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Citation
Ziermans, T. B., Kok, B., & Rijn, S. van. (2025). Response inhibition in Autistic children predicts positive psychotic symptoms in young adulthood: results from an 8-year follow-up study. *Review Journal Of Autism And Developmental Disorders*, 1-9. doi:10.1007/S10803-025-07015-3

Version:	Publisher's Version
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Downloaded from:	https://hdl.handle.net/1887/4290738

Note: To cite this publication please use the final published version (if applicable).

Journal of Autism and Developmental Disorders
https://doi.org/10.1007/s10803-025-07015-3

ORIGINAL ARTICLE

Response Inhibition in Autistic Children Predicts Positive Psychotic Symptoms in Young Adulthood—Results from an 8-Year Follow-up Study

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Accepted: 29 July 2025
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Abstract

Purpose Attenuated positive symptoms constitute the most validated vulnerability marker for psychosis in non-autistic young adults. Early deviations in executive functioning and social cognition are believed to contribute to the onset of these symptoms. This study evaluates the presence of psychotic symptoms in autistic young adults and their putative cognitive precursors.

Methods Thirty young adults diagnosed with an autism spectrum condition (ASC; M_{age}=20.1; 83.3% male) were assessed for psychotic symptoms. Their scores were compared to a typical peer comparison group (TC; M_{age}=22.1, 41.7% male) and, retrospectively, to their scores in childhood (M_{age}=12.1) to determine long term-stability. In addition, it was tested whether cognitive markers assessed in childhood could predict positive symptoms in young adulthood.

Results There was significant and moderate evidence for more negative symptoms in young adults with ASC compared to TC, but no difference in positive or disorganized symptoms. Furthermore, positive and negative symptoms did not differ significantly over time and displayed weak correlations between both assessments, while disorganized symptoms showed a modest decrease and a significant correlation. In addition, response inhibition accuracy in childhood was a significant cognitive predictor of positive symptoms at follow-up.

Conclusions Contrary to expectations, our results suggest that self-reported positive psychotic symptoms are not elevated in young adults with ASC. Psychotic symptoms remain relatively stable from childhood to young adulthood, although individual differences in symptom change are substantial. Response inhibition is a putative candidate risk marker for the development of positive symptoms in young, autistic adults that awaits further replication in large samples.

Keywords Autism · Psychosis · Positive symptoms · Executive functioning · Social cognition

Introduction

Autism spectrum conditions (ASC) are marked by an increased risk to be diagnosed with a psychotic disorder in adulthood (10.3% prevalence of schizophrenia versus 0.9% in the general population; 9.4% pooled prevalence of psychosis) (Hsu et al., 2022; Perälä et al., 2007; Varcin et al., 2022). Studies detailing the impact of psychosis above and beyond an autism diagnosis are scant. In two comparative studies, Larson and colleagues reported significantly more affective problems for ASC individuals with psychosis than those without (Larson et al., 2017, 2020). Furthermore, studies on co-occurring traits in autistic or psychotic samples emphasize that co-occurrence is generally associated with decreased (social) functioning (Bechi et al., 2021; Chisholm

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Published online: 01 September 2025

et al., 2019; Deste et al., 2020; Isvoranu et al., 2022; Ziermans et al., 2021), quality of life (Chisholm et al., 2019; Klang et al., 2022), and increased depression and suicidality (Upthegrove et al., 2018). Even though causality cannot be inferred from these data, the perceived unfavorable impact of co-occurring traits with increasing symptom severity in clinical populations demands a better understanding of psychosis prevention targets in autism.

Improved screening procedures can help identify which individuals are at most imminent risk for psychosis. The 'ultra-' or 'clinical high risk' (CHR) for psychosis paradigm has been the most successful approach in the past decades and predominantly centers around the presence of attenuated positive psychotic symptoms, such as mild delusional thinking and recurring hallucinatory experiences. For individuals reporting these symptoms, the transition rate to psychosis is approximately 20–30% within 3 years (Salazar et al., 2021), and slightly lower for young adolescents (Raballo et al., 2022). Not surprisingly, positive symptom severity ranks consistently among the strongest predictors for subsequent onset of psychosis (Mensi et al., 2021; Oliver et al., 2020a, b), even in young adolescent individuals (Ziermans et al., 2014). It has only been sparsely investigated whether psychosis vulnerability markers apply equally to young autistic individuals. However, a few preliminary studies suggest that transition rates to psychosis fall within the same range as the overall transition rates from CHR studies (Foss-Feig et al., 2019; Mammarella et al., 2024; Riccioni et al., 2022).

