

RESEARCH ARTICLE

Atypical development of social and nonsocial working memory capacity among preschoolers with autism spectrum disorders

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Abstract

Individuals with autism spectrum disorders (ASD) have shown impaired performance in canonical and nonsocial working memory (WM). However, no study has investigated social WM and its early development. Using biological motion stimuli, our study assessed the development of social and nonsocial WM capacity among children with or without ASD across the age span between 4 and 6 ($N = 150$). While typically developing (TD) children show a rapid development from age 5 to 6, children with ASD showed a delayed development for both social and nonsocial WM capacity, reaching a significant group difference at age 6. Furthermore, we found a negative correlation between social (but not nonsocial) WM capacity and the severity of autistic symptoms among children with ASD. In contrast, there is a positive correlation between both types of WM capacity and intelligence among TD children but not among children with ASD. Our findings thus indicate that individuals with ASD miss the rapid development of WM capacity in early childhood and, particularly, their delayed social WM development might contribute to core symptoms that critically depend on social information processing.

Lay Summary

We examined the developmental trajectory of social and nonsocial working memory (WM) capacity among children with autism spectrum disorders (ASD) in the age span from 4 to 6. Compared with normal children, children with ASD lack rapid improvement in both types of WM. The severity of core autistic symptoms but not IQ correlates with social WM capacity among children with ASD; in contrast, IQ but not the severity of core symptoms correlates with WM capacity among normal children.

KEYWORDS

autism spectrum disorders, biological motion, early development, social processing, social working memory

INTRODUCTION

Autism spectrum disorder (ASD) is a heritable neurodevelopmental disorder characterized by impaired social interaction, interpersonal communication, and restrictive and repetitive behavior (DSM-5, American Psychiatric Association, 2013). Though the core symptoms of autism are mostly related to social and communication aspects, researchers have found extensive cognitive deficits in this population, including working memory (Kercood et al., 2014; Wang et al., 2017), attention (Koolen

et al., 2014), and processing speed (Eack et al., 2013). In fact, cognitive deficits have been shown as a strong predictor of autistic symptoms, especially for language and communication skills (Kjellmer et al., 2012; Luyster et al., 2008; Thurm et al., 2007) and adaptive behaviors (Chang et al., 2013). Working memory (WM) refers to the ability to maintain and manipulate a limited set of information for a short period in an ongoing task (Baddeley & Hitch, 1974). It supports various cognitive abilities, such as fluid intelligence, scholastic aptitude, information selection, planning, reasoning, and language

processing (Baddeley, 2003; Conway et al., 2002; Cowan et al., 2012; Hollingworth et al., 2008; Unsworth et al., 2014). Most WM studies in autism have used canonical WM stimuli, for instance, letters, numbers, visual objects (e.g., colors, shapes), and object locations. Across the life span, individuals with ASD showed impaired WM performance (for reviews, Kercood et al., 2014; Wang et al., 2017), which is correlated with the severity of communication deficits (Gilotty et al., 2002; Oliveras-Rentas et al., 2012) and restrictive and repetitive behavior symptoms (Lopez et al., 2005; Sachse et al., 2013). To our best knowledge, previous studies only used nonsocial stimuli for assessing WM performance despite the fact that elucidating the atypicality in social information processing is essential for understanding the etiology and development of autism (Klin et al., 2009).

WM of social information have been recently investigated in neurotypical population (e.g., He et al., 2019; Heleven & Van Overwalle, 2016; Meyer et al., 2012; Schweizer et al., 2019; Sippel et al., 2021; Smith et al., 2017; Thornton & Conway, 2013; Tsukiura & Umeda, 2017; Xin & Lei, 2015). Social stimuli employed in these studies included human faces with emotion, people's names (together with trait words), and biological motion (BM) stimuli. WM with social information involves distinct neural substrates from WM with nonsocial information (Lu et al., 2016; Meyer et al., 2012). For example, WM with people's names recruits the mentalizing network, including the dorsomedial and ventromedial prefrontal cortex and the temporoparietal junction (Meyer & Lieberman, 2012; Meyer et al., 2012, 2015). This network is deactivated during canonical WM tasks with stimuli such as letters and locations, highlighting the importance of distinguishing the stimuli that are to be processed. Retaining BM in WM requires the involvement of the mirror neuron system (Gao et al., 2015; Lu et al., 2016), which has an intimate relation with social cognition (Rizzolatti & Fabbri-Destro, 2008). Important for autism research, the behavioral performance and its neural correlates in social WM tasks can predict critical social abilities, such as perspective taking (Meyer et al., 2012; Meyer et al., 2015), empathy (Gao et al., 2016; Xin & Lei, 2015), and theory of mind (He et al., 2019), which are of enormous importance for individuals with autism.

Here we opted to use BM as social stimuli for assessing WM capacity among children with ASD. BM, especially its common form of point-light displays (PLD, Johansson, 1973; Troje, 2013), carries movement information of animate entities without other reference information such as clothes, texture, and hairstyle. Individuals with ASD have shown impairments in BM processing (for a review, Todorova et al., 2019), for example, children with ASD exhibited poor BM perception (Blake et al., 2003; Koldewyn et al., 2011), which were related to

the severity of core autistic symptoms (Kaiser et al., 2010). BM also offered us a unique opportunity to probe the development of social WM in early childhood compared to other stimuli (i.e., human face with emotion and people's names; for a deliberation, see Introduction in He et al., 2019). Our recent work has revealed a critical development period among typically developing (TD) children: WM of BM, but not that of canonical stimuli, develops rapidly around age 4 and 5, and its capacity positively correlates with theory of mind (He et al., 2019). Interestingly, ages 4 and 5 are viewed as a critical period for the development of social ability, such as theory of mind (Liu et al., 2008; Wellman et al., 2001). Thus, it would be important to examine the development of social WM in this critical period and its association with core autistic symptoms.

