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## Special Issue “Neurodevelopmental Neurodiversity”: Research Report

Prepotent response inhibition in autism: Not an inhibitory deficit?<sup>☆,☆☆</sup>

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## ABSTRACT

Research outcomes on prepotent response inhibition in neurodevelopmental conditions during adulthood seem inconsistent, especially in autism. To gain further insight in these inconsistencies, the current study investigates inhibitory performance, as well as task strategies such as adaptive behavior during inhibitory tasks in autistic adults. As Attention-Deficit/Hyperactivity Disorder (ADHD) is often co-occurring in autism and associated with differences in both inhibition and adaptation, the role of ADHD symptoms is explored. Additionally, prior research is extended to middle- and late-adulthood, and the role of cognitive aging is assessed. Hundred-and-five autistic adults and 139 non-autistic adults (age: 20–80 yrs) were compared on a Go-NoGo task. No significant group differences in inhibitory difficulties (commission errors) or adaptation (post error slowing) were observed, and both did not relate significantly to ADHD symptoms. However, when controlling for reaction time autistic individuals made significantly more inhibitory errors than non-autistic individuals, yet the effect size was modest (Cohen's  $d = .27$ ). Exploratory analyses showed that adaptation significantly related to inhibition in non-autistic individuals only, possibly hinting at altered adaptive behavior during inhibitory tasks in autistic adults. ADHD symptoms related to response variability in the autism group only. Furthermore, task strategy changed with older age in both groups, with slower and more cautious responses at older age. Taken together, although minor differences may exist, autistic and non-autistic people show largely similar patterns of inhibitory behavior throughout

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adulthood. Differences in task timing and strategy seem relevant for future longitudinal studies on cognitive aging across neurodevelopmental conditions.

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## 1. Introduction

Cognitive models of neurodevelopmental conditions can be highly valuable as they help us bridge the gap between brain and behavior (Frith, 2012). Prepotent response inhibition is one of the most widely investigated domains of cognitive functioning in autism (Lai et al., 2017) and Attention-Deficit/Hyperactivity Disorder (ADHD; Pievsky & McGrath, 2018). It is defined as the deliberate suppression of dominant, motor responses (Nigg, 2000) and considered important for goal directed behavior by rapidly stopping motor actions, even after such actions have been activated (e.g., stopping to cross the street when a car approaches; Wessel, 2018). While in both ADHD and autism inhibition is worth studying in more detail, less consensus has been reached on inhibitory difficulties in autism, especially in adulthood (Hlavatá et al., 2018; Kuiper et al., 2016). This is why the current research focusses on autistic adults. Given that ADHD and autism often co-occur (Gonzalez-Gadea et al., 2014; Rommelse et al., 2011), are both characterized by a neurodivergent cognitive style (Pellicano & Houting, 2022), and may show similarities in inhibitory behavior (Karalunas et al., 2018), we also explore the role of ADHD symptoms.

In contrast to ADHD, in autism, prepotent response inhibition is not considered a core cognitive mechanism. The evidence for difficulties with prepotent response inhibition in autistic adults is rather mixed (Christ et al., 2011; Geurts, van den Bergh, & Ruzzano, 2014; Kuiper et al., 2016), and the association between inhibition and autistic characteristics is inconsistent (Hlavatá et al., 2018). However, some argue that inhibitory difficulties could be related to the repetitive behavior (Agam et al., 2010; Mosconi et al., 2009) or social difficulties observed in autistic individuals (Uzefovsky et al., 2016). For instance, not being able to suppress certain motoric expressions may result in repetitive behavior (i.e., *stimming/flapping*) or passing over social cues when one is passionately sharing insights/experiences. When assuming that these autistic traits stem from a lack of inhibitory capacities, one might ignore other mechanistic differences that might explain such traits, e.g., the aforementioned motoric expressions might be a deliberate action to reduce stress or anxiety (Leekam et al., 2011). Rather than assuming that inhibitory difficulties are a cause of autistic traits, we would argue that inhibitory difficulties might be a consequence of other cognitive differences in autistic individuals. Clinically, such a mechanistic explanation would be relevant as this provides additional evidence for the idea that autistic traits are not merely a consequence of some cognitive “deficit”, but might have a function on its own. If so, instead of trying to modify autistic traits in clinical treatment, one might want to consider the possibility that autistic traits are helpful for individuals with a different cognitive style.