The prognostic accuracy of positive symptoms can be enhanced by including cognitive assessments (Rosen et al., 2021). General cognitive abilities, executive functioning (EF), and social cognition are among the most commonly identified cognitive vulnerability markers in CHR cohorts (Cowman et al., 2021; Halverson et al., 2019; Tor et al., 2020, 2024; Ziermans et al., 2014), though some studies report mixed or negative findings (Lin et al., 2013; Schmidt et al., 2016; van Donkersgoed et al., 2015). This inconsistency between psychosis prediction models is likely due to large clinical and methodological heterogeneity (Sanfelici et al., 2020). While all high-risk individuals generally display positive symptoms, these may very well have different (cognitive) determinants.

So far, there is limited research available on cognitive faculties that may precede the onset of psychotic symptoms in autism. Initial studies in modest CHR-subgroups of autistic individuals have shown that they display more problems with social and cognitive functioning (Foss-Feig et al., 2019; Maat et al., 2020; Riccioni et al., 2022), and low-level information processing (Di Lorenzo et al., 2020; Foss-Feig et al., 2021) than at-risk individuals without autism. However, most CHR-studies contain clinically enriched samples (Fusar-Poli et al., 2016) and thereby minimize generalizability to, for example, the general autism population. Therefore, a more commonsense approach to detect autism-specific cognitive vulnerability markers for psychosis would be to focus on autism-only cohorts and cognitive domains that are typically diminished across both spectra. The domains of executive functioning (e.g. working memory, inhibition, shifting) and social cognition (e.g. emotion processing and theory of mind) are prime candidates given the extensive literature supporting the presence of impairments (e.g. Demetriou et al., 2018; Fusar-Poli et al., 2012; L. Oliver et al., 2020a, b; Velikonja et al., 2019) and their recurring prominence as cognitive vulnerability markers for psychosis in non-autistic populations, as stated above.

The current study examines 8-year follow-up data of a cohort of autistic children (9–18 years) from a child psychiatric outpatient department. Participants were contacted again around young adulthood and were asked to fill out mental health questionnaires online. The first two aims were to compare their psychotic symptoms in young adulthood with typical peer comparison data and their symptom scores at baseline. Based on the literature we expected higher scores in our ASC group than for comparisons. Furthermore, it was hypothesized that there would be a modest increase in psychotic symptoms over time, in particular for positive symptoms, based on the increased incidence of (risk for) psychosis in ASC and longitudinal observations of psychotic experiences for this age range in the general population (Sullivan et al., 2020). The third aim was to test whether positive symptoms in young adulthood could be predicted by cognitive markers assessed in childhood. We focused on EF and social cognition as these domains stand out most prominently in the autism-psychosis literature. Since the outcome is aimed at generating hypotheses, no domain- or task-specific hypotheses were formulated.

Method

Participants

The ASC group was recruited between 2009 and 2012 through a child psychiatric outpatient unit with specialized services for children with autism (Centrum Autisme, Rivierduinen) in the larger Leiden region, the Netherlands. Autism diagnoses were classified according to DSM-IV criteria at baseline, after extensive clinical assessments and consensus by board-certified child psychiatrists and a multidisciplinary team. Additional inclusion criteria at baseline were Dutch as the primary language and age between 9 and 18 years. Exclusion criteria were a recent history of substance abuse, intellectual disability (<60 IQ points) and neurological conditions. The total baseline sample consisted of 57

ASC children (M_{age}=12.2, SD=2.2, range=9.0-18.2, 81% male) of whom 30 individuals were included at follow-up (M_{age}=20.1, SD=2.3, range=17.1–25.8, 83% male). Follow-up assessments took place pre-pandemic, between August 2018-February 2019. The follow-up sample did not differ significantly from the baseline-only sample regarding age, sex, IQ, autistic or psychotic symptoms (see Supplemental Table S1).

