Schizophrenia Research: Cognition 28 (2022) 100241

Contents lists available at ScienceDirect

Schizophrenia Research: Cognition

journal homepage: www.elsevier.com/locate/scog

Episodic memory impairment in children and adolescents at risk for schizophrenia: A role for context processing☆

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ARTICLE INFO

Keywords:
Cognitive impairments
Context processing
Episodic memory
Schizophrenia

ABSTRACT

People with schizophrenia experience episodic memory impairments that have been theorized to reflect deficits in processing context (e.g., spatio-temporal features tied to a specific event). Although past research has reported episodic memory impairments in young people at-risk for schizophrenia, the extent to which these impairments reflect context processing deficits remains unknown. We addressed this gap in the literature by examining whether children and adolescents at risk for schizophrenia exhibit context processing deficits during free recall, a memory task with high contextual demands. Our sample included three groups ($N = 58$, 9–16 years old) varying in risk for schizophrenia:16 high-risk, unaffected first-degree relatives of patients with schizophrenia, bipolar disorder, and/or schizoaffective disorder, 22 clinical control participants with a comorbid disorder (ADHD and/or an anxiety disorder), and 20 healthy control participants. Participants first completed a free recall task and then completed a recognition memory task. Based on established theories of episodic memory, we assumed that context processing played a more pivotal role in free recall than recognition memory. Consequently, if schizophrenia risk is associated with context processing deficits, then memory impairment should be present in free recall measures that are most sensitive to context processing (i.e., recall accuracy and temporal contiguity). Consistent with this prediction, free recall accuracy and temporal contiguity were lower for the high-risk group than the healthy controls, whereas recognition memory was comparable across groups. These findings suggest that episodic memory impairments associated with schizophrenia in unaffected, first-degree relatives may reflect context processing deficits.

Schizophrenia is a debilitating mental disorder that impairs cognition (for a review, see Barch and Ceaser, 2012). Episodic memory, defined as memory for objects and events tied to a specific space and time, is an aspect of cognition that is consistently impaired in schizophrenia prodrome (for a review, see Valli et al., 2012), patients with schizophrenia (Heinrichs and Zakzanis, 1998; Mesholam-Gately et al., 2009), and adults genetically at-risk for schizophrenia (i.e., first- and second-degree relatives; Kremen et al., 1998; Toomey et al., 1998; Toulopoulou et al., 2003). Some studies have reported episodic memory impairments in young first-degree relatives (aged 7–25 years old; Cosway et al., 2000; Hemager et al., 2018) that later predict schizophrenia diagnosis (Erlenmeyer-Kimling et al., 2000; Johnstone et al., 2005). This suggests that those most at-risk for developing the disorder may show such memory impairments. To characterize this vulnerability to memory impairment and its underlying mechanisms, we examined the role of context processing in children and adolescents at risk for schizophrenia.

Context processing in episodic memory reflects the ability to encode and retrieve relationships among event features, such as when, where, and with whom they occurred (see e.g., Anderson and Bower, 1972).

☆ The stimuli, data, and analysis scripts are available on the Open Science Framework: https://doi.org/10.17605/OSF.IO/D6B8M.

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https://doi.org/10.1016/j.scog.2022.100241
Received 17 November 2021; Received in revised form 26 January 2022; Accepted 27 January 2022
Available online 11 February 2022

Disruptions in such processing may underlie the episodic memory impairments observed in schizophrenia (Ranganath et al., 2008; Keefe et al., 2002). Episodic memory tasks involving self-initiated memory search (e.g., free recall and source memory) are often more sensitive to schizophrenia-related impairment than tasks requiring recognition decisions (e.g., Aleman et al., 1999; Paulsen et al., 1995, but see Sahakyan and Kwapil, 2016). This differential sensitivity suggests that schizophrenia-related memory impairment should be more pronounced in tasks assumed to require more self-initiated generation of contextual cues, such as free recall, than tasks that require less cue generation, such as recognition.

