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Lever, A.G.; Ridderinkhof, K.R.; Marsman, M.; Geurts, H.M.

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Reactive and Proactive Interference Control in Adults With Autism Spectrum Disorder Across the Lifespan

Anne G. Lever, K. Richard Ridderinkhof, and Maarten Marsman
University of Amsterdam

Hilde M. Geurts
University of Amsterdam and Dr. Leo Kannerhuis

As a large heterogeneity is observed across studies on interference control in autism spectrum disorder (ASD), research may benefit from the use of a cognitive framework that models specific processes underlying reactive and proactive control of interference. Reactive control refers to the expression and suppression of responses and proactive control refers to the adjustment of response to previous situations. We administered a Simon conflict task in 2 independent adult samples (IQ >80) and applied distributional analyses to examine temporal dynamics of interference control in ASD. Along comparable interference effects in both reactive and proactive control, young men (n = 23, 18–36 years) diagnosed with ASD made as many fast errors on conflict trials as neurotypical controls (n = 19) and showed similar suppression on slow responses (Study 1). However, over the adult life span (19–79 years), individuals with ASD (n = 118) made fewer fast errors on conflict trials, and had overall slower and more accurate responses than controls (n = 160; Study 2). These results converge to the idea that individuals with ASD adopt a more cautious response bias over the adult life span, which is not yet observed among young adults. Our findings suggest that it is fruitful to distinguish different processes involved in interference control and contribute to an increased understanding of interference control mechanisms in adults with ASD.

Keywords: autism spectrum disorder, response inhibition, aging, reactive and proactive interference control, conflict adaptation

Autism spectrum disorder (ASD) is a heterogeneous, neurodevelopmental disorder that is thought to last a lifetime (American Psychiatric Association, 2000, 2013). Core symptoms of ASD include qualitative impairments in social communication and social interaction, and restricted, repetitive patterns of behavior, interests, or activities. ASD is also associated with difficulties in cognitive control (Solomon, Ozonoff, Cummings, & Carter, 2008). Cognitive control refers to those processes that allow for monitoring and regulating goal-directed behavior to flexibly adapt behavior to environmental requirements (Botvinick, Braver, Barch, Carter, & Cohen, 2001). The ability to contain prepotent behavioral responses when such responses are reflex-like, premature, inappropriate, or incorrect (Ridderinkhof, van den Wildenberg, Segalowitz, & Carter, 2004), or inhibition, is such a cognitive control process. A lack of inhibitory control is thought to underlie some of the core symptoms observed in ASD (Lopez, Lincoln, Ozonoff, & Lai, 2005). Interference control, or resistance to distractor interference, is a specific aspect of the multifaceted nature of inhibition (Friedman & Miyake, 2004; Nigg, 2000) and denotes the ability to suppress irrelevant information. It is often necessary for effective and appropriate communication and social interactions. For example, when the literal meaning of a message should be ignored to capture the figurative or metaphoric meaning or when the opportunity to start about a specific topic of interest should be suppressed to maintain a conversation ongoing. Hence, these abilities to control behavior are important in daily life actions and may be involved in the difficulties that individuals with ASD encounter.

The existing literature on interference control in ASD is rather inconsistent, with some studies demonstrating impairments among individuals with ASD (Adams & Jarrold, 2012; Christ, Holt, White, & Green, 2007; Christ, Kester, Bodner, & Miles, 2011; Henderson et al., 2006), and others showing no differences between individuals with ASD and typically developing controls (Geurts, Luman, & Van Meel, 2008; Larson, South, Clayson, & Clawson, 2012; Schmitz et al., 2006; Solomon et al., 2008, 2009). The adherence of findings in a recent meta-analysis point to the idea of interference difficulties in ASD, but substantial heterogeneity across studies was observed (Geurts, van den Bergh, &

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Anne G. Lever and K. Richard Ridderinkhof, Department of Psychology and Amsterdam Brain and Cognition, University of Amsterdam; Maarten Marsman, Department of Psychology, University of Amsterdam; Hilde M. Geurts, Department of Psychology and Amsterdam Brain and Cognition, University of Amsterdam; and Research, Development & Innovation, Dr. Leo Kannerhuis.

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Correspondence concerning this article should be addressed to Anne G. Lever, who is now at Department of Psychiatry, VU University Medical Center, A. J. Ernststraat 1187, 1081 HL Amsterdam, the Netherlands. E-mail: a.lever@vumc.nl

Ruzzano, 2014). First, the use of rather crude measures, such as mean reaction time (RT), common in the ASD cognitive control literature, was suggested to be one of the major reasons for this heterogeneity. Second, the question whether or not individuals with ASD present difficulties in this domain is based on the assumption that interference control is a coherent, unified process, while we know from the cognitive control literature that it is not (Ridderinkhof, Forstmann, Wylie, Burle, & van den Wildenberg, 2011). Using crude measures and considering interference control as a coherent, unified process may not capture different stages or components involved in cognitive control and, thus, may not identify potentially impaired or preserved facets of interference control in ASD. Assessment of temporal dynamics of cognitive control may overcome these drawbacks and may address and clarify more fine grained aspects. This type of research in the ASD population has, however, been limited. Hence, more elaborate models of (specific aspects of) cognitive control should, therefore, be applied to attempt to disentangle which underlying processes contribute to an overall decrease in performance (Geurts et al., 2014; see also Solomon et al., 2008, 2009, 2014, for such an application). In the current study, we will use such an elaborate, theoretical framework: a dual-process model.

Dual-Process Models

Interference control is often measured with conflict tasks, such as the Eriksen flanker task (Eriksen & Eriksen, 1974) or the Simon task (Simon, 1969). In these tasks, a conflict is induced between task-irrelevant and task-relevant information that needs to be resolved. Dual-process models have been proposed to explain the interference effects elicited in conflict tasks (De Jong, Liang, & Lauber, 1994; Kornblum, Hasbroucq, & Osman, 1990; Ridderinkhof, van der Molen, & Bashore, 1995) by assuming that stimulus information is processed along two separate pathways. Although the direct activation route handles information rapidly and semi-automatically, the deliberate decision route involves slower decision processes. In case of the Simon task, the irrelevant feature (i.e., spatial location of the stimulus) directly activates the corresponding spatial response via the direct route. The relevant feature (i.e., color of the stimulus) is processed along the deliberate route to correctly translate the stimulus-response mapping based on task instructions. On congruent trials, the irrelevant stimulus feature, activating the direct route, and relevant stimulus feature, activating the deliberate route, converge at the level of response activation, leading to fast and accurate responses. On incongruent trials, the irrelevant and relevant stimulus features do not correspond and cause interference, leading to slower and less accurate responses.

The Activation-Suppression Hypothesis

Although the mean interference or congruency or Simon effect (i.e., the difference in RT and accuracy between congruent and incongruent trials) is a useful measure to reflect the additional time and demands required to solve interference, it does not capture the temporal dynamics of information processing that are involved in conflict situations (see van den Wildenberg et al., 2010). The activation-suppression hypothesis provides an explicit account to explain these temporal aspects. According to this hypothesis, the activation of the response associated with the irrelevant stimulus feature via the direct route (i.e., automatic response capture) can be selectively inhibited by the deliberate route (i.e., selective response suppression), but this process needs time to build up and is, therefore, only efficient after some time (Ridderinkhof, 2002). Several predictions follow from these assumptions. First, fast responses on incongruent trials do not benefit from the selective inhibition process as there is not enough time to build it up, resulting in a large number of fast errors. Second, as slow responses on incongruent trials do have this advantage, these are associated with more accurate responses. Third, even though congruent trials are associated with faster and more accurate responses than incongruent trials, the activation of the suppression process tends to inhibit the automatically captured response, which in congruent trials happens to coincide with the correct response. Congruent trials will, thus, benefit from faster responses, whereas their facilitation is reduced on slower responses. In contrast, incongruent trials are facilitated on slower responses. As a result, the interference effect is more affected by selective response suppression on slow trials than on fast trials (van den Wildenberg et al., 2010).

These predictions can be examined with a related analytical technique that, thus, allows to study the temporal dynamics underlying the manifestation of fast, impulsive errors and its subsequent build-up of selective response suppression (Ridderinkhof, 2002). We focus on two types of these distributional analyses: conditional accuracy functions (CAFs) and delta plots. CAFs provide a way to study automatic response capture by plotting accuracy data as a function of the entire RT distribution. Typically, CAFs reveal a high number of errors on fast RTs on incongruent trails, indicating strong automatic response capture in conflicting situations. Delta plots provide a graphical representation of response suppression by plotting RT differences between congruent and incongruent trials (i.e., the Simon effect) as a function of the entire RT distribution. Typically, delta plots reveal a reduction of the Simon effect on slower RTs, eventually even becoming negative, indicating efficient response suppression as an act of top-down control.

Reactive and Proactive Control

The function of detecting and solving interference after the occurrence of a conflict situation within the same trial, including the mechanisms of selective response suppression, is often designated as within-trial or *reactive* control. It relies upon the transient activation of the lateral prefrontal cortex, in combination with a more extensive network of other brain regions (Braver, 2012; Ridderinkhof et al., 2011). After such a conflict situation, one can also decide to adjust behavioral settings before the next trial to anticipate and prevent interference before it occurs. This mechanism is called between-trial or *proactive* control and involves the use of goal-relevant information to bias attention, perception, and action systems. It relies upon sustained activation of the lateral prefrontal cortex (Braver, 2012). As a result of this proactive control mechanism, interference effects on RT and accuracy are typically reduced when current trials are preceded by conflict (i.e., incongruent) trials. More specifically, when a congruent trial is followed by another congruent trial, responses are typically fast and accurate, whereas when a congruent trial is followed by an incongruent trial, responses are slower and error prone because of

a low level of control. After an incongruent trial, however, control is enhanced, resulting in a smaller difference in RTs or errors between current congruent or incongruent trials, and, hence, a smaller interference effect. This effect is called the Gratton effect (Gratton, Coles, & Donchin, 1992), conflict adjustment effect (Botvinick et al., 2001), or congruency sequence effect (CSE; Egner, 2007). We will refer to the CSE effect because this is a theory-neutral, operational term.