The SPQ-BR data for a typical adult comparison (TC) group consisted of 72 young adults (M_{age}=22.1, SD=1.9, range=18–26, 41.7% male) and was collected in two rounds (2019 and 2020) of undergraduate research projects at the Department of Psychology, University of Amsterdam. Participants were excluded if they were diagnosed with autism or schizophrenia spectrum disorder, reported a severe visual or hearing impairment, or an existing neurological condition. Despite the similar age range, the TC were significantly older on average (t=4.33, p<.001), and had significantly lower male-to-female ratio, with 1 participant identifying as neither (X^2=14.85, p<.001).

After providing a complete description of the study to parents and subjects, we obtained written informed consent according to the Declaration of Helsinki at both timepoints. The baseline study was approved by the Ethical Committee of Leiden University Medical Center, the Netherlands. Approval for follow-up assessment was obtained from the local ethics committee of the Department of Education and Child studies at Leiden University (ECPW2018/195).

Psychotic Symptoms

At baseline children filled-out a translated and child-friendly version of the 74-item Schizotypal Personality Questionnaire (SPQ-C-D)(van Rijn et al., 2015). The SPQ is an often-used instrument to measure schizotypy and, as such, screens for putative risk for developing schizophrenia-spectrum disorders. The SPQ consists of self-descriptive, closed-ended questions, and each yes-answer is scored with one point (Raine, 1991). The total sum score can consist of three symptom dimensions: positive (cognitive-perceptual), negative (interpersonal), and disorganized. These can be further subdivided into nine subscales. The SPQ has a well-validated factor structure, also for the Dutch version (Vollema & Hoijtink, 2000).

At follow-up an abbreviated adult version of the SPQ was assessed. The SPQ-Brief Revised (SPQ-BR) contains 32 items of the original SPQ, but employs a 5-point Likert scale for scoring (from 0='strongly disagree' to 4='strongly agree') (Cohen et al., 2010; Davidson et al., 2016). The Dutch SPQ-BR was comprised of the designated 32 items from the original translation of the SPQ. The questionnaire contains 14 items for the positive (range 0–56), 10 items for the negative (range 0–40) and 8 items for the disorganized (range 0–32) dimension. These scores can be further subdivided into seven subscales.

SPQ-scores were transformed for comparisons between baseline and follow-up. The SPQ-C-D scores at baseline were summed for the 32 items of the SPQ-BR. Next, scores for the SPQ-BR at follow-up were recoded to binary scores (0–2=0, i.e. 'no'; 3–4=1, i.e. 'yes') and summed. Since a score of 2 ('neutral') was ambiguous, a conservative approach was taken to include such answers as 'no' and only code a symptom as present if participants filled out 'agree' or 'totally agree' on the SPQ-BR.

Cognitive Measures

Cognitive measures were collected at baseline only, using five computerized tasks from the Amsterdam Neuropsychological Tasks (ANT, version 2.0), one subtest (number repetition) of the Dutch version of the Clinical Evaluation of Language Fundamentals (CELF-4-NL), the Social Cognitive Skills Test (SCST), and the Karolinska Directed Emotional Faces. A brief description of each task is included in the supplemental information, and additional detail for all ANT-tasks is available online in the English handbook https://www.researchgate.net/publication/392017160_Handbook_ANT_March_2025.