Furthermore, we noticed that WM capacity was understudied in autism since most previous studies examined WM performance that reflected a combined effect of WM capacity and manipulation instead of capacity *per se* (Wang et al., 2017). The estimation of WM capacity demands the use of rigorous methods, for example, change detection or continuous-feature recall paradigms, to limit the role of information manipulation (Cowan, 2001; Zhang & Luck, 2008). The few studies that employed these methods revealed that individuals with ASD have intact or even better WM capacity for nonsocial stimuli (Bodner et al., 2019; Lynn et al., 2022; Stevenson et al., 2021), but its early development, especially in preschoolers, was left unchecked (see Cowan, 2016, for a review; Riggs et al., 2006; Simmering, 2012).

In the present study, we examined the capacity of social and nonsocial WM using a unified paradigm involving change detection of biological motion stimuli. We used both normal BM as social stimuli and inverted BM (IBM) as a nonsocial counterpart of BM, as widely accepted in WM and perception studies (Gu et al., 2019; He et al., 2019; for a review, Hemeren, 2008; Shi et al., 2018). Moreover, we recruited children with ASD and neurotypical children aged 4 to 6 ($n = 25$ for each age), a critical period for social WM development (He et al., 2019). We compared social and nonsocial WM capacities within and between children with and without ASD and examined possible associations between WM capacity and the severity of core symptoms of autism.

METHODS

Participants

The present study recruited 150 children between the ages 4 and 6, including 75 children with ASD and 75 typically-developed (TD) children. Considering that He et al. (2019) recruited 24 children for each age group, we

decided to recruit 25 children with ASD for each of the three age groups (4-year-olds, 5-year-olds, and 6-year-olds). They were from a special school providing autism intervention and education in Qingdao, China. Similarly, the TD group included 25 neurotypical children for each age group recruited from a kindergarten in the same city. Their IQ and age were matched with the ASD group (Table 1). All children with ASD in this study had been previously diagnosed by experienced clinicians based on the Diagnostic and Statistical Mental Disorders-Fifth Edition. We also obtained the Chinese version of the Autism Spectrum Quotient: Children's Version (AQ-Child; Auyeung et al., 2008) and the Social Responsiveness Scale (SRS; Constantino & Gruber, 2002) to confirm the diagnosis for children with ASD before the experiment. The AQ is a 50-item questionnaire rated by children's caregivers on a four-point Likert scale from 0 (representing definitely agree) to 3 (representing definitely disagree) based on children's behaviors in the last 6 months. The SRS is a 65-item parent-report questionnaire on a four-point Likert scale from 0 ("behavior does not occur") to 3 ("behavior always occurs") based on children's behaviors in the last 6 months. The cut-off scores of AQ and SRS are 76 and 59.9, respectively. For both, the larger the scores are, the more severe the autistic symptoms. We measured their IQ by the Chinese version of Wechsler Preschool and Primary Scale of Intelligence-Forth Edition (WPPSI-IV, Wechsler, 2014). Participants' characteristics are summarized in Table 1.

The research was approved by the Ethical Committee of the School of Psychological and Cognitive Sciences at Peking University. Children's oral consent and their parents' written consent were obtained before the experiment.

Stimuli and apparatus

We used seven types of PLDs of BM from the database created by Vanrie and Verfaillie (2004), including cycling, jumping, painting, spading, walking, waving, and chopping. Each PLD was depicted by 13 light points placed at distinct joints of the human body to form a meaningful human movement. Each PLD comprised 30 distinct frames, each displayed twice in succession, resulting in a 1-s animation with a 60 Hz refresh rate. The PLDs were presented on a gray (128, 128, 128 in RGB value) background on a 23-in LCD monitor. The PLDs subtended a visual angle of $3.68^\circ \times 2.05^\circ$ from a visual distance of 70 cm. Similar to the previous studies (He et al., 2019; Shen et al., 2014), we randomly presented one to five distinct stimuli in a WM trial. The stimuli were either in normal upright orientation (BM stimuli) or in an upper-side down orientation (inverted, IBM stimuli). Each stimulus was shown at one of the five possible locations that were evenly distributed on the periphery of a 4.88° -radius circle centered in the mid of the screen.

TABLE 1 Participants' characteristics

	4-year-olds			5-year-olds			6-year-olds			4 ~ 6-year-olds		
	ASD		TD	ASD		TD	ASD		TD	ASD		TD
	M (SD)	r-value	M (SD)	M (SD)	r-value	M (SD)	M (SD)	r-value	M (SD)	M (SD)	r-value	M (SD)
N (female)	25 (0)	N/A	24 (7)	25 (2)	N/A	25 (8)	25 (4)	N/A	25 (9)	75 (6)	N/A	74 (24)
Age	4.57 (0.29)	-1.46	4.68 (0.22)	5.51 (0.30)	0.32	5.48 (0.34)	6.46 (0.27)	0.32	6.32 (0.24)	5.51 (0.83)	1.96	5.50 (0.72)
VIQ	108.76 (15.77)	-1.56	114.25 (8.80)	106.60 (15.90)	-0.89	110.16 (12.02)	107.72 (13.22)	-0.89	111.20 (13.05)	107.69 (14.62)	-0.94	111.84 (11.44)
FSIQ	111.20 (12.08)	-0.96	114.08 (8.59)	109.92 (14.14)	0.50	108.00 (12.89)	107.60 (9.07)	0.50	111.32 (10.45)	109.57 (11.88)	-1.35	111.09 (10.96)
AQ	89.68 (14.17)	8.62***	62.17 (7.19)	88.44 (13.08)	53.32 (8.19)	53.32 (8.19)	57.56 (15.39)	6.76***	57.56 (15.39)	90.44 (14.19)	8.14***	59.31 (11.05)
SRS	87.64 (18.28)	10.44***	44.13 (9.82)	91.60 (18.83)	40.68 (11.69)	40.68 (11.69)	100.12 (26.71)	11.49***	40.00 (10.98)	93.12 (21.96)	10.41***	41.57 (10.87)

Abbreviations: AQ, autism spectrum quotient; FSIQ, full-scale intelligence quotient; SRS, social responsiveness scale; VIQ, verbal intelligence quotient. *** $p < 0.001$.