The assumption that context processing deficits underlie schizophrenia-related episodic memory impairments has inspired researchers to use free recall to identify such deficits. In free recall, participants study a list and later recall items in any order. The extent that items are recalled in the studied order, referred to as *temporal contiguity* (for a review, see Healey et al., 2019), is assumed to indicate effective context processing. Schizophrenia patients (Polyn et al., 2015) and people with first episode psychosis (Murty et al., 2018) who recall fewer words show less temporal contiguity than healthy controls. Similarly, young adults with high negative schizotypy symptoms, which predict schizophrenia-spectrum disorders (Kwapil et al., 2013), show poorer free recall accuracy and temporal contiguity than people in the normal range of negative symptoms (Sahakyan and Kwapil, 2016, 2018). Together, these studies suggest that temporal contiguity in free recall provides an assay of the contribution of context processing to schizophrenia-related episodic memory impairment.

The current study extends this research by testing the hypothesis that episodic memory impairments in young people at-risk for schizophrenia reflects context processing deficits. Children and adolescents varying in risk for schizophrenia completed tasks that differed in their context processing requirements. They first completed a free recall task and then completed a recognition task. Based on the context-deficit view of schizophrenia-related memory impairment, we expected that deficits associated with high risk for developing schizophrenia would appear selectively in the free recall measures.

1. Method

The current research was approved by the Institutional Review Board of the University of North Carolina at Chapel Hill. Participants were compensated for their time and travel.

1.1. Participants

We recruited 58 children and adolescents (24 female) aged 9 to 16 years from the UNC Psychiatry Child and Adolescent Clinic, the Outreach and Support Intervention Services, the Schizophrenia Treatment and Evaluation Program, public schools, and community clinics. Participants were assigned to one of three age-matched groups and the mean age was not significantly different across groups, $F(2, 55) = 0.35$, $p = 0.71$ (see Table 1). The high-risk group included 16 non-psychotic, first-degree relatives of a person with schizophrenia, bipolar disorder, and/or schizoaffective disorder. The clinical control group included 22 participants who were currently diagnosed with ADHD and/or an anxiety disorder with no family history of psychotic mental illnesses, as these disorders and schizophrenia share common disruptions in frontal-limbic brain circuitry (McTeague et al., 2017). These disruptions are associated with varying amounts of cognitive disorganization, reduced attention, and impaired cognitive control (Arnsten and Rubia, 2012), which are all critical for strategic retrieval of episodic context (Chun and Turk-Browne, 2007; Vatansever et al., 2021). The healthy control group included 20 participants with no family history of psychotic mental illnesses and no current clinical diagnoses. Note that all participants completed both tasks, but we excluded data from four participants in the recognition analyses due to computer error or confusion with response mapping. Clinical diagnoses were based on the Structured Clinical Interview for DSM-IV (SCID; First et al., 1995). We excluded people with chronic medical conditions, a history of psychotic or mood disorders, PTSD, substance abuse disorders, and current or previous use of psychotropic or cognition enhancing medications. Schizotypy was assessed with the Scale of Prodromal Symptoms (SOPS) to examine the relationship between schizotypy and memory scores (McGlashan et al., 2001).

1.2. Materials

The memory tasks included concrete nouns from the Medical Research Council (MRC) psycholinguistic database (Coltheart, 1981) with a maximum age of acquisition $\leq$9.1 years to ensure the youngest participants could recognize them. To maximize participants' reliance on temporal context in organizing their recall, we created a stimulus set with minimal inter-item associations. For more details about these and other stimulus characteristics, see the first section of the Supplementary Material document (SM1).

