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Developmental changes of visuospatial working memory in autistic children and adolescents

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Abstract

Background. Limited longitudinal research examining developmental changes in visuospatial working memory (WM) among children and adolescents with autism spectrum disorder (ASD) has prompted our investigation.

Methods. We assessed 123 autistic children and adolescents and 145 typically developing controls (TDC) using the Cambridge Neuropsychological Test Automated Battery at baseline (Time 1 [mean age ± SD]: ASD: 13.04 ± 2.86; TDC: 11.53 ± 2.81) and 2–9 years later (Time 2: ASD: 18.08 ± 3.17; TDC: 16.41 ± 3.09) to measure changes of visuospatial (working) memory over time. The linear mixed model was used to compare the differences between ASD and TDC and estimate the effect of changes over time, age, ASD diagnosis, and interactions of Time×Age×ASD. The overall Age×ASD effect was calculated in the spline regression.

Results. Autistic children and adolescents exhibited significantly poorer performance on all spatial tasks and some visual tasks than their TDC counterparts at Time 1 and Time 2, after adjusting for sex, age, attention deficit/hyperactivity disorder (ADHD), and full-scale intelligence quotient. There was an overall improvement from Time 1 to Time 2 across all tasks with significant Age×Time interactions. Significant Age×ASD interactions were observed in the delayed matching to sample, pattern recognition memory (PRM), spatial span (SSP), and spatial working memory (SWM) tasks with no significant Time×ASD interactions. In the quadratic nonlinear model, Age×ASD interactions were significant in PRM and SSP.

Conclusion. Despite significant improvements during the follow-up period, autistic children and adolescents continue to experience persistent deficits in SWM, with a weaker age-related improvement in visuospatial WM than TDC.

Introduction

Autism spectrum disorder (ASD) is characterized by social communication problems and restricted, repetitive behavior, interest, and activities in multiple contexts (American Psychiatric Association, 2013). Individuals with ASD consistently demonstrate impairments in visuospatial working memory (WM), particularly in tasks that require high executive or memory load (Barendse et al., 2013; Habib, Harris, Pollick, & Melville, 2019; Kaufmann et al., 2013; Seng et al., 2020; Steele, Minshew, Luna, & Sweeney, 2007; Van Eylen et al., 2015; Wang et al., 2017). Conflicting data existed concerning visuospatial memory in individuals with ASD: some reported impaired (Chien et al., 2015; Minshew & Goldstein, 2001; Salmanian, Tehrani-Doost, Ghanbari-Motlagh, & Shahrivar, 2012), some reported not impaired (Lynn, Luna, & O'Hearn, 2022; Williams, Goldstein, Carpenter, & Minshew, 2005), and some reported even superiority (O'Riordan, 2004; Ropar & Mitchell, 2001), compared to typically developing controls (TDC). The types and complexity of stimuli and the executive load (Barendse et al., 2013) used in different studies might contribute to different results.

According to Baddeley's model, visuospatial WM consists of a central executive, an episodic buffer for integrating multidimensional information, and a visuospatial sketchpad for short-term visuospatial data storage for encoding, storing, and recalling information (Baddeley, 2012). Different brain pathways are involved in processing visuospatial information such as object recognition (color and shape) and perception of location and action (Zachariou, Klatzky, & Behrmann, 2014). Therefore, visual (object recognition) and spatial (location) tasks should be addressed distinctly, with further differentiation within spatial memory between simultaneous, and sequential types (Mammarella, Pazzaglia, & Cornoldi, 2008). Research by Lynn, Luna, and O'Hearn (2022) suggests that visual WM is comparable between ASD and TDC across various ages, suggesting that deficits in ASD may primarily affect spatial WM. Additionally, Stevenson et al. (2021) found that while autistic children and adolescents replicated colors well, they

struggled with color-location binding memory, highlighting the need to explore further which aspects of visual memory (object recognition) or spatial information processing, and specifically which components of spatial working memory (SWM), are affected in ASD.

Previous cross-sectional, age-stratified studies have demonstrated that SWM improves from childhood through adolescence to adulthood in both autistic and non-autistic individuals (Demetriou et al., 2018; Habib, Harris, Pollick, & Melville, 2019; Wang et al., 2017). However, despite age-related improvements, ASD individuals consistently showed impairments in SWM at all ages, suggesting a delayed developmental trajectory into adulthood (Luna et al., 2007). Notably, few longitudinal follow-up studies have explored the developmental changes in visuospatial WM and visual memory (Vogan, Morgan, Smith, & Taylor, 2019).

Current study

The use of abstract shapes and patterns in visuospatial tasks, such as computerized neuropsychological tasks of the Cambridge Neuropsychological Test Automated Battery (CANTAB), helps minimize confounding effects from other memory types, semantic information, and language abilities, which are often impaired in ASD (Salmanian, Tehrani-Doost, Ghanbari-Motlagh, & Shahrivar, 2012). CANTAB is a well-established tool for assessing constructs such as general learning, memory, and response speed (Robbins et al., 1994). It incorporates visual search tasks to control for general attentional and visual perceptual functions and has been widely used in variable populations, with neural correlates of task performance extensively investigated (Owen et al., 1990). Our study aimed to explore the longitudinal changes in visuospatial memory and WM in autistic children and adolescents. Additionally, we aimed to determine whether visual/spatial WM impairments might be associated with deficits in the visuospatial sketchpad and executive control, considering the co-occurrence of ADHD and the influence of executive load.

Methods

Participants and procedures

The Time 1 and Time 2 evaluations of this longitudinal study were approved by the Research Ethics Committee of National Taiwan University Hospital (NTUH), respectively (Approval nos. 200903062R [Time 1] and 201403109RINC [Time 2]; ClinicalTrials.gov: NCT00916851 [Time 1] and NCT02233348 [Time 2]). After participants and their parents provided the written informed consent (child's assent for participants, if appropriate), we conducted diagnostic interviews, clinical evaluations, and neuropsychological tests using the CANTAB.

