

From hallucinations to delusions: A narrative review of psychotic-like experiences and their implications

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Abstract

Psychotic-like experiences (PLEs) refer to sub-threshold hallucinations and delusions observed in both clinical samples and the general population. Psychotic-like experiences have far-reaching implications for an individual's coping strategies and daily functioning. They are associated with both psychotic and non-psychotic disorders. This article presents a comprehensive review of the current literature on PLEs, incorporating a detailed exploration of the definition, prevalence, risk factors, functional impairments, and comorbid psychiatric disorders. Medline/PubMed and Embase were searched to establish and identify the literature. A total of 108 studies met our inclusion criteria. The genetic and biochemical backgrounds of PLEs are discussed, focusing on gene polymorphisms, changes in brain gyrification and hypothalamic-pituitary–adrenal (HPA) axis dysfunction. Psychological factors, such as trauma exposure, emotion regulation difficulties, cognitive biases, and attachment issues, were thoroughly examined, especially in terms of their impact on the emergence of PLEs. Here, we show how important the clinical aspects of developmental PLEs are, underlining the significance of an increased risk of self-harm and suicidal behaviors in those individuals and the comorbidity of psychiatric disorders in enabling clinicians to discern specific areas to observe. Although there is limited evidence on effective protocols for PLE management, various treatment approaches are explained. Despite increased research on PLEs in recent years, further investigation is needed to fully understand the nature of PLEs and to optimize therapeutic strategies. This article consolidates the current knowledge by synthesizing information on PLEs, including risk factors, comorbidities, treatments, and their impact on individual's lives.

Key words: psychosis, delusions, hallucinations, FK506 binding protein 5, hypothalamic–pituitary–adrenal axis

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Introduction

Psychotic-like experiences (PLEs) are sub-threshold hallucinations and delusions that cannot be classified according to international diagnostic systems due to their low severity, limited duration or absence of associated functional impairment.^{1,2} Positive PLEs include phenomena such as fleeting visual or auditory hallucinations, while negative PLEs include, e.g., blunted affect and physical and social anhedonia.³ In the last decades, the view of PLEs changed from being previously dichotomously diagnosed psychotic symptoms in clinical samples to a continuum of psychotic experiences, applying also to non-help-seeking individuals from the general population.³ The prevalence of PLEs is significantly higher among young people and more frequent among women, and those who are non-married, unemployed, have a high educational level, and low socioeconomic status.^{4–7} Indirect effects from traumatic life events (TLEs), i.e., physical, sexual and emotional abuse, as well as neglect experiences and exposure to PLEs through perceived stress, dissociation, external locus of control, negative self-schemas, and negative other-schemas were found. They include phenomena such as bizarre experiences, perceptual abnormalities, persecutory ideas, and magical thinking.⁸

Psychotic-like experiences have wide implications for an individual, including maladaptive coping strategies and reduced functioning. Many studies have shown a significant association with poorer social and global functioning,^{9,10} as well as impaired social and role functioning.¹¹ Individuals with PLEs have a lower health-related quality of life.¹² In a large study, PLEs correlated with both interpersonal violence and violence towards objects.^{13,14} Childhood PLEs are linked to lower self-esteem and optimism, avoidant coping and school misconduct. They are also associated with poorer school performance, as well as language and mathematical abilities.¹⁵ The presence of PLEs is strongly connected with poorer treatment outcomes, both with pharmacotherapy¹⁶ and psychotherapy.¹⁷

Furthermore, PLEs increase the risk of conversion to a psychotic disorder, and this conversion depends on the severity or persistence of PLEs.¹⁸ Psychotic-like experiences are associated not only with psychosis, but also with non-psychotic disorders such as depressive episodes, post-traumatic stress disorders or personality dysfunctions.¹⁹ In a 2019 meta-analysis, childhood and adolescent PLEs were associated with over a 3-fold increased risk of affective and substance use disorders.²⁰ The presence of PLEs in childhood predicts future mental health service use and psychotropic pharmacological treatment.²¹ Children with PLEs had higher average total healthcare costs during adolescence.¹² In a large study, PLEs were associated with an increased risk of mortality, especially due to suicide and neoplasms. For people experiencing PLEs, the predicted median lifespan was over 5 years shorter.²²

Over the years, various terms have been used when referring to non-clinical psychotic symptoms. Firstly, the term attenuated psychotic symptoms (APS) was introduced as a part of the ultra-high risk (UHR) criteria for developing psychosis in the future.²³ The UHR criteria, including APS and brief, limited intermittent psychotic symptoms (BLIPS), are assessed through clinical interviews. Unfortunately, various modifications of UHR criteria were introduced.²⁴ The term “psychotic-like experiences” was first used by Strauss,²⁵ but the concept has changed throughout the years. In general, there is no consensus regarding the definition and conceptualization of PLEs, which may create inconsistencies in comparing and interpreting the results of different studies.²⁶

Objectives

Recently, the number of publications regarding PLEs has significantly increased. This study aimed to undertake a comprehensive review with particular attention to recent advancements, notably in dynamic fields like genetics, imaging and biochemical studies. Psychological and clinical aspects, as well as the efficacy of the possible treatments are also discussed.

Genetics

There is a growing number of studies showing the association between genetic polymorphisms and prevalence of PLEs. Researchers have examined the effects of possible factors, that may increase the risk of experiencing PLEs, such as: the catechol-O-methyltransferase (COMT) rs4680, the dopamine D2 receptor (DRD2) rs6277 and the dopamine transporter (DAT1) rs28363170 variable number tandem repeat (VNTR) genes.²⁷ Higher severity of PLEs and a history of TLEs correlated with low levels of ASEs in *DAT1* 10R/10R homozygotes. The study implied a probability of increased PLE severity with a history of TLEs only in 10R/10R homozygotes of the *DAT1* 9R/10R VNTR polymorphism. The correlation between a history of TLEs and greater severity of PLEs was found among the *COMT* rs4680 Met allele carriers with high levels of cognitive biases.²⁸ In a study concerning the connection between *COMT* gene rs4680 polymorphism, executive dysfunction and PLEs, a recessive genetic model was found to moderate this relationship in both the overall sample and among women, but not in men.²⁹ The *DRD2* rs6277 C allele was associated with an increased occurrence of PLEs.²⁸ The relationship between zinc finger protein 804A (*ZNF804A*) polymorphisms (rs1344706 and rs7597593) and the positive dimensions of schizotypy and PLEs was investigated.³⁰ Results indicated a significant correlation between rs7597593 and both schizotypy and PLEs, specifically in women. Carriers of the C allele exhibited higher scores in the positive dimension of both

variables compared to TT homozygotes. Research also shows an association between the regulator of G protein signaling 4 (*RGS4*) polymorphisms and PLEs. Individuals carrying the T allele of rs951436 and/or the A allele of rs2661319 demonstrated higher scores on positive and negative PLEs.³¹

There have been numerous studies to investigate the influence of FK506 binding protein 5 (*FKBP5*) gene polymorphisms on PLEs. In a recent cross-sectional study,³² a *FKBP5* gene was found to moderate the correlation between perceived stress and attachment style in those with PLEs. Compared to C allele carriers, there was a higher severity of PLEs in TT homozygotes with the single nucleotide polymorphism rs4713902 and a worse self-efficacy and anxious attachment style. Moreover, rs3800373 GG homozygotes demonstrated a greater incidence of PLEs, compared to T allele carriers, which corresponded with the former findings on the association between an enhanced possibility of developing schizophrenia in G allele carriers.³³ One study³⁴ found that individuals with the rs737054 T allele who experienced emotional neglect exhibited a more severe level of PLEs. Additionally, the rs1360780 CC (rather than the T allele carriers observed in previous studies)^{35,36} and rs9296158 GG homozygotes with a history of physical abuse reported a higher severity of PLEs, which is consistent with prior findings.^{37,38} The interaction between the *FKBP5* haplotype and bullying was also examined and found to be associated with positive PLEs.³⁷

A mega-genome-wide association study³⁹ investigated the genetic overlap between PLEs in adolescents and schizophrenia, bipolar disorder and major depression in adulthood. A polygenic risk score (PRS) analysis showed that the schizophrenia PRS predicted all adolescent PLEs trait domains, while the major depression PRS predicted only 2 of them in adolescence – anhedonia and parent-rated negative symptoms.