Data Analysis

Data were analyzed with JASP software (version 0.18.3) utilizing both classical frequentist tests and their Bayesian alternatives to assess the probability of each outcome. After establishing internal consistency (Cronbach's alpha), follow-up data on psychotic symptoms were compared to the available SPQ-BR comparison data. Two-tailed Mann-Whitney U tests were applied for each dimension and subscale. Next, paired-sample Wilcoxon tests were performed to assess whether psychotic symptoms at baseline changed over time from baseline to follow-up and Kendall's Tau correlations to assess the strength of the associations. Lastly, multiple linear regression analyses were performed to investigate whether cognitive task performance at baseline could predict positive symptoms at follow-up. Inclusion of predictors was restricted to cognitive variables and covariates that met a firm linearity assumption (r=.30 or higher; Ratner, 2009) due to the limited number of cases in the model. IQ, Sex and age at baseline were considered as potential covariates. For all analyses p-values and Bayes Factor (BF) are reported. Results with p<.05 are indicated as significant findings and BF_{10} expresses the probability of the data given H1 relative to H0. Default settings for priors in JASP were maintained for all Bayesian analyses.

Table 1 Descriptives of autistic participants (N=30; M ± SD)

	Baseline	Follow-up
Sex (% male)[1]	83.3	83.3
Age (years)	12.2 ± 2.7	20.1 ± 2.3
- *Range*	9.0–18.2	17.2–25.8
IQ estimate[2]	99.4 ± 17.3	
- *Range*	65 − 129	
Autistic symptoms (ADI-R Total)[3]	39.9 ± 7.1	
- *Social interaction*	19.7 ± 4.4	
- *Communication*	15.8 ± 3.7	
- *Restricted behaviours*	4.5 ± 2.7	
Psychotic symptoms (SPQ-C-D Total)[4]	33.6 ± 18.3	
- *Positive symptoms*	8.9 ± 7.2	
- *Negative symptoms*	15.6 ± 9.4	
- *Disorganized symptoms*	9.2 ± 5.4	

[1]Two participants, 1 male and 1 female at baseline, identified as the opposite gender at follow-up

[2]Matrix reasoning/Vocabulary; one extreme outlier (IQ=196) was winsorized

[3]Autism Diagnostic Interview - Revised; data missing for 5 participants

[4]Schizotypal Personality Questionnaire for Children – Dutch; data missing for 2 participants

Table 2 SPQ-BR symptom dimensions and subscales (M ± SD) for the autism and typical comparison groups

	ASC (N=30)	TC (N=72)	p-value	BF_{10}
Positive Symptoms	13.30 ± 9.52	13.44 ± 8.70	0.889	0.225
- *Ideas of reference/ Suspiciousness*	8.07 ± 5.05	7.61 ± 4.94	0.630	0.236
- *Odd beliefs/Magical thinking*	1.70 ± 2.91	2.67 ± 3.04	0.060	0.492
- *Unusual perceptions*	3.53 ± 3.08	3.17 ± 3.15	0.498	0.264
Negative Symptoms	16.93 ± 8.00	11.13 ± 7.05	0.001	7.377
- *Social Anxiety*	7.47 ± 3.95	5.33 ± 3.70	0.009	1.672
- *Constricted Affect/No Close Friends*	9.47 ± 5.36	5.79 ± 4.60	0.002	5.928
Disorganized Symptoms	14.90 ± 7.32	13.88 ± 6.79	0.438	0.263
- *Eccentric Behaviour*	7.43 ± 4.20	5.60 ± 3.81	0.026	0.718
- *Odd Speech*	7.47 ± 4.18	8.28 ± 3.93	0.336	0.264

Results

Participants

Descriptive characteristics of the ASC group are included in Table 1. All average ADI-scores at baseline were above recommended clinical cut-off scores.

Psychotic Symptoms at follow-up Compared To Peer Data

Internal consistency for SPQ-BR scores was excellent overall (α=0.927), and good for all dimension subscales (α=0.833-0.888), Mann-Whitney U tests revealed a significant higher median for negative symptoms in ASC compared to TC (U=641.0, p<.001) with moderate evidence for a group difference (BF_{10}=7.38). Exploration on a subscale level showed that both negative subscales, constricted affect/no close friends and social anxiety, were significantly higher for ASC (both p<.01) with moderate evidence for the alternative hypothesis in constricted affect/no close friends (BF_{10}=5.93) and weak evidence for social anxiety (BF_{10}=1.67). Eccentric behavior, part of the disorganized dimension, was the only other subscale that differed significantly (U=776.5, p=.026), but according to its Bayesian counterpart not sufficient to provide meaningful evidence for a group difference (BF_{10}=0.72). Results are displayed in Table 2.