Design and procedure

The children were tested in two sessions, one for BM and one for IBM. The order of the two tests was counterbalanced among children. For each test, the children were encouraged to memorize stimuli of increasing set size (1, 2, 3, 4, or 5); each set size was tested in a block of 12 trials (Case et al., 1982), resulting in a total of 60 trials in a session (Case et al., 1982). The children were told that they would play an increasingly challenging game. To avoid verbal encoding of the BM stimuli, the experimenter never mentioned the words depicting actions of BM stimuli, such as “walking” and “jumping.” Following He et al. (2019), we did not add a digit articulatory task to the WM task to prevent participants from verbally encoding the stimuli. This is because children do not develop verbal encoding strategies until around 7 years old (Hitch et al., 1989). Before the formal data collection, children were required to perform set Size 1 and set Size 2 trials until they accumulated four successful trials for each set size.

Each trial began with a red fixation cross displayed at the center of the screen for 300 ms, and the participants were encouraged to fixate on it (Figure 1). Then the screen turned blank for a random interval of 150 ~ 350 ms before the memory array of a varying number of BM stimuli was simultaneously presented on the screen. Each stimulus was shown at one of the five possible random locations with a presentation time of N s (N was the number of stimuli) such that participants had enough encoding time. Next, after showing a blank interval for 1 s, we presented a recognition probe BM stimulus at the center of the screen for 1 s. The children were required to make a judgment on whether the probe had ever appeared in the memory array. The verbal responses of children were recorded by the experimenters with an external keyboard. The children’s judgments were not time constrained. The keyboard input would also trigger a trial-end feedback, a smiling cartoon face for a correct response or a frowning cartoon face for an incorrect response, presented at the center of the screen for 300 ms.

The probability that the probe was in the memory array was 50%, and the stimuli of the memory array and

the probe were randomized across trials. A trial would start when children attended and made a response in the previous trial; otherwise, the same trial would repeat. The children were allowed to decide the duration of their rest time between test blocks.

Data analysis

One 4-year-old TD child did not finish the working memory task due to discomfort during the experiment, and his data were not included in the analysis. We used Cowan’s formula (see Cowan, 2001 for formula derivation) to estimate the number of representations stored in WM, $K = S \times (H - F)$. Where S is the set size (ranging from 1 to 5 in different blocks of trials in the current study), H (hit rate) is the ratio that participants correctly detecting a changed probe among the trials containing a changed probe, F (false alarm rate) is the ratio that participants erroneously reporting a changed probe among the trials not containing a changed probe. It has been revealed that if the numbers of to-be memorized items is smaller than WM capacity or the number of to-be-memorized items still increases after WM reaching its capacity, the calculated K value will be smaller than the true WM capacity (e.g., Balaban et al., 2019). Therefore, to avoid underestimating the WM capacity (i.e., the maximum amount of information WM can hold in a time), researchers conventionally manipulated the number of to-be-memorized stimuli in the change detection task. Given our previous studies on TD children (He et al., 2019), we chose the set size from 1 to 5. For the same reason, we followed previous WM studies (e.g., Curby & Gauthier, 2007; Gao et al., 2015; Gao et al., 2016; He et al., 2019; Shen et al., 2014) and set the largest K -value among the K estimates under different set sizes (i.e., K -max) to represent the WM capacity, which we argue is a reliable index in estimating the WM capacity. A value of K -max indicates that a specific participant can retain at most K -max target items in WM. For each stimulus type (BM and IBM), we calculated the K for each set size and then obtained the maximum K (K -max) among the five set-size conditions as the children’s WM capacity (Shen et al., 2014). To compare the

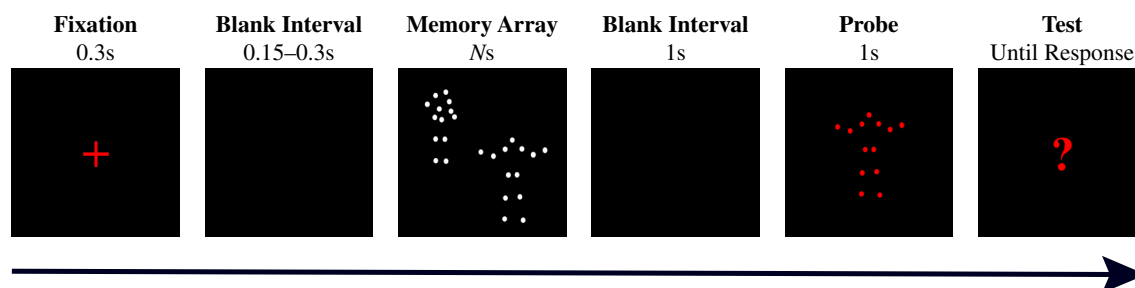


FIGURE 1 A schematic illustration of a trial. The stimuli are magnified here for clarity

difference between the two stimulus types, we performed a two-way, mixed-design analysis of variance (ANOVA) with stimulus type (BM vs. IBM) as a within-subject factor and age as a between-subject factor for each participant group separately. To compare group differences, we also performed a two-way ANOVA on *K*-max with the age and group (ASD vs. TD) as between-subject factors, separately for the two stimulus types.

