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Atypical Working Memory Decline Across the Adult Lifespan in Autism Spectrum Disorder?

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Whereas working memory (WM) performance in typical development increases across childhood and adolescence, and decreases during adulthood, WM development seems to be delayed in young individuals with autism spectrum disorder (ASD). How WM changes when individuals with ASD grow old is largely unknown. We bridge this gap with a cross-sectional study comparing age-related patterns in WM performance (*n*-back task: 3 load levels) among a large sample of individuals with and without ASD ($N = 275$) over the entire adult life span (19–79 years) as well as interindividual differences therein. Results demonstrated that, despite longer RTs, adults with ASD showed similar WM performance to adults without ASD. Age-related differences appeared to be different among adults with and without ASD as adults without ASD showed an age-related decline in WM performance, which was not so evident in adults with ASD. Moreover, only IQ scores reliably dissociated interindividual differences in age-gradients, but no evidence was found for a role of basic demographics, comorbidities, and executive functions. These findings provide initial insights into how ASD modulates cognitive aging, but also underline the need for further WM research into late adulthood in ASD and for analyzing individual change trajectories in longitudinal studies.

General Scientific Summary

It is largely unknown what happens to cognition when individuals with autism spectrum (ASD) grow old. This study focusses on working memory and suggests that age-related differences in working memory performance might be different among adults with and without ASD.

Keywords: autism spectrum disorder (ASD), working memory, aging, regression trees, executive functions

Supplemental materials: <http://dx.doi.org/10.1037/abn0000108.supp>

Autism spectrum disorder (ASD) is a heterogeneous neurodevelopmental disorder characterized by qualitative impairments in social interaction and communication, and restricted, repetitive behavior (American Psychiatric Association [APA], 2013), and is associated with impairments in executive functions (EF; Hill, 2004; Pennington & Ozonoff, 1996). EF is an umbrella term

referring to various cognitive functions involved in control and coordination that are necessary for complex, goal-directed behavior. At the same time, EF deficits are observed during typical aging (e.g., Friedman, Nessler, Cycowicz, & Horton, 2009; Verhaeghen & Cerella, 2002). Although ASD is a lifelong condition, surprisingly little is known about alterations in cognitive functioning in

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individuals with ASD when they grow old. Hence, the current study addresses the question whether cross-sectional age-gradients in a core EF function, namely working memory (WM), deviate in ASD clients in comparison to a typically developing control sample.

WM is the ability to maintain and manipulate information online in the absence of actual sensory information in order to guide goal-directed behavior (e.g., Baddeley, 2003; Cowan, 2014). As such, it is important for daily life functioning. In typical development, WM performance increases throughout childhood into adolescence (Conklin, Luciana, Hooper, & Yarger, 2007; Gathercole, Pickering, Ambridge, & Wearing, 2004; Tamnes et al., 2013) and decreases during adulthood (Borella, Carretti, & De Beni, 2008; Hasher & Zacks, 1988; Park et al., 2002; see Sander, Lindenberger, & Werkle-Bergner, 2012 for an overview). Although these observations derive mainly from cross-sectional studies, longitudinal evidence suggests nonlinear change-patterns with accelerated decline in older adulthood (Nyberg, Lövdén, Riklund, Lindenberger, & Bäckman, 2012; for further elaborations, see Lindenberger, von Oertzen, Ghisletta, & Hertzog, 2011; Raz & Lindenberger, 2011).

Although the developmental trajectory of WM in ASD is not well charted, there is preliminary evidence for it being deviant from typical development (see O'Hearn, Asato, Ordaz, & Luna, 2008). Cross-sectional studies demonstrated that WM improved from childhood to adolescence in both ASD and typically developing individuals (Happé, Booth, Charlton, & Hughes, 2006; Luna, Doll, Hegedus, Minshew, & Sweeney, 2007; but see Rosenthal et al., 2013), but that WM development from adolescence to young adulthood was delayed in ASD (i.e., maturity was reached at a later age; Luna et al., 2007). A recent longitudinal study over a 2-year period pointed out that WM development among children and adolescents might be arrested (Andersen et al., 2015). These findings suggest a delayed development of WM in individuals with ASD that protracts into young adulthood (O'Hearn et al., 2008). So far, the trajectory of WM development in middle adulthood is unknown. In late adulthood, an initial small cross-sectional study suggests comparable age-related decline in older individuals with ASD compared with typically developing elderly, but WM abilities in those with ASD still seem to be reduced in old age (Geurts & Vissers, 2012).

Whether WM is indeed impaired in individuals with ASD is, however, still a topic of debate: Studies comparing individuals with and without ASD of the same age on a group level show inconsistent results (e.g., Koshino et al., 2008; Ozonoff & Strayer, 2001; Williams, Goldstein, Carpenter, & Minshew, 2005; see Barendse et al., 2013 for a review). WM impairments are mainly found when individuals with ASD are compared with typically developing individuals rather than to other pathological groups (Russo et al., 2007); when spatial WM rather than verbal WM is examined (Steele, Minshew, Luna, & Sweeney, 2007; Williams et al., 2005; but see Ozonoff & Strayer, 2001); and when there are increased demands on WM, for example when the complexity of the task is high or when item manipulation is required instead of maintenance only (Koshino et al., 2008; Steele et al., 2007; Williams et al., 2005).

Whereas WM is sensitive to age-related decline, considerable interindividual differences exist between individuals of the same age (Eenshuistra, Ridderinkhof, & van der Molen, 2004; Vogel &

Awh, 2008) that tend to increase with advancing adulthood (e.g., Nagel et al., 2008; Werkle-Bergner, Freunberger, Sander, Lindenberger, & Klimesch, 2012). Similarly, among individuals with ASD, individual differences may partially explain the inconsistent WM findings. For example, de Vries and Geurts (2014) found that a relatively small subgroup of children with ASD that demonstrated WM deficits accounted for the WM impairment found on a group level when comparing children with and without ASD. These findings underscore that both ASD and aging are characterized by broad heterogeneity.