At Time 1, autistic children and adolescents were referred by senior child psychiatrists from the Children's Mental Health Center of NTUH and other hospitals or schools in Taiwan. The clinical diagnosis was initially based on criteria of the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DSM-IV) (American Psychiatric Association, 1994), including autistic disorder and Asperger's disorder at Time 1, and further confirmed by the corresponding author to meet DSM-5 ASD criteria (American Psychiatric Association, 2013). The TDC participants, recruited at schools in the same districts as the ASD group, and their siblings underwent thorough evaluations to ensure that they did not exhibit ASD traits. Parents of autistic children and adolescents received interviews with the Chinese version of the Autism Diagnostic Interview-Revised (ADI-R) (Gau et al., 2011). Also, they reported autistic symptoms of children on the Social Responsiveness Scale (SRS) and ADHD/oppositional defiant disorder (ODD) symptoms on the Swanson, Nolan, and Pelham Rating Scale (*SNAP-IV*) at Time 1.

At Time 2, children and adolescents who completed the assessments at Time 1 were invited to the follow-up assessments. After providing assent and written informed consent, all the participants and their parents were reassessed with the Kiddie Schedule for Affective Disorder and Schizophrenia–Epidemiological Version (K-SADS-E) interviews for DSM-5 (Chen, Shen, & Gau, 2017) for any lifetime and current psychiatric diagnosis. Participants with ASD also received an Autism Diagnostic Observation Schedule (ADOS) (Chang et al., 2023), and their parents reported on the ADI-R interview again. The corresponding author meticulously reviewed all the clinical records and ADI-R assessments to ensure that every participant with ASD met the DSM-5 diagnostic criteria for ASD (ADOS, ADI-R, and SNAP-IV descriptions are provided in the Supplementary Material).

All participants received the intelligence test by the *Wechsler Intelligence Scale for Children, 4th Edition* if they were younger than 17 years and the *Wechsler Adult Intelligence Scale, 4th Edition* if they were 17 years or older at Time 1, and visuospatial tests of CANTAB at both Time 1 and Time 2. Participants who used medication were asked to hold medication for at least 24 h. The participants were excluded from the study if their full-scale intelligence quotient (FIQ) was lower than 60 at Time 1 or if they did not complete the CANTAB tests either at Time 1 or Time 2.

One hundred twenty-five autistic children and adolescents and 160 TDC participants completed measurements at two time points. However, 2 autistic children and 15 TDC participants were excluded from the main analysis because the interval between their measurements was less than 2 years.

Measures

Social Responsiveness Scale

The SRS is a quantitative measure of autistic symptoms, including 65 items. It is designed to evaluate children and adolescents aged 4–18 in their natural social settings over the previous 6 months. The scale utilizes a 4-point Likert scale, ranging from '0' (not true) to '3' (almost always true). The higher scores indicate more autistic symptoms. The internal consistency of our study sample was very high (Cronbach's alpha: 0.84–0.95). The psychometrics of the Chinese version of SRS (Gau et al., 2013) are provided in the Supplementary Material.

The Chinese version of the Kiddie Schedule for Affective Disorder and Schizophrenia–Epidemiological Version

The K-SADS-E is a semistructured clinical interview used to diagnose psychiatric disorders for both lifetime and current episodes (Ambrosini, 2000). The Chinese version of *K-SADS-E* for DSM-IV and DSM-5 have demonstrated reliability and validity in assessing psychiatric disorders (Chen, Shen, & Gau, 2017; Gau, Chong, Chen, & Cheng, 2005; Gau & Soong, 1999). The details of the interviewer training are described in the Supplementary Material and elsewhere (Gau et al., 2010; Lin, Yang, & Gau, 2016).

Cambridge Neuropsychological Testing Automated Battery

The CANTAB (www.cambridgecognition.com) is a computerized testing system developed for assessing neuropsychological functions in clinical and research settings (Sahakian & Owen, 1992). Seven non-verbal visuospatial tasks were selected in this study.

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1. Matching to sample visual searching

This 7-minute task requires the participants to recognize an abstract pattern with a brief delay. The sample pattern is first shown in the middle of the screen; later, a varying number (1, 2, 4, or 8) of similar patterns (with one identical to the sample pattern) are shown around the edge of the screen. The abstract pattern is composed of four elements with four different colors. There are 12 trials at each level of difficulty, presented at random. Percent correct is used here as the outcome variable.

2. Delayed matching to sample

This 7-minute task evaluates the simultaneous and delayed recognition of abstract patterns consistent with four elements with four colors. A sample pattern shows on the upper half of the screen, and then four choice patterns show on the lower half of the screen simultaneously or with a delay (0, 4, or 12 s) after the sample pattern disappears. The participants were asked to touch the sample pattern that was identical to their choice pattern. This task consists of 20 counterbalanced trials, including 5 simultaneous trials and 5 trials for each of the 3 delay intervals. If the first choice is wrong, the participant must make the next one until the right one is chosen. The total correct of all delays is used here as the outcome variable.

3. Pattern recognition memory

This 4-minute task is designed to evaluate recognition memory of meaningless visual shapes. A series of 12 sample patterns are exhibited on the screen in the display stage; then, the participants are instructed to choose the pattern ever shown in the display stage in a series of 12 pairs of patterns (one sample pattern and one novel pattern). The test patterns are presented in reverse order to the order in the display stage. The result of the recognition portion is presented here.

4. Paired associates learning

This 8-minute task evaluates the recognition memory of patterns and locations. There are eight stages in this test. Six to eight boxes are exhibited on the screen and randomly opened in each stage. A particular pattern is present in one or more of the boxes. Next, the patterns are presented in the middle of the screen, one at a time, and the participants have to identify the box in which the pattern initially appeared. In case of an incorrect selection, the boxes are opened sequentially to help the participants recall the pattern's location. There are up to 10 trials at each stage. The participants will proceed to the next stage when they get all the correct locations. The test is terminated when the participant fails to pass the stage. The index of total adjusted errors (adjusting stages completed) is presented here.

5. Spatial Recognition Memory

This task assesses recognition memory of spatial locations using a two-choice forced discrimination paradigm. During the presentation phase, empty boxes appear in various screen locations, which participants must memorize. Afterward, five pairs of stimuli are shown sequentially in different locations, and participants must select the box from the presentation phase, in reverse order. This subtest is repeated four times, with five new locations each time, and the percentage of correct responses is presented here.

6. Spatial span

This is a 5-minute task similar to the Corsi blocks task, requiring the ability to remember the order of visual stimuli presented. The task begins with a level of two boxes and then gradually up to nine boxes. The color of the boxes is changed one by one in a predetermined sequence at the sample stage. A sound signals the end of the sequence. The participants must touch the boxes on the screen in the same order as the sequence at the sample stage. The test terminates when the participant fails in all three sequences at a particular level. Spatial span (SSP) (spatial span, span length [SSPsL]) and total usage errors (the number of times the box not in the sequence of locations pre-selected is selected [SSPtuE]) are presented.