Psychotic Symptoms from Baseline To follow-up

One individual was removed because their baseline item-scores for SPQ could not be retrieved. For the remaining ASC group (N=29) there was a significant decrease in disorganized symptoms over time (W=239.0, p=.040, BF_{10}=1.47) with marginal evidence for H1, but no change was detected for positive (W=139.5, p=.203, BF_{10}=0.41) or negative symptoms (W=230.0, p=.070, BF_{10}=1.35), respectively (see Fig. 1). The strength of associations between symptoms at baseline and follow-up showed a small and non-significant correlation for positive symptoms (τ=0.17, p=.256, BF_{10}=0.54) and negative symptoms (τ=0.25, p=.083, BF_{10}=1.36). However, there was a significant positive correlation between disorganized symptoms at both timepoints, with substantial evidence for a meaningful association (τ=0.31, p=.030, BF_{10}=3.62).

Predicting Positive Psychotic Symptoms at follow-up

In total, eight executive functioning, four social cognition, and IQ estimates were considered as cognitive predictors. Only two variables met the linearity assumption: accuracy on response inhibition (r=.36) and accuracy on cognitive flexibility (r=.32). For covariates, age at baseline correlated significantly with inhibition accuracy (r=.55, p=.002) and was added for model comparison. Model comparison with the three remaining predictors indicated that the model with inhibition and age at baseline was the best-fitting model (BF_M=2.98; BF_{10}=1.84). This final model was significant