Several factors have been proposed to drive age-related cognitive decline and WM performance, such as slowing speed of processing (Salthouse, 1996), worsening suppression of irrelevant information (i.e., interference control; Hasher & Zacks, 1988) degrading sensory functioning (Baltes & Lindenberger, 1997), changes in global intelligence (Hockey & Geffen, 2004), social participation status (Lövdén, Ghisletta, & Lindenberger, 2005), depressive symptoms (Paterniti, Verdier-Taillefer, Dufouil, & Alperovitch, 2002), and attention-deficit/hyperactivity disorder (ADHD; Engelhardt, Nigg, Carr, & Ferreira, 2008). Some of these factors are also known to be critical in ASD. For example, comorbid conditions are common in ASD (Hofvander et al., 2009), individuals with ASD show interference control difficulties (Geurts, van den Bergh, & Ruzzano, 2014) and response slowing (Travers et al., 2014), and societal participation, such as having a job and being satisfied with received environmental support, is generally low (Howlin, Moss, Savage, & Rutter, 2013; Magiati, Tay, & Howlin, 2014; van Heijst & Geurts, 2015). Given the substantial interindividual differences in typical aging as well as in ASD, and the overlap in factors contributing to both conditions, the present study addresses the additional question whether differential age-related patterns in WM performance could be observed in specific subgroups among adults with and without ASD.

In summary, the current cross-sectional study investigates WM in ASD over the entire adult life span (i.e., including middle and late adulthood) by means of an *n*-back task. In an *n*-back task, a continuous stream of stimuli is presented and the objective is to indicate whether the current stimulus matches a stimulus shown *n* trials previously. Stimuli used in the current study consisted of simple pictures (Severens, Van Lommel, Ratinckx, & Hartsuiker, 2005). An *n*-back task taps into core WM-processes such as maintenance of items in memory, updating of task relevant information, binding of items into a serial order, and resolution of proactive interference (Chatham et al., 2011). Hence, it is often used in cognitive neuroscience research to investigate WM (Jarrald & Towse, 2006; Smith & Jonides, 1997) by experimentally manipulating load parametrically (Jaeggi, Buschkuhl, Perrig, & Meier, 2010). The aims of the current study are threefold. First, we investigate WM performance across different load levels comparing adults with and without ASD. We hypothesize that, if there is WM impairment in ASD, this should become apparent in the cognitively more demanding condition (i.e., 2-back condition). Second, we study the effect of age on WM performance over the adult life span in ASD and non-ASD to examine developmental patterns. In typical development, age-related changes in WM performance are independent of modality (verbal or visuospatial) or span/nonspan (Conklin et al., 2007; Park et al., 2002). Therefore, given that age-related differences of spatial WM span were found to be similar among older adults with and without ASD (Geurts &

Visser, 2012) before, we hypothesize similar age-related differences in WM performance across groups in our study as well (that is, a parallel pattern of age-gradients across groups). Third, we explore whether we can find predictors of interindividual differences in age-related patterns of WM performance using regression trees.

Method

Participants

ASD group. Our sample consisted of 168 individuals with an ASD who were recruited through different mental health institutions across the Netherlands, and by means of advertisement on client organization websites. They were screened, based on self-reported information, for the following exclusion criteria: (a) no clinical ASD diagnosis according to *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; APA, 2000) criteria; (b) history of neurological disorders (e.g., epilepsy, stroke, cerebral contusion); and (c) diagnosed with schizophrenia, or having experienced more than one psychosis. Based on these criteria, 26 individuals were excluded, and the ASD diagnoses of the remaining 142 participants were verified by administering the Autism Diagnostic Observation Schedule Module 4 (ADOS; Lord et al., 2000) and the Autism-Spectrum Quotient (AQ; Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001). If participants did not score above the cutoff of 7 on the ADOS, a score above the AQ cutoff of 26 was required (Woodbury-Smith, Robinson, Wheelwright, & Baron-Cohen, 2005). Of the 39 participants who did not meet the ADOS criterion, only five did also not meet the AQ criterion and were excluded from further analysis. Of the remaining 138 participants, two were excluded as their IQ, estimated with two subtests of the Wechsler Adult Intelligence Scale (3rd ed.; WAIS-III; Wechsler, 1997) was below 80; none of the participants was excluded based on a Mini-Mental State Exam (MMSE; Folstein, Folstein, & McHugh, 1975) score below 26. Moreover, we excluded two participants due to a current alcohol- or drugs dependency and 14 participants due to having experienced more than one psychosis or not remembering how many psychoses were experienced during lifetime, revealed by administration of the Mini International Neuropsychiatric Interview (MINI; Sheehan et al., 1998), which were previously not indicated by self-report. Finally, we excluded one individual who could not be evaluated for screening due to noncompliance to answering MINI questions. The eligible ASD group consisted of 118 participants.

Comparison group. The comparison group (COM) consisted of 193 individuals without ASD who were recruited by means of advertisements on the university website and on social media, and within the social environment of the researchers. They were screened, based on self-reported information, for the following exclusion criteria: (a) clinical diagnosis of ASD or ADHD; (b) a history of neurological disorders; (c) diagnosed with schizophrenia, or having ever experienced a psychotic episode; and (d) ASD or schizophrenia in close family members (i.e., parents, children, brothers and sisters). Fourteen individuals were excluded and the remaining 179 participants filled out the AQ. If participants scored above the suggested AQ cutoff for the general population of 32 or higher (Baron-Cohen et al., 2001) they were excluded. One par-

ticipant did exceed the AQ cutoff and one participant had too many missing AQ responses (10.0%). Of the remaining 177 participants, two were excluded as their estimated IQ was below 80; none of the participants was excluded based on a MMSE score below 26. Finally, after administering the MINI, we excluded: (a) six participants due to a current alcohol- or drugs dependency and (b) two participants who could not be evaluated for screening due to noncompliance to answering questions. The eligible COM group consisted of 167 participants.

N-back data of six ASD participants were lost due to technical problems, two COM participants withdrew after the first session, and two participants (one ASD, one COM) did not complete the *n*-back task. Hence, 111 participants with ASD and 164 participants without ASD were included (see Figure 1 for an illustration of the inclusion process). The groups were matched on age and estimated IQ. However, the proportion of females was larger in the COM group than in the ASD group. As expected, the ASD group demonstrated higher levels of autism traits than the COM group (see Table 1).

Materials

Instruments used for ASD assessment and screening are reported in the online supplemental material eAppendix 1.

***N*-back.** *N*-back stimuli were black and white drawings of simple objects (Severens et al., 2005). These stimuli were chosen to be comparable with a previous study of our research group among children with ASD (de Vries & Geurts 2014). We employed an adapted version of their task. The task consisted of three different load levels representing increasing demand for WM: 0-back, 1-back, and 2-back. In the 0-back condition, serving as a baseline, participants had to respond “yes” when a car was depicted and “no” for every other image. In the 1-back condition, participants had to respond “yes” when the picture shown was identical to the previous picture and “no” when it was not. In the 2-back condition, participants had to respond “yes” when the picture shown matched the picture two trials before and “no” when it did not match.