1.3. Procedure

The free recall task began with one practice trial, followed by five critical trials. The procedure was mostly identical in the practice and critical trials, but the practice trial included fewer study items (5 vs. 10) and a shorter recall period (30 s vs. 60 s) than the critical trials. During study, words appeared individually for 4 s each (250 ms inter-stimulus interval; ISI). Participants read words aloud and studied them for a test. They then completed a math task for 30 s before starting recall. During recall, participants typed as many words as they could remember from the preceding list in any order. Responses were entered individually and disappeared after each entry. To facilitate comfortable responding, participants were told not to worry about spelling accuracy. Each participant received the same lists of words, and each list contained a unique set of words. The order in which words appeared in a given list was randomized anew for each participant.

Table 1
Risk group characteristics in free-recall and recognition tasks.

Characteristic	Free-recall task			Recognition task		
	High risk	Clinical control	Healthy control	High risk	Clinical control	Healthy control
N	16	22	20	15	21	18
N females (%)	7 (43.75)	10 (45.45)	7 (35.00)	6 (40.00)	10 (47.61)	6 (33.33)
Mean age (SD)	13.30 (2.26)	13.19 (2.44)	13.73 (1.73)	13.26 (2.34)	13.05 (2.41)	13.67 (1.82)
Age range in years	9.60–16.30	9.10–16.70	10.11–16.90	9.60–16.30	9.10–16.70	10.11–16.90

Note. The sample included people with the following self-reported race/ethnicity: 36 (62%) Caucasian/White, 13 (22%) African American/Black, 7 (12%) Hispanic/Latino, and 2 (4%) other. Most clinical control participants (N = 15) and some high-risk participants (N = 5) who had a comorbid diagnosis of ADHD and/or an anxiety disorder had a history of treatment with stimulant or antidepressant medication. Participants were matched for age, gender, and education across risk-groups. The sample sizes were larger for the free recall than recognition task because four participants were excluded from the recognition analyses. Three excluded participants had negative discriminability (d') scores, indicating confusion with response mapping, and one excluded participant did not complete the task due to computer error.

The recognition task included a novel word set that was not used in the free recall task. Participants first read 60 words aloud and studied them for a test. Words appeared individually in random order for 3 s each (500 ms ISI). Participants then completed a math task for 120 s before starting recognition. During recognition, 120 words (60 studied and 60 foils) appeared individually in random order for 3 s each (500 ms ISI). Participants were allowed 3 s to decide if each word was studied by pressing the V and N keys to indicate if words were "old" (studied) or "new" (foils), respectively. Participants rated the confidence of each response (within 4 s) as "Definitely", "Maybe", or "Guess" by pressing the 1, 2, and 3 keys, respectively. Participants failed to make both responses for 9.56% trials; these missing observations were not significantly different across risk groups, $F(2, 54) = 0.94$, $p = 0.40$.

2. Results

The statistical approach (described in SM2) included frequentist and Bayesian frameworks because we hypothesized group differences in recall but not recognition, and the Bayesian approach quantified evidence for a null effect (Kruschke, 2011).

2.1. Free recall

2.1.1. Correct recall

Overall correct recall was compared among groups by averaging performance across the five critical trials (Fig. 1A). Models including Risk Group as a factor indicated a significant effect, χ^2 (2) = 6.49, p = 0.04, with strong evidence for the alternative hypothesis, $BF_{10} = 24.24$. Recall was significantly lower for the high-risk than the healthy control group, $t(55) = 2.53$, $p = 0.04$; no other differences were significant, largest $t(55) = 1.66$, $p = 0.23$. Models including the factors Risk Group and Trial were used to examine the consistency of this pattern across trials (Fig. 1B). There was a significant effect of Trial, χ^2 (4) = 24.53, $p <$ 0.001, and strong evidence for the alternative hypothesis, BF_{10} = 9594.57. Recall was significantly lower on trial 3 than trials 1, 4, and 5, smallest $t(220) = 3.20$, $p = 0.01$. The interaction was not significant, χ^2 (8) = 10.13, $p = 0.26$, but there was strong support for the alternative hypothesis, $BF_{10} = 835.15$. The qualitative pattern across trials shows that the high-risk group consistently had the lowest recall. There were few intrusions per trial on average (< 0.25), and intrusions were not significantly different across risk groups, largest χ^2 (2) = 2.23, $p = 0.33$. The next section summarizes analyses of temporal contiguity measures assumed to be the most sensitive to context processing. Additional measures of recall dynamics upon which group differences in context processing can be inferred appear in SM3. The main findings from the additional measures are summarized in the Discussion section.