Numerous studies have linked various genetic polymorphisms, such as those in *COMT*, *DRD2*, *DAT1*, *ZNF804A*, *RGS4*, and *FKBP5* genes, to the severity and occurrence of PLEs. These associations are often moderated by factors like TLEs and ASEs. Specifically, certain genotypes, such as *DAT1* 10R/10R homozygotes, *COMT* rs4680 Met allele carriers and *FKBP5* TT homozygotes, are associated with increased severity of PLEs, especially in the presence of TLEs. Additionally, there is evidence of genetic overlap between PLEs in adolescents and schizophrenia, bipolar disorder and major depression in adulthood, as indicated in PRS analyses.

Biology/biochemistry

There are only a few studies in the literature regarding the biochemical aspects of PLEs. These studies concentrated mainly on the hypothalamic–pituitary–adrenal (HPA) axis, as its dysregulation is thought to be associated with psychosis.⁴⁰

A study conducted by Collip et al. investigated HPA axis functioning in people at above-average genetic risk for psychotic disorders.⁴¹ Diurnal cortisol profiles and the association between HPA axis activity and subclinical psychotic experiences were examined in the siblings of patients with psychotic disorders and a healthy control group.

The results showed that momentary PLEs were associated with increased cortisol levels, which can be interpreted in 2 directions. One interpretation involves the impact of distress connected with psychotic experiences on increased cortisol secretion. However, after the multi-level analyses, it seemed that subclinical PLEs may have a greater contribution than negative effects on increasing cortisol levels. Another interpretation is that the increased cortisol levels are either involved in the pathogenesis of psychotic experiences or reflect secondary processes. These results could be interpreted as evidence for an association between heightened cortisol secretion, the dopaminergic system and subclinical psychotic experiences, but whether it is causal could not be established in this study.

A study by Thompson et al. examined connections between the experience of stressful events, HPA axis activity, and hippocampal and pituitary volumes in young people who met the UHR criteria of developing a psychotic disorder.⁴² The criteria included the following groups: (a) experienced subthreshold positive psychotic symptoms during the past year, (b) experienced episodes of frank psychotic symptoms that have not lasted longer than a week and have been self-remitting, or (c) have a first degree relative with a psychotic disorder or the identified patient has a schizotypal personality disorder and they have experienced a significant decrease in functioning during the previous year.

The UHR participants who eventually developed psychosis had significantly lower cortisol levels at baseline than those who did not develop a psychotic episode. This confirms the fact that HPA axis dysfunction is involved in the development of psychosis. A significant correlation existed between experiencing minor stressful events and raised cortisol levels. Higher plasma cortisol levels were associated with higher levels of depression and anxiety but not with psychopathology, psychotic symptoms or general functioning. Hippocampal and pituitary volumes were not associated with either plasma cortisol levels or the number of glucocorticoid receptors.

In another study, the aspect of cortical gyrification in adolescents was investigated using magnetic resonance imaging (MRI). The results showed static gyrification changes in individuals with PLEs experiencing voice-hearing, unusual experiences of receiving messages, or persecutory ideas. The cortical gyrification was lower in the frontotemporal regions of the left hemisphere. This group also showed dynamic gyrification changes with higher gyrifications in the right parietal cortex during late adolescence.⁴³ A similar study was carried out by Evermann et al. in an adult sample. Psychotic-like experience distress was associated with lower gyrification of the left

precuneus. The PLE's depression dimension was correlated with lower gyrification in the right supramarginal and temporal region.⁴⁴ Another study, using deformation-based morphometry, demonstrated the role of the uncus in PLE pathogenesis. In this study, PLEs were associated with a reduced expansion of the uncus. The analysis revealed a developmental involvement of the right uncus in the cerebral basis of PLEs.⁴⁵

A limited amount of literature exists on the biochemical aspect of PLEs with a primary focus on the HPA axis. Studies suggest associations between heightened cortisol secretion and PLEs, potentially implicating HPA axis dysfunction in the pathogenesis of psychosis. Additionally, neuroimaging studies have found structural alterations in brain regions such as the frontal-temporal cortex, precuneus and uncus among individuals experiencing PLEs, suggesting a potential neurobiological correlate to these experiences.

Psychological aspects, trauma

There have been numerous studies investigating the association between childhood trauma (CT) and PLEs. Prior research suggests that witnessing violence,⁴⁶ suffering from emotional^{47,48} or sexual abuse, and having experienced 3 or more types of trauma⁴⁹ are predominantly correlated with PLEs. A recent study⁵⁰ comparing the prevalence of delusions and hallucinations in 30-year-olds who had experienced childhood maltreatment with those who had not, showed a higher likelihood of PLEs in self-reported abuse survivors.

As has been previously suggested, there is a strong connection between peer bullying and PLEs, both significantly increasing suicidality in adolescents.⁵¹ Furthermore, research on the correlation between implicit emotion regulation, discrimination and psychopathological symptoms in children showed that greater levels of discrimination predicted a higher endorsement of PLEs.⁵² The association between CT and PLEs during early adulthood was also investigated in terms of wisdom scores in college students. This study showed lower scores of wisdom in those reporting PLEs, wisdom in general was negatively correlated with CT and PLEs, while a decreased wisdom level mediated the association between CT and the occurrence of PLEs.⁵³

A number of authors have recognized the relationship between family functioning (FF) and PLEs. For example, Zhan et al.⁵⁴ examined the association between PLEs, emotion-regulation (ER) strategies among adolescents and the possibility of a parental relationship impact. It indicated that lower use of reappraisal, greater use of suppression and parental conflict or divorce correlated with the number of PLEs endorsed. Moreover, these factors notably predicted a higher level of distress from PLEs. This has also been explored in terms of the COVID-19 pandemic and lockdown in a study by Wu et al.⁵⁵ that not only determined a positive association between increased stress and PLEs but also

showed that better FF may alleviate the adverse influence of elevated perceived stresses on PLEs. Nevertheless, prior research⁵⁶ suggests there might be other aspects that have a mediating effect between FF and PLEs. A significant correlation was found among college students who reported, above all, trouble with sleep quality, interpersonal adaptation and loneliness. Studies concerning the relationship between PLEs and higher levels of loneliness are well documented.^{57–59} However, a recent cross-lagged panel analysis indicated that, although greater feelings of loneliness and less social support predicted more PLEs over time, these variables may be implicated at earlier stages of psychosis risk.⁶⁰ Those findings are consistent with the social deafferentation hypothesis,⁶¹ which suggests that social isolation can contribute to the development of hallucinations and delusions. Additionally, there has been an independent emotional processing pathway identified,⁶² associating higher PLEs and loneliness with lower efficiency of recognizing emotion states, further mediated by higher levels of perceived rejection. On the other hand, despite existing research on the connection between the need for closure (NFC) and the occurrence of delusionality and hallucinations in patients with psychotic disorders, 1 study tested this issue on a larger sample from the general population.⁶³ Psychotic-like experiences, jumping to conclusions (JTC) task, and a full abridged NFC scale, consisting of "Preference for Predictability", "Discomfort with Ambiguity" and "Decisiveness", were used to assess the connection. It identified an insignificant correlation between NFC and PLEs, along with no association with JTC results, as well as a negative relationship between "Decisiveness" scores and the severity of PLEs.

Another study⁶⁴ found that experiencing distress from positive PLEs was linked to more pronounced challenges with emotion regulation, lower reappraisal self-efficacy and less habitual acceptance use. Similarly, distress from negative PLEs correlated with greater difficulties in emotion regulation and less habitual acceptance use. In a retrospective study considering affective lability (AL), the prevalence of PLE subtypes and PLE's contribution to the specific use of emotion regulation strategies in adulthood were studied.⁶⁵ The study has provided evidence that men with "hearing voices" PLEs and women with "special messages" and "bodily changes" PLEs correlated with higher levels of AL, and the use of cognitive reappraisal mediated the relationship between hearing voices during PLEs and AL. These findings suggested a significant negative correlation between the severity of paranoia symptoms and the possibility of utilizing adaptive emotion regulation strategies in men, as well as a significant positive correlation between the severity of paranoia symptoms and the frequency of using maladaptive emotion regulation strategies in women. Prior studies reported corresponding data,^{66–68} indicating AL as a salient clinical characteristic for psychotic disorders. The findings of a prospective study revealed that affective dysregulation (AD) is linked

to an increased probability of developing and maintaining paranoid delusions and auditory hallucinations among adolescents and young adults.⁶⁹ Researchers also suggested a significant correlation between positive psychotic symptoms and higher levels of AL in patients diagnosed with schizophrenia.⁷⁰ Moreover, individuals with high-level PLEs were associated with greater use of emotion suppression and lesser use of acceptance,⁶⁴ which correlates with the former characteristics of psychotic disorders.^{67,71} These findings implied that emotion regulation skills play a protective role in reducing the distress associated with PLEs. Furthermore, they underscore the importance of early psychotherapeutic interventions aimed at addressing emotion regulation difficulties in individuals experiencing PLEs. There is the potential efficacy of cognitive reappraisal in alleviating the impact that PLEs have on AL, which signals the necessity of further studies regarding this subject.