Stimuli were presented on a computer screen each for 1,000 ms and were afterward replaced by a black mask for 750 ms or until response was given. During this time window, participants were instructed to respond by giving either a “yes” or a “no” response by pressing the corresponding button. The next stimulus was presented after a fixed 250-ms intertrial interval. To ensure the task was properly understood, we gave extensive task instructions for each load level. First, the task was orally explained and instructions were displayed on screen. Second, a paper-version practice block (15 trials) was administered in order to give participants time to familiarize themselves with the task and allow the experimenter to give additional instructions as needed. Third, participants performed a computerized practice block (24 trials). Moreover, task instructions were repeated before each experimental block. The task consisted of four experimental blocks per load level (24 trials each). Blocks consistently switched between load levels, that is, 0-back was followed by 1-back, which was followed by 2-back, which was followed by 0-back, etcetera. Stimuli were presented in a pseudorandomized order. To rule out the effect of interfering response mapping memory processes, two cues were provided: a “yes” card was presented in accordance of the associated “yes”

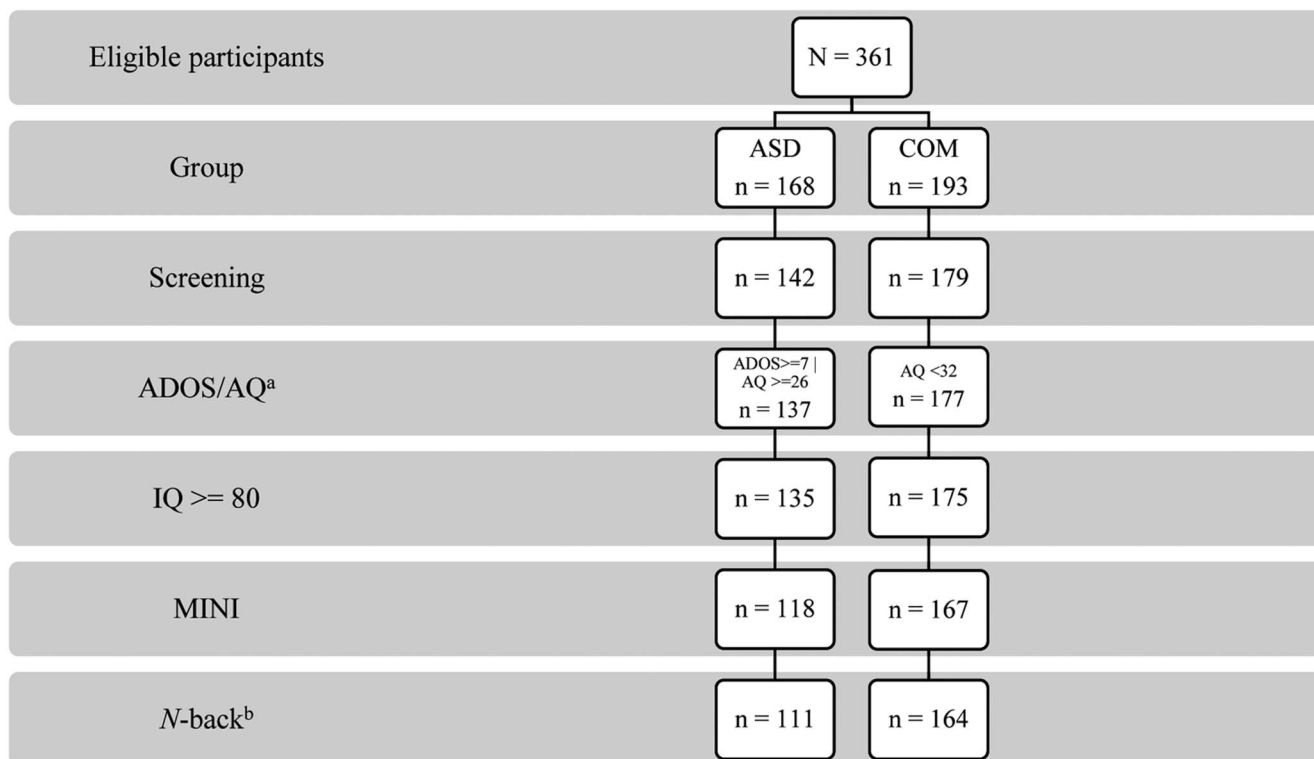


Figure 1. Diagram of the inclusion process. ASD = autism spectrum disorder; COM = comparison; ADOS = Autism Diagnostic Observation Schedule; AQ = Autism-spectrum Quotient; IQ = estimated IQ. ^a Only five participants of those scoring below the ADOS cutoff (<7; n = 35) did also score below the AQ cutoff (<26). ^b N-back data of some participants could not be obtained. See Method section for details.

key, and a “no” card in accordance of the associated “no” key. Participants were instructed to respond as fast and as accurately as possible. The task yielded two dependent variables: accuracy (proportion of correct responses), and mean reaction time (RT) on correct responses.

Predictor variables. To explore whether we could predict age-related differences in WM performance, we selected a series of potential predictor variables based on (a) a known relationship with WM decline in typical aging and (b) being critical in individuals with ASD. Therefore, we included, in addition to demographic and clinical variables (estimated IQ, diagnosis [ASD, no ASD], gender, education, AQ traits) measures of (a) processing speed (measured as mean RT on correct trials during a choice response task (Donders, 1969); see Supplemental material eAppendix 2); (b) interference control (measured as mean RT difference between compatible and incompatible trials during a Simon task, i.e., Simon effect; Simon, 1969; see Supplemental material, eAppendix 2); (c) comorbidity, by choosing the three most common comorbid conditions in ASD (Hofvander et al., 2009), that is depression, anxiety (measured with depression and anxiety subscales of the Symptom Checklist-90; Arrindell & Ettema, 2005; Derogatis, 1977), and ADHD (using the attention and hyperactivity, and inattention subscales of the ADHD list; Kooij et al., 2004); and (d) participation status, operationalized as satisfaction with and need for environmental support and professional employment (measured

with the environmental subscale of the abbreviated World Health Organization Quality of Life questionnaire; The WHO-QOL Group, 1998; Trompenaars, Masthoff, Van Heck, Hodiament, & De Vries, 2005; professional employment was encoded according to the International Standard Classification of Occupations-08).

Procedure

Participants were informed about the study purposes and its procedure and written informed consent was obtained. Thereafter, participants filled out a series of questionnaires and were tested in two sessions. In the first session, the ADOS (only ASD group), two subtests of the WAIS, MMSE, and MINI were administered. In the second session, the n-back, choice response task, and Simon task, among seven other tasks, were administered in counterbalanced order. Not all administered questionnaires and tests are of relevance for the current study, so these are discussed elsewhere (e.g., Lever & Geurts, 2015). Participants received compensation for their travel expenses; most COM participants also received a small amount of additional compensation (max. €20). The study was approved by the ethical review board of the Department of Psychology at the University of Amsterdam (2011-PN-1952); all procedures complied to relevant laws and institutional guidelines.