Reactive and Proactive Control in ASD

Although reactive and proactive control, as described above, have been investigated among clinical groups, such as attention deficit hyperactivity disorder (ADHD; Ridderinkhof, Scheres, Oosterlaan, & Sergeant, 2005), mild cognitive impairment (Wylie, Ridderinkhof, Eckerle, & Manning, 2007), and Parkinson's disease (e.g., Wylie, Ridderinkhof, Bashore, & van den Wildenberg, 2010), only a handful of studies examined these mechanisms among individuals with ASD. For example, Solomon and colleagues (2014) investigated the neural substrates underlying reactive and proactive control. Given that adolescents with ASD recruited brain regions associated with reactive control—anterior cingulate cortex and ventrolateral prefrontal cortex—rather than with proactive control—lateral prefrontal cortex—during a prepotent response task, they concluded that individuals with ASD prefer to rely on reactive rather than proactive control (Solomon et al., 2014). Nevertheless, at a behavior level, the authors only used a measure of reactive control and it is unclear whether these individuals with ASD showed intact or deficient CSEs. In an adapted version of the Eriksen flanker task, children and adolescents with ASD did not seem to show behaviorally deviant conflict monitoring and adaptation effects (i.e., CSEs), even though the neural processes underlying the detection and resolution of conflict were altered (Larson et al., 2012). Similar CSEs among individuals with and without ASD were also found when using social-emotional stimuli to induce conflict (Worsham, Gray, Larson, & South, 2015). Yet, despite these interesting findings, studies on temporal dynamics of interference control processes among individuals with ASD are lacking.

Present Study

In summary, in the current article, we rely on the above-described account to have a conceptual and more fine-grained model of cognitive control that may capture and explain the ASD-related heterogeneity observed in interference control. We present two studies in which we investigate reactive and proactive control and the temporal dynamics of interference control processes among intellectually able individuals with ASD. Automatic response capture and selective response suppression during reactive control are compared between individuals with and without ASD. In the first study, we examine these underlying cognitive control mechanisms in a group of adult men between 18 and 36 years old. Based on previous findings, we expect to observe deviant interference control during reactive control processes (Geurts et al., 2014), but an intact CSE (Larson et al., 2012; Worsham et al., 2015). In absence of literature on automatic response capture and selective response suppression in ASD, we do not have a specific prediction on this regard. In the second study, we aim to validate the results of Study 1 in an independent sample composed of adults between 19 and 79 years.

Study 1

Method

Participants. Twenty-four men aged 18–36 years with a clinical ASD diagnosis according to *Diagnostic and Statistical Manual for Mental Disorders-Fourth Edition-Text Revised* (*DSM–IV–TR*) criteria (American Psychiatric Association, 2000) determined by a multidisciplinary team, were recruited through Dr. Leo Kannerhuis Research, Development & Innovation, a specialized autism clinic in the Netherlands, and by advertisements on the website of the Dutch Autism Association. Twenty age-matched men without an ASD were recruited among acquaintances of Dr. Leo Kannerhuis's employees and formed the comparison group (COM). All non-ASD participants scored below 26 on the Autism-spectrum Quotient (AQ; Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001). Individuals with an estimated IQ below 80[1] were excluded, which resulted in the exclusion of one COM participant. Because of a stress reaction, one ASD participant was not able to finalize the Simon task and was, therefore, excluded from further analyses.

As these adults participated in a study assessing autonomic and endocrine activity (Smeekens, Didden, & Verhoeven, 2015), the following exclusion criteria were also applied: cardiac disease and complaints, respiratory problems, liver- and/or kidney failure, use of β-blockers or antidepressant medication. The final sample consisted of 23 adults with ASD and 19 adults without ASD (see Table 1).

Measures.

Simon task. Participants performed a classic visual Simon task (Broeders, Schmand, Wylie, de Bie, & Ridderinkhof, in prep). A square fixation point of 0.30 cm was presented at the center of the screen for a variable intertrial interval ranging from 1,750–2,250 ms. Next, a circle appeared on either the left or the right side of fixation (2.09 cm) until a response was made or the maximum time of 1,500 ms was exceeded. The circle had a diameter of 1.27 cm and was either green or blue. Two response keys were associated with the colors. The green circle required a left-hand response; the blue circle required a right-hand response. When the color of the circle was presented on the same side as the associated response button (e.g., the green circle requiring a left response appeared on the left side of the fixation point), the trial was considered congruent. When the color of the circle was presented on the nonassociated side (e.g., the green circle requiring a left response appeared on the right side of the fixation point), the trial was considered incongruent. Participants were instructed to respond as fast and accurate as possible. Each participant completed a practice block of 12 trials to learn the color-response association. Next, four experimental blocks of 60 trials each were presented. Color and response side were randomly varied across trials; congruent (n = 120) and incongruent (n = 120) trials were randomly assigned.

[1] This threshold was adopted to ensure that individuals were able to perform this kind of cognitive tasks and to provide informed consent themselves.

Table 1
Means (SDs), Demographic, and Clinical Scores of the ASD and COM Group (Study 1)

Variables	Group: ASD (n = 23)	Group: COM (n = 19)	Statistics
Education[a]	18/5/0	1/12/6	Fisher's test, $p < .001$
Diagnosis[b]	4/5/12/2	—	—
Age	23.3 (4.7)	26.0 (4.8)	$t(1,40) = -1.88$, $p = .067$, $\eta_p^2 = .08$
	Range 18–36	Range 18–35	
IQ	108.9 (13.6)	117.8 (13.7)	$t(1,40) = -2.10$, $p = .042$, $\eta_p^2 = .10$
	Range 83–137	Range 86–149	
AQ	24.4 (7.8)	8.5 (4.5)	$t(1,40) = 7.90$, $p < .001$, $\eta_p^2 = .61$
	Range 13–38	Range 2–17	
ADHD[c]	23.6 (9.5)	18.6 (8.9)	$t(1,38) = 1.70$, $p = .098$, $\eta_p^2 = .07$
	Range 7–42	Range 5–38	

Note. ASD = autism spectrum disorder group; COM = comparison group; IQ = estimated intelligence quotient; AQ = Autism-spectrum Quotient; ADHD = attention-deficit-hyperactivity disorder.
[a] The numbers between slashes indicate the educational level based on the Verhage coding system (Verhage, 1964): junior general secondary or vocation education/senior general secondary education or vocation colleges/university education. [b] The numbers between slashes indicate a diagnosis of Autism/Asperger Syndrome/Pervasive Developmental Disorder Not Otherwise Specified/ASD. [c] ADHD symptoms were assessed with the ADHD-SR (Kooij et al., 2005), a Self-Reported Questionnaire. Two ASD participants did not complete the ADHD-SR. Other self-reported Axis-I comorbidities included, among others, depression and anxiety.

Cognitive functioning. Cognitive functioning (estimated IQ) was assessed with two subtests of the Wechsler Adult Intelligence Scale-Third Edition (WAIS-III; Wechsler, 1997): Vocabulary and Block Design. Both subtests have very good internal consistency (α = .91/.89) and good test–retest reliability (r = .91/.88) and are in combination highly correlated with full scale IQ (e.g., Ringe, Saine, Lacritz, Hynan, & Cullum, 2002).

Diagnostic measures. All participating adults with ASD already had a diagnosis within the autism spectrum diagnosed by a multidisciplinary team including a psychologist and a psychiatrist according to *DSM–IV* criteria. Yet, the Dutch version of the AQ (Baron-Cohen et al., 2001; Hoekstra, Bartels, Cath, & Boomsma, 2008) was administered to assess the presence of autistic traits. The Dutch version of the AQ shows satisfactory internal consistency (α = .71/.81) and test–retest reliability (r = .78; Hoekstra et al., 2008).

Procedure. After written informed consent was obtained, the abbreviated version of the WAIS-III and the Simon task were administered. Additional tasks were administered as part of a larger study, but these are described elsewhere (Smeekens et al., 2015). Within three days after completing the experimental session, participants filled out some questionnaires online, including the AQ. The study was approved by the local ethical review board of the Faculty of Social Sciences of the Radboud University Nijmegen, the Netherlands (ECG 0601011), and complied with all relevant laws and institutional guidelines.

Statistical analyses. First, extreme RT values (>3 *SD*), either excessively slow or fast, were removed from the data of each participant (see, e.g., Wylie et al., 2010). This conservative trim procedure resulted in the elimination of less than 2.6% of trials per subject (ASD: M = 1.3%, SD = 0.7%; COM: M = 1.2%, SD = 0.6%). Second, fast (<100 ms) responses were also removed from the data, resulting in the elimination of 0.9% of trials per participant (ASD: M = 0.04%, SD = 0.2%; COM: M = 0.02%, SD = 0.1%). Third, mean RT and mean accuracy (i.e., mean percentage of correct responses) were calculated for each participant. As RTs and accuracy data were not normally distributed, RTs were log transformed and arcsine-square-root transformation was applied to accuracy to obtain normality.