Psychotic symptoms and PLEs are strongly influenced by negative emotions and cognitive biases. A study investigated the moderating effects of psychosis-related cognitive biases, negative affective states and PLEs,⁷² showing that emotional and cognitive processes promote the development of PLEs but do not have a cumulative effect. The study findings indicated that external attribution biases moderated the association between anxiety and positive PLEs, while attention to threat biases moderated the link between depression and positive PLEs, which is in line with former research.^{73–75} Additionally, JTC biases were associated with positive PLEs and served as a moderator for the connection between anxiety and depression, as well as negative PLEs, which corresponds with a previous meta-analysis⁷⁶ and earlier findings.⁷⁴ In a cross-sectional study regarding metacognition and its role in the occurrence of PLEs,⁷⁷ results led to the assertion that metacognitive functioning, as an independent element, negatively predicts PLEs, and therefore may prevent the emergence of PLEs.

Researchers recognized possible prodromal mechanisms that may momentarily precede PLEs, such as aberrant salience (i.e., assigning excessive importance to irrelevant stimuli) and ASEs.⁷⁸ Some authors have also examined the presence of AD, including low resiliency, low reactive control and negative emotionality prior to the development of PLEs.⁷⁹ Affective dysregulation in adolescence predicted the occurrence of PLEs 3 years later. Additionally, PLEs arising during late adolescence were associated with a subsequent increase in AD in young adults.

A connection between PLEs, co-occurring distress, cognitive functioning, and early developmental delays or difficulties in adolescents reporting distressing PLEs has been investigated.⁸⁰ Compared to children without PLEs and those with non-distressing PLEs, children experiencing distressing PLEs had lower receptive language and delayed recall, and were more likely to have speech/motor developmental delays or difficulties. These findings emphasize that integrating cognitive strategies that target mechanisms underlying PLE distress may be beneficial.

In a cross-sectional study investigating the association between the 3-dimensional (cognitive, affective and reflective) wisdom levels and subclinical psychotic symptoms,⁸¹ researchers found that high-level PLEs correlated with a lower wisdom level. Moreover, the occurrence and distress caused by PLEs were found to be negatively correlated with wisdom. Compared to affective and cognitive wisdom, reflective wisdom exhibited a negative correlation with the overall frequency and distress levels of PLEs. The findings to date suggest that individuals with PLEs may have an affective wisdom deficit, while affective wisdom is vital for establishing social connections with others. Therefore, further studies may hold the potential to prevent the progression from a high-risk state to full-blown psychosis. These results are consistent with previous research, showing an association between psychosis-proneness and lack of empathy⁸² or considerable dysfunction in emotion recognition and interpersonal skills.⁸³

Recently, it was examined whether daily-life executive function and attachment difficulties (avoidance and anxiety) can predict PLEs.⁸⁴ Positive PLEs were found to be predicted by greater trouble monitoring behavioral impact, less difficulty completing tasks, greater difficulty regulating emotional reactions, and greater difficulty controlling impulses. Negative PLEs were found to be predicted by greater difficulties in alternating attention, transitioning across situations and regulating emotional reactions. Higher attachment anxiety predicted both positive and negative PLEs, in contrast to individuals with schizophrenia, which was connected with attachment avoidance. This highlights the potential role of the distinction between these attachment difficulties in the maintenance of sub-threshold symptoms and the risk of transitioning to a full-fledged psychotic disorder.

To gain a deeper understanding of risk factors related to suicidal ideations (SIs) and suicidal behaviors (SBs) in youths experiencing PLEs, a study⁸⁵ was conducted indicating that the occurrence of SI and SB increased as participants grew older for those with higher PLE distress. Furthermore, there was a significant association between PLEs at baseline and a progressive deterioration of both SIs and SBs, which was observed to entail a transition from SI to SB. Another study assessed the correlation between Prodromal Questionnaire-Brief Child Version (PQ-BC)⁸⁶ items and PLE distress regarding the prediction of lifetime SIs and SBs.⁸⁷ A significant association was found among items indexing thought control, auditory hallucinations, suspiciousness, and particularly nihilistic thinking/dissociative experiences, which exhibited the most substantial effect. Distress was found to be a partial mediator, as well as a moderator, between overall PLEs and PQ-BC items with SI and SB, underlining the significance of addressing it in suicide prevention endeavors.

Numerous studies have explored the association between childhood traumas, FE, emotion regulation, cognitive biases, and PLEs. Childhood trauma, peer bullying

and discrimination predict a higher endorsement of PLEs, while lower levels of wisdom and emotion regulation difficulties are associated with increased PLE distress. Neurodevelopmental delays and difficulties, as well as attachment anxiety, may contribute to the occurrence and distress of PLEs. Additionally, distress from PLEs is linked to SI and behaviors, with distress acting as both a mediator and moderator in this relationship. These findings underscore the complex interplay of various psychosocial and cognitive factors in the development and maintenance of PLEs, highlighting the importance of early intervention and holistic approaches to address these experiences.

Clinical aspects

Most people with PLEs never develop a psychotic disorder, and approx. 75–90% of developmental PLEs are transitory and benign, but these experiences may become clinically relevant, depending on the level of environmental risk.^{88–90} According to various studies, 1–7% of people with PLEs develop a psychotic disorder.^{91–93} The frequency of transitions depends on PLE trajectories – the group characterized by stable low levels of PLEs had lower percentages of transitions compared to the group with progressively increasing PLEs (1.28% compared to 3.39%).⁹³ Predictors of conversion to psychosis are regular cannabis use in adolescence and a family history of mental illnesses.⁹⁴

What is more, PLEs are associated not only with an increased risk for psychosis but also with other, non-psychotic psychiatric disorders. In a study conducted by Bourgin et al., psychotic-like experiences were significantly correlated with 25 psychiatric disorders, such as mood disorders, anxiety disorders, post-traumatic stress disorder (PTSD), attention deficit hyperactivity disorder (ADHD), personality disorders, as well as substance use disorders and pathological gambling. The prevalence of psychiatric disorders was gradually associated with higher prevalence rates of PLEs.⁴ In a study by Knight et al., patients with depression or anxiety in the presence of PLEs had more severe symptoms of these disorders at the outset than patients without PLEs. The group with PLEs had significantly lower recovery rates and required many more sessions to reach the threshold for recovery.⁹⁵ Psychotic-like experiences are associated with distress and depression, with a strength of this association depending on PLE manifestation.⁹⁶

The meta-analysis from 2019 confirms that PLEs are correlated with a significantly increased possibility of SI, suicide attempts and suicide death.⁹⁷ In another study, the odds of suicide attempts were significantly higher in patients with an anxiety disorder, major depressive disorder or behavioral disorder who were experiencing PLEs compared with patients who did not report PLEs.⁹⁸ Systematic reviews and meta-analyses have shown that individuals with PLEs were at a 2–3-fold increased risk of self-harm and SBs.^{97,99}

Children experiencing PLEs are more likely to experience mental health problems in young adulthood than children without PLEs. Psychotic-like experiences at age 12 increased the risk of low life satisfaction, loneliness, social isolation, sleep problems, overweight, tobacco dependence, parenthood, and low educational attainment. However, many of the associations between childhood PLEs and poor outcomes were explained by familial risk factors.¹⁰⁰

For the time being, most studies did not differentiate between subtypes of PLEs, although they vary significantly according to distress, associated psychopathology, help-seeking and implications for mental health. Future research considering the heterogeneous character of different PLEs may help assess which PLEs are clinically relevant, improving risk screening and therapeutic strategies.¹⁰¹

Psychotic-like experiences are associated with an increased risk of various psychiatric disorders, including psychosis, mood disorders, anxiety disorders, PTSD, ADHD, and substance use disorders. Psychotic-like experiences also correlate with increased distress, depression and suicidality, with children experiencing PLEs more likely to face mental health challenges in adulthood. Differentiating between the subtypes of PLEs may aid in understanding their clinical relevance and improve risk screening and therapeutic interventions.