Table 1
Demographic, and Clinical Scores of the ASD and COM Group

Description	Group		Statistics
	ASD (<i>n</i> = 111)	COM (<i>n</i> = 164)	
Gender	79 M/32 F	93 M/71 F	Fisher's test, $p = .016$; OR = 1.88
Education ^a	0/1/0/3/31/51/25	0/0/1/5/28/80/50	Fisher's test, $p = .144$
Diagnosis ^b	16/57/33/5	—	
Age	47.5 (15.0) range = 20–79	46.0 (16.5) range = 19–77	$F(1, 273) = .58, p = .448, \eta_p^2 = .00$
IQ	115.2 (16.9) range = 84–155	113.3 (16.7) range = 80–155	$F(1, 273) = .87, p = .352, \eta_p^2 = .00$
MMSE	29.1 (1.0) range = 26–30	29.1 (1.0) range = 26–30	$F(1, 273) = .16, p = .687, \eta_p^2 = .00$
AQ	33.4 (8.1) range = 8–49	12.2 (5.1) range = 2–26	$F(1, 272)^b = 703.61, p < .001, \eta_p^2 = .72$
ADOS	8.59 (3.11) range = 1–19	—	

Note. ASD = autism spectrum disorder; COM = comparison group; M = male; F = female; IQ = estimated intelligence quotient; MMSE = Mini-Mental State Examination; AQ = Autism-Spectrum Quotient; ADOS = Autism Diagnostic Observation Schedule; OR = odds ratio.

^a The numbers between slashes indicate the educational level based on the Verhage (1964) coding system, ranging from 1 (primary education not finished) to 7 (university degree). ^b The numbers between slashes indicate a diagnosis of autism/asperger syndrome/pervasive developmental disorder not otherwise specified/ASD. ^c One ASD participant did not complete the AQ (but met the ADOS criterion and, hence, was included). ^d Of the final sample, 27 participants scored below the ADOS cutoff (<7). Excluding these participants from the analyses did not alter the pattern of results.

Statistical Analyses

Prior to *n*-back analyses, we removed RT outliers. At an individual level, trials with RTs deviating more than 3 standard deviations from the mean and RTs faster than 100 milliseconds were removed. This procedure resulted in the exclusion of less than 3.1% of all trials in each group; that is, the maximum percentage of removed outliers was 3.1% for the ASD group ($M = 1.6\%$, $SD = 0.5\%$) and 3.1% for the COM group ($M = 1.6\%$, $SD = 0.5\%$) and did not differ between groups, $F(1, 273) = 0.52, p = .472$.

At a group level, mean RTs were calculated over the remaining responses on correct trials. RTs were normally distributed and, therefore, not transformed. Accuracy was calculated as the proportion of correct responses (correct number of trials per total number); Arcsine-square-root transformation was applied to increase normality, but, to ease interpretation, accuracy rates are reported in raw score units.

To test whether the groups differed in their WM performance across load levels, we performed two mixed-design analyses of variance (ANOVAs) with repeated measures of load (0-back, 1-back, 2-back) as within-subject factor and group (ASD, COM) as between-subjects factor. As the ASD and COM group differed in their male to female ratio ($p = .016$ by Fisher's exact test), and gender may influence WM performance in either ASD or aging (e.g., Lejbak, Crossley, & Vrbancic, 2011), gender (male, female) was added as a between-subjects factor in the overall group analyses. Accuracy and RTs on correct trials constituted the dependent variables.

To investigate whether age-related differences in WM performance varied across groups, we composed a difference score by subtracting untransformed accuracy on the 0-back condition from untransformed accuracy on the 2-back condition.¹ Arcsine-square-

root transformation was applied to the difference score to increase normality. The resulting transformed difference score constituted the dependent variable for our regression analysis with (centered) age, group, and Age \times Group interaction as predictors. As age-related WM decline might accelerate with increasing age, we explored whether there were differential effects of a quadratic component of age on WM in the ASD and COM group. To this end, we tested an additional model including a quadratic age term as main effect (age²) and its interaction with group (Age² \times Group).

All group-level analyses were run both with and without outlier correction (i.e., data points more than three times the interquartile range above or below the first quartile). We report results with outlier correction and state results without outlier correction only if the pattern of results changed. To reduce the probability of Type I errors, alpha level was set at .01 for the group comparisons and the age-related regression analyses. Whenever the assumption of sphericity was violated, we used the Greenhouse–Geisser correction (but we report uncorrected degrees of freedom).

With Bayesian statistics, we explored the robustness of the group comparisons and age-related differences. Bayesian hypothesis testing allows assessing the strength of evidence for a hypothesis H_a over an alternative hypothesis H_b based on the observed data (Rouder, Speckman, Sun, Morey, & Iverson, 2009). Typically, hypothesis H_a is the hypothesis of interest (i.e., H_1) and H_b is the null hypothesis stating that there is no effect (i.e., H_0). We can calculate a Bayes factor to quantify the evidence in favor of the data supporting H_1 rather than H_0 , which is denoted as BF_{10} . We can also use the Bayes factor to express evidence in favor of H_0 ,

¹ This procedure was chosen to account for unspecific variance and to obtain the largest possible contrast in WM ability.

by using the relation $BF_{01} = 1/ BF_{10}$. For example, $BF_{10} = 5$ indicates that it is 5 times more likely that the data derived from H_1 than from H_0 , whereas $BF_{10} = 1/5$ indicates that it is 5 times more likely that the data derived from H_0 than from H_1 . A BF_{10} between 1 and 3 indicates anecdotal evidence, between 3 and 10 substantial evidence, between 10 and 30 strong evidence, between 30 and 100 very strong evidence, and above 100 extreme evidence in favor of H_1 (Jeffreys, 1961; Wagenmakers, Wetzels, Borsboom, & van der Maas, 2011). When $BF_{10} = 1$, there is no evidence in the data for either H_1 or H_0 and when $BF_{10} < 1$ there is evidence in favor of H_0 .