7. Spatial working memory

The task lasts 4 min and involves uncovering hidden tokens within boxes. It includes three difficulty levels (four-box, six-box, and eight-box), each comprising four trials. In each trial, one token is hidden in a box, which is not reused in subsequent trials of the same difficulty level. An error is recorded if the same box is opened again within a single trial (known as within error) or if a box previously discovered to have a token inside is opened again in subsequent trials of the same difficulty level (known as between errors). Strategy utilization (SWMS) and between errors (SWMbE) across all three levels are presented.

Statistical analysis

We used SAS 9.4 (SAS Institute Inc., Cary, NC, USA) for statistical analysis. We compared continuous and categorical variables between ASD and TDC using the general linear model and chi-square test (or Fisher's exact test if needed), respectively. A large standard deviation was noted in the PAL; therefore, 11 outliers, which meant total adjusted error > 45 (four standard deviations of TDC away from ASD group mean), at Time 1 or Time 2 in the ASD group, were excluded from all analyses of PAL. The ASD and TDC group comparisons of CANTAB tasks were calculated by univariable and multivariable analyses adjusting for age, sex, lifetime ADHD, and/or FIQ at Time 1, as well as follow-up interval at Time 2. Interactions of the ASD×Difficulty level in SWM (4-, 6-, and 8-box) and delayed matching to sample (DMS) (0-, 4-, and 12-s delay) were analyzed. Cohen's d was calculated for CANTAB performance between ASD and TDC at Time 1 and Time 2.

The mixed model was used to compare age and time effects between groups, controlling for within-person correlations in repeated measures. We analyzed interactions including Time×ASD, Time×Age, Age×ASD, and Time×Age×ASD after adjusting for sex, lifetime ADHD, FIQ, and follow-up interval. Subsequently, spline regression was utilized to assess the fit of visuospatial (working) memory trajectory by age. Our data fitted the quadratic nonlinear curve better than the linear model. The overall Age×ASD effect was calculated in the spline regression (de Boor, 1978) without considering the measurement time. Due to multiple comparisons of multiple CANTAB metrics, the false discovery rate (FDR; Benjamini–Hochberg method)-adjusted p-values were applied, with an FDR threshold set at 0.05.

Results

Demographic data

Table 1 describes the demographic data of our study sample – significantly more boys with ASD than TDC. Autistic participants were significantly older than TDC. Therefore, we adjusted for age and sex in all analyses. The FIQ, VIQ, and PIQ profiles were lower

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Table 1. Demographic and clinical data of ASD and TDC

N (%) or mean (SD)	ASD (*n* = 123)	TDC (*n* = 145)	Statistics χ^2 or *F* (*p-value*)
Males, N(%)	114 (92.68)	101 (69.66)	22.24 (<0.01)
Age			
Time 1	13.04 (2.86)	11.53 (2.81)	18.91 (<0.001)
(age range)	(8–18)	(8–18)	
Time 2	18.08 (3.17)	16.41 (3.09)	18.93 (<0.001)
(age range)	(10–25)	(12–26)	
Follow-up interval	5.04 (1.81)	4.88 (1.36)	0.66 (0.11)
Intelligence at Time 1			
FIQ	101.07 (18.54)	110.4 (11.27)	25.56 (<0.001)
VIQ	102.34 (18.27)	110.93 (11.22)	21.82 (<0.001)
PIQ	100.94 (18.6)	108.31 (13.14)	14.02 (<0.001)
SRS			
Social communication	37.06 (14.76)	8.32 (7.32)	377.38 (<0.001)
Stereotyped behavior	19.17 (8.67)	3.76 (4.05)	321.92 (<0.001)
Social awareness	20.28 (4.7)	12.09 (5.62)	152.07 (<0.001)
Social emotion	11.57 (4.66)	3.34 (3.01)	271.31 (<0.001)
SNAP-IV			
Inattention	15.15 (6.74)	5.37 (5.04)	165.43 (<0.001)
Hyperactivity/impulsivity	10.5 (6.33)	2.79 (3.39)	140.77 (<0.001)
Oppositional	9.45 (6.11)	3.46 (3.97)	82.48 (<0.001)
Any psychiatric disorder	94 (76.42)	35 (24.14)	72.87 (<0.001)
ADHD, lifetime	65 (52.85)	16 (11.03)	55.17 (<0.001)
Medication use			
Previous	51 (41.46)	3 (1.88)	Fisher exact test $p < 0.001$
Current	32 (38.89)	2 (1.38)	Fisher exact test $p < 0.001$
Father's education	(*N* = 108)	(*N* = 137)	
Senior high school and below	35 (32.41)	42 (30.66)	0.09 (0.77)
College and above	73 (67.59)	95 (69.34)	
Mother's education	(*N* = 113)	(*N* = 138)	
Senior high school and below	38 (33.63)	51 (36.96)	0.30 (0.58)
College and above	75 (66.37)	87 (63.04)	

Note: ASD, autism spectrum disorder; FIQ, full-scale intelligence quotient; PIQ, performance intelligence quotient; SD, standard deviation; SNAP, Swanson, Nolan, and Pelham Rating Scale; SRS, Social Responsiveness Scale; TDC, typically developing control; VIQ, verbal intelligence quotient.

in ASD than TDC. No difference in parental education was noted between ASD and TDC. Autistic children and adolescents were more likely to have co-occurring ADHD and more likely to use psychotropic medications than TDC. Children and adolescents with ASD scored higher in all SRS subscales and had more ADHD and ODD symptoms than TDC.

Visuospatial (working) memory performance: ASD vs. TDC

Table 2 shows the comparisons of visuospatial tasks between ASD and TDC at Time 1 and Time 2. Autistic children and adolescents performed significantly worse than their TDC counterparts in spatial tasks (SRM, SSP, and SWM) at both Time 1 and Time 2, after adjusting for sex, age, lifetime ADHD, and FIQ. The visual tasks with significant ASD–TDC difference were DMS at both Time 1 and Time 2, PAL at Time 1, and pattern recognition memory (PRM) at Time 2 after adjusting for sex, age, and lifetime ADHD, and the significance disappeared after adjusting for FIQ.