Psychotherapy and pharmacotherapy

High-risk criteria for psychotic disorders are based on the presence of PLEs.¹⁰² Considerable research efforts have been invested in developing methods of delaying or preventing psychotic illness onset. Intervening earlier may offset the accumulation of damaging personal, social and economic effects.¹⁰³ Intervention during the prodromal stage and the first 3 years, defined as the critical period of illness, has the potential to reduce the ultimate severity.¹⁰⁴

In a meta-analysis from 2013, cognitive behavioral therapy (CBT) had a moderate effect on reducing the transition to psychosis at 12 months. Low-quality evidence for complex psychosocial interventions also suggested that these interventions are associated with a reduced transition to psychosis. Very low-quality evidence suggests a beneficial effect of the supplementation of omega-3 fatty acids during long-term follow-up,¹⁰⁵ which was also confirmed in a meta-analysis from 2019.¹⁰⁶ The therapeutic effect of omega-3 intake may result from altered membrane fluidity and receptor responses following their incorporation into cell membranes. Omega-3 may also interact with the dopaminergic and serotonergic systems through the modulation of arachidonic acid release. Furthermore, there may be an increase in glutathione levels in the temporal lobes, which protects neurons from excitotoxicity and oxidative stress.¹⁰⁷ Although no other treatments have shown any clear effects, most of the studies

included in this review had several problems, mainly an unclear risk of selection bias, a high risk of detection bias, and a high risk of analytics bias owing to incomplete outcome data.

Two meta-analyses from 2018 indicate that, to date, there is no evidence that any specific intervention is particularly effective over the others in preventing the transition to psychosis. The treatments tested were needs-based interventions (NBI) and their combination with omega-3, ziprasidone, olanzapine, aripiprazole, family therapy, D-serine, CBT, and the French & Morrison protocol (CBT-F). Studies investigating integrated psychological interventions, CBT-F + risperidone + NBI and CBT-van der Gaag protocol (CBT-V) + CBT-F + NBI were also included.^{108,109}

In a meta-analysis from 2019, no clear difference between the groups was found in the comparison of antipsychotic drugs (amisulpride, risperidone and olanzapine) connected with specific care packages (non-drug interventions like supportive, empathic listening and counseling) and specific care packages alone. Although a lower number of participants in the intervention group treated with olanzapine and supportive interventions transitioned to psychosis during follow-up compared to the control group (~25% compared to ~40%), these results were imprecise and did not meet levels of statistical significance. Data for the amisulpride-NFI comparison were so few and low-quality that no conclusion could be stated. There was no evidence that adding risperidone to CBT makes a difference in any outcome, including transition to psychosis. All of the studies were rated as a very low quality of evidence by the authors of the meta-analysis using the GRADE approach. The risk of bias was rated as very serious (regarding the randomization method not being described, allocation concealment method not described and high attrition rates). Furthermore, the imprecision in studies was reported. No evidence was found to support the effectiveness of NBIs, CBT, integrated psychological interventions, or family-focused therapy (FFT) in comparison with each other.¹⁰⁶

The most recent meta-analysis from 2020 investigated psychological interventions and analyzed the proportion of remissions from PLEs as a primary outcome and changes in psychotic symptoms, depression, anxiety, functioning, distress, and quality of life as a secondary outcome. The findings were primarily null, except that CBT may reduce the distress associated with PLEs, but it was not effective for any other secondary outcome. The limitations included a lack of studies on this issue, a small number of participants and variable study quality. Only 2 studies provided randomized controlled trial evidence that CBT was effective in remission of PLEs. Generally, no strong evidence was found for the supremacy of any intervention. The authors suggest that, despite its limited effectiveness in preventing transitions to psychosis, CBT may be used to reduce the distress connected with PLEs. The lack of consequential evidence for clinical and functional improvements indicates

a necessity for further research into psychological interventions for PLEs.¹⁰²

Research into delaying or preventing psychotic illness onset emphasizes early intervention during the prodromal stage and the critical period of the first 3 years, potentially reducing ultimate severity. Cognitive behavioral therapy shows promise in reducing the transition to psychosis and alleviating distress associated with PLEs. Omega-3 fatty acid supplementation may have beneficial effects. However, evidence for other interventions is inconclusive, with no clear superiority of any specific treatment. Further research is needed to evaluate the efficacy of interventions for PLEs and their impact on clinical and functional outcomes.

Discussion

Psychotic-like experiences present a significant challenge for both researchers and clinicians due to their varying severity and duration and the absence of associated functional impairments. Numerous studies have connected genetic variations, such as those in *COMT*, *DRD2*, *DAT1*, *ZNF804A*, *RGS4*, and *FKBP5* genes, to the severity and occurrence of PLEs. Certain genotypes, like *DAT1* 10R/10R homozygotes and *COMT* rs4680 Met allele carriers, are linked to an increased PLE severity. Additionally, there is evidence of genetic overlap between PLEs in adolescence and later psychiatric disorders like schizophrenia and depression, as seen through PRS analyses. Research on the biochemical aspect of PLEs, mainly focusing on the HPA axis, suggests an association between heightened cortisol secretion and PLEs. Neuroimaging studies have identified structural brain alterations in individuals with PLEs, particularly in regions like the frontal-temporal cortex, precuneus and uncus.

The association between childhood trauma, FE, emotion regulation, cognitive biases, and PLEs is well documented. Childhood trauma, peer bullying and discrimination predict higher PLE endorsement, while lower wisdom and emotion regulation difficulties contribute to increased PLE distress. Additionally, PLE distress is linked to SIs and SBs, emphasizing the need for early intervention and holistic approaches. Psychotic-like experiences elevate the risk of various psychiatric disorders, including psychosis, mood disorders, anxiety disorders, PTSD, ADHD, and substance use disorders. Further research is crucial to evaluate the efficacy of psychological interventions for PLEs, although CBT and omega-3 fatty acids have demonstrated some efficacy.

This article builds upon previous research by synthesizing findings on the complex interplay of genetic, neurobiological, psychosocial, and cognitive factors in the development and implications of PLEs. It extends our understanding of how various genetic polymorphisms, trauma, family dynamics, emotion regulation, and

cognitive biases contribute to the severity and occurrence of PLEs. Highlighting the association between PLEs and a range of psychiatric disorders, distress and suicidality, it underscores the importance of early intervention and holistic approaches to address these experiences.

The article holds significant importance for both research and clinical practices. It emphasizes the need for a comprehensive understanding of PLEs. The research presented in this paper offers valuable insights into the nature and impact of PLEs on an individual's life. The findings indicate that PLEs have significant functional and psychological implications, including their association with distress, reduced quality of life, and an increased risk of various psychiatric conditions. To address these limitations and advance our understanding of PLEs, several key actions should be considered. Efforts should be made to establish a consensus on the definition and diagnostic criteria of PLEs. A standardized framework will enhance the comparability of research findings and improve the accuracy of PLE diagnoses in clinical settings. Further research is needed to explore the underlying biological mechanisms of PLEs. This may include in-depth investigations into genetic polymorphisms, biochemical pathways and neural correlates associated with PLEs. Continued interdisciplinary collaboration between geneticists, neuroscientists, psychologists, and clinicians is crucial for improving our understanding of the complex etiology and development of PLEs. Leveraging advancements in neuroimaging techniques, genetic sequencing technologies and biomarker identification tools can facilitate the elucidation of biological correlates and pathways associated with PLEs. High-quality, randomized clinical trials are pivotal in assessing the efficacy of diverse treatment approaches for managing PLEs. The inclusion of diverse populations ensures that findings are broadly applicable. Understanding the trajectory of PLEs and their predictive value in the development of psychiatric conditions is crucial. This knowledge informs early intervention strategies and enhances outcomes for individuals at risk. Implementing early intervention strategies that target individuals at risk for PLEs, such as those with a history of trauma or familial predisposition, can help alleviate the progression of symptoms and reduce the likelihood of developing psychiatric disorders. This article underlines the importance of adopting a holistic approach to intervention and prevention efforts.