Prior research points out that behavioral and neural phenomena during prepotent response inhibition in autistic individuals are not aligned. That is, behavioral performance differences between autistic and non-autistic individuals on inhibitory tasks are often non-significant, while the autistic brain clearly seems to respond differently when confronted with such tasks (Duerden et al., 2013; Goldberg et al., 2011; Kana et al., 2007). This could suggest that autistic people differ in their approach to such tasks rather than having an inhibition problem per se. Indeed, most authors who observed a brain/behavior discrepancy argue that this is due to the recruitment of additional processes to complete the task correctly (Duerden et al., 2013; Goldberg et al., 2011; Kana et al., 2007). Behavioral differences in prepotent response inhibition observed in some studies with larger sample sizes (Uzefovsky et al., 2016; Van Eylen et al., 2015) and meta-analyses (Geurts, van den Bergh, & Ruzzano, 2014; Kuiper et al., 2016) suggest that difficulties in prepotent response inhibition in autism do occur, but that the sensitivity of the behavioral instruments is low. At the very least, these inconsistencies indicate that the robustness of the finding of difficulties in prepotent response inhibition in autism is rather poor (Hlavatá et al., 2018; Høyland et al., 2017), and that if inhibitory difficulties exist, the nature of these difficulties are poorly understood. Possibly, differences between autistic and non-autistic people in inhibitory behavior are the result of an alternative cognitive strategy.

Differences in cognitive strategies during inhibitory tasks may be found in adaptive behavior, which can be observed in the form of post-error slowing (PES)—an increase in reaction time right after erroneous responses. The exact mechanism behind PES remains unknown because no robust correlations between PES and accuracy are observed (Dutilh et al., 2012). Yet, it is thought that at least part of the process can be viewed as a micro-adaptation steering task performance (Ridderinkhof, 2002). In individuals with ADHD, PES has been extensively investigated in explaining reduced performance on prepotent response inhibition tasks (for a meta-analysis see: Balogh & Czobor, 2016). Research shows a clear reduction in PES in individuals with ADHD compared to individuals without ADHD during inhibitory and attention tasks (Balogh & Czobor, 2016). By contrast, in autistic- and non-autistic children, differences in PES during an inhibitory task were non-significant (Goldberg et al., 2011). Yet, when using an attention task (visual oddball task) in autistic young adults significantly reduced PES was observed (Bogte et al., 2007). Other studies even observed post-error *speeding* in autistic children and young adults (Sokhadze et al., 2010, 2019). This suggests not only the absence of adaptation, but also the presence of anti-adaptive behavior during an attention task in autistic individuals. Therefore, insufficient adaptation might be a likely mechanism for reduced inhibitory abilities in autism, as

well as a potentially shared cognitive characteristic of ADHD and autism.<sup>1</sup>

Given that previous research on prepotent response inhibition in autism have predominantly focused on children (Kuiper et al., 2016), it is important to consider the potential influence of age. Our research not only extends previous studies by including autistic adults, but also specifically investigates the effects of age on these processes. Meta-analytic evidence indicates modest declines in performance with age on Stop-Signal tasks, but not on Go-NoGo tasks (Maldonado et al., 2020). Others observed no effects on prepotent response inhibition, and even increased adaptation compared to younger adult (Staub et al., 2014). By exploring age-related effects in a wide age range (20–79 years) we can obtain richer insights in the linearity of these effects, as well as their interactions with autism.