To explore whether we could predict interindividual differences in age-related trends, we used regression trees (see Strobl, Malley, & Tutz, 2009 for an overview; also see Brandmaier, von Oertzen, McArdle, & Lindenberger, 2013). Regression trees are a nonparametric regression approach based on model-based recursive partitioning: in a hierarchical fashion, predictors are selected that partition the sample best into homogeneous subgroups with different parameter estimates of an initially specified regression model. Membership to the resulting subgroups is determined by predictors in the form of a hierarchy of decisions forming a tree: Inner nodes of the tree represent decision nodes, terminal nodes (or leaves) represent regression models. A tree is created by recursively selecting the predictor that best explains heterogeneity in the sample. In other words, at each level of growing a tree, the predictor that predicts maximal differences in the regression model is selected as a splitting variable. The exact splitting point is selected by maximizing the difference of the fit between the current node (i.e., parent node) and its two daughter nodes. The parent node is split into two daughter nodes if they represent better fit of the model to the data than the parent node. This process is repeated until a stopping criterion (e.g., a specified minimal number of observations or a specified threshold for the minimum improvement of a split's model fit) is met. The result is a tree with a set of leaves, each containing a subset of observations associated with different parameters of the initially specified regression models.

To build our regression tree, we (a) set up the initial regression model regressing the accuracy difference score on age as baseline model, and (b) determined potential predictors as candidates for the decision nodes in a tree. These candidates included a set of demographic variables (group, gender, education, profession, IQ, environmental support), comorbidities (depression, ADHD, anxiety, ASD), and EFs (interference control, processing speed). The tree was grown using the "party" package (Hothorn, Hornik, & Zeileis, 2006) in R. We set our stopping criterion to a minimum number of cases per terminal node of 20 and used Bonferroni correction for multiple comparisons at each node of the tree.

The baseline model was specified as a linear regression model with arcsine-square-root accuracy difference score regressed on age. Thus, the tree was geared up for exploring subgroups with differential age-gradients in WM performance. Although the regression tree was run with R 3.0.2 (R Core Team, 2012), the Bayes factors were calculated with JASP 0.7.0, an open source statistical package (Love et al., 2015). The other analyses were run with SPSS 22.0.

Results

Group Differences

As expected, there was a main effect of load level on the proportion of correct responses. Post hoc tests using Bonferroni correction revealed that accuracy decreased with increasing WM load. Accuracy was higher on 0-back (97.3%) than on 1-back (95.4%; $p < .001$) condition and higher on 1-back than on 2-back (88.9%; $p < .001$) condition. The main effects of group and gender were not significant. Also, none of the interactions were significant (see Table 2). These results showed that decline in performance due to increasing WM load was similar for individuals with and without ASD.

Analyses on RTs revealed the expected significant main effect of load level, indicating that RTs increased with increasing WM load. Post hoc pairwise comparisons using Bonferroni correction showed that RTs on the 0-back condition (513 ms) were faster than responses on the 1-back condition (607 ms; $p < .001$), and that RTs on the 1-back condition were faster than RTs on the 2-back condition (712 ms; $p < .001$). There was a significant main effect of group. The ASD group showed higher RTs (629 ms) than the COM group (596 ms; $p = .002$). None of the interactions reached significance (see Table 2).

To quantify evidence in favor of the data supporting the null findings on accuracy, we ran Bayesian exploratory ANOVAs with arcsine transformed accuracy as dependent variable and group and gender as independent variables: $BF_{10} = 1/7.2$ for the 0-back (please note that $BF_{10} < 1$ and, thus, there is evidence in favor of H_0 , indicating that it is 7.2 times more likely that the data derived from H_0 than from H_1), $BF_{10} = 1/1.4$ for the 1-back, and $BF_{10} =$

Table 2
Statistics of the Repeated Measures ANOVAs With Load as Within-Subject Factor, and Group and Gender as Between-Subjects Factors, Assessing WM Accuracy and RTs of the ASD and COM Group

Dependent variable and factor	Statistics		
	F	p	η_p^2
Correct responses			
Load	350.49	<.001	.56
Group	1.30	.256	.01
Gender	1.26	.264	.01
Group × Gender	.90	.345	.00
Load × Group	2.70	.070	.01
Load × Gender	.90	.406	.00
Load × Group × Gender	.28	.749	.00
Reaction times			
Load	1154.49	<.001	.81
Group	10.07	.002	.04
Gender	.43	.514	.00
Group × Gender	.41	.522	.00
Load × Group	1.94	.149	.01
Load × Gender	.33	.699	.00
Load × Group × Gender	.49	.594	.00

Note. Degrees of freedom are 2 and 542 for all within-group analyses, and 1 and 271 for all between-groups analyses. Significant values ($p < .01$) are indicated in bold script. ANOVAs = analysis of variance; WM = working memory; RTs = reaction times; ASD = autism spectrum disorder; COM = comparison.

1/1.3 for the 2-back. This indicates that the data provides substantial evidence for H_0 (i.e., group does not have an effect) on the baseline condition and only anecdotal evidence for H_0 on the 1-back and 2-back condition.

Age Effects

As gender did not have any influence on the results shown above, we excluded gender as a predictor from further regression analyses.² The regression model investigating differences in accuracy over age explained 9% of the observed variance. There was a main effect of age, demonstrating that increasing age was associated with larger difference scores (see Table 3). The main effect of group and the Age \times Group interaction were nonsignificant at the corrected alpha level, which indicated that the groups did not significantly differ in their difference scores and that age had a similar impact on WM decline in the ASD and COM group, when a linear pattern was considered. However, adding age² and Age² \times Group improved the model, $F_{\text{change}}(2, 269) = 4.19, p_{\text{change}} = .016$, and changed our findings. The model explained 12% variance and both interaction terms were significant, indicating differential age-related patterns, linear and quadratic, across the ASD and COM group. Post hoc regression analyses per group indicated a linear pattern in the COM group— $F(1, 162) = 19.79, p < .001, R^2 = .11$; $F_{\text{change}}(1, 161) = 2.62, p_{\text{change}} = .108, R^2_{\text{change}} = .01$ —and a combined linear and quadratic pattern in the ASD group— $F(1, 109) = 2.94, p = .089, R^2 = .03, F_{\text{change}}(1, 108) = 5.46, p_{\text{change}} = .021, R^2_{\text{change}} = .05$ (also see Figure 2).³

To assess the evidential strength for an interaction between age and group, we ran a Bayesian exploratory regression analysis with the difference score as dependent variable and group, age, and Age \times Group as predictors. We tested the hypothesis that the interaction model was preferred (H_1) over the model with only main effects (H_0). This comparison resulted in a $BF_{10} = 1/2.7$, indicating anecdotal evidence against the hypothesis that group and (linear) age interact in accuracy difference score. When adding a quadratic term and its interaction with group to the regression analysis, both the interaction models were preferred to the model without the linear interaction term ($BF_{10} = 6.8$) or without the

quadratic interaction term ($BF_{10} = 11.6$). Hence, the data provided substantial and strong evidence in favor of the hypothesis that group and age interact in the accuracy difference score when allowing for a nonlinear pattern. We followed-up on this result by running also Bayesian regressions per group, as we did in the frequentist analyses above. In the ASD group, the combined linear and quadratic model (H_1) was preferred to the model with only linear age (H_0 ; $BF_{10} = 5.0$). Nevertheless, comparing the combined model to the model without any age effects (i.e., the null model; H_0) yielded a $BF_{10} = 2.2$, indicating only anecdotal evidence for an age effect in the ASD group. In the COM group, the model with only linear age was preferred to the combined model ($BF_{01} = 1/1.5$) and the model with linear age was preferred to the null model ($BF_{10} > 100$), indicating extreme evidence for a (linear) age effect in the COM group.