2.2. Temporal contiguity

Temporal contiguity in recall can be assessed as response probabilities conditionalized on the lag between study list input positions (Lag-CRPs) that estimate the direction and distance of recall transitions (Kahana, 1996). The temporal contiguity effect presents as higher probabilities of forward than backward transitions between adjacent input positions. This context processing measure is assumed to indicate the extent that retrieved context cues subsequent retrievals (Howard and Kahana, 2002). Temporal contiguity can also be quantified as a single number by computing temporal factor scores that summarize the positional lag of each transition as the percentile of that transition within the distribution of all possible transitions (see Polyn et al., 2009). A score of 0.5 indicates recalling in random order and a score of 1.0 indicates perfect temporal organization. If the high-risk group has impaired context processing, then they should show lower probability of positive-near transitions and lower temporal factor scores.

Models comparing Lag-CRPs (Fig. 2) included Lag and Risk Group as factors. There was a significant effect of Lag, χ^2 (5) = 131.15, $p < 0.001$,

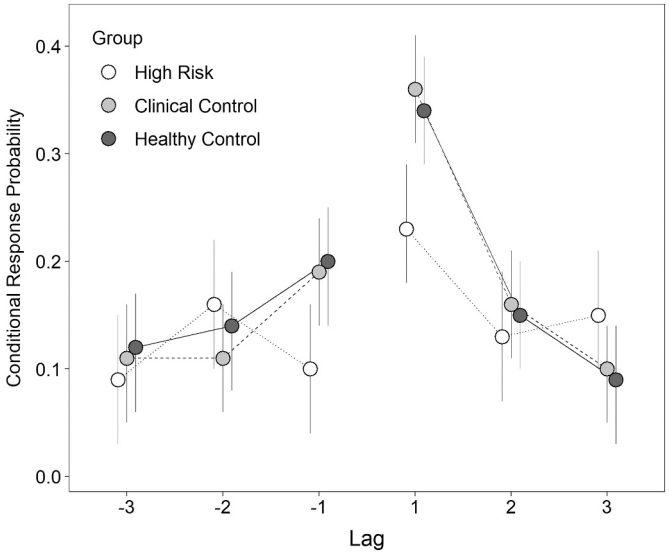

Fig. 2. Lag conditional response probabilities across risk groups.
Note. Conditional response probabilities estimated from a mixed effect model. The lags only ranged from −3 to 3 because there were sparse observations at longer lags. Error bars are 95% confidence intervals. The probabilities for recalls from adjacent input positions (−1 and 1) were significantly lower for the high-risk group than the clinical control and healthy control groups.