To reveal the influence of age, IQ, and the severity of autistic symptoms (SRS) on WM capacity, we first examined simple Pearson correlations among ASD and TD separately and then with collapsed data across groups. We also performed multiple linear regression on the *K*-max with age, IQ, and SRS as predictors. The contribution of the three predictors was further examined by partial correlation. The α level was set at 0.05.

RESULTS

We measured both social and nonsocial WM capacity among children with ASD and their age- and IQ-matched controls by requiring them to memorize a varying number of BM stimuli. We found that both social and nonsocial WM capacity for TD children increased with age between 4 and 6. However, for children with ASD, both social and nonsocial WM capacity showed poor development during the same age span. Besides the age effect, we found that WM capacity of both BM and IBM stimuli correlated with IQ among TD children but children with ASD did not show a similar IQ correlation. Instead, their WM capacity of BM but not IBM correlated with the severity of core autistic symptoms.

The comparison of WM capacity between social and nonsocial stimuli

We first explored whether BM and IBM stimuli made a difference for our participants. We conducted two-way repeated-measures ANOVAs on *K*-max with age as a between-subject factor and stimulus type as a within-subject factor for the ASD and the TD group separately. For the TD group, the two-way ANOVA showed a significant main effect of age ($F[2, 71] = 11.90, p < 0.000, \eta_p^2 = 0.25$), a nonsignificant main effect of stimulus type ($F[1, 71] = 0.70, p = 0.17, \eta_p^2 = 0.026$), and a nonsignificant interaction ($F[2, 71] = 0.86, p = 0.48, \eta_p^2 = 0.024$). Children with ASD showed a similar lack of difference between BM and IBM. The two-way ANOVA yielded an insignificant main effect of stimulus type ($F[1, 72] = 0.35, p = 0.56, \eta_p^2 = 0.005$) and interaction effect ($F[2, 72] = 1.75, p = 0.18, \eta_p^2 = 0.046$). Importantly, the main effect of age failed to reach significance ($F[2, 72] = 1.71, p = 0.19, \eta_p^2 = 0.043$), indicating that the development of WM capacity was not evident for the age span we examined among children with ASD. Overall,

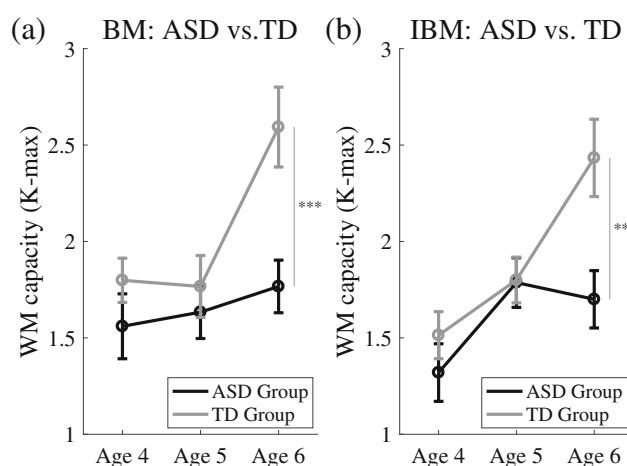


FIGURE 2 WM capacity as a function of age and participant group, shown separately for BM (a) and IBM (b) stimuli. The TD group showed significantly higher capacity than the ASD group only at age 6 for both types of stimuli. The error bars indicate the standard errors. *** $p < 0.001$, ** $p < 0.01$

we did not observe a significant difference in WM capacity for social and nonsocial stimuli for both groups.

Group comparisons for WM capacity of social stimuli

Within-group analyses above appeared to indicate that WM capacity increased with age among TD children but not among children with ASD¹ (Figure 2a). We further examined possible group differences by using the two-way group $\times$ age ANOVA on *K*-max for BM stimuli. For BM stimuli, the group by age interaction was marginally significant with a small effect size ($F[2, 143] = 2.83, p = 0.06, \eta_p^2 = 0.038$). We also found a significant main effect of group ($F[1, 143] = 9.67, p = 0.002, \eta_p^2 = 0.063$), suggesting that on average children with ASD had a lower WM capacity than TD. The average *K*-max was 1.65 ± 0.73 and 2.06 ± 0.90 for ASD and TD groups, respectively. The main effect of age was also significant ($F[2, 143] = 6.50, p = 0.002, \eta_p^2 = 0.083$). Post hoc comparisons (Bonferroni-corrected) indicated that 6-year-olds had a higher *K*-max than both 4- and 5-year-olds ($p = 0.005$ and $p = 0.008$, respectively), while there was no difference between 4- and 5-year-olds ($p = 1.00$). As the interaction was marginally significant, the main effect of age was mainly driven by the data from the TD group. We then analyzed the simple main effect among different groups. For TD children, the 6-year-olds showed significantly larger *K*-max than the 5- and 4-year-olds ($p = 0.001$ and $p = 0.002$, respectively), while there was no difference between 4- and 5-year-olds ($p = 1.00$). The

¹We further analyzed *K*-value of each set size, and found no group difference across each set size.