Exploratory Regression Trees

Participants with missing values in one or more predictor variables were excluded from the regression tree analyses (remaining $n = 257$; 105 ASD, 152 COM). Exploratory regression tree analyses yielded a tree with a single decision node suggesting that IQ is a predictor of differential age-gradients on the accuracy difference score. The resulting two terminal nodes (IQ = 94 constituted the splitting point, thus there was one leaf with participants with IQ ≤ 94 , and one leaf with participants with IQ > 94) differed in their parameters of the initially specified model. Follow-up regression analysis with (centered) age, group (IQ ≤ 94 , IQ > 94), and Age \times Group as predictors, revealed a main effect of group. Participants with an IQ over 94 ($n = 227$; 93 ASD, 134 COM) had smaller difference scores ($p < .001$) than participants with an IQ of 94 or lower ($n = 30$; 12 ASD, 18 COM). Also the Age \times Group interaction was significant ($p = .035$). Post hoc tests showed that age impacted those with higher IQs— $F(1, 225) = 28.83, R^2 = .11, p < .001, \beta = 0.34$ —but did not have an impact in those with lower IQs— $F(1, 28) = 0.02, R^2 = .00, p = .902, \beta = -0.02$. In other words, participants with higher IQs showed overall better relative performance, but declined with increasing age. Participants with lower IQs performed poor overall, without any significant age-related differences. Individuals in the two terminal nodes did not differ in their mean age or gender ratio. None of the other predictors predicted age-related differences in WM performance after Bonferroni correction.

Table 3

Betas and p Values for the Regression Models Assessing the Difference Scores Between 2- and 0-Back for Correct Responses

Model and predictor	Accuracy difference score	
	β	p
Model 1 ^a		
Age	-.311	.000***
Group	-.121	.038*
Age \times Group	.082	.261
Model 2 ^b		
Age	.400	.383
Group	-.296	.001**
Age \times Group	-1.232	.008**
Age ²	-.730	.117
Age ² \times Group	1.365	.004**

^a $F(3, 271) = 8.95, p < .001, R^2 = .09$. ^b $F(5, 269) = 7.17, p < .001, R^2 = .12$.

* $p < .05$. ** $p < .01$. *** $p < .001$.

Discussion

In the current study, we investigated age-related patterns of cognitive functioning in ASD in one essential EF, namely WM.

² However, we cross-checked whether gender indeed did not influence the results by running all regression analyses with gender and Gender \times Group as additional predictors. In none of the analyses, gender or Gender \times Group were significant predictors; the pattern of findings did not change.

³ We explored whether the ASD and COM group differed in their error patterns and the impact of age. Analyses of the proportion of commission errors (i.e., erroneous responses) yielded similar results to those obtained with accuracy. Analyses of the proportion of omission errors (i.e., missed responses) revealed no group differences and no different impact of age between groups. Hence, participants with and without ASD demonstrated similar (age-related) error patterns across n -back WM performance.

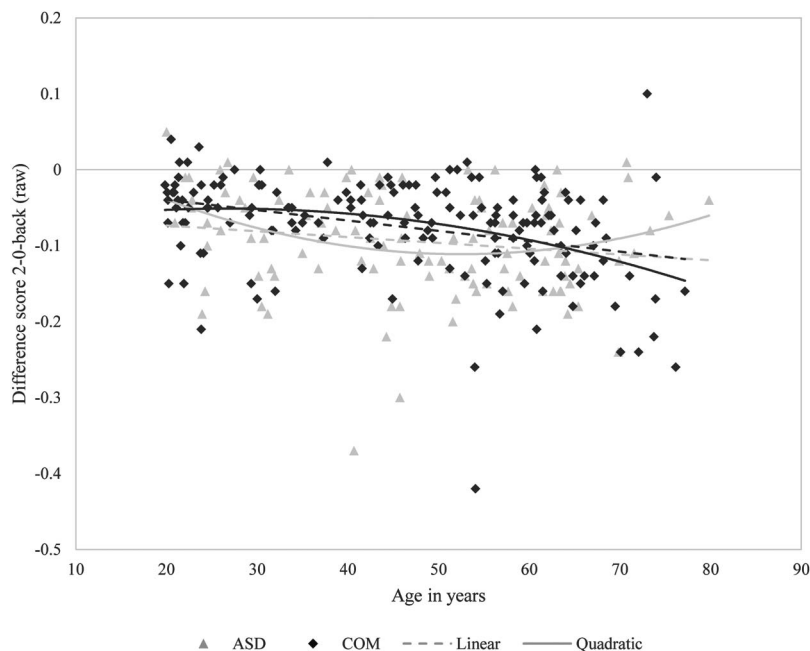


Figure 2. The impact of age (linear and quadratic) on the difference scores of correct trials in the autism spectrum disorder (ASD) and the comparison (COM) group.

EFs are known as a major challenge in ASD and deteriorate in typical aging. So far, the question of whether age-related cognitive decline follows a different pattern in ASD has been highly under-investigated. The present cross-sectional findings suggest, despite longer RTs, similar WM performance, but a differential age-related WM pattern in ASD clients compared with individuals without ASD.

The n -back task results revealed the typical decrease in performance with increasing WM load (e.g., Smith & Jonides, 1997). N -back performance did not significantly differ between adults with and without ASD on neither load level, as revealed by both conventional frequentists and Bayesian analyses. There are three possible explanations for this unpredicted result. First, the version of our task may not have been as challenging for adults with ASD as we expected. Even though a 2-back task involves manipulation and updating of information (Chatham et al., 2011), a further increment of n might have been necessary to sufficiently challenge all individuals and to eventually detect subtle WM difficulties in ASD. Second, the used stimuli were simple pictures, but as they were easy to name, verbal WM might have been invoked. Adults with ASD perform generally well on n -back tasks using obvious verbal stimuli, such as letters, and our findings are in line with these studies (Koshino et al., 2005; Williams et al., 2005). Third, individuals with ASD present a heterogeneous group and also their WM performance reveals large interindividual differences. Although the overall group may perform similarly to individuals without ASD, it does not preclude that a small subgroup of adults with ASD does have WM difficulties, as previously found in children (de Vries & Geurts, 2014).