To investigate reactive control of interference, two mixed design analyses of variance (ANOVAs)[2] were computed with Congruency (congruent, incongruent) as within-subject factor and Group (ASD, COM) as between-subjects factor and log transformed RT and arcsine-square-root transformed accuracy as dependent variables. The strength of automatic response capture was examined by means of conditional accuracy functions (CAFs). In a CAF, accuracy rates are plotted as a function of the entire RT distribution. Therefore, RTs of congruent and incongruent trials are rank-ordered and divided into five approximately equal-sized segments, called bins. Next, accuracy rates are calculated for each bin, resulting in five accuracy values for congruent trials and five accuracy values for incongruent trials. These values are plotted against the mean RT for each bin. The accuracy values within the first bin (i.e., fastest responses, automatically driven) are considered a measure of strength of automatic response capture (fast responses are error-prone because under control of the direct reflex-like route leading to activation of incorrect responses when trials are incongruent). These accuracy values of the ASD and COM group are compared by means of a paired sample t test. Selective response suppression was examined with delta plots. Delta plots show the Simon effect as a function of the entire RT distribution. Also for this measure, RTs are rank-ordered and divided into five bins, but now for correct responses only. Mean RTs are calculated for both congruency levels in each bin. Next, the Simon effect is calculated for each bin, resulting in five Simon effect values. These are plotted against the mean RT for each bin. The delta slope of the slowest segment, that is the difference between the Simon effect of the fourth and the fifth bin, is considered a measure of proficiency of suppression (selective suppression needs time to build up and is, thus, reflected on slower responses). These slopes of the ASD and COM group are compared with a paired sample t test.

To investigate proactive control of interference, two mixed design ANOVAs were computed with Congruency (congruent, incongruent), Group (ASD, COM) and trial sequence (preceding trial congruent [PTC], preceding trial incongruent [PTI]) as experimental factors and log transformed RT and arcsine-square-root transformed accuracy as dependent variables.

Next to conventional p values, we used Bayes factors (Jeffreys, 1935, 1961; Kass & Raftery, 1995) to quantify evidence for a hypothesis H_a against an alternative hypothesis H_b, based on the

[2] The groups differed on their mean IQs. However, as IQ was not correlated with the Simon effect, RTs, or accuracy on (in)congruent trials (all rs < .2, all ps > .16), IQ was not considered as covariate in the analyses.

observed data. Typically, H_a is the hypothesis of interest (denoted here as H_1) and H_b the null-hypothesis stating that there is no effect (denoted here as H_0). We indicate the Bayes factor expressing evidence for H_1 over H_0 as BF_{10}, which can also be used to quantify evidence in favor of the null-hypothesis H_0 by using the relation $BF_{01} = 1/BF_{10}$. For instance, when $BF_{10} = 3$, it is three times more likely that the data derived from H_1 than from H_0, and when $BF_{10} = 1/3$, it is three times more likely that the data derived from H_0 than from H_1. To aid the interpretation of Bayes factors, Wagenmakers, Wetzels, Borsboom, & van der Maas (2011) suggested to use the following scale: "anecdotal evidence" in favor of H_1 when $1 < BF_{10} \leq 3$, "substantial evidence" when $3 < BF_{10} \leq 10$, "strong evidence" when $10 < BF_{10} \leq 30$, "very strong evidence" when $30 < BF_{10} \leq 100$, and "extreme evidence" when $BF_{10} > 100$. Note that $BF_{10} = 1$ indicates that there is no evidence for or against H_1 (meaning that it is equally likely that the data derived from H_1 or H_0), and that a $BF_{10} < 1$ indicates evidence in favor of H_0.

We computed Bayes factors for the *t* tests and ANOVA models described above. In the Bayesian *t* tests, we compare the (null) hypothesis that the groups do not differ with the (alternative) hypothesis that the groups differ by comparing a model with the main effect of group to the null model. In the Bayesian mixed design ANOVAs, we compare the most complex model that includes the effect we are interested in with the model that excludes this effect. For example, by determining the evidential strength for an interaction between group and congruency, we compare a model with the main effects of group and congruency to a model with the main effects of group and congruency and the interaction term. This procedure yields a Bayes factor that indicates to which extent which model is preferred and, thus, indicates the evidence in favor of or against the hypothesis that group and congruency interact.

Bayes factors were computed using the freely available statistical software program JASP (Love et al., 2015, submitted), which can be downloaded from https://jasp-stats.org/. All other analyses were run with SPSS 22.0 (IBM Corp., 2013). There were no outliers (i.e., data points more than three times the interquartile range above or below the first quartile) on reactive control, whereas there was one outlier in the ASD group in the proactive control analyses. As removing this outlier did not change the pattern of findings, we reported the results including this outlier.

Results

Reactive control. On reactive control (see Table 2), as predicted, there was a pronounced effect of congruency on both RT and accuracy: Congruent trials were associated with faster RTs ($BF_{10} > 100$) and more accurate responses ($BF_{10} = 69.07$) than incongruent trials. This congruency effect did not interact with group (RT: $BF_{10} = 1/2.47$; accuracy: $BF_{10} = 1/3.31$), nor was there a main effect of group on accuracy ($BF_{10} = 1/3.03$). For RT, there was a slight preference against a main effect of group, although the amount of evidence was very small and, therefore, inconclusive ($BF_{10} = 1/1.39$; see Figure 1). Hence, the two groups presented a comparable Simon effect (i.e., the difference between congruent and incongruent trials: $RT_{incongruent} - RT_{congruent}$, $accuracy_{congruent} - accuracy_{incongruent}$).

Table 2
Statistics of Group Comparisons on Reactive Control (Study 1)

	RTs			Accuracy		
Factors	*F*	*p*	η_p^2	*F*	*p*	η_p^2
Congruency	**121.88**	**<.001**	**.75**	**13.65**	**.001**	**.25**
Group	1.36	.251	.03	.02	.891	.00
Group × Congruency	.22	.641	.01	.03	.859	.00

Note. RTs = reaction times. Degrees of freedom are (1, 40) for all group analyses. Significant values ($p < .05$) are indicated in bold script.

Accuracy rates of the fastest responses on incongruent trials did not differ between groups, $t(1,40) = 0.50$, $p = .620$, $\eta_p^2 = .01$, $BF_{10} = 1/2.98$ indicating that the strength of response capture was similarly expressed across the ASD and COM group (Figure 2a). Likewise, there was no effect of group on the delta slope of the slowest responses, $t(1,40) = 1.72$, $p = .094$, $\eta_p^2 = .07$, indicating that the strength of response suppression was comparable between the ASD and COM group (Figure 2b). Nevertheless, evidence was rather inconclusive as the Bayes factor in favor of the null hypothesis was close to one ($BF_{10} = 1/1.03$).

Proactive control. On proactive control, as predicted, we found that responses were faster ($BF_{10} > 100$) and more accurate ($BF_{10} > 100$) when congruent trials were preceded by congruent trials rather than when preceded by incongruent trials, and when incongruent trials were preceded by incongruent trials rather than when preceded by congruent trials (Table 3, Figure 3). In other words, the Simon effect was larger after congruent trials than after incongruent trials. This effect did not differ between groups (RT: $BF_{10} = 1/3.83$; accuracy: $BF_{10} = 1/2.87$). Hence, proactive control is similarly enhanced after a conflict situation in individuals with and without ASD.

Discussion

In line with earlier clinical studies (Ridderinkhof et al., 2005; Wylie et al., 2007, 2010), we applied distributional techniques, designed to test the activation-suppression hypothesis (Ridderinkhof, 2002), and examined CSEs to study the underlying mechanisms of interference control in ASD. With regard to reactive control, Study 1 demonstrated that the congruency effect elicited by conflict and the number of fast errors on incongruent trials was comparable among young adults with and without ASD. Fast responses on incongruent trials are prone to errors as they activate a direct reflex-like route that leads to the activation of the incorrect response and are considered a measure of automatic response capture (Ridderinkhof, 2002). Furthermore, the selective suppression of responses by means of the deliberate route, revealed by a reduction of the Simon effect on slow responses (van den Wildenberg et al., 2010), was similar in individuals with ASD and controls.

Study 1 also indicated that the proactive mechanism adopted to detect and adjust behavior in reaction to conflict situations seems to be intact in individuals with ASD. As in typically developing adults (Botvinick et al., 2001; Egner, 2007; Gratton et al., 1992), we observed a reduced interference effect after incongruent trials compared with congruent trials, indicating enhanced cognitive control after conflict. This behavioral result is in line with previous studies in ASD (Larson et al., 2012; Worsham et al., 2015).

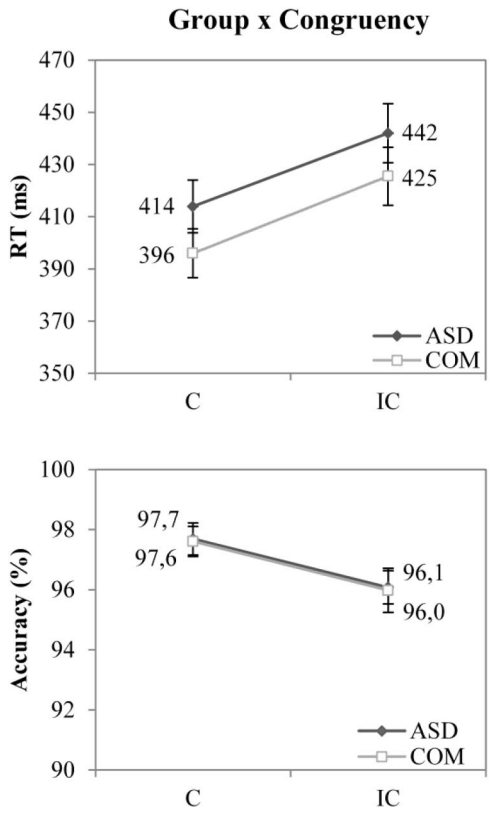

Figure 1. Mean reactions times (RTs) and accuracy rates for congruent and incongruent trials per group (Study 1). *Note:* ASD = autism spectrum disorder group; COM = comparison group; C = congruent; IC = incongruent. Error bars present *SE*s.