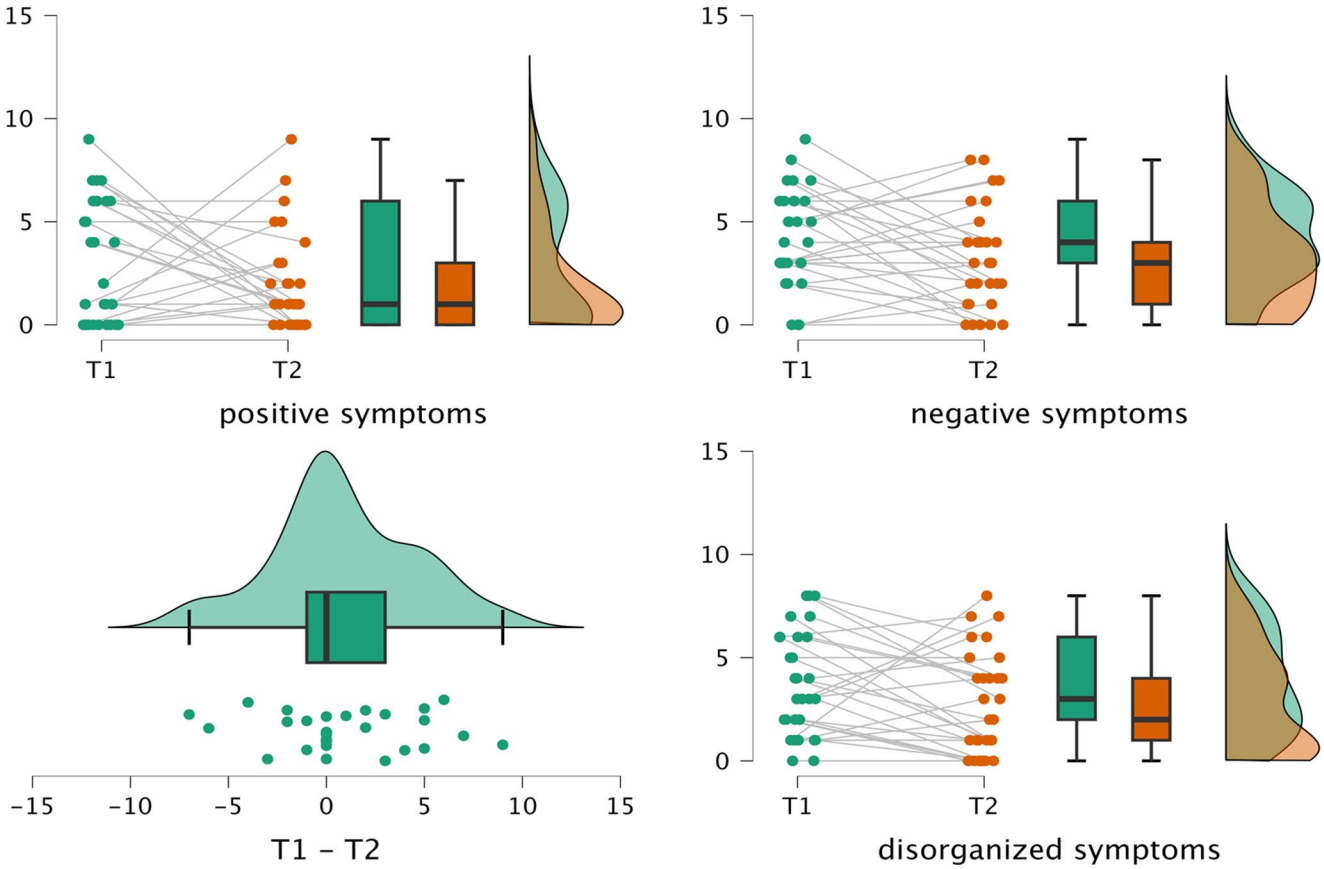

Fig. 1 Rainbowplots of psychotic symptoms at baseline and 8-year follow-up. This figure illustrates individual psychotic symptom scores (positive, negative, disorganized) for ASC participants at baseline (T1) and 8-year follow-up (T2). Boxplots are shown with boxes representing the interquartile range (IQR; Q1-Q3), bold horizontal stripe as the median and whiskers ± 1.5 IQR. The respective data distributions of T1 and T2 are displayed on the right of the boxplots. In the bottom left panel difference scores for positive symptoms and their distribution can be seen. A negative score on the x-axis indicates an increase in symptoms over time and vice versa

(F=4.85, p=.016, BF_{10}=3.77) and explained a moderate-to-large amount of variance (R^2=0.27). Both predictors were significant with substantial evidence for inclusion of inhibition in the model and modest evidence for age (inhibition: ß=0.61, t=3.03, p=.005, $BF_{inclusion}$=3.74; age: ß=−0.45, t=−2.27, p=.032, $BF_{inclusion}$=2.29). In other words, ASC children that were more error prone during a response inhibition task scored higher on positive symptoms as young adults, after controlling for age.

Discussion

Contrary to our hypotheses, results showed that positive psychotic symptoms were not increased in ASC adults compared to their typically developing peers. In addition, while psychotic symptoms remained relatively stable from childhood to young adulthood on a group level, the data also showed substantial individual differences in symptom change over time. Furthermore, response inhibition was the sole cognitive predictor in childhood that showed a significant relation with future positive symptoms and might represent an early cognitive marker for psychosis vulnerability in autism.

The relatively modest presence and long-term stability of psychotic symptoms were unexpected findings. While it is conceivable that our follow-up participant-group consisted mostly of individuals with lower symptom severity, attrition analyses indicated no group differences in psychotic symptoms at baseline. Another relevant factor could be that our study group consisted of autistic individuals diagnosed in childhood, whose risk for developing psychotic disorders in adulthood is only marginally elevated compared to the general population (Schalbroeck et al., 2019; Selten et al., 2015). However, even children diagnosed with autism appear to have increased odds (OR=2.81) to report psychotic experiences in early adolescence (Sullivan et al., 2013). The finding of elevated negative psychotic symptoms in autism is not unexpected, given the interpersonal nature of these symptoms and their overlap with social-communicative features common to autism. Indeed, recent studies suggest that positive psychotic symptoms may best distinguish autistic

and psychotic phenotypes (e.g., Pablo et al., 2025; Corbera et al., 2024). Interestingly, however, upon checking relations with parent-reported ADI-R subscales, these did not correlate significantly with self-reported negative psychotic symptoms. This suggests that perceived symptom overlap may vary by informant and developmental stage.

