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Review

The onset of delusion in autism spectrum disorder: a psychopathological investigation

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SUMMARY

Autism spectrum disorder (ASD) is a neurodevelopmental disorder that affects social communication and behavioral routines. Diagnosis is complicated due to the age of symptom onset and the diverse symptomatology. ASD frequently occurs with comorbid psychiatric disorders, including psychotic disorders, that affect the individual's quality of life, health, and prognosis. The presence of psychosis in ASD is still a debatable topic. We conducted a narrative review to investigate the psychopathological factors that may contribute to the onset of delusion in individuals with ASD, focusing on Attributional style and Theory of Mind (ToM). Although an external Attributional style for negative events is a risk factor for the pathogenesis of delusion in individuals with psychotic spectrum disorders, it seems not to play a pivotal role in the onset of delusions in ASD patients. On the other hand, there is stronger evidence for the lack of ToM in delusional genesis in both psychotic and ASD subjects. To date, the available literature on ToM is still contradictory, and more research is needed, including consideration of social-cognitive deficits and a deeper understanding of the timing of ToM deficit onset in these conditions.

Future studies should also investigate the prevalence and type of delusions in ASD patients and their correlation with the severity of autistic symptoms. It is important for modern psychopathology to address these aspects systematically due to the increasing number of ASD diagnoses.

Key words: autism, psychosis, schizophrenia, theory of mind, attributional style

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Introduction

Autism spectrum disorder (ASD) is a neurodevelopmental disorder that affects social communication and behavioral routines. In 1943, Leo Kanner, an Austrian psychiatrist, laid the groundwork for the first definition of childhood autism, describing 11 children, 8 boys and 3 girls, which he presented with "Innate Autistic Disorders of Affective Contact". In 1980 Autism was first included in DSM-III, which defined it as a lack of responsiveness to other people (autism), a gross impairment in communicative skills, and bizarre responses to various aspects of the environment, all developing within the first 30 months of age. At the time of this publication, the latest version of DSM-5 defined two main criteria for the diagnosis of ASD: the presence of "persistent deficits of social communication and social interaction" and "restricted and repetitive patterns of behavior, interests or activities". In addition, it is necessary for the symptoms to manifest in early childhood. Ultimately, emphasis is placed on the effect that these symptoms can have on an individual's daily social and interpersonal interactions [1].

ASD poses a significant challenge in diagnosis, primarily due to the age of symptom onset and its diverse symptomatology. The heterogeneity is marked by varying degrees of severity and considerable fluctuations in symptoms. Additionally, the utilization of a wide range of evaluation methods in the diagnostic procedure further complicates the diagnosis [2]. The prevalence of ASD among children in Western societies is estimated to be around 100/10000 but with significant regional differences [3]. The incidence is likely to increase as a result of improved detection through screening and diagnostic tests and advancements in clinical evaluation techniques [4,5]. In a regional longitudinal record-linkage study conducted in Sweden, including all children up to age 17, they found that the prevalence of ASD increased from 4.2% in 2001 to 14.4% in 2011 [6]. In Poland, a study conducted between 2010 and 2014 estimated the prevalence of ASD in children up to age 17 to be 5.3% [7]. In the province of Pisa, Italy, a prevalence of 0.86% was found in children aged 7 to 9 years [8]. Another Italian study found that the cumulative prevalence of autism remained stable from 2001 to 2015, but more cases were diagnosed at an earlier age. The authors emphasized the importance of using cumulative prevalence by time period for a better understanding of ASD occurrence [9].
ASD is frequently found in comorbidity with several clinical and psychiatric disorders [10]. Some symptoms are present from birth or evolve during the first years of life, while others may not become apparent until later. In the latter case, socio-economic, cultural, and family contexts may play an important role in the development of ASD. Several reviews have investigated the comorbidity between ASD and various psychiatric and nonpsychiatric disorders. Among the most common health problems are intellectual disability, structural brain abnormalities with or without epilepsy, structural brain changes or abnormalities, and dysmorphic characteristics [9,11,12].
Behavioral and psychiatric comorbidities play a crucial role in the quality of life, health, and prognosis [13]. They may include hyperactivity, impulsiveness, aggression, mood disorders, self-harm, and psychotic disorder [14].
Detecting comorbid psychosis in patients with ASD poses a multifaceted dilemma, particularly during the initial stages of psychotic illness and among individuals with ultra-high risk for psychosis (UHR-P), including those with Attenuated Psychosis Syndrome (APS) [15-18].
The presence of psychosis in ASD individuals is still an argument of debate. Kiyono and colleagues performed a meta-analysis published in 2020 showing in ASD a pooled prevalence of psychotic experiences up to 37%, while in the general population a prevalence of psychotic episodes has been reported between 5% to 12%. However, in their subgroup analysis, only 6% of the pooled patients experienced hallucinations, the same as the general population, while 45% experienced delusions [9-21].
Our goal is to investigate the psychopathological factors that may contribute to the onset of delusion in individuals with ASD, with a particular focus on Attributional style and Theory of Mind (ToM). Since our investigation is centered on the mechanisms that involve language, our examination was restricted to individuals diagnosed with level 1 ASD, encompassing those previously diagnosed with Asperger's Syndrome (AS) or high-functioning autism, according to the diagnostic criteria in effect before the publication of the DSM-5.

