex trauma and controls and explore whether the timing of trauma (i.e., its onset and recency) moderated this association. Children with complex trauma had poorer overall cognitive functioning than controls, and the timing of trauma (early onset and, to a greater extent, recency of trauma) moderated this association. Thus, findings suggest that children with complex trauma are at risk of cognitive difficulties quickly after trauma exposure.

Psychodynamic perspectives:

From a psychodynamic perspective, the experience of psychosis has long conceptualized as a defense against unbearable or unmanageable emotions (Martindale and Summers 2013) and psychotic symptoms understood to have

meaning in the context of people's lived experiences (Martindale 2007). In some studies, for example, the content of psychotic experiences has found to reflect specific aspects of traumatic and adverse life experiences among both high-risk and clinical populations (Velthorst et al. 2013).

The growth of the hearing voices movement and the rejection of the assumption that psychotic experiences are inherently pathological, have also resulted in the development of new theories about the mechanisms and meaning of auditory verbal hallucinations (AVHs) in particular. For example, in their extensive review on AVHs, Longden , Madill & Waterman (2012) propose that AVHs may be understood, not necessarily as symptoms of a psychotic disorder, but as unconscious dissociative responses to the experience of trauma (Longden et al. 2012), a protective mechanism through which individuals cognitively and affectively disconnect from traumatic events that they are unable to process

Mechanisms linking childhood trauma and psychosis:

Several studies have explored other proposed models explaining the connection between traumatic events and psychotic symptoms (Michel et al.,2022). Models that focus on psychological factors highlight the role of dysfunctional cognitive schemata and affective dysregulation as the primary

psychological mechanisms. Cognitive models suggest that traumatic events drastically alter cognitive schemata in children and adolescents, promoting maladaptive beliefs of self, the world, and their future (Beck's cognitive triad) so that the world is perceived as negative and threatening. These distortions of thought significantly influence the interpretation of internal and external experiences and lay the foundation for persecutory delusions experienced by those struggling with psychosis (Misiak et al.,2017).

Associations between paranoia and negative cognitions about self and others have been well established in clinical and nonclinical samples and it posited that these cognitive distortions may be one of the primary mediators in the relationship between psychosis and trauma (Michel et al.,2022). More specifically, a statistically significant correlation found between negative attributional styles associated with trauma and positive symptoms of psychosis, most notably, hallucinations (Stanton, Denietolis, Goodwin &, Dvir ,2020).

Further, there is a salient relationship between the content of childhood trauma and the subsequent themes of delusions and hallucinations reported. Additionally, recent cognitive models suggest an association between negative schematic

beliefs and a predisposition to both paranoia and hallucinations, with negative beliefs about self and others being most strongly associated with a predisposition to paranoid thoughts. Therefore, persecutory delusions can view as threat beliefs, which may emerge as a response to the experience of interpersonal stress and trauma (Michel et al.,2022).

The link between childhood traumatic events and affect dysregulation might explain the high rates of comorbidity, particularly in disorders with affect dysregulation problems (such as depression, anxiety, and substance use) present in those who experienced childhood trauma with or without psychosis (Michel et al.,2022). The link between childhood traumatic events and affect dysregulation has well established and recent research has also proposed an affective model to describe the connection between trauma and psychosis (Misiak et al.,2017). Emotion regulation is considered a developmental task highly influenced by a safe, emotionally attuned, and regulated caregiving environment. Interpersonal trauma, especially traumatic events occurring within the caregiving environment, disrupt the development of adaptive emotion regulation strategies, resulting in increased risk for emotional dysregulation (Stanton et al.,2020) and therefore, an increased risk of psychosis.

The perception, identification, and interpretation of social cues—are one set of core mechanisms that contribute to the strong association between childhood trauma and transdiagnostic psychopathology (McLaughlin et al.,2020). Children who have experienced trauma can identify expressions of anger or fear with less perceptual information than children who have never experienced trauma; this heightened perceptual sensitivity is specific to threat cues and does not exist for other emotions (e.g., happiness and sadness) (Pollak & Sinha,2002). The magnitude of perceptual sensitivity to threat increases as the severity of trauma increases and persists into adulthood following trauma exposure in childhood (McLaughlin et al.,2020).