In the current research, we use a Go-NoGo task to compare inhibitory performance (commission errors) and adaptation (PES) between a group of autistic adults and non-autistic adults. We expect worse performance of autistic individuals than non-autistic individuals on prepotent response inhibition and adaptation. Furthermore, we expect that adaptation relates less strongly to inhibitory performance in autistic individuals than in non-autistic individuals. We also explore group differences in other features of task strategy, namely response speed (MRT) and response variability (SD RT). Furthermore, we expect worse, slower, and more variable responses at older age independent of group, but no age-related differentiation in adaptive behavior. We also do not expect age-related differences for autistic and non-autistic adults given, that the majority of findings in other cognitive domains suggest a parallel development (e.g., Torenvliet et al., 2021; Tse et al., 2019). Exploratorily, we investigate the role of ADHD-symptoms in both autistic- and non-autistic individuals. We expect that ADHD symptoms relate to prepotent response inhibition and adaptation in both individuals with and without autism.

## 2. Methods

We report how we determined our sample size, all data exclusions, all inclusion/exclusion criteria, whether inclusion/exclusion criteria were established prior to data analysis, all manipulations, and all measures in the study. Our experimental tasks are available at: <https://doi.org/10.17605/OSF.IO/3HRZG>.

### 2.1. Participants

Participants ( $n = 249$ ) were between 20 and 79 years and were recruited via several clinical institutions across the Netherlands, (social) media advertisements of (autism) networks, and the social network of the researchers. The

exclusion criteria were 1) a history of neurological disorders (e.g., epilepsy, stroke), schizophrenia or having experienced more than one psychotic episode; 2) Wechsler Adult Intelligence Scale III (WAIS-III; Wechsler, 1997)  $IQ < 80$  or Mini Mental State Examination (MMSE; Folstein et al., 1975; Kok & Verhey, 2002)  $< 26$ ; and 3) current alcohol or drugs dependency as indicated by the Mini International Neuropsychiatric Interview (MINI; Sheehan et al., 1997). For the autism group, two additional exclusion criteria were 1) no registered diagnosis of autism according to the DSM-IV (American Psychiatric Association, 2013), and 2) Autism Diagnostic Observation Schedule (ADOS; Bastiaansen et al., 2011; Lord et al., 2000)  $< 7$  and the Autism Quotient (AQ; Baron-Cohen et al., 2001)  $< 26$ . For the no-autism group, three additional criteria were: 1) a history of autism, or ADHD; 2) close family-members (i.e., parents, children, and siblings) with autism or schizophrenia; and 3)  $AQ > 32$ . While we did not explicitly inquire about race/ethnicity, it is worth noting that our participants were mostly White.

### 2.2. Measures

Details of our descriptive measures can be found in S1. In short, we administered the ADOS, MMSE, an abbreviated WAIS-III, AQ, and ADHD-Rating Scale (ADHD-SR; Kooij et al., 2005). Legal copyright restrictions prevent public archiving of these measures which can be obtained from the copyright holders in the cited references.

#### 2.2.1. Three-stimuli Go-NoGo task (Go-NoGo task [in-house development author AGL])

In each trial, one of two Go-stimuli or a No-Go stimulus was presented after which participants either pressed the space bar as fast as possible (Go) or withheld their response (NoGo). The two Go-stimuli had a probability of .50 and .25 for the common-Go and rare-Go respectively. The NoGo stimulus had a probability of .25. Stimuli were three red-colored Disney cartoons, and were counter-balanced between participants (Goofy/Donald—common-Go or NoGo, Minnie -rare-Go). Trial sequences were fixed. Stimulus presentation time was 400 ms in the practice trials and 250 ms in the experimental trials. The response window was 1400 ms in practice trials and 1250 ms. in experimental trials. Inter-trial intervals varied between 350 and 650 ms. to increase task difficulty. Responses during the ISI were not recognized. Eighty practice trials, and two blocks of 160 experimental trials were presented. See S2, Figure S1, left panel.