Attributional style

Attributional style is a personal and typical way of finding and explaining the causes of events. Abnormalities in attributing meanings can exist in many conditions, such as psychosis and ASD.
Craig and colleagues evaluated ToM and Attributional style in schizophrenia patients with persecutory delusions and compared them to subjects with AS and a healthy control group [22]. They used the "Attributional Style Structured Interview", which asks the participants to describe how they would explain 20 events in four categories: negative events involving a human agent, positive events involving a human agent, negative events not involving a human agent, and positive events not involving a human agent. The "Leeds Attributional Coding System" was utilized to register, transcribe, and codify the answers on five binary dimensions: internal or external, stable or unstable, global or specific, personal or universal, and controllable or uncontrollable. The authors found that subjects with paranoid delusions tended to make external-personal attributions for negative events more than subjects with AS and controls. Both paranoid and AS groups scored worse on the ToM tasks than the control group. Also, the paranoia-related scores were higher compared to the control group. This suggests that ToM and the attributional abnormalities contribute to paranoid delusions. However, the lack of attributional abnormalities in the AS group implies that paranoid delusion results from different mechanisms than those involved in the delusions of schizophrenic patients [22].
Another study by Aakre and colleagues compared patients who suffer from paranoid delusion with those without paranoid delusion and a healthy control group. They similarly revealed a greater tendency to make external-personal attributions for negative events in patients with paranoid delusion [23].
Didehbani and colleagues conducted a study in 2012 examining insight into illness and social attributional styles in individuals with AS compared with healthy controls. There was an inverse relationship between externalizing bias and level of insight in individuals with AS, i.e., a high

level of insight was associated with a tendency for self-blame. This association was particularly evident when assessed with a clinician-administered insight measure compared to a self-reported measure and may be related to an increased awareness when externally assessed [24].
Blackshaw and colleagues investigated Attributional styles in a cohort of individuals with AS. The authors found that subjects with AS scored lower points on ToM tests and higher points on paranoia tests compared to the control group. However, the two groups had no significant differences regarding the dimension of meaning attribution. The paranoia observed in individuals with AS is therefore believed to stem from different factors compared to those that contribute to paranoia in individuals diagnosed with schizophrenia. The latter may arise as a strategy used as a defense from a subject-threatening plot, while the former may stem from confusion about not understanding the subtleties of social interactions and rules. This would explain why abnormal meaning attributions were not found in subjects with AS [25].