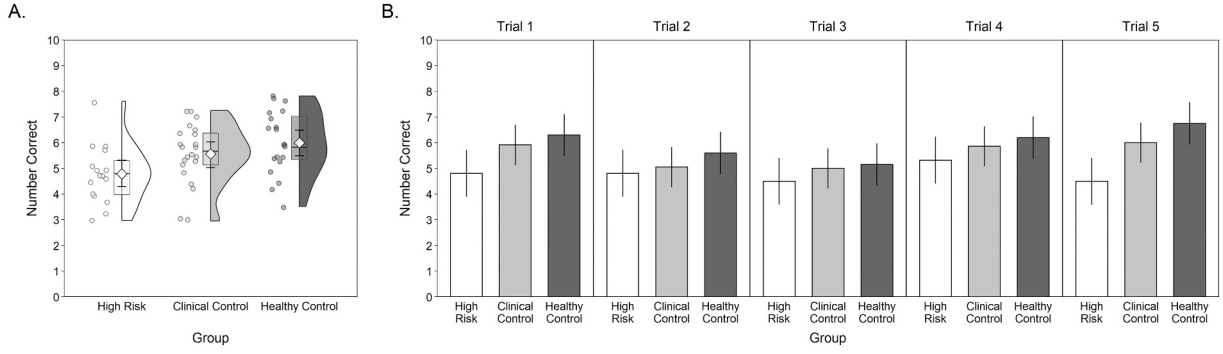

Fig. 1. Correct recall across risk groups and trials.
Note. (A) Correct recall averaged across trials was significantly lower for the high-risk than other groups. (B) This nominal pattern was consistent across all five trials. Group means are shown as the heights of white diamonds (A) and bars (B), and corresponding error bars are 95% confidence intervals. (A) Medians and interquartile ranges are displayed in boxplots. Distributional information is shown as individual participant estimates (dots) and the approximated frequencies of those estimates displayed as kernel probability densities (the width of corresponding half violin plots).

with strong support for the alternative hypothesis, $BF_{10} = 6.12 \times 10^6$, no significant effect of Risk Group, χ^2 (2) = 3.73, $p = 0.16$, with moderate support for the null hypothesis, $BF_{10} = 0.16$, and a significant interaction, χ^2 (10) = 22.13, $p = 0.01$, with strong support for the alternative hypothesis, $BF_{10} = 3020.96$. The interaction showed significantly lower probabilities at the nearest transitions (lags 1 and −1) for the high-risk than clinical control and healthy control groups, smallest $t(1559) = 2.43$, $p = 0.04$, and no other significant differences, largest $t(996) = 1.72$, $p = .20$. Models comparing temporal factors score with Risk Group as a factor indicated a significant effect, χ^2 (2) = 9.98, $p < 0.01$, and moderate support for the alternative hypothesis, $BF_{10} = 7.37$, showing that the score for the high-risk group (0.54, 95% *CI* = [0.47, 0.60]) was significantly lower than the scores for the clinical control (0.64, 95% *CI* = [0.58, 0.70]) and healthy control (0.63, 95% *CI* = [0.57, 0.69]) groups, largest $t(54.2) = 2.51$, $p = 0.04$, which were not significantly different, $t(55) = 0.47$, $p = 0.90$. These results suggest that the high-risk group showed impaired poorer context processing during recall.

The level of temporal contiguity can be affected by primacy, recency, and other serial position effects (Kahana, 1996; Healey et al., 2019). These effects can be controlled for by calculating chance-adjusted temporal factor scores (Healey, 2018) that compare the non-adjusted temporal factor scores to the scores that would be expected if transitions between items were random rather than determined by temporal contiguity (for details see Mundorf et al., 2021). Models comparing chance-adjusted scores with Risk Group as a factor indicated no significant effect, $\chi^2(2) = 2.56$, $p = 0.28$, with a weak support for the null hypothesis, $BF_{10} = 0.60$, even though the numerical trend was similar to the non-adjusted scores (high-risk = 0.33, 95% CI = [0.01, 0.66], clinical control = 0.53, 95% CI = [0.27, 0.82], healthy control = 0.62, 95% CI = [0.31, 0.93]). Notably, adjusted temporal factor scores were noisy due to our small sample size, which likely explains the discrepancy with the non-adjusted scores.

2.3. Recognition

Table 2 displays the overall probabilities of "old" responses to studied and foils items. Group differences in sensitivity (d') and response bias (c) were assessed using standard signal detection theory equations (Green and Swets, 1966). More information about the statistical approach used to assess recognition memory performance is available in SM4. The models for these measures included Risk Group as a factor. The models for sensitivity (Fig. 3A) indicated no significant effect of Risk Group, $F(2, 51) = 1.51$, $p = 0.23$, and weak support for the null hypothesis, $BF_{10} = 0.43$. The models for response bias (Fig. 3B) indicated no significant effect of Risk Group, $F(2, 51) = 0.65$, $p = 0.52$, and moderate support for the null hypothesis, $BF_{10} = 0.23$. Collectively, these results indicate that there were no group differences in memory sensitivity or response bias in the task with lower context processing demands and that most participants responded conservatively.