TABLE 2 Correlation coefficients from simple Pearson correlation analysis

	WM capacity of BM	WM capacity of IBM	Age	Score of AQ	Score of SRS	FSIQ
	All (ASD and TD) ^a	All (ASD and TD)	All (ASD and TD)	All (ASD and TD)	All (ASD and TD)	All (ASD and TD)
WM capacity of BM	-	-	-	-	-	-
WM capacity of IBM	0.52*** (0.47*** and 0.52***)	-	-	-	-	-
Age	0.24*** (0.10 and 0.41***)	0.32*** (0.21 and 0.45***)	-	-	-	-
Score of AQ	-0.26** (-0.17 and -0.08)	-0.18* (-0.07 and 0.01)	-0.05 (0.02 and -0.24*)	-	-	-
Score of SRS	-0.28*** (-0.31** and 0.07)	-0.17* (-0.06 and 0.06)	0.05 (0.20 and -0.15)	0.85*** (0.75*** and 0.22)	-	-
FSIQ	0.21** (0.16 and 0.23*)	0.15 (0.11 and 0.17)	-0.11 (-0.20 and 0.13)	-0.03 (0.05 and 0.01)	-0.11 (-0.10 and -0.08)	-

^aCorrelation performed with pooled data as well as with data from each group (in the parenthesis).

*** $p < 0.001$; ** $p < 0.01$; * $p < 0.05$.

average K -max was 1.80 ± 0.56 , 1.77 ± 0.68 , and 2.59 ± 1.04 for 4-, 5-, and 6-year-olds, respectively. For the ASD group, there was no significant difference between age groups, with an average K -max of 1.56 ± 0.84 , 1.633 ± 0.68 , and 1.77 ± 0.68 for 4-, 5-, and 6-year-olds, respectively. Furthermore, simple main effect of group showed that TD children performed better than children with ASD only at age 6 ($p < 0.001$), not at age 4 ($p = 0.29$) or 5 ($p = 0.55$). In sum, social WM capacity increased with age among TD children, especially from age 5 to 6, while the same trend was not significant among children with ASD.

Group comparisons for WM capacity of nonsocial stimuli

For WM capacity, IBM showed similar group and age effects as BM² (Figure 2b). The same two-way ANOVA on K -max of IBM revealed a significant main effect of group ($F[1, 143] = 6.77$, $p = 0.01$, $\eta_p^2 = 0.045$), showing that children with ASD had a lower WM capacity than TD (average K -max of 1.60 ± 0.73 and 1.92 ± 0.84 for the ASD and the TD group, separately). The age $\times$ group interaction was significant ($F[2, 143] = 3.24$, $p = 0.04$, $\eta_p^2 = 0.043$). The simple main effect of age among the TD group showed that 6-year-olds had significantly larger K -max than both 4- and 5-year-olds ($p < 0.001$ and $p = 0.008$, respectively). Four-year-olds did not significantly differ from 5-year-olds ($p = 0.53$). The K -max was 1.51 ± 0.60 , 1.80 ± 0.59 , and 2.43 ± 1.00 for 4-, 5-, and 6-year-olds, respectively. Thus, similar to BM, we observed a rapid development in the WM

capacity of IBM from age 5 to 6 among TD children. In contrast, the simple main effect of age among children with ASD showed that the K -max of IBM was not significantly different between age groups (4 vs. 5, $p = 0.076$; 4 vs. 6, $p = 0.21$; 5 vs. 6, $p = 1.00$). The K -max was 1.32 ± 0.75 , 1.79 ± 0.64 , and 1.70 ± 0.74 for 4-year-olds, 5-year-olds, and 6-year-olds, respectively. Simple main effects of group showed that TD children showed a higher K -max than children with ASD at age 6 ($p = 0.001$) but not at age 4 ($p = 0.36$) or 5 ($p = 0.95$). These results indicated that TD significantly increased the WM capacity of IBM from age 5 to age 6, but children with ASD failed to show the same age effect.

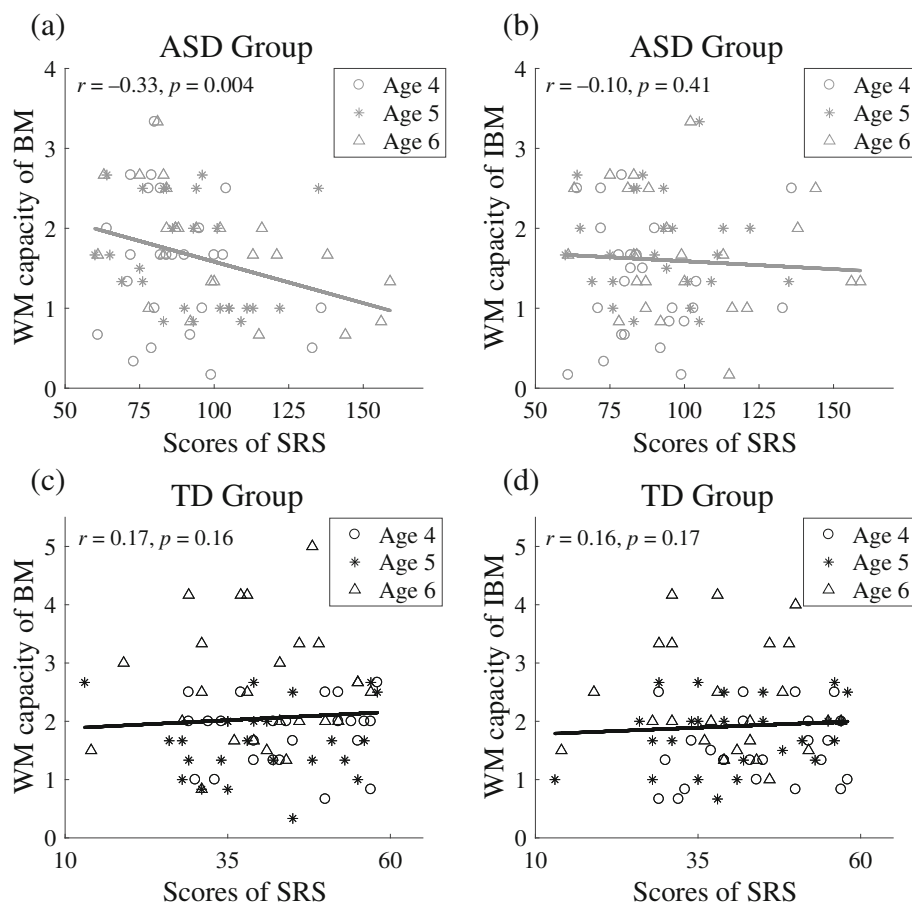
Correlations between WM capacity, age, IQ, and severity of core autistic symptoms