ToM in ASD and delusions

ToM explains the ability to infer other individuals' mental states and is a fundamental element of human social interactions [26,27]. Some mental states lead to deficiencies in the ability to perceive any perspective other than their own. ToM-deficits have been described in various neurodevelopmental disorders, such as ASD, attention-deficit hyperactive disorder (ADHD), and schizophrenia, as well as in acquired neuropsychiatric disorders, such as various brain injuries and dementia [28,29]. Current models have a differentiated approach to ToM, separating cognitive ToM, affective ToM, and a spectrum of a continuum from hypo- to hyper-ToM [30-35]. Cognitive ToM explores the cognitive ability to recognize the mental state of others, including their thoughts and intentions, while affective ToM is an empathic evaluation of another individual's emotional state [34]. Hyper-ToM is described as an excessive presence of ToM that leads to an over-attribution of knowledge to others, resulting in inaccurate conclusions about their mental state [35].
In individuals with ASD, the primary features include difficulties in shared social interaction with verbal and non-verbal communication deficits and stereotyped repetitive behavior. To a various degree, there is an inability to assess the behavior of other individuals based on their mental states. Thus in ASD, a deficit in ToM has been linked to the core autistic symptomatology [36,37]. An early study by Baron-Cohen and colleagues performed on children with and without ASD showed a lack of ToM in children with ASD [37]. Further studies on adults and children on more demanding ToM tasks showed an association with inferior social cognitive performance [23,38-41]. Also, there are differences along the ASD spectrum as there is high individual variance on tasks designed to assess the ToM, and individuals performing better on ToM tests are better socially integrated [23,42,43]. Not all aspects of ASD can be credited to a deficit in ToM [44,45], but its effect on social skills is significant [46].
The notion of a dysfunctional ToM in individuals with ASD is not without controversies. As discussed in a recent review, deficits in ToM are due to impaired communication skills – a criterion for the DSM-5-TR diagnosis of ASD – and the authors argue that the evidence for dysfunction in ToM is based on a poor methodological foundation [47].
The first mention of a relationship between ToM and delusion came from Frith in 1992. Frith postulated that a lack of ToM was a predisposing cognitive factor for delusion and stated that paranoid delusions occur because the patient makes incorrect assumptions about other people's intentions [48]. Since then, ToM has been extensively studied in patients with psychotic disorders establishing an association with dysfunction in ToM [49-52]. In several studies, altered ToM abilities were linked not only with the diagnosis of schizophrenia but also with specific psychotic delusional symptoms. More specifically, some studies investigated impaired ToM in patients with persecutory delusions showing that those patients presented pronounced debts in various ToM capacities, including the ability to infer emotions and intentions and to understand second-order false beliefs [31,53-56].
Regarding hyper-ToM, most studies support an association between hyper-ToM errors with positive symptoms and delusion [32,53,56,57]. However, Dorn and colleagues recently found no association between hyper-ToM errors and psychotic symptoms, whether positive, negative, or disorganized [58].
Establishing a connection between ToM, delusion, and ASD is challenging due to the lack of literature on the topic. The notion of delusional beliefs in patients with ASD has initially been limited to case reports [59-62]. However, in the last two decades, experimental studies have established a meaningful relationship, which was discussed in our recent review [17]. Furthermore, individuals with ASD regularly present dysfunctions in ToM [63]. Assuming that dysfunction in ToM is a predictor of delusion in patients with psychotic disorder, one could presume its importance in delusion also in individuals with ASD.
As previously mentioned, Blackshaw with associates showed higher scores on measures of paranoia and lower scores on ToM tasks in subjects diagnosed with AS [25]; on the contrary, Abell and Hare found no association between ToM ability and general delusion or paranoia in individuals with AS [64]. Instead, they propose a model of development and maintenance of delusional beliefs in people with AS in which ToM indirectly influences individual life experiences and renders them vulnerable to developing delusional beliefs.

When do ToM impairments and biased attributional styles appear in psychosis?