Hence, we demonstrated in Study 1 similar reactive and proactive interference control abilities in young adults with ASD compared with those without ASD. Despite that the exploratory Bayesian analyses show support for these frequentist results as they indicate some evidence against H_1 (i.e., a group effect), the amount of evidence ranges from small ($BF_{10} \leq 1/3.83$) to no evidence at all ($BF_{10} \leq 1/1.03$). In addition, there are some potential methodological caveats suggesting that we need to be careful in making strong claims based on this single study.

First, although the task we used has proven its validity in a sample of Parkinson's disease patients (e.g., Broeders et al., in prep), it was not yet administered to individuals with ASD. The interstimulus interval of the Simon task had a rather long duration and the colored circles appeared close to the fixation point. Adults with ASD are sensitive to event presentation rate (i.e., variation of the interstimulus interval), showing similar performances on slow or medium event rate, but decreased performance on fast event rates (Raymaekers, van der Meere, & Roeyers, 2004). Moreover, Adams and Jarrold (2012) showed that increasing size of the target and increasing distance between distractors in a Flanker task reduced the interference effect in typically developing controls, but not in children with ASD. Also in the Simon task, increasing the distance between fixation and the stimulus (i.e., a larger eccentricity) reduced the Simon effect (Hommel, 1993). If individuals with ASD are less sensitive to distractor salience, then they should demonstrate a larger interference effect compared with controls when distractor salience is large. These observations suggest that diminishing the interstimulus interval and increasing the stimulus-fixation distance should facilitate the occurrence of an effect between individuals with and without ASD when difficulties in interference control indeed exists in ASD. Therefore, we changed these parameters of the Simon task in a second study.

Second, only 12 practice trials were administered before starting the test session. This small number may suffice to acquaint the participants with the global properties of the task, but perhaps not to train them to attain asymptote RTs, in particular when responding to incongruent stimuli.

Third, the low number of self-reported ASD traits caught our attention. It may indicate that the ASD participants presented mild symptoms, which could be a potential argument for absent interference control deficits, but it also may illustrate poor introspection (see Frith, 2004). As these AQ scores did not deviate from those previously reported by participants with the same mean age (Bishop & Seltzer, 2012; Ketelaars et al., 2008; Kurita, Koyama, & Osada, 2005), it seems plausible that young adults tend to report low ASD traits. Furthermore, although the sample consisted of individuals who were diagnosed with ASD by a specialized mental health institution, their diagnoses were not independently verified by the researchers with a standardized diagnostic instrument to assess the quality and quantity of current ASD symptomatology. Therefore, in the second study, we administered one of the most commonly used instruments in ASD research: the Autism Diag-

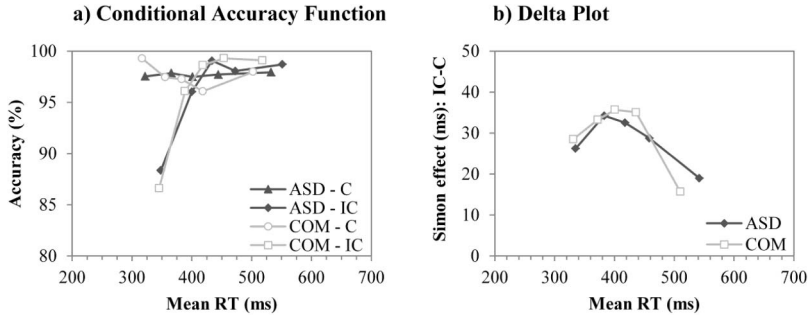

Figure 2. (a) Conditional accuracy functions and (b) delta plots per group (Study 1). *Note:* ASD = autism spectrum disorder group; COM = comparison group; C = congruent; IC = incongruent.

Table 3
Statistics of the Group Comparison on Proactive Control (Study 1)

Factors	RTs			Accuracy		
	F	*p*	η_p^2	*F*	*p*	η_p^2
Congruency	**128.88**	**<.001**	**.76**	**9.37**	**.004**	**.19**
Trial sequence	**7.48**	**.009**	**.16**	**4.57**	**.039**	**.10**
Group	1.33	.256	.03	.00	.973	.00
Congruency × Trial sequence	**152.57**	**<.001**	**.79**	**74.45**	**<.001**	**.65**
Group × Congruency	.12	.727	.00	.35	.559	.01
Group × Trial sequence	.05	.826	.00	.55	.465	.01
Group × Congruency × Trial sequence	.13	.717	.00	.00	.995	.00

Note. RTs = reaction times. Degrees of freedom are (1, 40) for all analyses. Significant values ($p < .05$) are indicated in bold script.

nostic Observation Schedule (ADOS; Lord et al., 2000) to assess the current presence of ASD symptoms to validate the clinical diagnosis as determined by ASD experts.

Finally, despite the observation that age does not seem to be a relevant moderator in interference control among individuals with ASD (Geurts et al., 2014), only a few studies took adults with ASD into account and it is, thus, unclear whether the absence of age-related effects protracts into adulthood. Typically developing adults experience age-related decline in several cognitive domains (e.g., Friedman, Nessler, Cycowicz, & Horton, 2009; Verhaeghen & Cerella, 2002). Although aging is not systematically associated with impairments in interference control (Nieuwenhuis et al., 2002; Wild-Wall, Falkenstein, & Hohnsbein, 2008) and proactive control of interference seems to be spared (Puccioni & Vallesi, 2012; Yano, 2011), older adults generally show a larger Simon effect compared to younger adults (Kawai, Kubo-Kawai, Kubo, Terazawa, & Masataka, 2012; Pick & Proctor, 1999; Van der Lubbe & Verleger, 2002; see Proctor, Pick, Vu, & Anderson, 2005, for an overview). Whether automatic response capture and selective response suppression are sensitive to age-related differences is yet unknown. Hence, we set out to examine the role of age in interference control processes among individuals with and without ASD across adulthood in a new experiment, extending the age range of the sample to the adult life span.

In summary, to determine whether we can corroborate our null findings in an independent ASD sample, we conducted Study 2 with an adapted visual Simon task in a larger sample with an extended age range to investigate also age-related differences in underlying processes of interference control across adulthood in ASD.

Study 2

Method

Participants. Individuals between 19 and 79 years with a diagnosis within the autism spectrum according to *DSM–IV* criteria (American Psychiatric Association, 2000) were diagnosed by a

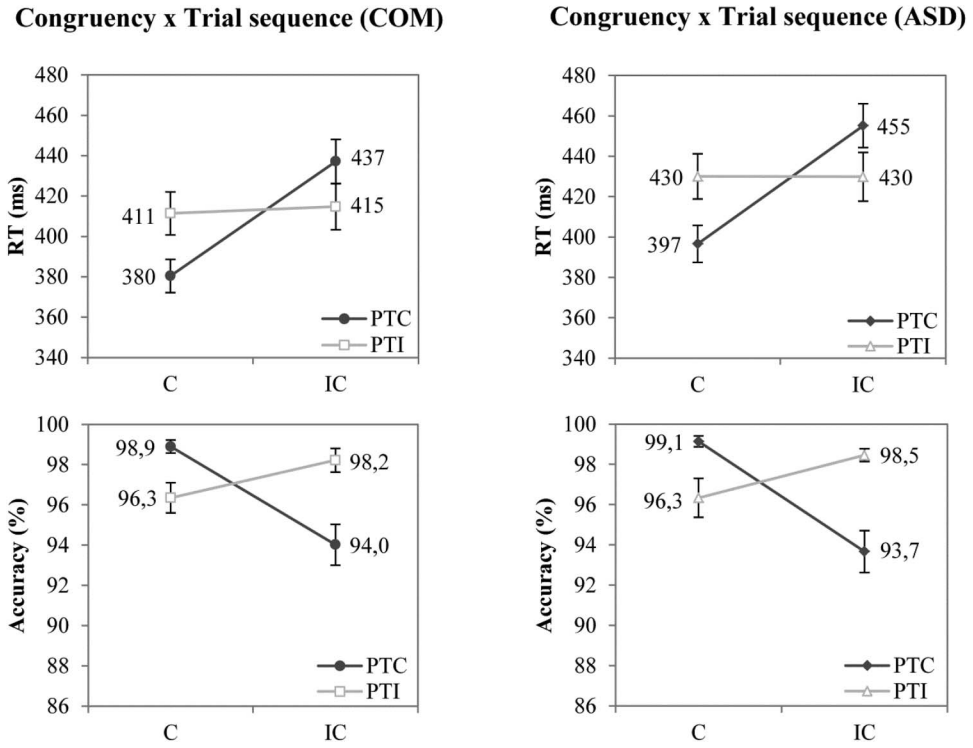

Figure 3. The congruency sequence effect per group (Study 1). *Note:* ASD = autism spectrum disorder group; COM = comparison group; C = congruent; IC = incongruent; PTC = previous trial congruent; PTI = previous trial incongruent. Error bars present *SE*s.

multidisciplinary team including a psychologist or psychiatrist and were recruited through several mental health institutions across the Netherlands and by advertisements on client organization websites. Of the 168 individuals, 45[3] were excluded because of (a) the absence of a clinical ASD diagnosis, (b) the current or former presence of neurological problems (e.g., epilepsy, stroke, and cerebral contusion), schizophrenia, or psychoses, (c) a current alcohol- or drugs dependency, or (d) an estimated IQ below 80 (criterion 1 and 4 are similar to Study 1). ADOS module 4 (Lord et al., 2000) and AQ (Baron-Cohen et al., 2001) were administered to verify the participants' clinical diagnosis. Participants who scored above the ADOS threshold (≥7) or AQ (≥26) threshold were included in the current study. Of the 35 participants who did not meet the ADOS criterion, only five did also not meet the AQ criterion and were excluded from further analysis. This resulted in an eligible ASD sample of 118 participants, of whom all completed the Simon task (for a description of the sample, see also Lever & Geurts, 2016a; Lever, Werkle-Bergner, Brandmaier, Ridderinkhof, & Geurts, 2015; for information on psychiatric comorbidities, see Lever & Geurts, 2016b).