3. Discussion

The current study tested the hypothesis that young people at risk for schizophrenia should experience episodic memory impairment reflecting a context processing deficit. Supporting this hypothesis, children and adolescents at high genetic risk for schizophrenia showed impaired recall and reduced temporal contiguity relative to lower-risk groups but no recognition memory deficit. These results suggest that young people at high risk for schizophrenia experience context processing deficits in episodic memory. This finding is consistent with the broader literature showing comparable memory deficits associated with vulnerability to schizophrenia (Sahakyan and Kwapil, 2016, 2018), first-episode psychosis (Murty et al., 2018), and clinically diagnosed schizophrenia (Polyn et al., 2015).

Converging evidence for the proposal that episodic memory impairment associated with schizophrenia reflects context processing deficits also comes from computational modeling work identifying the mechanisms underlying free recall deficits present in other populations assumed to experience context processing deficits. For example, one computational model of free recall suggests that similar patterns of recall deficits and reduced temporal contiguity associated with healthy aging reflect impaired context retrieval during the recall period (e.g., Healey and Kahana, 2016). We further tested predictions from this computational model about context processing deficits by examining potential group differences in other dynamics of free recall. A complete description of the measures and results is available in SM3.

A comparison of serial position functions across groups (SM3.1, Fig. S1) showed recency effects for only the high-risk group, which was inconsistent with the uniform recall deficits across positions associated higher vulnerability for schizophrenia (Sahakyan and Kwapil, 2018) and clinically diagnosed schizophrenia (Polyn et al., 2015). According to a context-based view, such recency could indicate that the context associated with the end-of-list study items was most accessible after the study-test delay for the high-risk group. This could reflect slower processing of context changes across study items, but this result should be replicated and formally modeled before drawing strong conclusions. Notably, first recall probabilities (SM3.2, Fig. S2) did not differ across risk groups, which was inconsistent with the lower recall initiation probabilities from the first-studied items observed in people with higher schizophrenia vulnerability (Fig. S1) and with the prediction that context processing deficits should reduce recall initiation from those items (Sahakyan and Kwapil, 2018). This suggests that the particular context processing deficits associated with schizophrenia risk impacted context retrieval most after the first recall attempt. Finally, interresponse times between recalls (SM3.3, Fig. S3) revealed no group differences, which was also inconsistent with a context deficit view. But the nominal pattern did indicate the most rapid slowing for later recalls in the high-risk group, which paralleled findings in people with higher schizophrenia vulnerability (Sahakyan and Kwapil, 2018). Together, with the results reported in the main manuscript above, these additional findings suggest that if schizophrenia risk is associated with context processing deficits, there may be nuanced differences in the exact

Table 2
Hit and false alarm rates in recognition memory as a function of confidence level for each risk group.

Group	Measure							
	Hits				False alarms			
	All	Definitely	Maybe	Guess	All	Definitely	Maybe	Guess
High risk	0.75 [0.73, 0.78]	0.66 [0.63, 0.70]	0.06 [0.05, 0.08]	0.03 [0.02, 0.04]	0.17 [0.14, 0.19]	0.09 [0.07, 0.11]	0.06 [0.04, 0.07]	0.02 [0.01, 0.03]
Clinical control	0.74 [0.71, 0.76]	0.61 [0.58, 0.63]	0.10 [0.08, 0.12]	0.03 [0.02, 0.04]	0.15 [0.13, 0.18]	0.07 [0.06, 0.08]	0.05 [0.04, 0.06]	0.03 [0.02, 0.04]
Healthy control	0.68 [0.65, 0.71]	0.53 [0.50, 0.56]	0.11 [0.09, 0.13]	0.04 [0.03, 0.06]	0.18 [0.15, 0.20]	0.07 [0.06, 0.09]	0.05 [0.04, 0.07]	0.06 [0.04, 0.07]

Note. 95% confidence intervals appear in brackets.