The influence of executive load

Figure 1 shows the interaction of ASD and different SWM and DMS executive load levels. There was a significant ASD×difficulty interaction in SWM at both Time 1 and Time 2 and in DMS at Time 1. Autistic children and adolescents exhibited significantly worse performance in SWM across three difficulty levels at Time 1 and Time 2 after adjusting for age, sex, lifetime ADHD, and FIQ (Supplementary Table S1; all $p < 0.01$). Significantly worse

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Table 2. Comparisons of visuospatial (working) memory of ASD and TDC stratified by measurement time

	ASD	TDC	Cohen's *d*	Group comparisons[a]	Group comparisons[b]	Group comparisons[c]
Mean (SD)	(*N* = 123)	(*N* = 145)			*F* (*p-value*[d])	
Time 1						
MTS, Percent correct	93.85 (5.04)	95.18 (4.33)	−0.28	6.03 (0.02)	0.77 (0.38)	0.16 (0.78)
DMS, Total correct (all delays)	22.84 (4.35)	24.11 (4.12)	−0.30	15.66 (<0.001)	7.86 (0.01)	2.65 (0.16)
PRM, Percent correct	89.3 (12.32)	91.67 (8.11)	−0.23	2.28 (0.13)	3.29 (0.09)	0.11 (0.74)
PAL, Total errors (adjusted)	7.66 (7.40)	4.94 (6.19)	0.40	9.62 (0.01)	7.85 (0.02)	3.35 (0.28)
SRM, Percent correct	78.21 (12.13)	84.45 (9.41)	−0.58	30.11 (<0.001)	16.99 (<0.0001)	8.63 (0.01)
SSP, Span length	6.38 (1.74)	7.08 (1.56)	−0.43	29.36 (<0.001)	17.42 (<0.0001)	8.65 (0.01)
SSP, Total usage errors	1.88 (1.78)	1.72 (1.57)	0.10	5.22 (0.03)	2.69 (0.12)	0.46 (0.64)
SWM, Strategy utilization	34.86 (5.77)	32.25 (5.07)	0.48	23.04 (<0.001)	16.29 (<0.0001)	10.21 (0.007)
SWM, Between errors (total)	36.11 (20.34)	22.51 (18.24)	0.70	49.74 (<0.001)	34.14 (<0.0001)	21.36 (<0.0001)
Time 2[e]						
MTS, Percent correct	97.82 (23.09)	96.56 (3.54)	0.08	0.26 (0.61)	0.00 (0.95)	0.13 (0.72)
DMS, Total correct (all delays)	24.66 (3.94)	26.78 (2.49)	−0.66	21.34 (<0.001)	9.45 (0.004)	2.72 (0.18)
PRM, Percent correct	88.66 (12.30)	93.16 (7.18)	−0.46	8.71 (0.01)	6.47 (0.01)	1.15 (0.36)
PAL, Total errors (adjusted)	6.93 (8.01)	3.88 (5.26)	0.46	5.1 (0.03)	2.62 (0.12)	0.18 (0.75)
SRM, Percent correct	81.91 (13.34)	88.24 (8.89)	−0.57	17.84 (<0.001)	13.21 (<0.001)	5.6 (0.06)
SSP, Span length	6.76 (1.78)	7.55 (1.53)	−0.48	15.25 (<0.001)	9.37 (0.004)	2.37 (0.19)
SSP, Total usage errors	1.58 (1.62)	0.93 (1.15)	0.47	17.18 (<0.001)	14.86 (<0.001)	9.37 (0.01)
SWM, Strategy utilization	32.62 (5.76)	29.76 (5.42)	0.51	19.77 (<0.001)	10.86 (0.002)	4.49 (0.08)
SWM, Between errors (total)	26.94 (22.2)	13.07 (13.56)	0.77	30.83 (<0.001)	21.68 (<0.001)	10.41 (0.01)

Note: CANTAB, Cambridge Neuropsychological Test Automated Battery; DMS, delayed matching to sample; FDR, false discovery rate; FIQ, full-scale intelligence quotient; MTS, matching to sample visual searching; PAL, paired associates learning; RPM, pattern recognition memory; SRM, spatial recognition memory; SSP, spatial span; SWM, spatial working memory. Due to multiple comparisons stemming from multiple CANTAB metrics, the FDR (Benjamini–Hochberg method)-adjusted *p*-values were used. The allowed FDR is 0.05.
[a]Adjusting for sex and age.
[b]Adjusting for sex, age, and lifetime ADHD diagnosis.
[c]Adjusting for sex, age, FIQ, and lifetime ADHD diagnosis.
[d]False discovery rate-adjusted *p*-value (Benjamini–Hochberg method).
[e]We added the follow-up interval into the analysis in addition to covariates at Time 1.

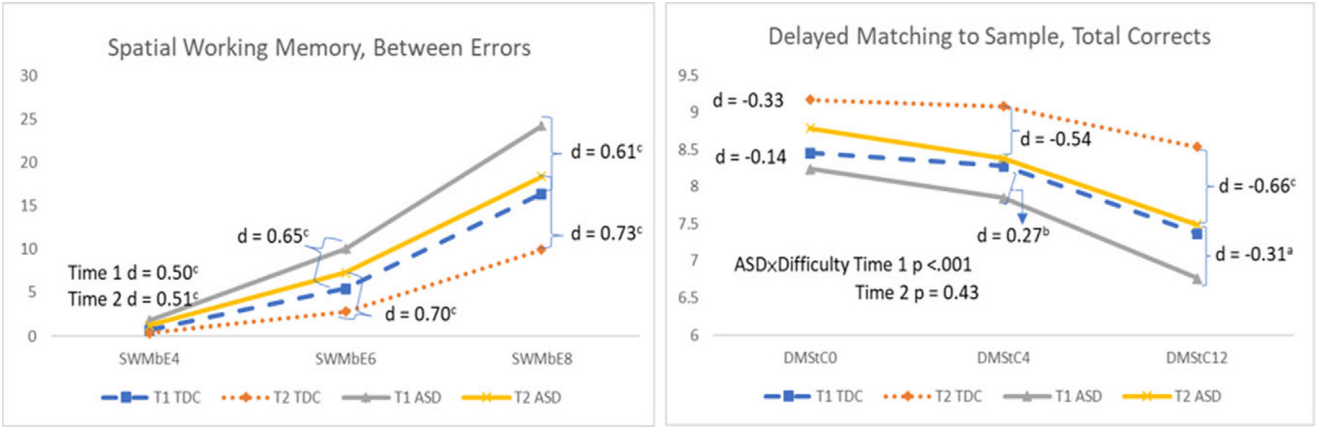

Figure 1. ASD×Difficulty interactions. [a]$p < 0.05$; [b]$p < 0.01$; [c]$p < 0.001$. *d* = Cohen's *d*.