To examine the effect of age, IQ, and the severity of core autistic symptoms (measured by SRS and AQ) on the WM capacity, we first examined simple Pearson correlations with collapsed data samples from the two groups and with data from the two groups separately (Table 2). The three factors (age, IQ, and scores of SRS or AQ) showed some correlations with K -max, and importantly they were not significantly correlated with each other either within each group or with collapsed data.

We then analyzed how these three factors affect WM capacity among ASD and TD groups separately by multiple linear regression. We found that for the TD group the WM capacity of BM can be predicted by age ($\beta = 0.41$, $t = 7.72$, $p < 0.001$) and IQ ($\beta = 0.22$, $t = 2.11$, $p = 0.04$, Figure 4c), but not by severity of autistic symptoms (AQ: $\beta = 0.01$, $t = 0.13$, $p = 0.90$; SRS: $\beta = 0.15$, $t = 1.43$, $p = 0.16$, Figure 3c). The linear model was significant ($F[3, 70] = 7.23$, $p < 0.001$) with a

²We further analyzed K -value of each set size, and found no group difference across each set size.

FIGURE 3 WM capacity plotted as a function of SRS score. Data are shown in separate plots based on group and stimulus types, including (a) BM for ASD group, (b) IBM for ASD group, (c) BM for TD group, and (d) IBM for TD group. Each data point is from a participant; different symbols denote different age groups. The partial correlation coefficients and the p -values from multiple linear regression analyses are shown for each plot



partial r^2 of 23.6%. Similarly, the WM capacity of IBM could also be predicted by age ($\beta = 0.47, t = 4.51, p < 0.001$) and IQ ($\beta = 0.18, t = 1.74, p = 0.08$, Figure 4d), but not by severity of autistic symptoms (AQ: $\beta = 0.12, t = 0.14, p = 0.26$; SRS: $\beta = 0.15, t = 1.38, p = 0.17$, Figure 3d). The linear model was also significant with a partial r^2 of 25.2% ($F[3, 70] = 7.88, p < 0.001$). The age effect for TD children was further supported by a significant partial correlation of age and WM capacity (BM: $r[70] = 0.41, p < 0.001$; IBM: $r[70] = 0.45, p < 0.001$) when IQ and SRS were controlled as covariates. The IQ effect was also supported by a significant partial correlation of IQ and WM capacity using age and SRS as covariates, though the correlation with the K -max of IBM was only marginally significant (BM: $r[70] = 0.26, p = 0.03$; IBM: $r[70] = 0.20, p = 0.087$).

In contrast, for the ASD group, multiple linear regression analyses revealed that only severity of autistic symptoms (SRS: $\beta = -0.33, t = -2.96, p = 0.004$, Figure 3a) could predict the WM capacity of BM, while age ($\beta = 0.20, t = 1.41, p = 0.23$) and IQ ($\beta = 0.20, t = 1.72, p = 0.09$, Figure 4a) could not. The linear model was significant ($F[3, 71] = 4.17, p = 0.009$) with a partial r^2 of 15.0%. Multiple linear regression was not significant for predicting WM capacity of IBM (Figures 3b and 4b), though ($F[3, 71] = 2.04, p = 0.12$). The specific effect of

the severity of autistic symptom on social WM was further supported by a significant partial correlation between SRS and WM capacity (BM: $r[71] = -0.33, p = 0.004$; IBM: $r[71] = -0.10, p = 0.41$) using IQ and age as covariates. The nonsignificant effect of age was also corroborated by a nonsignificant partial correlation between age and WM capacity of IBM, after controlling the effect of IQ and SRS (BM: $r[71] = 0.01, p = 0.40$; IBM: $r[71] = 0.21, p = 0.065$).

In sum, age had a significant effect on WM capacity for TD children but not for children with ASD, consistent with the ANOVA results above. IQ could predict TD children's WM capacity for both social and nonsocial WM, but not for children with ASD; the severity of core autistic symptoms can predict the performance of children with ASD only and specifically for their social but not for their nonsocial WM capacity.

DISCUSSION

Using BM stimuli, our study examined the WM capacity for social and nonsocial stimuli among children with ASD across an age span that potentially contains a critical development period. We found that our TD children showed rapid development of WM capacity for both BM