Trials faster than 100 ms were removed. Outlier trials (2.5 median absolute differences [MAD]  $\pm$  individual mean; Leys et al., 2013) were also removed. Dependent measures were mean reaction time (MRT), standard deviation reaction time (SD RT), % omission errors, % commission errors, and PES (as adaptation measure). MRT and SD RT were computed using correct trials. Omission and commission errors were computed as the percentages incorrect of the total number of trials after outlier removal. For PES we used the difference between the mean reaction time from trials directly following a correct trial (RTcc) and the mean reaction time from trials directly following an error (RTec), following standard practice

<sup>1</sup> The study was initially designed to also conclude on possible differences in context monitoring: scanning the environment for contextual cues to start or stop an action. However, due to a large ceiling effect in our task, we could not inspect the role of context monitoring in inhibitory behavior.



in AD(H)D-research (Balogh & Czobor, 2016). We used trials after NoGo trials (i.e., commission errors) only, as they are the key errors in the task.

The Oddball task was designed to be orthogonal to the GoNoGo task, and is described in S3, and Figure S1. We do not report on this task here, as, although we piloted the task extensively and in both autistic and non-autistic adults, on actual performance we detected a ceiling effect. Almost 80% of participants obtained a perfect score. All pre-registered analyses on this task are in S4, Tables S1, S2 and S3, and are therefore, not included here.

### 2.3. Procedure

After written informed consent was obtained, participants completed a screening procedure, including two sets of questionnaires, and a 2–2.5-hour interview session including the ADOS (autism only). Next, a 2.5 h cognitive session was planned, including Go-NoGo and Oddball task and Oddball tasks (for details see: Geurts et al., 2021). Both tasks were administered in counterbalanced order. Travel expenses were compensated; most participants also received additional compensation (max. €20). The study was approved by the ethical review board of the Department of Psychology of the University of Amsterdam (2011-PN-1952).

### 2.4. Analyses

Our analysis plan consisted of three parts.

1. We reported the following outcome measures MRT, SD RT, % omission errors, % commission errors, and mean PES.
2. Age-effects were analyzed using separate multiple regressions for each outcome variable. Group and the interaction between group and (centered) age were added in a second step. Non-linear age effects were explored by adding  $age^2$  as a predictor. Alpha was adjusted using Bonferroni-Holm corrections.
3. To test whether adaptation predicted prepotent response inhibition we used hierarchical regression analyses. Pre-potent response inhibition (% commission errors) was the outcome variable. Centered adaptation (PES) was the predictors in the first step. In a second step, group and the interaction between the predictors and group were added. In a third step, we added centered age (and  $age^2$ , in case  $age^2$  provided better model fit than age in step 2), the interaction between the predictors and  $age/age^2$ , and a three-way interaction with  $group*age/age^2$ .

We used Akaike Information Criteria (AIC) and Bayesian Information Criteria (BIC) for model comparisons. Lower AIC/BIC indicated better model fit. When contradictory, we followed AIC, which allows more leniency for extra predictors compared to BIC (Burnham & Anderson, 2004).

Next to our planned analyses (pre-registered at: <https://osf.io/ayn7e/>), exploratory correlation analyses between the ADHD-SR subscales (attention, and hyperactivity/impulsivity)

and the main outcome measures on the Go-NoGo task were performed to investigate whether ADHD symptoms related to specific aspects of prepotent response inhibition.

## 3. Results

All analyses, outcomes and conclusions can be verified in our R-markdown, through: <https://osf.io/jg45w/files/osfstorage>. Anonymized data are archived at: <https://doi.org/10.17605/OSF.IO/GW3QT>.

### 3.1. Sample characteristics

Two-hundred-forty-four participants were included in the analyses. On average, 14 outlier trials per individual were removed (range: 1–41 trials). Fast responses (<100 ms) occurred rarely, and on average less than one fast trial per individual was removed in both groups (range: 0–13 trials) The number of outliers and fast responses did not differ significantly between groups ( $p$ -values >.55).