Research on PLEs has expanded considerably in recent years, fueled by the growing recognition of their prevalence and clinical significance. Studies have shown various factors contributing to the occurrence and persistence of PLEs. They are relatively common in the general population; therefore, understanding them is crucial for several reasons. While most individuals who experience PLEs do not go on to develop a psychotic disorder, these experiences can still cause significant distress and impairment in daily functioning. Psychotic-like experiences can have

a significant impact on the individual's quality of life, affecting various domains, including social relationships, academic or occupational functioning, and overall well-being. Research aimed at understanding and addressing PLEs can lead to an improved quality of life. Moreover, PLEs are considered to be an important risk factor for the development of psychotic disorders. Research on PLEs can help in the early detection of individuals at risk for developing psychosis. Early intervention strategies, informed by research findings, can potentially prevent or mitigate the progression of symptoms and improve long-term outcomes. Psychotic-like experiences also represent important risk factors for non-psychotic disorders, including a number of conditions such as depressive episodes, PTSDs, personality dysfunctions, and substance use disorders. Research on the co-occurrence of PLEs with non-psychotic disorders can improve the development of comprehensive treatment approaches that address both psychotic and non-psychotic symptoms simultaneously. This holistic approach can improve treatment outcomes and the overall wellbeing of individuals experiencing these comorbidities. Psychotic-like experiences have also been linked to an increased risk of SBs. By investigating the relationship between PLEs and SIs, attempts and completed suicides, researchers can identify factors that contribute to this risk and develop targeted interventions to prevent suicide and promote mental health. Furthermore, understanding the biological, psychological and social factors involved in PLEs can help reduce the stigma surrounding this mental illness. Increased awareness and knowledge can foster empathy, support and acceptance for individuals experiencing PLEs and related conditions.

Limitations




This review also has several limitations that need to be considered. One of the major limitations in studying PLEs is the heterogeneity of experiences, ranging from mild perceptual abnormalities to more distressing hallucinations and delusions. First, the lack of a consensus regarding the definition and diagnostic criteria for PLEs further complicates matters and hinders the comparability of research findings. Second, many studies in this review focused on specific subpopulations, and while these studies provide valuable insights into potential risk factors, they may not fully represent the diversity of individuals with PLEs in the general population, limiting the generalizability of the findings. Third, although the review highlights some genetic and biochemical factors associated with PLEs, the field is still in the early stages of understanding these mechanisms and further research is needed. Fourth, the paper discusses the potential effectiveness of CBT in alleviating distress related to PLEs but notes the lack of robust evidence for other treatment methods. This limitation underscores the need for further research to identify effective interventions for individuals experiencing PLEs.

Conclusions

This review highlights the complex nature of PLEs and their significant impact on individuals. Psychotic-like experiences have profound functional implications, leading to maladaptive coping strategies, impaired social and global functioning, lower health-related quality of life, and higher violence risk. They are associated with psychosis and a variety of non-psychotic psychiatric disorders. The genetic and biochemical aspects of PLEs have garnered attention, with studies revealing associations between specific gene polymorphisms and dysregulated HPA axis activities, especially elevated cortisol levels. Imaging studies have shown lower cortical gyrification in frontal-temporal regions of the left hemisphere, left precuneus, right supramarginal and temporal regions, as well as the uncus. While CBT has shown promise in managing PLEs, there is a lack of strong evidence supporting the efficacy of other procedures.

Future studies are required to better understand the trajectory of PLEs and their predictive value in developing psychotic disorders and other psychiatric conditions. Efforts should be made to establish a consensus on the definition and diagnostic criteria of PLEs. Furthermore, high-quality, randomized clinical trials are necessary to evaluate the efficacy of different treatment approaches in managing PLEs and improving outcomes. In conclusion, while this study presents several challenges and limitations, it also offers promising avenues for research and clinical practice. The continued exploration of the biological, psychological and clinical aspects of PLEs is essential for providing effective interventions and improving the lives of individuals experiencing these phenomena.

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References

- Kelleher I, Cannon M. Psychotic-like experiences in the general population: Characterizing a high-risk group for psychosis. *Psychol Med*. 2011;41(1):1–6. doi:10.1017/S0033291710001005
- Misiak B, Frydecka D, Kowalski K, Samochowiec J, Jabłoński M, Gawęda Ł. Associations of neurodevelopmental risk factors with psychosis proneness: Findings from a non-clinical sample of young adults. *Compr Psychiatry*. 2023;123:152385. doi:10.1016/j.comppsy.2023.152385
- Van Os J, Hanssen M, Bijl RV, Ravelli A. Strauss (1969) revisited: A psychosis continuum in the general population? *Schizophr Res*. 2000;45(1–2):11–20. doi:10.1016/S0920-9964(99)00224-8
- Bourgin J, Tebeka S, Mallet J, Mazer N, Dubertret C, Le Strat Y. Prevalence and correlates of psychotic-like experiences in the general population. *Schizophr Res*. 2020;215:371–377. doi:10.1016/j.schres.2019.08.024
- Kelleher I, Connor D, Clarke MC, Devlin N, Harley M, Cannon M. Prevalence of psychotic symptoms in childhood and adolescence: A systematic review and meta-analysis of population-based studies. *Psychol Med*. 2012;42(9):1857–1863. doi:10.1017/S0033291711002960
- Stainton A, Chisholm K, Woodall T, et al. Gender differences in the experience of psychotic-like experiences and their associated factors: A study of adolescents from the general population. *Schizophr Res*. 2021;228:410–416. doi:10.1016/j.schres.2021.01.008
- Loch AA, Chianca C, Alves TM, et al. Poverty, low education, and the expression of psychotic-like experiences in the general population of São Paulo, Brazil. *Psychiatry Res*. 2017;253:182–188. doi:10.1016/j.psychres.2017.03.052
- Yung AR, Nelson B, Baker K, Buckby JA, Baksheev G, Cosgrave EM. Psychotic-like experiences in a community sample of adolescents: Implications for the continuum model of psychosis and prediction of schizophrenia. *Aust N Z J Psychiatry*. 2009;43(2):118–128. doi:10.1080/00048670802607188
- Carey E, Gillan D, Healy C, et al. Early adult mental health, functional and neuropsychological outcomes of young people who have reported psychotic experiences: A 10-year longitudinal study. *Psychol Med*. 2021;51(11):1861–1869. doi:10.1017/S0033291720000616
- Healy C, Campbell D, Coughlan H, Clarke M, Kelleher I, Cannon M. Childhood psychotic experiences are associated with poorer global functioning throughout adolescence and into early adulthood. *Acta Psychiatr Scand*. 2018;138(1):26–34. doi:10.1111/acps.12907
- Chun CA, Cooper S, Ellman LM. Associations of psychotic-like experiences, related symptoms, and working memory with functioning. *Eur Psychiatry*. 2020;63(1):e20. doi:10.1192/j.eurpsy.2020.21
- Rimvall MK, Wolf RT, Olsen EM, et al. Healthcare costs, school performance, and health-related quality of life in adolescence following psychotic experiences in preadolescence: A longitudinal cohort study. *Schizophr Bull*. 2021;47(3):682–691. doi:10.1093/schbul/sbaa175
- Kinoshita Y, Shimodera S, Nishida A, et al. Psychotic-like experiences are associated with violent behavior in adolescents. *Schizophr Res*. 2011;126(1–3):245–251. doi:10.1016/j.schres.2010.08.028
- Dolphin L, Dooley B, Fitzgerald A. Prevalence and correlates of psychotic like experiences in a nationally representative community sample of adolescents in Ireland. *Schizophr Res*. 2015;169(1–3):241–247. doi:10.1016/j.schres.2015.09.005
- Steenkamp LR, Bolhuis K, Blanken LME, et al. Psychotic experiences and future school performance in childhood: A population-based cohort study. *J Child Psychol Psychiatry*. 2021;62(3):357–365. doi:10.1111/jcpp.13281
- Perlis RH, Uher R, Ostacher M, et al. Association between bipolar spectrum features and treatment outcomes in outpatients with major depressive disorder. *Arch Gen Psychiatry*. 2010;68(4):351. doi:10.1001/archgenpsychiatry.2010.179
- Wigman JT, Van Os J, Abidi L, et al. Subclinical psychotic experiences and bipolar spectrum features in depression: Association with outcome of psychotherapy. *Psychol Med*. 2014;44(2):325–336. doi:10.1017/S0033291713000871
- Kaymaz N, Drukker M, Lieb R, et al. Do subthreshold psychotic experiences predict clinical outcomes in unselected non-help-seeking population-based samples? A systematic review and meta-analysis, enriched with new results. *Psychol Med*. 2012;42(11):2239–2253. doi:10.1017/S0033291711002911
- Bhavsar V, Dorrington S, Morgan C, et al. Psychotic experiences, psychiatric comorbidity and mental health need in the general population: A cross-sectional and cohort study in Southeast London. *Psychol Med*. 2021;51(1):147–157. doi:10.1017/S0033291719003106
- Healy C, Brannigan R, Dooley N, et al. Childhood and adolescent psychotic experiences and risk of mental disorder: A systematic review and meta-analysis. *Psychol Med*. 2019;49(10):1589–1599. doi:10.1017/S0033291719000485
- Rimvall MK, Van Os J, Verhulst F, et al. Mental health service use and psychopharmacological treatment following psychotic experiences in preadolescence. *Am J Psychiatry*. 2020;177(4):318–326. doi:10.1176/appi.ajp.2019.19070724
- Sharifi V, Eaton WW, Wu LT, Roth KB, Burchett BM, Mojtabai R. Psychotic experiences and risk of death in the general population: 24–27 year follow-up of the Epidemiologic Catchment Area study. *Br J Psychiatry*. 2015;207(1):30–36. doi:10.1192/bjp.bp.113.143198
- Yung AR, McGorry PD, McFarlane CA, Jackson HJ, Patton GC, Rakkar A. Monitoring and care of young people at incipient risk of psychosis. *Schizophr Bull*. 1996;22(2):283–303. doi:10.1093/schbul/22.2.283
- Schultze-Lutter F, Schimmelmann BG, Ruhrmann S. The near Babylonian speech confusion in early detection of psychosis. *Schizophr Bull*. 2011;37(4):653–655. doi:10.1093/schbul/sbr039
- Strauss JS. Hallucinations and delusions as points on continua function: Rating scale evidence. *Arch Gen Psychiatry*. 1969;21(5):581. doi:10.1001/archpsyc.1969.01740230069010