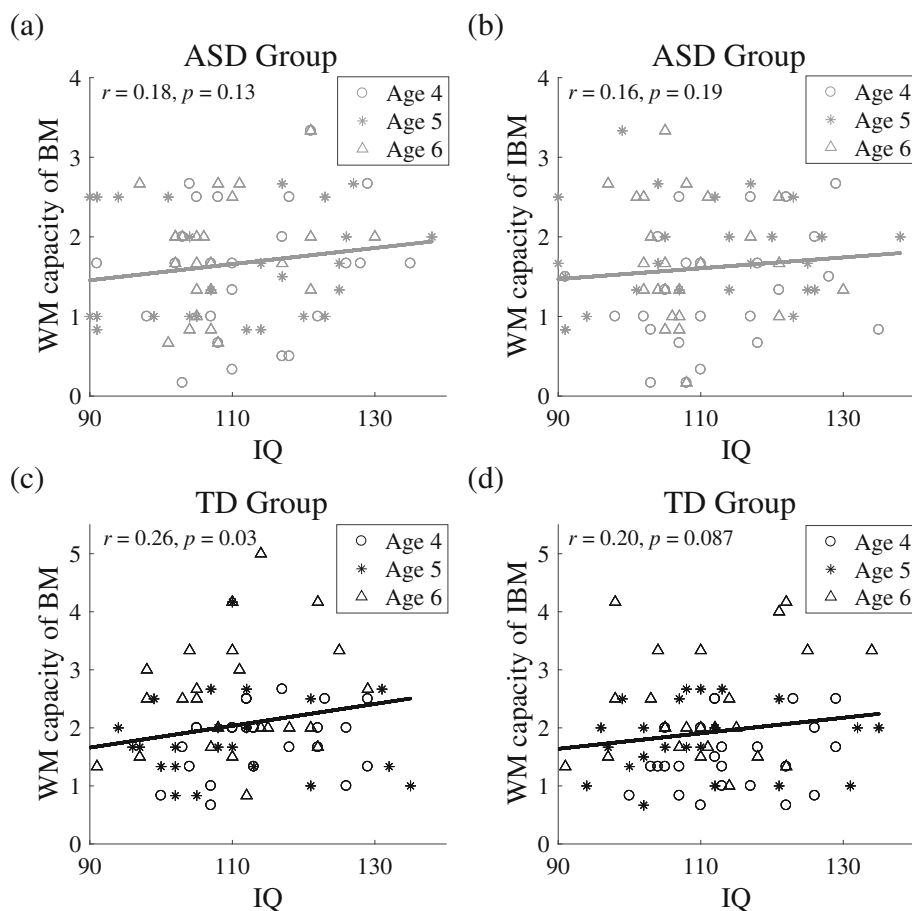


FIGURE 4 WM capacity as a function of IQ. Data are shown in separate plots based on group and stimulus types, including (a) BM for ASD group, (b) IBM for ASD group, (c) BM for TD group, and (d) IBM for TD group. Each data point is from a participant; different symbols denote different age groups. The partial correlation coefficients and the p -values from multiple linear regression analyses are shown for each plot

and IBM stimuli from age 5 to 6. In contrast, the fast development was absent among children with ASD. Regression analyses revealed that IQ could predict the WM capacity among TD children. In contrast, the severity of autistic symptoms could predict the WM capacity among children with ASD, but this association was specific for social WM and not for nonsocial WM. Furthermore, IQ could not predict the WM capacity among children with ASD. Our findings thus indicate that children with ASD missed the rapid WM development for social information around age 6, and their impairment is related to the severity of autistic symptoms.

Previous ASD studies have examined the WM performance of canonical stimuli such as numbers or spatial locations (for reviews, Habib et al., 2019; Wang et al., 2017). Our study, instead, examined social WM and its early developmental trajectory and compared it to nonsocial WM with physically comparable stimuli. The finding of low WM capacity only at age 6 differs from the developmental pattern of other types of WM in ASD. For spatial WM (e.g., change detection in complex scenes) and object WM, researchers typically found a truncated developmental trajectory with improvement arrested in adolescence, followed by a substantial group difference in adulthood (Luna et al., 2007; Lynn

et al., 2022; O'Hearn et al., 2008; O'Hearn et al., 2010; O'Hearn et al., 2014). The emergence of social WM deficits thus appears early, possibly associated with developmental deviance in social cognition among children with ASD. Consistent with this possible association, we also observed a positive correlation between SRS and WM capacity after controlling the confounds of IQ. Relatedly, our recent study on TD children revealed that WM of BM correlates with social cognition development (He et al., 2019).

Interestingly, we also found that nonsocial WM capacity, assessed by inverted BM stimuli, showed similar developmental deviance as social WM capacity. This result contradicts the intact or even better WM capacity reported by the three recent studies that rigorously measured WM capacity with continuous cued-recall or change detection, the same paradigm employed here (Bodner et al., 2019; Lynn et al., 2022; Stevenson et al., 2021). We postulate that differences in visual features of memory items, the age of participants, or both, might lead to this discrepancy. The three studies presented simple visual items simultaneously at different locations and varied their visual features, i.e., colors, colored shapes, and visual patterns. Pertinent to our study, Lynn et al. (2022) found that children (mean age 11, range 7 ~ 12 years), adolescents, and adults all showed

comparable WM capacity when tested with different visual features, including object color, shape, and pattern. Similarly, (Bodner et al., 2019) reported intact WM capacity for colored objects among adolescents and adults with ASD. Instead of using simple geometric shapes, our study used BM stimulus, which is arguably a more complex and dynamic visual object whose encoding emphasizes holistic processing (Blake & Shiffrar, 2007). Given their insensitivity to global features (Happé & Frith, 2006), individuals with ASD might have difficulty encoding BM items, evidenced by extensively reported impaired BM perception (Blake et al., 2003; Koldewyn et al., 2011). Interestingly, the importance of feature encoding for the WM test is indicated by a rare finding of *superior* WM capacity among adults with ASD: when adults with ASD showed perceptual advantage (hue discrimination) over the TD participants for the remembered color stimuli, they also performed better in the recall of these stimuli in the WM test (Stevenson et al., 2021). The other factor that might contribute to the discrepant findings is age difference since the lowest age span reported previously was 7 to 12 years (Lynn et al., 2022), and we examined between ages 4 and 6. It is possible that the developmental trajectory of WM is different for different memory items; future studies are warranted to comprehensively examine the joint effect of visual features and age on WM capacity.