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performance of ASD than TDC was noted in DMS at the 4-s ($F = 9.11$, $p = 0.003$) and 12-s ($F = 4.84$, $p = 0.03$) delays at Time 1 and at the 12-s delay ($F = 14.82$, $p < 0.001$) at Time 2 after adjusting for age, sex, and lifetime ADHD. After adjusting for FIQ, a significant ASD–TDC difference in DMS remained in a 4-s delay ($F = 4.55$, p = 0.03) at Time 1 and a 12-s delay ($F = 6.78$, $p = 0.01$) at Time 2.

To further explore the role of executive load in SWM, we also controlled for SSPsL and SRM in addition to age, sex, lifetime ADHD, and FIQ in the group comparison of SWMbE. ASD still had significant impairments in SWM at Time 1 ($F = 13.17$, $p < 0.001$) and Time 2 ($F = 4.15$, $p = 0.04$).

Using a mixed model, we identified a significant ASD×SWMS interaction ($F = 5.81$, $p = 0.02$) in predicting SWMbE after controlling for age, sex, measurement time, and lifetime ADHD, with the higher correlation between SWMS and SWMbE in ASD than TDC. However, this interaction was no longer significant after adjusting for FIQ ($F = 2.77$, $p = 0.10$).

The correlations of binding memory (PAL) and visual/spatial memory (PRM/SRM)

Significant correlations were observed between PAL and both PRM ($r = -0.51$ in ASD, -0.27 in TDC, both $p < 0.001$) and SRM ($r = -0.44$ in ASD, -0.22 in TDC, both $p < 0.001$). In the mixed model, after adjusting for age, sex, measurement time, and lifetime ADHD, there was a significant ASD×PRM interaction ($F = 12.47$, $p < 0.001$) but a non-significant ASD×SRM interaction ($F = 1.64$, $p = 0.20$) in their correlation with PAL. The significance of these interactions remained unchanged after controlling for FIQ. These findings suggest that autistic children and adolescents rely more on visual memory to complete the paired learning task than TDC.

Developmental changes of visuospatial memory and moderating effect of time and ASD diagnosis (mixed linear regression)

Children and adolescents, both with and without ASD, significantly improved (time effect) in almost all CANTAB visuospatial tasks from Time 1 to Time 2, except for MTS and PRM (Table 3). A significant age effect was observed across all spatial tasks and DMS, with better performance associated with increasing age (Table 3). Additionally, a significant Age×Time interaction was found in all visuospatial tasks except MTS and PRM, suggesting a weaker positive association between age and performance at Time 2 compared with Time 1. Significant Age×ASD interactions were observed in most spatial tasks (except SRM) and some visual tasks (DMS and PRM), indicating that the positive association between performance and age was less pronounced in autistic children and adolescents compared with TDC. No significant Time×ASD or Time×Age×ASD interactions were observed.

To further explore the Age×Time and Age×ASD effects, we separately analyzed the age effect and Age×ASD interactions at Time 1 and Time 2 (Supplementary Table S3). At Time 1, significant Age×ASD interactions were observed in PRM ($F = 8.06$, $p = 0.01$), SSPsL ($F = 6.92$, $p = 0.02$), SSPtuE ($F = 9.11$, $p = 0.01$), SWMS ($F = 11.06$, $p = 0.01$), and SWMbE ($F = 8.11$, $p = 0.01$). Among these tasks, TDC exhibited a stronger association between age and visuospatial functions (except PRM). SSPsL performance was positively correlated with age in both ASD ($t = 4.41$, $p < 0.001$) and TDC ($t = 8.56$, $p < 0.001$), whereas positive associations with age were found in PRM ($t = 2.71$, $p = 0.007$), SRM ($t = 4.61$, $p < 0.001$), and SWMbE ($t = -7.35$, $p < 0.001$) exclusively in TDC. At Time 2, PAL performance ($t = 3.26$, $p = 0.001$) showed a significant inverse correlation with age in ASD.

Trajectory of visuospatial memory as a function of age (quadratic nonlinear model)

Figure 2 illustrates the spline regression fit across all tasks, improving model fitting from linear to quadratic models. In the quadratic nonlinear model, the age effect was significant in all tasks (all $p < 0.05$), and the Age×ASD interactions were significant in PRM ($p = 0.03$) and SSPtuE ($p = 0.02$).

Discussion

This study's longitudinal design enabled us to identify distinct developmental differences between ASD and TDC across adolescence and young adulthood. Autistic children and adolescents consistently performed worse on spatial (working) memory tasks than TDC at both Time 1 and Time 2, regardless of sex, age, and lifetime ADHD. Visual memory impairment in ASD was not as significant as spatial memory deficits, particularly after adjusting for FIQ. Overall, both groups showed significant improvement in visual and spatial WM over the follow-up period, with a stronger positive correlation between age and visuospatial WM at younger ages (Time 1) than at elder ages (Time 2).

Partially consistent with Lynn, Luna, and O'Hearn (2022), who reported that autistic individuals might have impaired spatial (working) memory while maintaining intact visual (working) memory, we also found greater ASD–TDC differences in tasks involving memory and retrieval of locations (SRM, SSP, and SWM) than those involving memory of color and pattern (MTS, PRM, and PAL), even with relatively high memory loads (e.g. 12 patterns in PRM) and with longer temporal delays (e.g.12 s in DMS). Differences between ASD–TDC visual (working) memory were largely explained by general intelligence. The CANTAB visual memory tasks used in our study primarily relied on local processing, which was relatively preserved in ASD, as opposed to deficits involving global processing (Mottron, Belleville, & Menard, 1999; Stevenson et al., 2021).