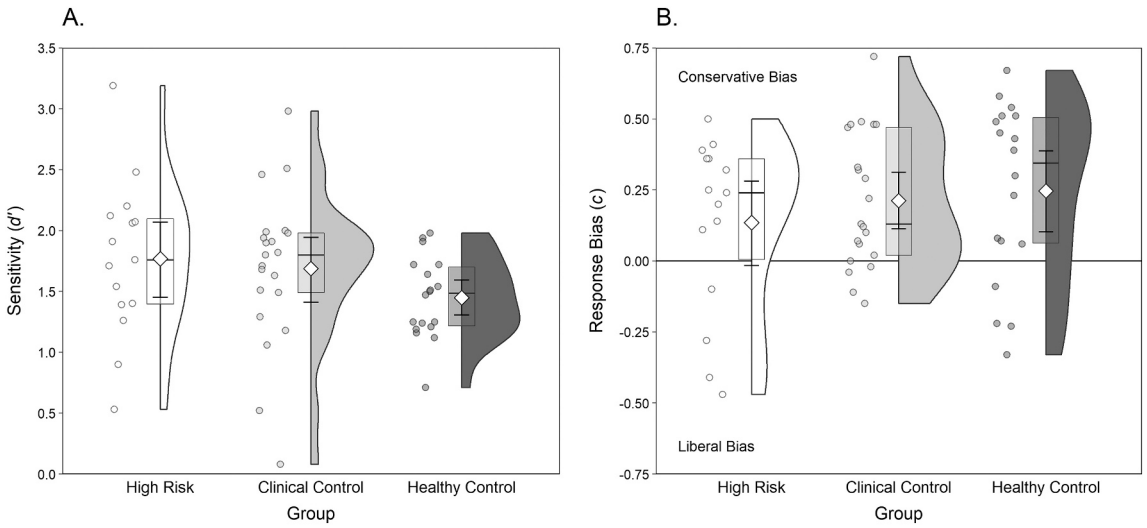

Fig. 3. Recognition sensitivity and response bias across risk groups.
Note. There were no significant between-group differences in (A) sensitivity (computed as *d'*) or (B) response bias (computed as *c*). Group means are shown as white diamonds and corresponding errors bars are 95% confidence intervals. Medians and interquartile ranges are displayed in boxplots. Distributional information is shown as individual participant estimates (dots) and the approximated frequencies of those estimates displayed as kernel probability densities (the width of corresponding half violin plots).

contextual mechanisms underlying group differences reported here relative to studies comparing other at-risk populations. These discrepancies among populations could be clarified in replication attempts including more recall trials, larger samples, and formal computational modeling.

As predicted, recognition memory did not differ across groups. This bolsters the assertion that the risk-related cognitive impairments reflect deficits in some aspects of context processing. However, the available literature is mixed as some studies of patients with schizophrenia report no recognition deficits (Mathews and Barch, 2004), while others report impairment as fewer hits (Heckers et al., 2000) and more false alarms (Weiss et al., 2004). Additionally, higher negative symptom schizotypy is associated with fewer hits, while higher positive symptom schizotypy is associated with more false alarms (Sahakyan and Kwapil, 2019). The inconsistency across tasks and populations could partly reflect variation in context processing requirements across tasks. Here, the high-risk group may have shown intact recognition because the task had low context processing requirements (i.e., one study list). Alternatively, the observed differential deficit in memory performance may not have truly reflected a differential deficit in ability. Instead, the true-score variance may have been smaller for recognition than recall, thus obscuring detection ability differences among groups using the recognition task (cf. Miller et al., 1995). This limitation could be addressed in future studies by matching mean performance levels in the recall and recognition tasks. If context processing deficits were observed here, they may have been less pronounced and/or qualitatively different in at-risk youth than adults vulnerable to or diagnosed with schizophrenia. Finally, since recognition always appeared after recall, the task order could have interacted with context processing differences. Note that the more sensitive test of context processing (i.e., free recall) first appeared to improve detection of group differences.