Similar patterns have been observed using tasks that assess attentional processes involved in orienting towards and disengaging from emotionally salient stimuli. Children who have experienced trauma exhibit faster attentional orienting to angry facial expressions and vocal cues, but not other emotions, suggesting that their attention is more easily captured by threatening stimuli than children who have never experienced trauma (McLaughlin et al.,2020). Once their attention has captured, children with trauma histories also have more difficulty disengaging from anger cues than children who have not experienced trauma (McLaughlin et al.,2020).

Finally, trauma-exposed children appear to use more liberal criteria for classifying emotional expressions and social situations as threatening (i.e., involving anger); specifically, they are more likely than children who have never experienced trauma to misclassify other negative emotions like sadness and fear and even neutral facial expressions as anger.

Altered patterns of emotional processing, including heightened emotional reactivity, low emotional awareness, and difficulties with emotion regulation, are among well-established transdiagnostic mechanisms linking childhood trauma with psychopathology (McLaughlin et al.,2020). One of the most consistently observed emotional patterns among children with trauma exposure is heightened emotional reactivity, such that salient negative cues in the environment (e.g., angry or fearful faces; social situations depicting people experiencing negative emotions) elicit greater emotional responses in children with trauma histories as compared to children who have never encountered trauma (Sheridan & McLaughlin ,2014).

This heightened emotional reactivity has been observed in studies utilizing behavioral tasks, self-report measures, and experience sampling (i.e., ecological momentary assessment) methods (Weissman et al.,2019) as well as neurobiological

responses, including greater activation in the amygdala and anterior insula, brain regions that encode emotional salience, to negative relative to neutral stimuli (McLaughlin et al.,2020). These patterns have been observed inconsistently in children exposed to other forms of adversity, particularly those involving deprivation (McLaughlin et al.,2015).

Young children exposed to trauma exhibit an earlier emergence of aversive learning—as indexed by the ability to generate a conditioned fear response to a previously neutral cue that predicts an aversive stimulus—than children without such exposure (Machlin et al.,2019). However, by adolescence, trauma-exposed youth exhibit difficulty discriminating between cues that predict threat and safety. For example, during aversive learning, trauma-exposed youth showed less differentiated physiological response between conditioned fear cues and unconditioned safety cues compared to youth without trauma histories (McLaughlin et al.,2020).

The role of neural substrate

Neuroimaging studies in established schizophrenia indicate that socioemotional dysfunction is associated with functional (Anticevic et al., 2012; Hooker et al.,2011; Taylor et al.,2012) and structural alterations within a corticolimbic circuit that includes the medial prefrontal cortex (MPFC),

amygdala, hippocampus, and insula, consistent with postmortem evidence implicating these regions in the disorder.

Preclinical studies in animal models of psychosis suggest that targeting corticolimbic dysregulation during the premorbid phase may prevent the emergence of schizophrenia-like features in adulthood. Human participants at clinical high risk (CHR) for psychosis also show deficits in emotion processing and hyperactivation within corticolimbic regions during emotional tasks that are qualitatively like those seen in patients with schizophrenia (Modinos et al.,2020).

Heightened dopamine activity is related to psychosis. Increased dopamine activity is associated with inducing or exacerbating psychotic symptoms; dopamine agonists produce such effects and dopamine antipsychotics, including typical antipsychotics (which act primarily on dopamine D2 receptors) and atypical antipsychotics (which act in part on D2 receptors), are used to treat psychotic symptoms (Ruby et al.,2014).

Decreased activation of NMDA (N-methyl-D-aspartate) receptors is associated with schizophrenia. In healthy individuals, the administration of ketamine (an NMDA receptor antagonist) induces positive (Adler et al.,1999), negative, and cognitive symptoms. Reductions in the expression of genes involved in glutamate and GABA

neurotransmission have been observed in the prefrontal cortex (PFC) in schizophrenia populations (Mirnics et al.,2000), as decreased expression of GAD67, which plays a role in GABA synthesis via catalyzing decarboxylation of glutamate.