Despite comparable WM accuracy rates, adults with ASD needed more time to respond. Although in previous studies using an n -back task no RT differences were found (e.g., Williams et al.,

2005), diminished processing speed is often observed in individuals with ASD (Travers et al., 2014). Furthermore, response slowing in ASD occurred independent of WM load, and seems, hence, a general feature rather than specific for WM. Nonetheless, WM accuracy apparently comes with a speed penalty that is greater for individuals with than without ASD. Whether these longer RTs are a result of a different strategy, which favors accuracy over speed (speed-accuracy trade-off), or part of a differential processing style and unrelated to accuracy, should be tested in a future study in which speed/accuracy instructions are experimentally manipulated.

Consistent with previous cross-sectional studies in typical aging, WM performance gradually declines with increasing age in adults without ASD (see Sander et al., 2012). This age-related pattern seemed, however, differentially expressed in individuals with ASD: The pattern was both linear and quadratic, with increasing age being associated with better performance, revealed by smaller difference scores. The difference score takes baseline performance (i.e., 0-back) into account and aims at filtering out unspecific variance. Smaller (compared with larger) difference scores indicated that increased load had a smaller detrimental effect on performance and, thus, designate better (relative) performance. Alternatively, one may argue that smaller differences scores are due to relatively poor baseline performance. We explored this possibility, but did not find any evidence in favor of this alternative. Individuals with ASD had similar baseline performance compared with those without ASD, $F(1, 273) = .13, p = .723, \eta_p^2 = .00$, and age had a comparable effect in both groups on baseline ($p = .400, \beta = -0.06$). Hence, adults with ASD had relatively good performance at increased load, rather than relatively poor performance at baseline, irrespective of age. More specifically, closer inspection of the age-related differences in WM perfor-

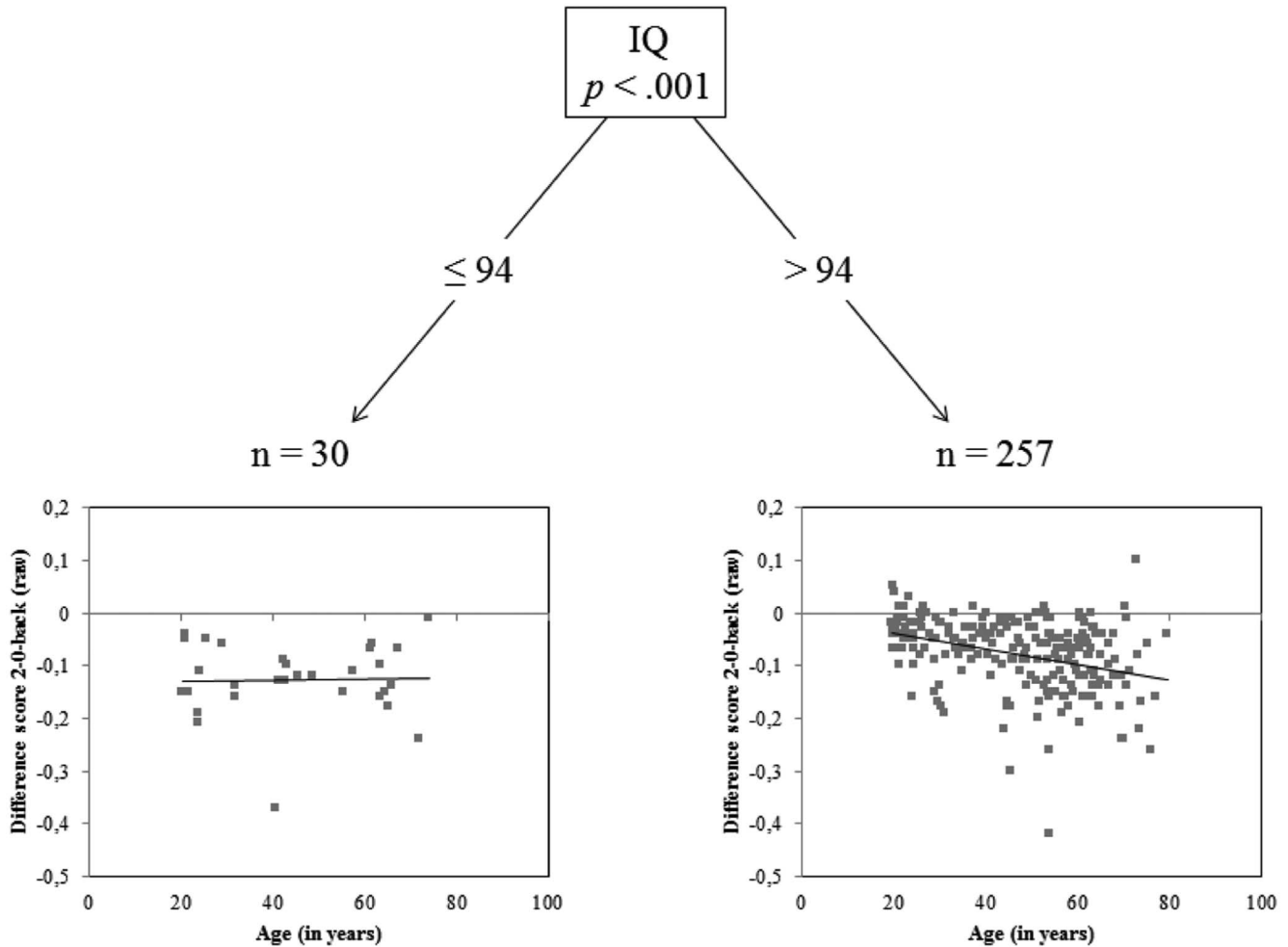


Figure 3. Visual representation of the regression tree with IQ as predictor.

mance among adults with ASD (see Figure 2) revealed that especially the oldest individuals with ASD demonstrated relatively small difference scores and, thus, exhibited relatively good WM performance at increased load. Nevertheless, there are two reasons why this pattern should be interpreted with caution. First, the inverted U-shape, suggesting improvement in old age, seems to be mainly driven by the oldest adults. Fjell et al. (2010) warn against overinterpreting outcomes that are driven by extremes of the age-range as they could be misleading about the true shape of the distribution. Second, although the Bayesian explorations indicated that there is substantial and strong evidence for differential age-related patterns, there is only anecdotal evidence that the data support an age effect when allowing for a nonlinear pattern in the ASD group. Hence, although the pattern could fit with the idea of ASD being a “safeguard” for typical age-related decline in WM performance (Geurts & Vissers, 2012; Lever & Geurts, 2015; Oberman & Pascual-Leone, 2014), careful interpretation about the pattern among older adults with ASD is warranted and further research is needed.