Individuals without ASD were recruited by means of advertisements on the university website and on social media, and within the social network of the researchers. Of the 193 individuals, 24 were excluded because of (a) the presence of ASD or schizophrenia in close relatives, (b) a diagnosis of ADHD, (c) the current or former presence of neurological problems (e.g., epilepsy, stroke, and cerebral contusion), schizophrenia, or a psychosis, (d) a current alcohol- or drugs dependency, or (e) an estimated IQ below 80 (criterion 5 is similar to Study 1). COM participants with an incomplete AQ (≥10% missing values, $n = 1$) or an AQ score above the threshold proposed for the general population (≥32, $n = 1$; Woodbury-Schmidt et al., 2005) were also excluded. This resulted in an eligible COM sample of 167 participants, of whom 160 completed the Simon task.

ASD and COM participants were matched on age and estimated IQ. However, the proportion of women was larger in the COM group than in the ASD group (see Table 4). Please note that we administered the Mini Mental State Examination to check adequate general cognitive functioning; all participants scored at least 26.

Measures.

Simon task. Participants performed a modified visual Simon task compared with Study 1. A fixation cross (0.90 cm) was presented at the center of the screen for a variable intertrial interval ranging from 1,250–1,750 ms. Next, a circle (diameter 2.11 cm) appeared on either the right or the left side (4.23 cm) of fixation. As in Study 1, the circle was displayed until response was made for a maximum of 1,500 ms and was either green or blue. Also, each color was associated with a left or right response key and participants were instructed to respond as fast and accurate as possible. Four experimental blocks were preceded by two practice blocks, instead of one short practice block in Study 1, during which participants could familiarize with the task. The first practice block consisted of 30 only congruent trials. The second practice block consisted of a mixture of 60 congruent and incongruent trials. As participants had difficulties to memorize the color-response association, two colored cues were provided in concordance with the color-response mapping. Color and response side were again counterbalanced across trials resulting in an equal probability of congruent ($n = 120$) and incongruent trials ($n = 120$). In addition, the color-response mappings were counterbalanced across participants (i.e., half of the participants associated the green circle with the left response button and the blue circle with the right response button; the other half associated the blue circle with the left response button and the green circle with the right response button).

Cognitive functioning. Cognitive functioning (estimated IQ) was assessed with two subtests of the WAIS-III (Wechsler, 1997): Vocabulary and Matrix Reasoning, instead of Block Design in Study 1. Both subtests have very good internal consistency ($\alpha = .91/.91$) and good test–retest reliability ($r = .91/.78$) and are in combination highly correlated with full scale IQ (e.g., Ringe et al., 2002).

Diagnostic measures. The Dutch version of the ADOS Module 4 (de Bildt & de Jonge, 2008; Lord et al., 2000) was administered to assess the presence of ASD symptoms. The ADOS is a standardized semistructured instrument designed for the assessment of ASD. Social interaction, communication, and play are elicited by means of 10–15 small conversations and activities. A client's behavior is observed and scored according to 31 criteria. A subset of criteria are used to compute the "original" diagnostic algorithm. We used a threshold of 7 for the classification of ASD. The ADOS was administered and scored by a trained and certified psychologist. Module 4 has moderate sensitivity (0.61), good specificity (0.82), and good predictive value (0.81) when administered to high-functioning adults (Bastiaansen et al., 2011). As in Study 1, the Dutch version of the AQ (Baron-Cohen et al., 2001; Hoekstra et al., 2008) was administered to assess the presence of autistic traits.

Procedure. After written informed consent was obtained, participants underwent an extensive screening during which the ADOS (only ASD participants) and the abbreviated version of the WAIS-III were administered. A few weeks later, the participants returned for an experimental session, including the Simon task. As the current study is part of larger project on aging in ASD, more tasks and questionnaires were administered, but these are described elsewhere (e.g., Lever & Geurts, 2016a; Lever et al., 2015). The order of tasks in the experimental session was counterbalanced across participants. The study was approved by the local ethical review board of the Department of Psychology of the University of Amsterdam, the Netherlands (2011-PN-1952), and complied with all relevant laws and institutional guidelines.

Statistical analyses. Study 2 used the same procedure to analyze the data as described in Study 1, but gender was added as a between-subjects factor as the ASD and COM group differed on their gender ratio. In addition, to investigate the effect of age on reactive and proactive control, centered age was added as a covariate to the mixed design ANOVAs and the interaction between centered age and group was inspected. Furthermore, we computed stepwise regressions with centered age in the first step, and group, group-by-centered age, and gender in the second step as predictors on accuracy of the first bin and on the slowest segment of the delta slope to examine the effect of age on response capture and sup-

[3] This number is mainly because of the second criterion. Psychoses (e.g., Croen et al., 2015; Hofvander et al., 2009) and neurological problems (e.g., Croen et al., 2015) frequently occur among individuals with ASD. However, there were no differences in AQ scores between those who were included and those who were not ($p > .4$).

Table 4
Means (SDs), Demographic, and Clinical Scores of the ASD and COM Group (Study 2)

Variables	Group: ASD (n = 118)	Group: COM (n = 160)	Statistics
Gender	83 M/35 F	91 M/69 F	Fisher's test, p = .024, odds ratio = 1.79
Education[a]	0/1/0/3/35/53/26	0/0/1/5/25/79/50	Fisher's test, p = .032
Diagnosis[b]	18/60/35/5	—	—
Age	47.6 (14.9) Range 20–79	46.1 (16.5) Range 19–77	$F(1, 276) = .66$, $p = .419$, $\eta_p^2 = .00$
IQ	114.8 (16.9) Range 84–155	114.0 (16.5) Range 80–155	$F(1, 276) = .16$, $p = .695$, $\eta_p^2 = .00$
MMSE	29.1 (1.0) Range 26–30	29.2 (1.0) Range 26–30	$F(1, 276) = .56$, $p = .457$, $\eta_p^2 = .00$
AQ	33.8 (8.3) Range 8–49	12.1 (5.2) Range 2–26	$F(1, 275)$[c] $= 708.90$, $p < .001$, $\eta_p^2 = .72$
ADHD[d]	21.2 (8.4) Range 5–46	11.5 (6.1) Range 0–32	$F(1, 273) = 122.6$, $p < .001$, $\eta_p^2 = .31$
ADOS[e]	8.6 (3.1) Range 1–19	—	

Note. ASD = autism spectrum disorder group; COM = comparison group; M = male; F = female; IQ = estimated intelligence quotient; MMSE = Mini Mental State Examination; AQ = Autism-spectrum Quotient; ADHD = attention-deficit-hyperactivity disorder; ADOS = Autism Diagnostic Observation Schedule.
[a] The numbers between slashes indicate the educational level based on the Verhage coding system (Verhage, 1964), 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] ADHD symptoms were assessed with the ADHD-SR (Kooij et al., 2005), a Self-Reported Questionnaire. Two ASD participants and one COM participant did not complete the ADHD-SR. Please see Lever and Geurts (2016b) for other psychiatric comorbidities. [e] Of the final sample, 30 participants scored below the ADOS cut-off (<7). Excluding these participants from the analyses did not alter the conclusions.

pression, respectively. In addition to the previously mentioned Bayesian analyses, we ran Bayesian (mixed design) ANCOVAs with centered age as covariate and Bayesian regressions to assess the evidential strength for the data supporting the hypothesis of a differential age-related effect in the two groups on reactive and proactive control by comparing two models, as described in the Methods section of Study 1.

Applying the conservative trim procedure to remove extreme RT values (>3 *SD*) resulted in the elimination of less than 2.6% trials per subject (ASD: $M = 1.2\%$, $SD = 0.6\%$; COM: $M = 1.1\%$, $SD = 0.5\%$). Removing fast (<100 ms) responses resulted in the elimination of less than 4.7% of trials per participant (ASD: $M = 0.05\%$, $SD = 0.2\%$; COM: $M = 0.1\%$, $SD = 0.5\%$). RTs were again log transformed and arcsine-square-root transformation was applied to accuracy to increase normality.

Again, Bayes factors were computed with JASP (Love et al., submitted, 2015), whereas all other analyses were run with SPSS 22.0 (IBM Corp., 2013). As removing one outlier (i.e., data points more than three times the interquartile range above or below the first quartile) in the COM group for the reactive control analyses and six outliers (5 COM, 1 ASD) for the proactive control analyses did not change the pattern of results, we reported the results including these outliers.