Groups ( $n_{\text{autism}} = 105$ ,  $n_{\text{no-autism}} = 139$ ) did not significantly differ in age, IQ, or MMSE-score (see Table 1). However, the no-autism group had a significantly higher number of women. Therefore, we added sensitivity analyses with sex. Average AQ scores were significantly higher in the autism group than in the no-autism group. Some autistic adults scored below the ADOS cut-off (yet above the AQ cut-off,  $n_{\text{ADOS-}} = 26$ ). Therefore, sensitivity analyses with the ADOS scores on the main outcome variables were performed. Average ADHD-SR subscale scores also significantly differed between groups, with higher scores in the autism group than in the no-autism group. Therefore, correlations of the ADHD-SR subscales with the main outcome measures were estimated for both groups separately. Six autistic adults reported a current ADHD diagnosis, two non-autistic adults were excluded for having a current ADHD diagnosis.

### 3.2. Groups differences

As shown in Table 2, group wise comparisons (independent  $t$ -tests) on any of the outcome measures revealed no significant group differences. Most importantly, we observed no significant difference in inhibitory performance or adaptation.

Group differences between non-autistic adults and those individuals who scored above the ADOS cut-off ( $n_{\text{ADOS+}} = 79$ ), are provided in S5 Table S4, and were similar to the total sample. However, as effect sizes on some outcome measures clearly increased (i.e., from .11 to .25 on inhibitory performance), we also directly compared groups scoring above/below the ADOS cut-off, see S5 Table S5. Autistic adults scoring above the cut-off (ADOS + group) made significantly more omission and commission errors than autistic adults scoring below the cut-off (ADOS- group). Reaction times were also faster in the ADOS + group compared to the ADOS- group, though not significantly. Although this provides evidence for the idea that the extent of autistic traits in autistic people

**Table 1 – Participant characteristics: group means, standard deviations, and statistics for age, IQ, MMSE, AQ and ADOS (Autism only).**

Measure	Group		Statistics	Cohens <i>d</i> [CI]
	Autism ( <i>n</i> = 105)	No-autism ( <i>n</i> = 139)		
Sex (M/W, M %)	74/31, 70.5%	82/57, 59.0%	$\chi^2 = 6.353, p = .012$	
	Mean (SD); Range	Mean (SD); Range	t-value (p-value)	
Age	47.3 (15.1); 20-79	45.8 (16.5); 20-77	.76 (.45)	.09 [-.16, .34]
IQ	115.5 (16.8); 84-155	112.0 (16.4); 80-155	1.59 (.11)	.20 [-.05, .45]
MMSE	29.1 (1.0); 26-30	29.2 (1.0); 26-30	-.38 (.71)	-.11 [-.36, .14]
AQ	33.4 (8.1); 8-49	12.2 (5.3); 2-26	23.07 (<.01)	3.10 [2.73, 3.47]
ADOS	8.6 (3.2); 1-19	–	–	–
ADHD-SR A	3.0 (2.4); 0-9	.7 (1.2); 0-7	8.65 (<.01)	1.21 [.94–1.15]
ADHD-SR HI	3.2 (2.3); 0-9	1.2 (1.6); 0-7	7.78 (<.01)	1.01 [.74–1.28]

Note. M, men, W, women; SD, standard deviation; IQ, estimated intelligence quotient; MMSE, Mini Mental State Examination; AQ, Autism-spectrum Quotient; ADOS, Autism Diagnostic Observation Schedule; ADHD-SR A, Attention Deficit Hyperactivity Disorder Rating Scale Attention subscale; ADHD-SR HI, ADHD-SR hyperactivity/impulsivity subscale.