Longitudinally, psychotic symptoms on a group level did not change significantly from childhood to adulthood, except for a small decrease in disorganized symptoms. In contrast, we expected a general increase in symptoms because participants in the current study were re-assessed around the time of peak onset for psychosis. In addition, a large longitudinal follow-up study in a general population-cohort reported an increased incidence of psychotic symptoms between 12 and 24 years with a peak incidence between 17 and 19 years (Sullivan et al., 2020) and in a more recent meta-analysis this peak was estimated to occur between 13 and 17 with a persistence rate of 35.8% (Staines et al., 2023). However, there are also reports of a modest decrease in symptoms with age during this period (Isaksson et al., 2022; Kelleher et al., 2012). In the current study, symptom reports between both timepoints indicated relatively weak associations and considerable interindividual variability in change over time, which suggests low persistence and limited predictive capacity of early psychotic symptoms. This could point to, e.g., a more transitory nature of mild psychotic symptoms or perhaps a different nature of underlying (pathogenic) factors in autism. Clearly, this highlights the need for large-scale, longitudinal and standardized assessments of psychotic symptoms in autistic cohorts to better understand their nature and dynamics.

In terms of cognitive predictors, our results yielded substantial evidence for response inhibition (accuracy) as a significant predictor of positive psychotic symptoms in autistic young adults. Impairments in response inhibition are common in autism (Geurts et al., 2014), schizophrenia, and high-risk individuals, though not necessarily related to positive symptoms (Fryer et al., 2019). One study in autistic children reported that response inhibition is related to formal thought disorder, a symptom that is often considered a core aspect of executive control of language skills in different contexts (Solomon et al., 2008). However, we were unable to confirm this in our earlier baseline study of the current ASC group (Ziermans et al., 2017). Together these findings imply that response inhibition represents a potential early vulnerability marker for psychosis risk in autism, which awaits further replication in larger (longitudinal) study samples. Moreover, while our sample size was sufficiently large to detect a relatively large effect ($\beta = 0.61$) using linear regression, we calculated that detecting a more moderate effect size ($\beta \approx 0.30$), with 80% power at $\alpha = 0.05$, would require a minimum sample of 85 participants to detect a significant effect. Assuming a base rate of psychosis incidence of approximately 10% in autistic individuals, this implies that at least 850 autistic individuals would need to be followed longitudinally to yield 85 cases who will continue to develop psychosis. This underscores the need for large-scale, collaborative, or registry-based studies to robustly identify risk factors. One way to achieve this is by implementing continuous psychosis-risk follow-up assessments in ongoing large-scale data collections, such as the Netherlands Autism Register initiative (https://nar.vu.nl/en/research).

In addition to a modest sample size and substantial attrition from baseline inclusions, interpretation of our results is further hampered by restricted participant information at follow-up. No information on medical history, education or other potentially relevant factors between baseline and follow-up was acquired, which could have provided additional insight. Another major issue in the autism-psychosis research literature is the lack of validated transdiagnostic symptom assessments (Schalbroeck et al., 2023). As such, it is uncertain whether SPQ-BR items are well-suited to assess psychotic symptoms in ASC, and future studies could benefit from better validated instruments to help differentiate between autistic and schizotypal symptom dimensions (Parvaiz et al., 2023). Finally, transforming the SPQ data to a singular format for better comparison could also have had a negative influence on interpretability of our symptom stability results.

In conclusion, this study includes unique 8-year follow-up data of psychotic symptoms in young autistic individuals from childhood to young adulthood. Our findings were unable to confirm previous reports of elevated, persistent psychotic symptoms, but pinpointed response inhibition as a putative cognitive vulnerability marker for developing positive symptoms in ASC. While this result should be considered preliminary and hypothesis-generating, it demonstrates the potential of early cognitive assessments as an aid in predictive modeling for future mental health conditions in autism and hopefully encourages more rigorous and voluminous research efforts for further confirmation and finetuning.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s10803-025-07015-3.

Funding This research received no specific grant from any funding agency, commercial or not-for-profit sectors.

Declarations

Competing Interests The authors declare none.

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