Results

Reactive control. On reactive control (see Table 5), as expected, there was again a marked effect of congruency on both RT and accuracy: Congruent trials were associated with faster RTs ($BF_{10} > 100$) and more accurate responses ($BF_{10} > 100$) than incongruent trials. Adults with ASD showed longer RTs ($BF_{10} = 19.89$) and were more accurate ($BF_{10} = 15.89$) than adults without ASD. These longer and more accurate responses were independent of trial type (i.e., congruent/incongruent trials; RT: $BF_{10} = 1/1.73$; accuracy: $BF_{10} = 1/2.74$) and longer RTs were not affected by gender (main effect: $BF_{10} = 1/1.66$, interaction: $BF_{10} = 1/7.12$). Nevertheless, women were more accurate than men ($BF_{10} = 1.98$), and accuracy was differently influenced by gender in the two groups ($BF_{10} = 2.03$). Follow-up analyses revealed that the accuracy congruency effect (i.e., Simon effect) was similarly expressed

Table 5
Statistics of the Group Comparisons on Reactive Control (Study 2)

Factors	RTs: F	RTs: p	RTs: η_p^2	Accuracy: F	Accuracy: p	Accuracy: η_p^2
Congruency	**828.18**	**<.001**	**.75**	**272.33**	**<.001**	**.50**
Group	**8.02**	**.005**	**.03**	**7.03**	**.009**	**.03**
Gender	.42	.517	.00	**4.04**	**.046**	**.02**
Group × Gender	.67	.412	.00	1.60	.207	.01
Group × Congruency	1.62	.205	.01	.41	.524	.00
Gender × Congruency	3.14	.078	.01	**5.51**	**.020**	**.02**
Group × Gender × Congruency	.32	.575	.00	**5.56**	**.019**	**.02**

Note. RTs = reaction times. Degrees of freedom are (1, 276) for all group analyses. Significant values ($p < .05$) are indicated in bold script.

in women with and without ASD ($F(1, 102) = 1.15$, $p = .285$, $\eta_p^2 = .01$, $BF_{10} = 1/2.92$) whereas men without ASD demonstrated a larger Simon effect than men with ASD ($F(1, 172) = 6.37$, $p = .013$, $\eta_p^2 = .04$, $BF_{10} = 3.06$; see Figure 4).

In contrast to Study 1, accuracy rates of the fastest responses on incongruent trials differed between groups ($F(1, 274) = 4.10$, $p = .044$, $\eta_p^2 = .02$, $BF_{10} = 3.69$). The COM group demonstrated more fast errors, indicating stronger response capture, than the ASD group (Figure 5a–c). There was no main effect of gender ($F(1, 274) = 0.02$, $p = .904$, $\eta_p^2 = .00$, $BF_{10} = 1/7.11$) nor an interaction effect ($F(1, 274) = 2.82$, $p = .095$, $\eta_p^2 = .01$), even though the Bayes factor of this interaction effect indicates that evidence is inconclusive ($BF_{10} = 1/1.35$). The gradient of the delta slope of the slowest responses was comparable across groups ($F(1, 274) = 1.52$, $p = .219$, $\eta_p^2 = .01$, $BF_{10} = 1/5.07$), indicating similar response suppression (Figure 5d–f). Gender did not seem to influence this result (main effect: $F(1, 274) = 3.24$, $p = .073$, $\eta_p^2 = .01$, $BF_{10} = 1.23$ [i.e., is inconclusive]; interaction: $F(1, 274) = 1.63$, $p = .203$, $\eta_p^2 = .01$, $BF_{10} = 1/2.51$).

Proactive control. On proactive control, as in Study 1, responses were faster ($BF_{10} > 100$) and more accurate ($BF_{10} > 100$) when congruent trials were preceded by congruent trials rather than when preceded by incongruent trials, and when incongruent trials were preceded by incongruent trials rather than when preceded by congruent trials (see Table 6). In other words, the Simon effect was larger after congruent trials than after incongruent trials. Although this effect was again similar across groups on RTs ($BF_{10} = 1/4.85$), it was more pronounced in the COM group on accuracy ($BF_{10} = 1/1.39$; see Figure 6). Hence, albeit individuals without ASD might more strongly release control after a nonconflict situation when accuracy is considered, the Bayes factor shows that the evidence for this effect is only anecdotal. Yet, cognitive control is enhanced after a conflict situation in both groups, revealed by a reduction of the Simon effect after incongruent trials.

Role of age in reactive control. When examining the effect of age on reactive control, increasing age was associated with longer RTs ($F(1, 273) = 73.33$, $p < .001$, $\eta_p^2 = .21$, $BF_{10} > 100$), and higher accuracy rates ($F(1, 273) = 14.59$, $p < .001$, $\eta_p^2 = .05$, $BF_{10} > 100$). Whereas RTs were longer overall, independently of whether congruent or incongruent trials were presented (i.e., the RT Simon effect was not affected by age; $F(1, 273) = 0.17$, $p = .680$, $\eta_p^2 = .00$, $BF_{10} = 1/23.26$), age interacted with congruency on accuracy ($F(1, 273) = 5.11$, $p = .025$, $\eta_p^2 = .02$), although there is little evidence for (or against) this effect ($BF_{10} = 1.03$). The association between increasing age and higher accuracy rates was significant on incongruent trials ($B = .002$, $SE = .001$, $t(273) = 2.62$, $p = .009$) but not on congruent trials ($B = .000$, $SE = .001$, $t(273) = 0.92$, $p = .359$; i.e., the accuracy Simon effect became smaller with increasing age). Nevertheless, the role of age on reactive control did not differ across groups (RT: $F(1, 273) = 2.47$, $p = .117$, $\eta_p^2 = .01$, $BF_{10} = 1/9.66$; accuracy: $F(1, 273) = 1.09$, $p = .298$, $\eta_p^2 = .00$, $BF_{10} = 1/3.11$).

Although increasing age was related to a lower percentage of fast errors ($F(1, 276) = 5.04$, $p = .026$, $\beta = 0.13$, $R^2 = .02$, $BF_{10} = 1.43$), it was not when the whole model was considered ($p = .262$, $\beta = 0.08$, $BF_{10} = 1/2.03$), suggesting the effect of age to be small (Figure 7a). Furthermore, the Bayesian analysis provide little evidence for or against an age effect. However, increasing age yielded a steeper downward slope of the delta plot at longer RTs (Figure 7b; $F(1, 276) = 6.28$, $p = .013$, $\beta = -0.15$,

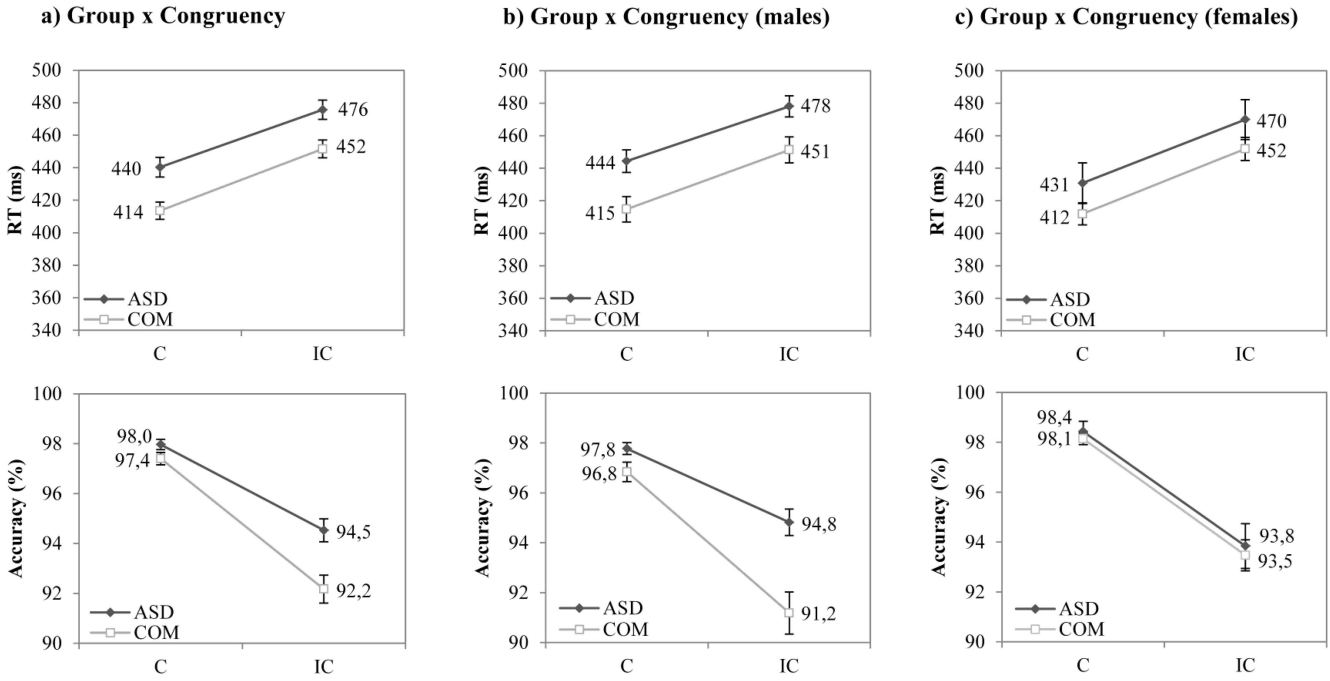

Figure 4. Mean reactions times (RTs) and accuracy rates for congruent and incongruent trials per group: (a) overall, (b) only men, and (c) only women (Study 2). *Note:* ASD = autism spectrum disorder group; COM = comparison group; C = congruent; IC = incongruent. Error bars present *SE*s.