**Table 2 – Group means and effect sizes for the Go-NoGo task.**

Group	Autism (N=)	No-autism (N=)	Statistics	Cohens <i>d</i> [CI]
	Mean (SD)	Mean (SD)	t-value ( <i>p</i> )	
MRT (msec.)	398.3 (62.2)	386.8 (67.9)	1.37 (.17)	.18 [-.07, .43]
SD RT (msec.)	70.2 (20.3)	65.5 (19.0)	1.86 (.06)	.24 [-.01, .49]
PES (msec.)	5.1 (39.1)	8.5 (42.0)	-.66 (.51)	-.08 [-.33, .17]
% omissions <sup>a</sup>	1.5 (2.7)	1.3 (3.1)	.3 (.85)	.07 [-.18, .32]
% commissions <sup>b</sup>	28.1 (17.5)	26.2 (17.1)	.85 (.40)	.11 [-.14, .36]

Note. MRT, mean reaction time (in milliseconds); SD RT, standard deviation reaction time (in milliseconds); PES, post-error slowing (in milliseconds); % omissions, percentage of omission errors; % commissions, percentage of commission errors.

<sup>a</sup> Score ranges of the percentage of omission errors were autism: 0–14%, no-autism: 0–25%.

<sup>b</sup> Score ranges of the percentage of commission errors were autism: 3–84%, no-autism: 0–81%.

relate to inhibitory performance, correlations between inhibitory performance and the AQ total score were practically zero, see S5 Table S6.

Nearly all correlations of the ADHD-SR subscales (inattention, hyperactivity/impulsivity) with the outcome measures were not significant, see S6, Table S7. The only significant correlation that was observed was between the hyperactivity/impulsivity scale and response variability in the autism group ( $r(103) = .20, p = .048$ ). Correlations were not corrected for multiple comparisons as we regard these analyses as exploratory.

Since the task was designed to elicit quick, automatic responses, we explored whether a speed-accuracy trade-off (slower responses to avoid errors) in one or both groups (autism or no-autism) had introduced noise to our data, possibly explaining the null-findings in inhibitory performance. We indeed observed a significant negative relation between MRT and commission errors (autism:  $r(104) = -.709, p < .01$ , no autism:  $r(138) = -.679, p < .01$ ), indicating that slower MRT was associated with more commission errors in both groups. To this end, we conducted an ANCOVA analysis to investigate whether commission errors differed between groups when controlling for MRT. These analyses revealed a large effect for MRT (partial  $\eta^2 = .48$ ), and that the percentage of commission errors differed significantly between groups ( $F(1,241) = 6.04, p = .01, d [95\%CI] = .32 [.07, .57]$ ) when partialing out the covariance with MRT. A higher adjusted percentage of

commission errors was observed in autistic individuals (29.27%) than non-autistic individuals (25.28%). No significant interaction effect between group and MRT was observed<sup>2</sup> in accordance with the assumption of homogeneous regression slopes ( $\beta = .32, t(243) = 1.10, p = .27$ ). This suggests that autistic adults as compared to non-autistic adults did not show an altered speed-accuracy trade-off, but autistic adults do make significantly more inhibitory errors when MRT was included to reduce the unexplained variance (nuisance) in our data.

Taken together, no major group differences were observed. However, when controlling for MRT, the autism group had a higher percentage of commission errors than the no-autism group, but not an altered speed-accuracy trade off. Relations with the ADHD-SR subscales were small and mostly non-significant.

### 3.3. Age effects, and age-related differences

We used multiple regression analysis to test whether age, group, sex and/or their interactions significantly predicted

<sup>2</sup> Note. Even though MRT did not differ significantly between groups, MRT still removed nuisance variance in the dependent variable (commission errors), therefore gaining explanatory power (see for example: Miller & Chapman, 2001). Please also note that PES and MRT were not related ( $r = .01$  in autism and  $r = .05$  in no autism).