26. Hinterbuchinger B, Mossaheb N. Psychotic-like experiences: A challenge in definition and assessment. *Front Psychiatry*. 2021;12:582392. doi:10.3389/fpsy.2021.582392
27. Frydecka D, Kotowicz K, Gawęda Ł, et al. Effects of interactions between variation in dopaminergic genes, traumatic life events, and anomalous self-experiences on psychosis proneness: Results from a cross-sectional study in a nonclinical sample. *Eur Psychiatry*. 2020;63(1):e104. doi:10.1192/j.eurpsy.2020.103
28. Kotowicz K, Frydecka D, Gawęda Ł, et al. Effects of traumatic life events, cognitive biases and variation in dopaminergic genes on psychosis proneness. *Early Interv Psychiatry*. 2021;15(2):248–255. doi:10.1111/eip.12925
29. Gong J, Zhang T, Zhou L, et al. Gender divergent effect of COMT gene rs4680 polymorphism on the association between executive dysfunction and psychotic-like experiences. *Behav Brain Res*. 2023; 439:114215. doi:10.1016/j.bbr.2022.114215
30. De Castro-Catala M, Mora-Solano A, Kwapil TR, et al. The genome-wide associated candidate gene *ZNF804A* and psychosis-proneness: Evidence of sex-modulated association. *PLoS One*. 2017;12(9): e0185072. doi:10.1371/journal.pone.0185072
31. De Castro-Catala M, Cristóbal-Narváez P, Kwapil TR, et al. Association between *RGS4* variants and psychotic-like experiences in nonclinical individuals. *Eur Arch Psychiatry Clin Neurosci*. 2017;267(1):19–24. doi:10.1007/s00406-016-0676-7
32. Stramecki F, Misiak B, Gawęda Ł, et al. The moderating role of the *FKBP5* gene polymorphisms in the relationship between attachment style, perceived stress and psychotic-like experiences in non-clinical young adults. *J Clin Med*. 2022;11(6):1614. doi:10.3390/jcm11061614
33. Mihaljevic M, Zeljkic K, Soldatovic I, et al. The emerging role of the *FKBP5* gene polymorphisms in vulnerability–stress model of schizophrenia: Further evidence from a Serbian population. *Eur Arch Psychiatry Clin Neurosci*. 2017;267(6):527–539. doi:10.1007/s00406-016-0720-7
34. Stramecki F, Frydecka D, Gawęda Ł, et al. The Impact of the *FKBP5* gene polymorphisms on the relationship between traumatic life events and psychotic-like experiences in non-clinical adults. *Brain Sci*. 2021;11(5):561. doi:10.3390/brainsci11050561
35. Alemany S, Moya J, Ibáñez MI, et al. Research Letter: Childhood trauma and the rs1360780 SNP of *FKBP5* gene in psychosis: A replication in two general population samples. *Psychol Med*. 2016;46(1):221–223. doi:10.1017/S0033291715001695
36. King DC, Taylor J, Elnitski L, Chiaromonte F, Miller W, Hardison RC. Evaluation of regulatory potential and conservation scores for detecting cis-regulatory modules in aligned mammalian genome sequences. *Genome Res*. 2005;15(8):1051–1060. doi:10.1101/gr.3642605
37. Cristóbal-Narváez P, Sheinbaum T, Myin-Germeys I, et al. The role of stress-regulation genes in moderating the association of stress and daily-life psychotic experiences. *Acta Psychiatr Scand*. 2017; 136(4):389–399. doi:10.1111/acps.12789
38. Cristóbal-Narváez P, Sheinbaum T, Rosa A, et al. The interaction between childhood bullying and the *FKBP5* gene on psychotic-like experiences and stress reactivity in real life. *PLoS One*. 2016;11(7): e0158809. doi:10.1371/journal.pone.0158809
39. Pain O, Dudbridge F, Cardno AG, et al. Genome-wide analysis of adolescent psychotic-like experiences shows genetic overlap with psychiatric disorders. *Am J Med Genet B Neuropsychiatr Genet*. 2018;177(4): 416–425. doi:10.1002/ajmg.b.32630
40. Ciufolini S, Dazzan P, Kempton MJ, Pariante C, Mondelli V. HPA axis response to social stress is attenuated in schizophrenia but normal in depression: Evidence from a meta-analysis of existing studies. *Neurosci Biobehav Rev*. 2014;47:359–368. doi:10.1016/j.neubiorev.2014.09.004
41. Collip D, Nicolson NA, Lardinois M, Lataster T, Van Os J, Myin-Germeys I. Daily cortisol, stress reactivity and psychotic experiences in individuals at above average genetic risk for psychosis. *Psychol Med*. 2011;41(11):2305–2315. doi:10.1017/S0033291711000602
42. Thompson KN, Phillips LJ, Komesaroff P, et al. Stress and HPA-axis functioning in young people at ultra high risk for psychosis. *J Psychiatr Res*. 2007;41(7):561–569. doi:10.1016/j.jpsychires.2006.05.010
43. Maitra R, Horne CM, O'Daly O, et al. Psychotic like experiences in healthy adolescents are underpinned by lower fronto-temporal cortical gyrification: A study from the IMAGEN Consortium. *Schizophr Bull*. 2023;49(2):309–318. doi:10.1093/schbul/sbac132
44. Evermann U, Gaser C, Besteher B, Langbein K, Nenadić I. Cortical gyrification, psychotic-like experiences, and cognitive performance in non-clinical subjects. *Schizophr Bull*. 2020;46(6):1524–1534. doi:10.1093/schbul/sbaa068
45. Yu T, Jia T, Zhu L, et al. Cannabis-associated psychotic-like experiences are mediated by developmental changes in the parahippocampal gyrus. *J Am Acad Child Adolesc Psychiatry*. 2020;59(5):642–649. doi:10.1016/j.jaac.2019.05.034
46. Mojtabai R. Psychotic-like experiences and interpersonal violence in the general population. *Soc Psychiat Epidemiol*. 2006;41(3):183–190. doi:10.1007/s00127-005-0020-4
47. Alemany S, Arias B, Aguilera M, et al. Childhood abuse, the BDNF-Val66Met polymorphism and adult psychotic-like experiences. *Br J Psychiatry*. 2011;199(1):38–42. doi:10.1192/bjp.bp.110.083808
48. Kessler RC, McLaughlin KA, Green JG, et al. Childhood adversities and adult psychopathology in the WHO World Mental Health Surveys. *Br J Psychiatry*. 2010;197(5):378–385. doi:10.1192/bjp.bp.110.080499
49. Wei Q, Pan Y, Zhang S, et al. Epidemiology of childhood trauma and its association with insomnia and psychotic-like experiences in Chinese Zhuang adolescents. *Front Psychiatry*. 2022;13:974674. doi:10.3389/fpsy.2022.974674
50. Kisely S, Strathearn L, Najman JM. A comparison of psychosis-like symptoms following self-reported and agency-notified child abuse in a population-based birth cohort at 30-year-follow-up. *Schizophr Res*. 2022;239:116–122. doi:10.1016/j.schres.2021.11.029
51. Csillag C, Nordentoft M, Mizuno M, et al. Early intervention services in psychosis: From evidence to wide implementation. *Early Interv Psychiatry*. 2016;10(6):540–546. doi:10.1111/eip.12279
52. Vargas TG, Mittal VA. Testing whether implicit emotion regulation mediates the association between discrimination and symptoms of psychopathology in late childhood: An RDoC perspective. *Dev Psychopathol*. 2021;33(5):1634–1647. doi:10.1017/S0954579421000638
53. Zhang J, Liu Z, Long Y, et al. Mediating role of impaired wisdom in the relation between childhood trauma and psychotic-like experiences in Chinese college students: A nationwide cross-sectional study. *BMC Psychiatry*. 2022;22(1):655. doi:10.1186/s12888-022-04270-x
54. Zhan C, Mao Z, Zhao X, Shi J. Association between parents' relationship, emotion-regulation strategies, and psychotic-like experiences in adolescents. *Children (Basel)*. 2022;9(6):815. doi:10.3390/children9060815
55. Wu Z, Zou Z, Wang F, et al. Family functioning as a moderator in the relation between perceived stress and psychotic-like experiences among adolescents during COVID-19. *Compr Psychiatry*. 2021;111:152274. doi:10.1016/j.comppsych.2021.152274
56. Liu S, Hu Y. The relationship between family functioning and psychotic-like experiences of college students: Chain multiple mediating effects. *Early Interv Psychiatry*. 2023;17(6):608–616. doi:10.1111/eip.13355
57. Lim MH, Gleeson JFM, Alvarez-Jimenez M, Penn DL. Loneliness in psychosis: A systematic review. *Soc Psychiatry Psychiatr Epidemiol*. 2018;53(3):221–238. doi:10.1007/s00127-018-1482-5
58. Narita Z, Stickley A, DeVlyder J. Loneliness and psychotic experiences in a general population sample. *Schizophr Res*. 2020;218:146–150. doi:10.1016/j.schres.2020.01.018
59. Granholm E, Holden JL, Mikhael T, et al. What do people with schizophrenia do all day? Ecological momentary assessment of real-world functioning in schizophrenia. *Schizophr Bull*. 2019;46(2):242–251. doi:10.1093/schbul/sbz070
60. Tan M, Barkus E, Favelle S. The cross-lagged relationship between loneliness, social support, and psychotic-like experiences in young adults. *Cogn Neuropsychiatry*. 2021;26(6):379–393. doi:10.1080/13546805.2021.1960156
61. Hoffman RE. A social deafferentation hypothesis for induction of active schizophrenia. *Schizophr Bull*. 2007;33(5):1066–1070. doi:10.1093/schbul/sbm079
62. Leathem LD, Currin DL, Montoya AK, Karlsgodt KH. Socioemotional mechanisms of loneliness in subclinical psychosis. *Schizophr Res*. 2021;238:145–151. doi:10.1016/j.schres.2021.10.002
63. Daren A, Mętel D, Bładziński P, et al. The relationships of need for closure dimensions with psychotic-like experiences and jumping to conclusions: A study of a large cross-population sample. *Psychiatr Pol*. 2021;55(2):287–307. doi:10.12740/PP/131533