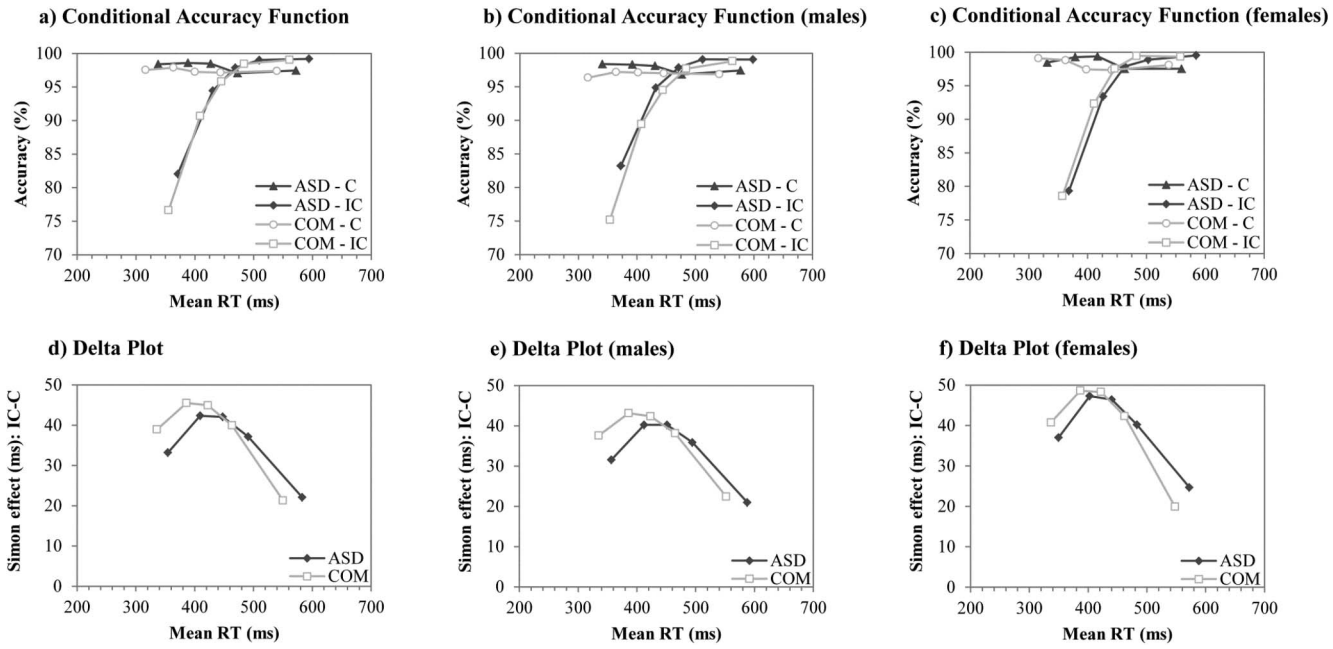

Figure 5. Conditional accuracy functions (a) overall, (b) only men, and (c) only women and delta plots (d) overall, (e) only men, and (f) only women per group (Study 2). *Note:* ASD = autism spectrum disorder group; COM = comparison group; C = congruent; IC = incongruent.

R^2 = .02, BF_{10} = 2.55), which was even more pronounced when the whole model was considered (p = .007, β = −0.20, BF_{10} = 8.01). Hence, the strength of response capture is likely to be constant across the adult life span, whereas the efficiency of response suppression was increased in older adults. Both effects did not differ across groups (respectively, $t(273) = 0.97$, p = .333, BF_{10} = 1/2.49, and $t(273) = 0.86$, p = .391, BF_{10} = 1/2.78).

Role of age in proactive control. Age also affected the efficiency of proactive control (see Figure 8). Older adults demonstrated a larger Simon effect after congruent trials than after incongruent trials compared to younger adults on RT ($F(1, 273) = 9.24$, p = .003, η_p^2 = .03, BF_{10} = 8.73), but not on accuracy ($F(1, 273) = 0.96$, p = .328, η_p^2 = .00, BF_{10} = 1/4.46). The role of age was similar in the two groups on both RT ($F(1, 273) = 2.83$, p = .094, η_p^2 = .01, BF_{10} = 1/4.15) and accuracy ($F(1, 273) = 1.07$, p = .302, η_p^2 = .00, BF_{10} = 1/2.64).

Exploratory analyses. Given the somewhat contrasting findings between Study 1 and 2, we explored whether a sub-

Table 6
Statistics of the Group Comparisons on Proactive Control (Study 2)

Factors	RTs			Accuracy		
	F	p	η_p^2	F	p	η_p^2
Congruency	**838.85**	**<.001**	**.75**	**258.92**	**<.001**	**.49**
Trial sequence	**41.75**	**<.001**	**.13**	**26.76**	**<.001**	**.09**
Group	**8.10**	**.005**	**.03**	**6.21**	**.013**	**.02**
Gender	.43	.513	.00	**4.61**	**.033**	**.02**
Group × Gender	.73	.394	.00	2.48	.116	.01
Congruency × Trial sequence	**1178.13**	**<.001**	**.81**	**499.23**	**<.001**	**.65**
Group × Congruency	1.57	.211	.01	.53	.469	.00
Gender × Congruency	3.32	.069	.01	2.60	.108	.01
Group × Trial sequence	.37	.546	.00	.05	.821	.00
Gender × Trial sequence	.01	.918	.00	3.44	.065	.01
Group × Gender × Congruency	.43	.510	.00	**4.16**	**.042**	**.02**
Group × Gender × Trial sequence	.34	.561	.00	.23	.632	.00
Group × Congruency × Trial sequence	1.23	.268	.00	**4.51**	**.035**	**.02**
Gender × Congruency × Trial sequence	.78	.377	.00	.06	.814	.00
Group × Gender × Congruency × Trial sequence	1.13	.289	.00	.53	.469	.00

Note. RTs = reaction times. Degrees of freedom are (1, 274) for all analyses. Significant values ($p < .05$) are indicated in bold script.

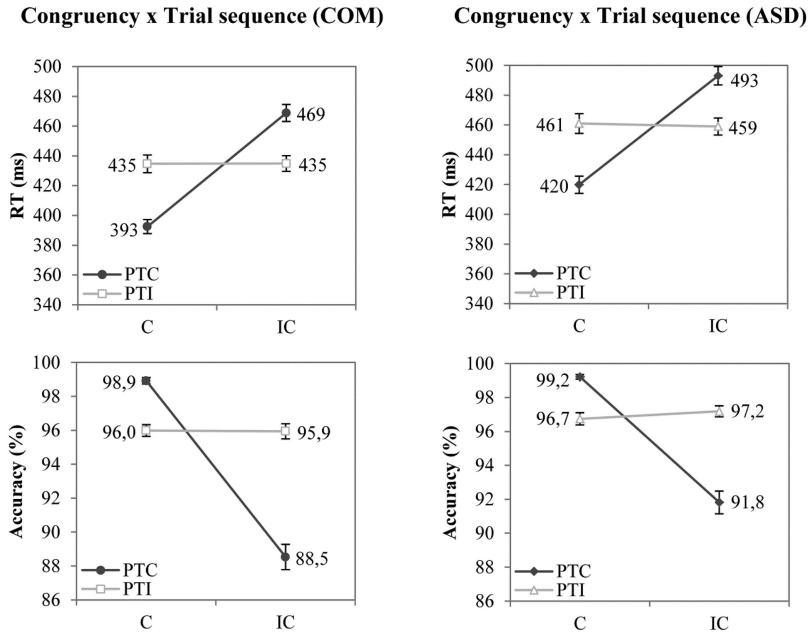

Figure 6. The congruency sequence effect per group (Study 2). *Note:* ASD = autism spectrum disorder group; COM = comparison group; C = congruent; IC = incongruent; PTC = previous trial congruent; PTI = previous trial incongruent.

group with the same gender and age characteristics as in Study 1 would demonstrate a similar pattern as found in Study 1. Therefore, we selected only male participants between 19 and 36 years of age (ASD: $n = 22$; COM: $n = 32$) and reran all analyses. We replicated all results of Study 1. The Bayes factors were also comparable with those entailed in Study 1, ranging from $BF_{10} = 1/3.97$ (RT interaction reactive control) to $BF_{10} = 1.86$ (delta slope).

Discussion

Despite slower RTs, adults with ASD showed more accurate responses compared with age- and IQ-matched controls and were not differently affected by interference from incongruent trials. Automatic response capture was reduced in adults with ASD, whereas selective response suppression was similar across groups. Exploratory Bayesian analyses supported these frequentist results and provided substantial to strong evidence in favor of or against the group-related hypotheses. Furthermore, women were more accurate than men, but this was mainly explained by the performance of the men without ASD who showed larger interference effects than men with ASD. Women with and without ASD performed similarly. Bayesian evidential strength for these results were, however, only anecdotal.

The proactive control mechanism of detecting and adjusting responses to previous trials, which results in a reduced interference effect on RT after conflict trials (Botvinick et al., 2001; Egner, 2007; Gratton et al., 1992), was also in Study 2 similar between adults with and without ASD (Larson et al., 2012; Worsham et al.,

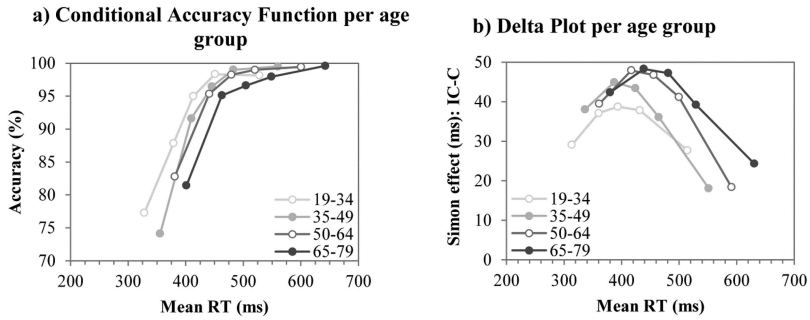

Figure 7. Exploratory (a) conditional accuracy functions for only incongruent trials and (b) delta plots per age group in years (Study 2). *Note:* C = congruent; IC = incongruent. Please note that age was split into age groups to provide a visual impression of age effects, while analyses were run with age as a continuous factor. However, participants were evenly distributed across ages: 19–34 years: $N = 70$ (25 ASD, 45 COM), 35–49 years: $N = 69$ (34 ASD, 35 COM), 50–64 years: $N = 70$ (30 ASD, 40 COM), 65–79 years: $N = 69$ (29 ASD, 40 COM).