We found that the severity of autistic symptoms, as indexed by SRS, was significantly correlated with WM capacity among children with ASD, only for BM but not for IBM stimuli. This selective correlation is consistent with our working hypothesis that BM can be viewed as social stimulus while IBM can be viewed as nonsocial stimulus (Gu et al., 2019; He et al., 2019; Shi et al., 2018). Indeed, those who scored lower in social cognition, social communication, social motivation, and autistic mannerisms exhibited worse social WM capacity. Among neurotypicals, social WM has been found to be closely tied to social-cognitive abilities, including theory of mind, perspective taking, and empathy (He et al., 2019; Meyer et al., 2012; Meyer et al., 2015). Social information processing is central for autistic deficits, including face recognition (Davis et al., 2017; Weigelt et al., 2012; Wolf et al., 2008), facial emotion identification (Humphreys et al., 2007; Trevisan & Birmingham, 2016), and atypical eye contact with others (Delbruck et al., 2019; Toth et al., 2006), and the ability to infer others' intention, emotion, and mental states (Jones et al., 2018; Joseph & Tager-Flusberg, 2004). We show that the development of short-term storage of social information, indexed by WM capacity, was delayed; how this contributes to or interacts with various social tasks would be of great theoretical and practical importance for autism research.

IQ can predict TD children's WM capacity for both BM and IBM stimuli, but not for children with ASD. The results of TD children are not surprising:

neurotypical adults exhibited a positive correlation between intelligence and WM capacity of canonical stimuli (Fukuda et al., 2010; Hicks et al., 2015; Kane et al., 2005; Unsworth et al., 2014) as well as of BM (Ye et al., 2019); our findings extended this correlation to preschool children. The lack of correlation in children with ASD is not surprising: first, the impaired BM perception was not correlated with intelligence among individuals with ASD (Todorova et al., 2019). Second, previous studies also failed to find a consistent relationship between intelligence and WM of canonical stimuli in ASD (for reviews, Habib et al., 2019; Wang et al., 2017). As our social WM task combined WM and BM processing, the lack of correlation with intelligence is thus expected. The group difference in the dependence of WM capacity on IQ suggests that the deficits in WM capacity are related to autism *per se*.

We did not detect any difference in WM capacity between the two types of BM stimuli, seemingly contradictory to our previous report that neurotypical children aged between 4 and 5 had a larger capacity for BM than for IBM stimuli (He et al., 2019). The advantage of BM also manifests itself among adults in a perceptual detection task (Pavlova & Sokolov, 2000; Troje, 2003) and has been attributed to a relatively low cognitive demand for sensory integration of moving dots (Pavlova, 2012; Pavlova & Sokolov, 2000). We postulate that the lack of difference observed here might be related to the relatively low developmental stage of our TD children compared with the previous sample. Our study recruited children from a normal kindergarten, while the previous study recruited children from one of the elite kindergartens in Hangzhou city, one of the largest cities in China. We could not back up this conjecture by comparing their IQ since the previous study did not measure IQ. However, the WM capacity we obtained supports this explanation: the *average* *K*-max of BM was 1.54, 1.97, and 2.85 for 3, 4, and 5-year-olds in the previous study, while it was 1.80, 1.77, and 2.59 for 4, 5, and 6-year-olds in the current study. A similar trend was seen in the WM capacity of IBM: the *K*-max was 1.20, 2.00, and 2.29 in 3, 4, and 5-year-olds in the previous study, while it was 1.51, 1.80, and 2.49 for 4, 5, and 6-year-olds in the current study. Thus, it appears that the social WM capacity in our sample lagged behind the previous sample by at least a year. Nevertheless, the lack of the difference between BM and IBM stimuli among TD children does not affect our main conclusions about abnormal development in social WM among children with ASD. If the environment did have some influence on the WM capacity, it could be the case that children with ASD improve their WM capacity by enriching academic environment. Furthermore, the performance of TD children hints that the advantage of BM over IBM might depend on the development stage (Kaiser & Shiffrar, 2009).

The delayed development in social WM can be further investigated by using brain imaging. For instance,

the abnormal face processing in ASD is accompanied by gradual deviance from the normal maturation of related neural circuits (Mamashli et al., 2018): the local and long-range functional connectivity involving fusiform face areas were intact among pre-adolescent children (ages 7 to 13) but significantly reduced among adolescents and young adults with ASD (ages 14–21). We expect that the abnormal development of social WM is accompanied by functional connectivity abnormalities involving the neural circuits associated with BM (Grezes et al., 2001; Grossman & Blake, 2002; Krakowski et al., 2011; Peuskens et al., 2005).

We observed that TD children had a rapid developmental improvement of social WM, which implies a window of plasticity and an opportunity for intervention for children with ASD. Comparing WM capacity between the present sample of TD children and our previous sample (He et al., 2019), it appears that the environmental factors could accelerate WM development of social and nonsocial stimuli. Thus, children with ASD might also benefit from enriching the academic environment, an important consideration for intervention of ASD targeting at improving WM capacity. Previous studies found that executive function interventions had great potential for improving social function and developmental outcomes in ASD (Vogan et al., 2018). Particularly, WM training for individuals with ASD not only improves the memory capacity itself (for a review, Morrison & Chein, 2011) but also benefits social interaction and social awareness (Weckstein et al., 2017). Moreover, the efficacy of WM intervention among ASD has been found to have a substantial effect as early as 5-year-olds (Traverso et al., 2015). Given our finding that children with ASD miss the rapid developmental gain in social WM at 5- to 6-year-olds, interventions combining social information processing and WM training during the pre-school period might be a potentially effective revenue.

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DATA AVAILABILITY STATEMENT

Research data are not shared.

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