64. Osborne KJ, Willroth EC, DeVlyder JE, Mittal VA, Hilimire MR. Investigating the association between emotion regulation and distress in adults with psychotic-like experiences. *Psychiatry Res.* 2017;256: 66–70. doi:10.1016/j.psychres.2017.06.011
65. Gong J, Luo Y, He Y, Zhou L, Zhao L, Liu J. Epidemiology of psychotic-like experiences by subtypes and their relationship with emotional regulation and affective lability among Chinese college students. *Schizophr Res.* 2022;244:39–45. doi:10.1016/j.schres.2022.02.037
66. Westermann S, Rief W, Lincoln TM. Emotion regulation in delusion-proneness: Deficits in cognitive reappraisal, but not in expressive suppression. *Psychol Psychother.* 2014;87(1):1–14. doi:10.1111/papt.12000
67. O'Driscoll C, Laing J, Mason O. Cognitive emotion regulation strategies, alexithymia and dissociation in schizophrenia: A review and meta-analysis. *Clin Psychol Rev.* 2014;34(6):482–495. doi:10.1016/j.cpr.2014.07.002
68. Van Der Velde J, Opmeer EM, Liemburg EJ, et al. Lower prefrontal activation during emotion regulation in subjects at ultrahigh risk for psychosis: An fMRI-study. *NPJ Schizophr.* 2015;1(1):15026. doi:10.1038/npschz.2015.26
69. Van Rossum I, Dominguez MdG, Lieb R, Wittchen HU, Van Os J. Affective dysregulation and reality distortion: A 10-year prospective study of their association and clinical relevance. *Schizophr Bull.* 2011;37(3): 561–571. doi:10.1093/schbul/sbp101
70. Høegh MC, Melle I, Aminoff SR, et al. Affective lability across psychosis spectrum disorders. *Eur Psychiatry.* 2020;63(1):e53. doi:10.1192/j.eurpsy.2020.44
71. Van Der Meer L, Wout MV, Aleman A. Emotion regulation strategies in patients with schizophrenia. *Psychiatry Res.* 2009;170(2–3):108–113. doi:10.1016/j.psychres.2009.07.010
72. Prochwicz K, Kłosowska J. The moderating role of cognitive biases on the relationship between negative affective states and psychotic-like experiences in non-clinical adults. *Psychiatry Res.* 2018;265: 118–127. doi:10.1016/j.psychres.2018.04.023
73. An SK, Kang JI, Park JY, Kim KR, Lee SY, Lee E. Attribution bias in ultrahigh risk for psychosis and first-episode schizophrenia. *Schizophr Res.* 2010;118(1–3):54–61. doi:10.1016/j.schres.2010.01.025
74. Gawęda Ł, Prochwicz K, Cella M. Cognitive biases mediate the relationship between temperament and character and psychotic-like experiences in healthy adults. *Psychiatry Res.* 2015;225(1–2):50–57. doi:10.1016/j.psychres.2014.10.006
75. Kinderman P, Bentall RP. Causal attributions in paranoia and depression: Internal, personal, and situational attributions for negative events. *J Abnorm Psychol.* 1997;106(2):341–345. doi:10.1037/0021-843X.106.2.341
76. McLean BF, Mattiske JK, Balzan RP. Association of the jumping to conclusions and evidence integration biases with delusions in psychosis: A detailed meta-analysis. *Schizophr Bull.* 2016;43(2):344–354. doi:10.1093/schbul/sbw056
77. Giugliano M, Contrada C, Foglia L, et al. Metacognitive abilities as a protective factor for the occurrence of psychotic-like experiences in a non-clinical population. *Front Psychol.* 2022;13:805435. doi:10.3389/fpsyg.2022.805435
78. Meyer MS, Cohn JR, Hayashi K, Cicero DC. Momentary assessment of aberrant salience, anomalous self-experiences, and psychotic-like experiences. *Personal Disord.* 2022;13(6):641–653. doi:10.1037/per0000530
79. Bornheimer LA, Martz ME, Suzuki T, et al. Affective dysregulation precedes emergence of psychosis-like experiences in a community sample of young adults. *Schizophr Bull.* 2022;48(3):664–672. doi:10.1093/schbul/sbac015
80. Barnes GL, Stewart C, Browning S, et al. Distressing psychotic-like experiences, cognitive functioning and early developmental markers in clinically referred young people aged 8–18 years. *Soc Psychiatry Psychiatr Epidemiol.* 2022;57(3):461–472. doi:10.1007/s00127-021-02168-9
81. Wu Z, Jiang Z, Wang Z, et al. Association between wisdom and psychotic-like experiences in the general population: A cross-sectional study. *Front Psychiatry.* 2022;13:814242. doi:10.3389/fpsyg.2022.814242
82. Derntl B, Michel TM, Premph P, et al. Empathy in individuals clinically at risk for psychosis: Brain and behaviour. *Br J Psychiatry.* 2015; 207(5):407–413. doi:10.1192/bjp.bp.114.159004
83. Glenthøj LB, Fagerlund B, Hjørthøj C, et al. Social cognition in patients at ultra-high risk for psychosis: What is the relation to social skills and functioning? *Schizophr Res Cogn.* 2016;5:21–27. doi:10.1016/j.scog.2016.06.004
84. Blair MA, Nitzburg G, DeRosse P, Karlsgodt KH. Relationship between executive function, attachment style, and psychotic like experiences in typically developing youth. *Schizophr Res.* 2018;197:428–433. doi:10.1016/j.schres.2018.02.026
85. Karcher NR, O'Hare K, Jay SY, Grattan R. Strengthening associations between psychotic like experiences and suicidal ideation and behavior across middle childhood and early adolescence. *Psychol Med.* 2023;53(13):6002–6010. doi:10.1017/S0033291722003166
86. Karcher NR, Barch DM, Avenevoli S, et al. Assessment of the Prodromal Questionnaire-Brief Child Version for Measurement of Self-reported Psychotic-like Experiences in Childhood. *JAMA Psychiatry.* 2018;75(8):853. doi:10.1001/jamapsychiatry.2018.1334
87. Jay SY, Schiffman J, Grattan R, et al. A deeper dive into the relation between psychotic-like experiences and suicidal ideation and behaviors in children across the United States. *Schizophr Bull.* 2022; 48(6):1241–1251. doi:10.1093/schbul/sbac090
88. Van Os J, Linscott RJ, Myin-Germeys I, Delespaul P, Krabbendam L. A systematic review and meta-analysis of the psychosis continuum: Evidence for a psychosis proneness–persistence–impairment model of psychotic disorder. *Psychol Med.* 2009;39(2):179–195. doi:10.1017/S0033291708003814
89. Hui C, Morcillo C, Russo DA, et al. Psychiatric morbidity, functioning and quality of life in young people at clinical high risk for psychosis. *Schizophr Res.* 2013;148(1–3):175–180. doi:10.1016/j.schres.2013.05.026
90. Yung AR, Yuen HP, Berger G, et al. Declining transition rate in ultra high risk (Prodromal) services: Dilution or reduction of risk? *Schizophr Bull.* 2007;33(3):673–681. doi:10.1093/schbul/sbm015
91. Linscott RJ, Van Os J. An updated and conservative systematic review and meta-analysis of epidemiological evidence on psychotic experiences in children and adults: On the pathway from proneness to persistence to dimensional expression across mental disorders. *Psychol Med.* 2013;43(6):1133–1149. doi:10.1017/S0033291712001626
92. Sullivan SA, Kounali D, Cannon M, et al. A population-based cohort study examining the incidence and impact of psychotic experiences from childhood to adulthood, and prediction of psychotic disorder. *Am J Psychiatry.* 2020;177(4):308–317. doi:10.1176/appi.ajp.2019.19060654
93. Zhang W, Zhu Y, Sun M, et al. Longitudinal trajectories of psychotic-like experiences and their relationship to emergent mental disorders among adolescents: A 3-year cohort study. *J Clin Psychiatry.* 2019;80(4):18m12437. doi:10.4088/JCP.18m12437
94. Adewuya AO, Oladipo OE, Imarah T, Asmal L, Emsley R. The 3-year progression of clinically significant psychotic-like experiences in a general adult population in Lagos, Nigeria. *Soc Psychiatry Psychiatr Epidemiol.* 2023;58(1):91–103. doi:10.1007/s00127-022-02358-z
95. Knight C, Russo D, Stochl J, et al. Prevalence of and recovery from common mental disorder including psychotic experiences in the UK Primary Care Improving Access to Psychological Therapies (IAPT) programme. *J Affect Disord.* 2020;272:84–90. doi:10.1016/j.jad.2020.04.015
96. Armando M, Nelson B, Yung AR, et al. Psychotic-like experiences and correlation with distress and depressive symptoms in a community sample of adolescents and young adults. *Schizophr Res.* 2010;119(1–3): 258–265. doi:10.1016/j.schres.2010.03.001
97. Yates K, Lång U, Cederlöf M, et al. Association of psychotic experiences with subsequent risk of suicidal ideation, suicide attempts, and suicide deaths: A systematic review and meta-analysis of longitudinal population studies. *JAMA Psychiatry.* 2019;76(2):180. doi:10.1001/jamapsychiatry.2018.3514
98. Kelleher I, Devlin N, Wigman JT, et al. Psychotic experiences in a mental health clinic sample: Implications for suicidality, multimorbidity and functioning. *Psychol Med.* 2014;44(8):1615–1624. doi:10.1017/S0033291713002122
99. Honings S, Drukker M, Groen R, Van Os J. Psychotic experiences and risk of self-injurious behaviour in the general population: A systematic review and meta-analysis. *Psychol Med.* 2016;46(2):237–251. doi:10.1017/S0033291715001841
100. Trotta A, Arseneault L, Caspi A, et al. Mental health and functional outcomes in young adulthood of children with psychotic symptoms: A longitudinal cohort study. *Schizophr Bull.* 2019;46(2):261–271. doi:10.1093/schbul/sbz069
101. Unterrassner L, Wyss TA, Wotruba D, Haker H, Rössler W. The intricate relationship between psychotic-like experiences and associated subclinical symptoms in healthy individuals. *Front Psychol.* 2017;8:1537. doi:10.3389/fpsyg.2017.01537