The role of biological mechanisms:

One of the dominant current perspectives on the etiology of psychotic disorders is that of a complex gene environment interaction that influences both the emergence and the persistence of psychotic symptoms over time (Coughlan & Cannon, 2017). From this perspective, no single pathway seen as responsible for the range of psychotic phenomena that individuals experience (Bentall et al. 2014). Rather, it is a dynamic interplay between genetics, epigenetics, exposure to childhood trauma, and the experience of suboptimal environmental factors that cause the activation and later pathologization of psychosis (Millan et al. 2016).

In relation to early trauma and adversity, existing evidence supports the view that exposure to stress, including childhood trauma, maltreatment and living in sub-optimal environmental conditions, can affect human brain, biological, social, cognitive, and emotional development. Certain permutations of these exposures and their effects may sensitize certain individuals to become more vulnerable or prone to

hallucinations, delusions, and psychotic disorders (Coughlan & Cannon, 2017). Along with any preexisting genetic loading, it is the experience of repeated exposure to multiple risk factors from the pre-natal to the early adult years that may place individuals at highest risk for progression to pathological manifestations of psychosis (Millan et al. 2016).

For evolutionary biology, the learning and extinguishing of fear, as well as avoidance behaviors, correspond to response mechanisms indicative of human evolutionary history and are part of an adaptative system whose purpose lies in management of the environment's dangerousness; that is, dealing with the threat of danger and the consequences of an imagined violence in its dimension of triviality, as a quotidian fact and inescapable condition of existence. The epistemological paradigm orienting this perspective of violence wagers on a reversal of the rationality that allowed Lift on to conjure the very disease category of PTSD, since within its territory, trauma was reputed to be "an expected part of our environment of evolutionary adaptation" (Kirmayer , Lemelson &Barad ,2007, p. 12)

Conclusion and recommendation:

There is a dynamic interplay between childhood trauma and other exposures and risk factors; the role of childhood

trauma needs to consider across the continuum of psychotic outcomes and in the context of a range of psychopathological trajectories and outcomes (Coughlan & Cannon, 2017).

Overall, childhood trauma increases the risk for psychosis, affects severity and type of psychotic symptoms, frequency of comorbid conditions, including depression and substance use, and linked to more severe functional impairment in individuals with psychosis (Misiak et al.,2017). Psychotic experiences, especially hallucinatory—and trauma, predict persistence of psychosocial problems in adolescents, underscoring the need to assess psychotic experiences and trauma in mental health screening programs (Michel et al.,2022). The exposure to multiple adverse childhood experiences is associated with linear increases in risk and severity of positive psychotic symptoms, which collectively support a dose-response effect.

From a clinical perspective, the complexity of the relationship between childhood trauma, psychosis and other psychopathology means that precise etiological models for the spectrum of psychotic symptoms and disorders remain elusive.

Whereas most screening programs for psychosocial problems in adolescence focus on internalizing and externalizing problems, the need for additional items concerning psychotic and traumatic experiences is evident

(Mayo et al.,2017) and using evidence-based assessments of trauma to help identify their impact is critical. Assessment of psychotic experiences in children and adolescents can be challenging and the need for more in-depth studies into developmental peculiarities in the early detection and intervention of psychoses in youth remains (Michel et al.,2022).

Several interventions hold promise, in particular psychological interventions and pharmacological approaches that, unlike antipsychotics, target abnormal glutamatergic function (Kantrowitz et al.,205) or oxidative stress (Amminger et al.,2010), as these may be more relevant to the pathophysiology of the early stages of schizophrenia than the abnormal dopaminergic function that underlies later full-blown psychosis. In the coming years, individualized risk assessment for psychosis might follow the lead of personalized medicine, such that risk could be stratified by severity or quantity, especially with the emergence of biomarkers and greater understanding of underlying neural mechanisms. This development should lead to both a reduction in the false positive rate and the development of more effective intervention strategies (Cheryl & Corcoran,2016).

Funding and/or Conflicts of interests/Competing interests:

The author declares that he has no Conflicts of interests.

Data availability statement: Not applicable

Data Availability Statement (DAS)

Data sharing is not applicable (for reviewing articles or theory-based articles).

“No datasets were generated or analyzed during the current study.”

Informed Consent: Not applicable

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