In children with ASD, WM development from childhood to young adulthood seems to be delayed (see O’Hearn et al., 2008),

and preliminary evidence suggests that WM difficulties persist into older adulthood (Geurts & Vissers, 2012). Our current results depart from these previous findings by demonstrating that WM development in middle and late adulthood does not necessarily continue to be deviant. There was no evidence for a WM deficit across adulthood in ASD, as measured by an n -back task, and no evidence for a pattern of increased age-related difficulties, which would result in an even larger difference between individuals with and without ASD in old age. Although speculative, this would suggest that some WM capacities, such as the ability of updating, matures after adolescence into adulthood, at a later stage than typically developing individuals (Andersen et al., 2014; Luna et al., 2007), and finally catch-up across adulthood. Nevertheless, there are two important distinctions to be made with the previous study on WM in late adulthood. First, in contrast to us, Geurts and Vissers (2012) used a spatial span task. Span tasks and n -back tasks both rely on WM related functions, such as the online maintenance of information, but they might tap into different processes (Redick & Lindsey, 2013). Although (simple or complex) span tasks involve the brief retention of stimuli (simple) and additional processing tests (complex), n -back tasks also involve

the updating of information. Second, their task relied on spatial WM and individuals with ASD present more difficulties with spatial WM than with verbal WM (Steele et al., 2007; Williams et al., 2005). Whether our task taps into verbal or more visual WM processes remains a topic of debate. Hence, despite the fact that both studies found a parallel age-related pattern (when allowing for only linear age-related differences), it is unclear if the discrepancy on group comparisons is due to different WM modality or to different underlying WM processes. Therefore, whether deficient span performance protracts into late adulthood in ASD whereas nonspan performance does not, or spatial WM difficulties protract into late adulthood, whereas verbal WM capacities do not, remains a question to be answered—ideally with longitudinal designs (e.g., Lindenberger et al., 2011; Raz & Lindenberger, 2011).

With regression trees, we explored whether we could distinguish subgroups of participants with different age-gradients indicating increased or reduced differences in WM performance with age. This exploratory method revealed that IQ constitutes a predictor of separate subgroups with different WM performances and/or differential age effects. Participants with lower IQs ($IQ \leq 94$) performed worse than participants with higher IQs ($IQ > 94$); the former did not show age-related WM decline, whereas the performance of the latter participants decreased with increasing age. An explanation for these nonintuitive results can be found in the data distribution, rather than in a floor effect, which one might expect: Visual exploration revealed that those with lower IQs show large heterogeneity, with participants of approximately the same ages ranging widely in difference scores. Hence, this could be a non-systematic relationship rather than the absence of linear age-related change (see Thomas et al., 2009). With regard to the exact splitting point, Brandmaier, von Oertzen, McArdle, and Lindenberger (2014) warned against the reification of splits of continuous variables; the reported IQ cutoff of 94 is of course subject to sampling error and, rather than reifying two distinct groups, we recommend to interpret it as a change point estimate, which might approximate a smooth underlying function.

Strengths, Limitations, and Future Directions

Given the large interindividual differences among individuals with ASD on the one hand (e.g., Towgood, Meuwese, Gilbert, Turner, & Burgess, 2009) and among older adults on the other (e.g., Werkle-Bergner et al., 2012), it seems crucial to study individual age-related processes over time (e.g., Lindenberger et al., 2011; Raz & Lindenberger, 2011). Even though this large cross-sectional study represents a significant initial attempt in the understanding of aging processes involved in individuals with ASD and provides, therefore, unique insights, it does not take into account how an individual ages. Therefore, longitudinal studies will be an important next step to examine the nature of age-related changes in WM performance among individuals with ASD.

The aim of our study was to understand age-related differences in adults with and without ASD. Arguably, to investigate typical aging, samples should involve individuals with normal-to-high intelligence. One could claim that, therefore, our sample was not representative of the general ASD population, which includes also individuals with intellectual disabilities (APA, 2013). In fact, our results may not apply to individuals with ASD and co-occurring intellectual disability. However, in contrast to many studies, other

psychiatric comorbid conditions did not constitute an exclusion criterion. This is crucial, as a large proportion of individuals with ASD suffer from at least one comorbid condition (Hofvander et al., 2009). Although comorbidities, such as depression or ADHD, may influence WM performance (Engelhardt et al., 2008; Paterniti et al., 2002), this is unlikely in our study, given our main findings and the fact that these conditions did not constitute predictors in the regression trees. Instead of compromising our findings, we believe it represents a strength of our study by augmenting the validity of our findings.

Although our ASD participants had a prior ASD diagnosis based on extensive diagnostic assessment in which, generally, developmental history is inquired, not all diagnoses could be verified by the ADOS (Lord et al., 2000), which is a recurrent problem when administering the ADOS to intellectually able adults with ASD (see Bastiaansen et al., 2011). To make sure that those who did not meet ADOS criteria did not influence our findings, we reran the group comparison and age-related regression analyses without those individuals. The pattern of results did not change. Furthermore, we did not administer the ADOS to the comparison group and cannot, thus, ensure that none of these participants had an undiagnosed ASD. Nevertheless, we inquired about ASD in participants themselves and in close family members and screened for ASD traits with the AQ. Therefore, the presence of ASD in the comparison group seems unlikely.

Conclusions

In sum, the present study provides unique cross-sectional evidence about age-related differences in WM performance among a large group of adults with and without ASD. Individuals with ASD, despite longer RTs, showed comparable WM performance across adulthood. The age-related gradual decline observed in typical individuals was differentially expressed in ASD when allowing for a nonlinear pattern. Albeit old age in ASD seemed to be associated with better WM performance, we argued that this finding should be interpreted with caution. Furthermore, additional exploratory Bayesian analyses suggested that age-related differences in WM performance among adults with ASD were barely worth mentioning. These findings provide initial insights into how ASD modulates cognitive aging, but also underlie the need for further WM research into late adulthood in ASD and for analyzing individual change trajectories in longitudinal studies.

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