Although we observed several deficits associated with high risk for schizophrenia, we did not observe any significant differences associated with the clinical control group. As a reminder, the clinical control group included participants with ADHD and anxiety disorders due to high-rates of such disorders being present in both schizophrenia patients (Braga et al., 2013; Ross et al., 2006) and their first-degree relatives who subsequently developed schizophrenia (Johnstone et al., 2005; Parnas et al., 1982). However, including two comorbid disorders in this group may have limited our ability to detect impairments if they exist because children with ADHD sometimes have intact (Skowronek et al., 2008) and other times impaired episodic memory (Groom et al., 2008), whereas people with anxiety disorders are at times impaired in free recall (Airaksinen et al., 2005). Here, the clinical control group showed better temporal contiguity than the high-risk group, indicating that the present sample of children with ADHD and/or an anxiety disorder were not impaired in this type of temporal context processing.

Finally, we examined group differences in schizotypy symptoms and the relationship between those symptoms and memory measures varying in sensitivity to context processing (see SM5). Regardless of risk-group status, there was a selective negative association between negative schizotypy symptoms and temporal factor scores (the most sensitive measure of context processing in these analyses; Fig. S5). This relationship is conceptually consistent with findings suggesting that high negative symptom schizotypy is associated with context processing deficits in episodic memory (Sahakyan and Kwapil, 2018).

In summary, the present study provides the first characterization of the similarities and differences in aspects of episodic memory in children and adolescents at-risk for schizophrenia. We tested the hypothesis that context deficits associated with risk of schizophrenia would lead to episodic memory deficits in a high-risk group on recall measures most sensitive to context processing. Consistent with this hypothesis, high-risk, first-degree relatives of people with schizophrenia and related disorders had impaired free recall performance and temporal organization of subsequent recalls indicative of impaired context processing. This is consistent with patterns of episodic memory previously observed in schizophrenia patients (Murty et al., 2018; Polyn et al., 2015; Toulopoulou et al., 2003) and young adults with high negative schizotypy symptoms (Sahakyan and Kwapil, 2016, 2018). However, we also detected some inconsistencies, primarily in first recall probabilities and interresponse times, suggesting that context-processing deficits observed in this population have nuanced on retrieval dynamics in free recall. The exact nature of context processing deficits could be identified in future work using variants of a context-based computational model of free recall (e.g., Healey and Kahana, 2016). This model-based approach could be used to identify deficits in specific contextual mechanisms within participants, thus enabling prediction of schizophrenia development.

Funding

This work was supported by the National Institute of Mental Health (NIMH) under Grant 5R01MH103790-05, the David Bray Peele and the Lindquist Undergraduate Research Awards from the University of North Carolina, Chapel Hill.

CRediT authorship contribution statement

Aslıhan İmamoğlu: Formal analysis, Investigation, Writing – original draft, Writing – review & editing, Visualization, Project administration. **Claudia Foubert:** Conceptualization, Methodology, Software, Investigation, Writing – original draft, Writing – review & editing, Project administration, Funding acquisition. **M. Karl Healey:** Formal analysis, Data curation, Writing – original draft, Writing – review & editing. **Stephanie Langella:** Methodology, Software, Writing – review & editing. **Aysenil Belger:** Conceptualization, Methodology, Resources, Project administration, Funding acquisition. **Kelly S. Giovanello:** Conceptualization, Methodology, Writing – original draft, Writing – review & editing, Supervision, Project administration, Funding acquisition. **Christopher N. Wahlheim:** Conceptualization, Methodology, Software, Formal analysis, Data curation, Writing – original draft, Writing – review & editing, Visualization, Supervision.

Declaration of competing interest

The authors have no conflicts of interest to declare that are relevant to the content of this manuscript.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.scog.2022.100241.

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