Children and adolescents with both ASD and ADHD exhibited greater impairments in WM, planning, organizing, inhibition, and attention than those with ASD alone (Benallie et al., 2021), raising the question of whether these deficits are primarily due to co-occurring ADHD or are intrinsic to ASD. Controlling for co-occurring ADHD significantly reduced the magnitude of ASD–TDC differences in visuospatial WM, but impairments in ASD remained. These findings, aligned with a meta-analysis (Lai et al., 2017), suggest visuospatial WM deficits are intrinsic to ASD.

Numerous studies have consistently reported impairments in visuospatial WM among individuals with ASD (Funabiki & Shiwa, 2018; Jiang, Capistrano, & Palm, 2014; Steele, Minshew, Luna, & Sweeney, 2007; Wang et al., 2017), although the specific components underlying these deficits remain elusive (Jiang, Capistrano, & Palm, 2014). Our findings revealed significant ASD×Difficulty interactions in DMS, highlighting the critical role of memory load in visual memory deficits in ASD (Barendse et al., 2013; Steele, Minshew, Luna, & Sweeney, 2007). Alongside decreased spatial memory (SRM and SSP), reduced central executive function independently contributed to the SWM deficits observed in ASD. The executive dysfunction was further supported by impaired SWM

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Table 3. Effects and interactions of measurement time, age, and ASD diagnosis on visuospatial functions (mixed linear regression)

	Estimate		Fixed effects	
	Estimate	95% CI	*F*-value	*p*-value[a]
MTS, Percent correct				
Time *(Time 2 vs. Time 1)*	2.24	(−10.64, 15.11)	0.91	0.38
Age	0.28	(−0.40, 0.95)	1.18	0.36
Age×Time	−0.14	(−1.04, 0.77)	0.4	0.53
ASD *(vs. TDC)*	−1.61	(−14.00, 10.78)	0.06	0.81
ASD×Time	5.64	(−14.54, 25.82)	0.3	0.75
Age×ASD	0.04	(−0.93, 1.02)	0.01	0.91
Age×ASD×Time	−0.17	(−1.50, 1.17)	0.06	1.00
DMS, Total correct (all delays)				
Time *(Time 2 vs. Time 1)*	9.53	(5.83, 13.23)	54.52	<0.001
Age	0.76	(0.57, 0.95)	45.74	<0.001
Age×Time	−0.64	(−0.90, −0.39)	51.34	<0.001
ASD *(vs. TDC)*	0.86	(−2.67, 4.40)	2.71	0.23
ASD×Time	3.02	(−2.65, 8.69)	1.1	0.67
Age×ASD	−0.15	(−0.42, 0.13)	4.69	0.046
Age×ASD×Time	−0.12	(−0.50, 0.25)	0.4	0.79
PRM, Percent correct				
Time *(Time 2 vs. Time 1)*	5.32	(−4.87, 15.50)	0.34	0.56
Age	0.75	(0.22, 1.27)	0.52	0.47
Age×Time	−0.46	(−1.17, 0.26)	0.47	0.55
ASD *(vs. TDC)*	13.60	(3.90, 23.31)	7.05	0.04
ASD×Time	−5.78	(−21.66, 10.11)	0.51	0.71
Age×ASD	−1.11	(−1.87, −0.34)	9.98	0.005
Age×ASD×Time	0.53	(−0.51, 1.58)	1.01	0.71
PAL, Total errors (adjusted)				
Time *(Time 2 vs. Time 1)*	−9.41	(−0.96, −0.24)	19.79	<0.001
Age	−0.60	(0.20, 1.17)	0.77	0.43
Age×Time	0.69	(−7.79, 5.83)	19.17	<0.001
ASD *(vs. TDC)*	−0.98	(−17.63, 4.16)	2.47	0.21
ASD×Time	−6.74	(−0.34, 0.74)	1.48	0.67
Age×ASD	0.20	(−0.44, 1.01)	3.55	0.08
Age×ASD×Time	0.29	(−0.96, −0.24)	0.61	0.78
SRM, Percent correct				
Time *(Time 2 vs. Time 1)*	18.91	(7.69, 30.13)	20.59	<0.001
Age	1.43	(0.85, 2.01)	11.23	<0.001
Age×Time	−1.35	(−2.13, −0.56)	19.63	<0.001
ASD *(vs. TDC)*	1.32	(−9.37, 12.01)	0.55	0.52
ASD×Time	3.96	(−13.54, 21.46)	0.2	0.74
Age×ASD	−0.51	(−1.35, 0.33)	2.93	0.10
Age×ASD×Time	0.02	(−1.14, 1.17)	0.00	1.00
SSP, Span length				
Time *(Time 2 vs. Time 1)*	2.31	(0.73, 3.90)	19.1	<0.001

(*Continued*)

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Table 3. (*Continued*)

	Estimate		Fixed effects	
	Estimate	95% CI	*F*-value	*p*-value[a]
Age	0.36	(0.27, 0.44)	63.19	<0.001
Age×Time	−0.22	(−0.33, −0.11)	26.35	<0.001
ASD *(vs. TDC)*	1.33	(−0.20, 2.85)	8.69	0.004
ASD×Time	0.99	(−1.45, 3.43)	0.64	0.76
Age×ASD	−0.16	(−0.28, −0.04)	14.53	0.002
Age×ASD×Time	0.001	(−0.16, 0.16)	0	0.99
SSP, Total usage errors				
Time *(Time 2 vs. Time 1)*	−2.20	(−3.83, −0.57)	4.33	0.049
Age	−0.29	(−0.38, −0.21)	39.44	<0.001
Age×Time	0.17	(0.06, 0.29)	5.93	0.02
ASD *(vs. TDC)*	−2.29	(−3.85, −0.74)	5.35	0.06
ASD×Time	1.65	(−0.86, 4.15)	1.68	0.88
Age×ASD	0.21	(0.08, 0.33)	10.72	0.005
Age×ASD×Time	−0.13	(−0.30, 0.03)	2.57	0.50
SWM, Strategy utilization				
Time *(Time 2 vs. Time 1)*	−8.83	(−14.71, −2.65)	4.57	0.05
Age	−0.90	(−1.20, −0.59)	22.29	<0.001
Age×Time	0.65	(0.24, 1.07)	4.36	0.049
ASD *(vs. TDC)*	−6.63	(−12.24, −1.02)	1.58	0.27
ASD×Time	7.51	(−1.50, 16.51)	2.69	0.92
Age×ASD	0.72	(0.28, 1.17)	6.77	0.02
Age×ASD×Time	−0.66	(−1.25, −0.06)	4.69	0.28
SWM, Between errors				
Time *(Time 2 vs. Time 1)*	−39.00	(−57.31, −20.69)	24.34	<0.001
Age	−3.71	(−4.67, −2.75)	38.4	<0.001
Age×Time	2.91	(1.62, 4.20)	23.89	<0.001
ASD *(vs. TDC)*	−13.02	(−30.50, 4.47)	2.17	0.21
ASD×Time	5.08	(−22.96, 33.12)	0.13	0.72
Age×ASD	2.00	(0.62, 3.38)	9.69	0.005
Age×ASD×Time	−1.06	(−2.92, 0.80)	1.27	0.78