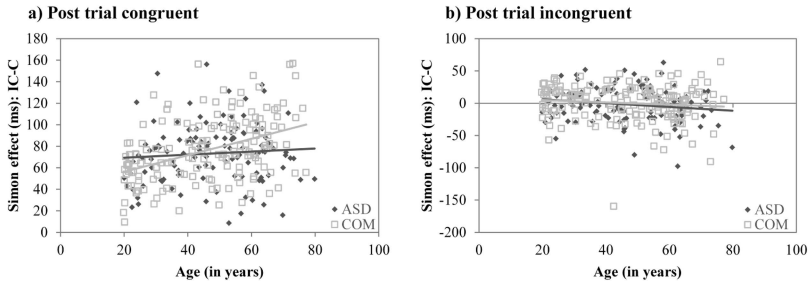

Figure 8. The (linear) effect of age plotted against the mean Simon effect for (a) post congruent trials and (b) post incongruent trials per group (the darkest line indicates the ASD group). *Note:* ASD = autism spectrum disorder group; COM = comparison group.

2015). This indicates that both groups enhanced control after incompatible trials. Even though controls were more sensitive to interference after congruent trials, suggesting that they more strongly released control when a previous trial was a nonconflict trial, exploratory Bayesian analyses indicated no group effect. Hence, this latest finding should be interpreted with caution.

Slower and more accurate responses, and reduced response capture fit well together and converge to the idea of a more cautious response strategy among adults with ASD. Although the task instructions were to respond as fast and accurate as possible, individuals with ASD reported that they preferred to be accurate rather than fast, despite several attempts of the researchers to emphasize the importance of speed. Hence, adults with ASD seem to adopt a conservative response criterion.

As ASD in older adulthood is largely under investigated (e.g., Mukaetova et al., 2011) and older adults may show larger interference effects than younger adults in the general population (e.g., see Proctor et al., 2005 for an overview), we examined the effect of age on reactive and proactive interference control. Increasing age was associated with slower and more accurate responses, but we did not find evidence for a larger RT Simon effect in older adults. In regular Simon tasks, age-related differences have previously been reported to be absent (see Proctor, Miles, & Baroni, 2011; Proctor et al., 2005; Vu & Proctor, 2008), although in tasks that used spatial features for both the relevant and irrelevant stimulus dimensions, age changes have been reported (Castel, Balota, Hutchison, Logan, & Yap, 2007; Kawai et al., 2012; Pick & Proctor, 1999; Van der Lubbe & Verleger, 2002). This would suggest that older adults present problems suppressing irrelevant information (i.e., stimulus location) when the relevant stimulus dimension also contains spatial information, such as an arrow (Proctor et al., 2011).

Although age-related RT prolongation did not result in significantly fewer fast errors on incongruent trials, selective suppression on the slowest RTs was enhanced in older adults. These findings suggests that a more conservative approach is adopted with increasing age during reactive control. However, on proactive control, while age did not influence the RT Simon effect after incongruent trials (see also Puccioni & Vallesi, 2012; Yano, 2011), it did after congruent trials. Increasing age was related to greater interference when the congruent trial was followed by an incongruent trial. Yet, the CSE remains intact across adulthood (Puccioni & Vallesi, 2012; Yano, 2011).

General Discussion

The aim of the current studies was to investigate the temporal dynamics underlying reactive and proactive interference control processes among intellectually able adults with ASD. In the first study, we examined these processes in young adults by using a visual Simon task. In the second study, we tried to validate the findings in an independent sample and, moreover, examined to role of age.

Study 1 demonstrated that young adults with ASD present comparable interference control performance compared with young adults without ASD as measured with a Simon task. The findings of Study 1 and 2 converge, despite changing task parameters, when considering only young adults (18–36 years). Young adults with and without ASD performed similarly on reactive and proactive control, and on the underlying reactive control processes of response capture and response suppression. When considering large part of the adult life span (19–79 years) in Study 2, our results provide a partially different perspective. On reactive control, adults with ASD were slower but more accurate, and had reduced response capture but similar response suppression. On proactive control, as in Study 1, there were no differences between groups.

Partially, these findings indicate typical age-related differences, also shown by similar age-related effects among adults with and without ASD. Typical aging is associated with diminished processing speed (e.g., Salthouse, 1996) and older adults take more time in making decisions and avoiding errors, whereas younger adults decide more quickly and find making errors more acceptable (Rabbitt, 1979; Salthouse, 1979; Smith & Brewer, 1995). Indeed, older adults adjust their behavior to minimize the number of errors against the cost of speed (Starns & Ratcliff, 2010). They might also be less able to *estimate* the time or *control* the time of their responses and, therefore, provide slower responses (Rabbitt, 1979). As a similar suggestion has been proposed for individuals with ASD (Falter, Noreika, Wearden, & Bailey, 2012), it seems that there are some similarities between the behavior of individuals with ASD and typically developing older adults (see Bowler, 2007, for the aging analogy in ASD). Nevertheless, more specifically, our findings also suggest that middle-aged and older adults with ASD use a quantitatively different response strategy than young adults with ASD, reflected by longer response duration, higher accuracy rates, and fewer fast errors. Slowing of RTs has been

previously reported for individuals with ASD compared with individuals without ASD (Travers et al., 2014), but increased accuracy also suggests a shift in the balance between speed and accuracy.

The current results appear inconsistent with those entailed by a meta-analysis indicating that individuals with ASD present interference control difficulties (Geurts et al., 2014). It should be noted, however, that an increase in IQ was associated with smaller effect sizes of interference control. Given that individuals in our sample presented high IQs, this could account for absent interference control differences in the current studies. Furthermore, although in the meta-analysis no evidence for age affecting effect sizes was found, this might be because of the inclusion of only a few adult studies. The number of included adult studies may not have been sufficient to detect age-related differences. In addition, the type of task used might have affected the results. While the Simon task taps into processes related to response interference, the Eriksen flanker task also involves perceptual interference (Egner, 2007; van den Wildenberg et al., 2010). As our results suggest that response interference is not impaired among adults with ASD, the possibility that perceptual interference is affected in ASD should be evaluated. Indeed, individuals with ASD seem to demonstrate perceptual enhancement (e.g., Lever & Geurts, 2016a; Mottron, Dawson, Soulieres, Hubert, & Burack, 2006; but see van der Hallen, Evers, Brewaeys, Van den Noortgate, & Wagemans, 2015) and it has been suggested that, therefore, they get more easily distracted (Adams & Jarrold, 2012).

Several limitations should be mentioned. First, we only included individuals with a normal-to-high intelligence. Whether our results generalize to the entire autism spectrum, including those individuals with an intellectual disability, remains unknown. Second, the cross-sectional nature of our study provides initial insights into age-related differences in interference control across adulthood in ASD, but does not allow to investigate changes over time (Raz & Lindenberger, 2011). Third, the literature documents the possible role of feature priming effects in CSEs in conflict tasks such as the Eriksen flanker task. However, there is consensus in the field that these priming effects play much less of a role in CSEs in Simon tasks than in other conflict tasks (see, e.g., Stürmer et al., 2002; Wühr & Ansorge, 2005). Hence, in accordance with mainstream accounts, the present findings can be attributed to proactive control settings. Therefore, despite the suggestion of a more conservative response bias in ASD, there was an insufficient number of trials to examine speed–accuracy trade-off by means of, for example, diffusion models (Ratcliff & McKoon, 2008).

The present findings may give rise to future hypotheses and directions. Middle-aged and older adults with ASD appear to show a shift in their speed/accuracy balance toward slower and more accurate responding, with fewer (rather than more) fast response capture errors than controls. By analogy, similar findings in patients with Parkinson's disease point to an important interaction between strategic and computational aspects of interference control in accounting for cognitive impairments among patients (Wylie et al., 2010). Beyond cognitive control research, it has also been suggested that individuals with ASD need more time to solve interference during global-local visual processing (van der Hallen et al., 2015) or during language processing (Koolen, Vissers, Hendriks, Effer, & Verhoeven, 2012), which indicates that differences in information processing are not limited to interference control as measured with conflict tasks. A speed/accuracy manipulation in the Simon task may thus be instructive for examining whether middle-aged and older individuals with ASD experience increased problems with the expression and suppression of impulse response capture when they are under pressure for speed. In addition, diffusion drift modeling may help understand the nature of a more conservative response bias in ASD, although this will require a more massive number of trials. These approaches may also answer the question whether individuals with ASD control their response latency to maintain accuracy or whether they are unable to respond quickly and, therefore, have sufficient time to avoid impulsive fast errors (see Yano, 2011, for a similar discussion on aging). Put differently, is being more cautious an efficient strategy for individuals with ASD as this strategy when applied allow proficient functioning in daily life? And under which circumstances does dealing with conflict cause difficulties for individuals with ASD?

In summary, we used a cognitive framework (i.e., the dual-route model and its extension, the activation-suppression hypothesis) to investigate interference control among adults with ASD. This provided the opportunity, in contrast to previous studies, to not only examine overall measures but also underlying mechanisms involved in interference control processes. Across the adult life span, our findings do not support the idea of behaviorally impaired reactive and proactive interference control processes and indicate efficient cognitive control processes, such as automatic response capture and selective response suppression, to resolve response conflict in intellectually able adults with ASD. Given our findings, it seems premature to conclude that the application of this cognitive dual-process model leads to an explanation for the observed heterogeneity among ASD studies on interference control (Geurts et al., 2014) and further research is, therefore, warranted. However, it does suggest that the framework is useful to disentangle different processes involved in interference control and contributes to an increased understanding of interference control among individuals with ASD. Hence, we would recommend the application of theoretical frameworks for the study of cognitive control in ASD in the future.

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