102. Sonesson E, Russo D, Stochl J, et al. Psychological interventions for people with psychotic experiences: A systematic review and meta-analysis of controlled and uncontrolled effectiveness and economic studies. *Aust N Z J Psychiatry*. 2020;54(7):673–695. doi:10.1177/0004867420913118
103. Michel C, Ruhrmann S, Schimmelmann BG, Klosterkötter J, Schulze-Lutter F. Course of clinical high-risk states for psychosis beyond conversion. *Eur Arch Psychiatry Clin Neurosci*. 2018;268(1):39–48. doi:10.1007/s00406-016-0764-8
104. Birchwood M, Todd P, Jackson C. Early intervention in psychosis: The critical period hypothesis. *Br J Psychiatry Suppl*. 1998;172(33):53–59. PMID:9764127.
105. Stafford MR, Jackson H, Mayo-Wilson E, Morrison AP, Kendall T. Early interventions to prevent psychosis: Systematic review and meta-analysis. *BMJ*. 2013;346:f185. doi:10.1136/bmj.f185
106. Bosnjak Kuharic D, Kekin I, Hew J, Rojnic Kuzman M, Puljak L. Interventions for prodromal stage of psychosis. *Cochrane Database Syst Rev*. 2019;2019(11):CD012236. doi:10.1002/14651858.CD012236.pub2
107. Amminger GP, Schäfer MR, Papageorgiou K, et al. Long-chain ω -3 fatty acids for indicated prevention of psychotic disorders: A randomized, placebo-controlled trial. *Arch Gen Psychiatry*. 2010;67(2):146. doi:10.1001/archgenpsychiatry.2009.192
108. Davies C, Cipriani A, Ioannidis JPA, et al. Lack of evidence to favor specific preventive interventions in psychosis: A network meta-analysis. *World Psychiatry*. 2018;17(2):196–209. doi:10.1002/wps.20526
109. Davies C, Radua J, Cipriani A, et al. Efficacy and acceptability of interventions for attenuated positive psychotic symptoms in individuals at clinical high risk of psychosis: A network meta-analysis. *Front Psychiatry*. 2018;9:187. doi:10.3389/fpsyt.2018.00187