Note: ASD, autism spectrum disorder; CANTAB, Cambridge Neuropsychological Test Automated Battery; CI, confidence interval; DMS, delayed matching to sample; FIQ, full-scale intelligence quotient; MTS, matching to sample visual searching; PAL, paired associates learning; PRM, pattern recognition memory; SRM, spatial recognition memory; SSP, spatial span; SWM, spatial working memory; TDC, typically developing control. The statistics were adjusted for sex and lifetime ADHD and the reference group for Time was Time 1 and for ASD was TDC. The formula of analysis: CANTAB task = intercept + β_1 * Time + β_2 * Age + β_3 * Age×Time + β_4 * ASD + β_5 * ASD×Time + β_6 * Age×ASD + β_7 * Age×Time×ASD + β_8 * Sex + β_9 * ADHD + β_{10} * Follow-up interval + β_{11} Time 1 FIQ + Error.
[a]False discovery rate-adjusted *p*-value (Benjamini–Hochberg method).

strategy utilization in ASD and the stronger correlation between SWM between error and strategy utilization in ASD than TDC. Strategy utilization in the SWM task refers to the extent to which predetermined repetitive search sequences are used to tackle more difficult task problems. Employing an organized search strategy reduces the cognitive load and enhances SWM task accuracy (Owen et al., 1990). The poor strategy use exhibited by autistic children and adolescents resembles patterns observed in individuals with frontal lobe lesions but not those with medial lobe lesions (Owen et al., 1995) and is correlated with SWM deficits, implying a frontal lobe dysfunction in ASD (Hawco et al., 2020).

Autistic children and adolescents showed significantly worse visuospatial binding memory (PAL). Moreover, PAL performance was reversely correlated to age at Time 2 in ASD but not TDC, implying a possible developmental arrest in this ability. PAL is the task requiring one to simultaneously manage visual (patterns) and spatial (location) information (Owen et al., 1995) and might involve the episodic buffer of the visuospatial subsystem in WM (Baddeley, Allen, & Hitch, 2011). This interface processes a different set of coding systems and was considered to have a limited capacity and might require executive effort to work (Baddeley, 2012). Earlier studies have shown that autistic adults encounter challenges in

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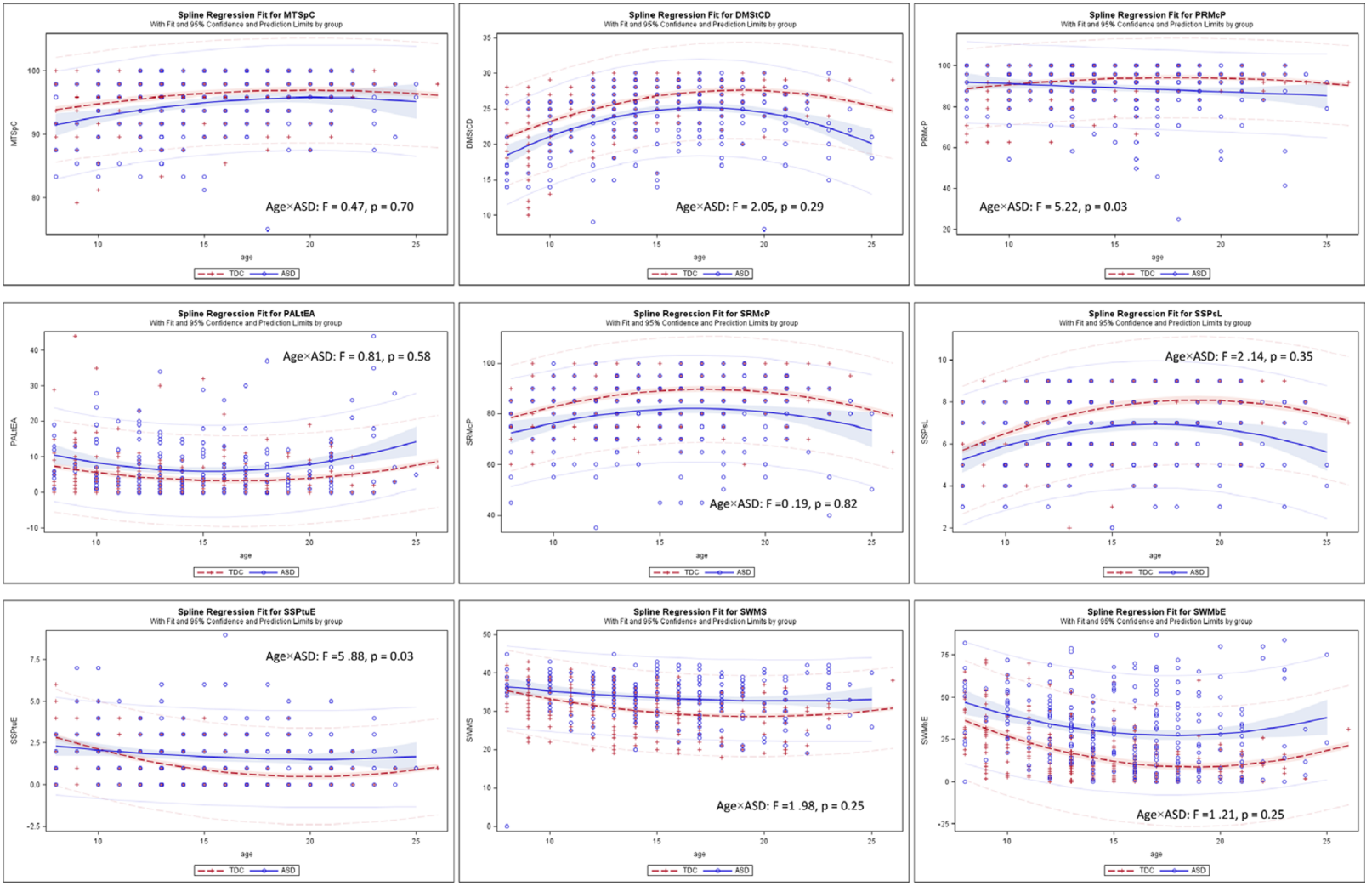