Several studies examined ToM deficits in psychosis [51,65]. However, the timing of ToM impairment onset in psychosis is not completely clear. While ASD children never seem to acquire ToM abilities [48], schizophrenic patients and brain-injured patients start showing severe ToM impairments only after their disease outbreak. Understanding when ToM deficits occur in schizophrenia might provide important information on schizophrenia pathogenesis and delusional formation. Bora and colleagues conducted a meta-analysis evaluating impairments in first-episode psychosis (FEP), ultra-high risk (UHR) subjects, and first-degree healthy relatives of schizophrenic patients [66]. The analysis showed that ToM was significantly impaired in UHR subjects and unaffected relatives compared to healthy controls. On the other hand, the severity of ToM impairment in FEP was close to the chronic patients' score reported in a precedent meta-analysis [49]. These findings seem to suggest that ToM impairments gradually appear during the early stages of schizophrenia and definitely erupt with large effect sizes after the explosion of the psychotic onset. Consequently, ToM-deficits do not seem to be explained by the effects of illness progression, chronicity, and long-term pharmacotherapy. These results are still not conclusive, and more recent research was not able to replicate them [67].

Concerning Attribution styles, Parks and colleagues investigated whether UHR individuals show increased hostility perception and blaming bias and explored the associations of these biased styles of attribution with the factor structure of multifaceted self-related psychological variables and neurocognitive performances. Similar to full-blown psychotic patients, UHR individuals showed increased hostility perception and blaming bias when compared with normal controls, supporting the emergence of attribution biases even in the putative prodromal phase of schizophrenia [68].

Ultimately, while emerging results are in favor of ToM-defictis playing a role in the pathogenesis of delusions in both ASD and schizophrenia, biased Attributional style seems to play a major role in paranoid delusions in psychotic patients. The dual role of ToM deficits in delusion formation could be attributed to the tight overlap and similarity between AS and schizotypy, as a significant association between positive and negative schizotypy and lower ToM performance has been demonstrated [49]. Additionally, since different aspects of the self have been reported to serve as accounting factors for attributional styles [68], further studies should evaluate the correlation between these dimensions and abnormalities in attributional style in level 1 or high-functioning ASD subjects.

Conclusions

The pathogenesis of delusions in patients with ASD is complex, and no clear psychopathological explanations exist. Unfortunately, few data are available in the literature.

According to our review, Attributional style does not appear to be related to the onset of delusions in ASD patients compared to psychotic patients.

Regarding ToM, there is stronger evidence for the lack of ToM in ASD individuals. However, the data are contradictory, and no definitive conclusions can be drawn. To better assess the relationship between ToM and delusion, it would be important to consider social-cognitive deficits such as cognitive and affective ToM, as well as measure the spectrum of a continuum from hypo- to hyper-ToM.

There are several considerations to be made. First, recognizing psychotic symptoms in ASD patients is complex and requires in-depth psychopathological knowledge. It is, therefore, possible that delusional symptoms are frequently not recognized. Furthermore, non-persecutory delusional symptoms may be more present in ASD patients, such as "expansive delusions" [59] or "unusual thoughts" [61]. These non-persecutory delusions are generally not related to any bias in meaning attributions, which could explain the lack of data about the role of Attribution style in the onset of delusions in ASD patients. Likewise, ToM deficits are more commonly associated with persecutory delusions.

In this regard, it would be helpful to conduct future studies to better characterize the prevalence and type of delusions in ASD patients. Furthermore, it would be necessary to investigate the correlation between the severity of autistic symptoms, particularly deficits in social communication, and the prevalence of delusional symptoms. In light of the exponential increase in ASD diagnoses, it is mandatory for modern psychopathology to address these aspects systematically.

Conflict of interest statement

The authors declare no conflict of interest.

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Authors' contributions

MR and GDL: study conceptualization and design; EE, FFN, CF, SF, GA: literature search; MR: wrote the first draft of the manuscript; AS, CN, MR and GDL: supervision of the manuscript; all the Authors reviewed and approved the final version of the manuscript.

Ethical consideration

Not applicable.

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