Figure 2. Spline regression of visuospatial (working) memory by age in ASD and TDC. *Note:* The colored region along the regression line is the 95% confidence limit, and the semitransparent line outside is the prediction limit. MTSpC, matching to sample visual searching, percent correct; DMStCD, delayed matching to sample, total correct (all delay); RPMcP, pattern recognition memory, percent correct; PALtEA, paired associates learning, total error (adjusted); SRMcP, spatial recognition memory, percent correct; SSPsL, spatial span, span length; SSPtuE, spatial span: total usage error; SWMS, spatial working memory, strategy utilization; SWMbE, spatial working memory between errors; ASD: autism spectrum disorder; TDC: typically developing control.

relational memory, particularly regarding the spatial relationship between objects, instead of the object memory per se (Maister, Simons, & Plaisted-Grant, 2013; Ring, Gaigg, & Bowler, 2015), and have inferior binding memory of color and location than non-autistic ones (Stevenson et al., 2021). Autistic individuals often struggle to integrate various information into a Gestalt perception, affecting their ability to interpret social cues across modalities like facial expressions, voices, and communication contents (Stevenson et al., 2021). Our study found significant correlations between PAL and both visual and spatial memory in autistic children and adolescents, mirroring patterns seen in individuals with frontal lobe dysfunctions (Owen et al., 1995). Additionally, autistic children and adolescents relied more on visual memory to complete the PAL task than TDC, who likely benefit from a more effective episodic buffer for efficient performance. At Time 2, PAL deficits in ASD were no longer significant after controlling for ADHD, suggesting that the executive deficit required to buffer locations and patterns in this CANTAB task (Maister, Simons, & Plaisted-Grant, 2013) may be partially mediated by co-occurring ADHD. Further studies using tasks that integrate different sensory modalities with varying executive loads across age groups will help elucidate the development and nature of episodic buffer deficits in ASD.

The was no significant developmental change from Time 1 to Time 2 in complex pattern recognition with brief delays (MTS) in either ASD or TDC, suggesting that visual recognition memory with brief interruptions may mature by late childhood and is not significantly impaired in ASD (Lynn, Luna, & O'Hearn, 2022). In contrast, the spatial (working) performance significantly improved with age and between Time 1 and Time 2 in both groups. However, the positive association between age and spatial (working) memory at Time 2 was not as striking as that at Time 1, potentially reflecting a performance plateau in CANTAB visuospatial tasks during mid to late adolescence (De Luca et al., 2003). Although earlier studies reported developmental arrest in spatial WM among individuals with ASD (Luna et al., 2007), our stratified analyses also revealed significantly weaker associations between age and both PRM and spatial (working) memory in ASD compared to TDC (significant Age×ASD interactions). However, the absence of a significant ASD×Time interaction in the mixed model precludes drawing definitive conclusions.

Due to the varying trends in age-performance associations observed at Time 1 and Time 2 for certain tasks, a linear model did not adequately capture the data across a wide age range. Instead, a quadratic polynomial nonlinear model provided a better fit for the developmental curve. The absence of an Age×ASD interaction in the overall quadratic model suggests that the developmental trajectories from ages 8 to 26 were similar in shape for both individuals with ASD and TDC. Visualizations revealed a U-shaped developmental trajectory for most of CANTAB visuospatial WM tasks, with performance plateauing by early adulthood. However, linear analyses showed significant Age×ASD interactions, indicating variations in developmental rates between ASD and TDC at various developmental stages. Our study demonstrated that, on average, individuals with ASD had persistent impairment in spatial (working) memory, visual memory storage and retrieval, and visuospatial binding memory from mid-childhood to early adulthood. However, heterogeneity exists, with only a subset of autistic individuals exhibiting a deviant cognitive profile (Torenvliet et al., 2023). Further longitudinal studies with larger sample sizes and repeated measurements are required to investigate the heterogeneous developmental trajectories in ASD.

Limitations

Our results should be interpreted considering the following limitations. First, our data predominantly included males, limiting the representation of the female population. Second, the wide age range and follow-up intervals posed challenges. Although age was controlled for in all analyses, age effects cannot be precisely linked to specific developmental periods. The limited sample size prevented further division into narrower age groups or matching follow-up intervals, though the follow-up interval distribution was relatively comparable between ASD and TDC groups (see Supplementary Figure for the boxplot of the two groups). Furthermore, no significant interaction was found between Time 1 CANTAB performance and the Time 1–Time 2 interval in predicting Time 2 performance across all CANTAB tasks (Supplementary Table S5). Third, participants were clinical cases from metropolitan regions, excluding those who could not complete CANTAB (mainly because of low IQ). Thus, the findings cannot be generalized to rural populations or individuals with low IQ. Fourth, autistic symptoms were assessed using subjective questionnaires of SRS rather than objective measures like ADOS, introducing potential reporting bias due to expectations and social norms. Fifth, some participants were taking ADHD medication during recruitment and follow-up, which could influence CANTAB performance. Medication use was associated with higher levels of inattentive symptoms in adolescents with ASD and poorer CANTAB performance in children, suggesting self-selection bias. To mitigate this issue, participants were asked to discontinue medication 24 h before evaluation. Finally, detailed information on participants' non-pharmacological interventions or service uses was not collected, preventing analysis of the impact on visuospatial performance changes.

Conclusion

Despite significant improvements during the follow-up period, autistic children and adolescents still had persistent spatial WM impairments, with a weaker age-related performance improvement in spatial WM and visual memory with a higher memory load than TDC. These deficits did not extend to simple visual memory tasks. Autistic children and adolescents also exhibited binding memory deficits, relying more on visual memory for paired tasks than TDC. Notably, these spatial WM deficits were not explained by co-occurring ADHD but were likely attributed to deficits in visuospatial sketchpad and central executive control.

Supplementary material. The supplementary material for this article can be found at http://doi.org/10.1017/S0033291725000133.

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