**Table 3 – Age effects on dependent variables in the Go-NoGo.**

		Outcome				
		MRT	SD RT	PES	% omission	% commission
Age	$\beta$ -value [CI]	.43 [-.04, .90]	.25 [.10, .40]	.24 [-.08, .55]	-.12 [-.15, -.10]	-.25 [-.38, -.12]
	t-value	7.48**	3.99**	3.76**	-1.92	-3.99**
	$R^2$ (adj)	.18	.06	.05	.01	.06
	AIC	2688.50	2135.42	2482.49	1216.27	2072.16
	BIC	2699.00	2145.91	2492.97	1226.77	2082.65

Note. MRT, mean reaction time (in milliseconds); SD RT, standard deviation reaction time (in milliseconds); PES, post-error slowing (in milliseconds); % omissions, percentage of omission errors; % commissions, percentage of commission errors; CI, confidence interval (95%); adj, adjusted; AIC, Akaike Information Criterion; BIC, Bayesian Information Criterion; \* =  $p_{Holm} < .05$ , \*\* =  $p_{Holm} < .01$ ;  $p_{Holm}$ , Holm corrected  $p$ -values which were calculated by dividing the observed  $p$ -value by an adjustment factor (AF;  $AF_{max} = 5$ ,  $AF_{min} = 1$  based on (adjusted)  $R^2$  for each outcome.

performance. Older age was significantly associated with fewer commission errors and slower as well as more variable response speed (see Table 3). Adding group and sex did not improve the models, see S7 Tables S8 and S9. So, generally, the age effects were not different between autistic and non-autistic adults or between men and women. However, a significant interaction between age, group, and sex was observed, with older autistic women showing higher rates of PES than others (see S7 Figure S2 [ $\beta = -.40$ ,  $t(243) = -2.85$ ,  $p_{Holm} = .01$ , adj.  $R^2 = .08$ , AIC = 2484.27, BIC = 2515.71]). No, or only marginal improvements of model fit when adding age,<sup>2</sup> indicating an absence of quadratic age effects, see Table S10.

Taken together, no differential age effects were observed except for higher rates of PES of older autistic women.

### 3.4. Predicting prepotent response inhibition using adaptation

We created a model containing PES, group, and age to explain the percentage of commission errors (prepotent response inhibition). Older age was associated with a significantly lower percentage of commission errors, but PES and group showed no such association, see Table 4.

We explored whether these null findings could have been due to an unreliability in the PES measure, because the initial analysis also included participants whose PES was only based

on a few trials (i.e., participants who only made a few mistakes). Therefore, we reran the analyses in a subsample only containing persons whose PES was based on at least eight trials; corresponding to the first quartile (Q1) and higher of the PES trials in both groups (see S8, Figure S3). In this subsample ( $n_{autism} = 78$ ,  $n_{no-autism} = 102$ ), the overall fit of the model was slightly better ( $R^2$  full sample = .05 vs.  $R^2$  subsample = .08). A significant interaction between PES and group on the percentage of commission errors was observed, see Table 4, and S8 Figure S4. Longer PES (i.e., greater adaptation) was significantly associated with higher commission error rates (i.e., inhibition failures) in non-autistic individuals, but not in the autistic individuals. This indicates that inhibitory performance is less related to adaptation in the autistic individuals compared to non-autistic individuals. However, these results should be interpreted with caution, given the low explained variance of the model.

Taken together, prepotent response inhibition was explained by adaptive behavior in non-autistic individuals and not in autistic individuals, but only when excluding participants with few PES trials.

## 4. Discussion

The current study aimed to gain insight in prepotent response inhibition in neurodevelopmental conditions, with a specific

**Table 4 – Prepotent response inhibition with post-error slowing, group, and age as predictors in the full sample, and subsample.**

	Full sample ( $n = 244$ )		Subsample ( $n = 180$ )	
	$\beta$ -value [CI]	t-value	$\beta$ -value [CI]	t-value
PES	-.08 [-.17, .01]	-.86	-.13 [-.22, -.04]	-1.12
Group	-.06 [-4.38, 4.24]	-.99	-.05 [-4.67, 4.56]	.73
Age	-.33 [-.55, -.11]	-3.18**	-.34 [-.58, -.09]	2.69*
PES x Group	.13 [.02, .24]	1.34	.28 [.16, .40]	2.40*
PES x Age	-.05 [-.05, -.04]	-.53	-.07 [-.08, -.07]	-.68
Group x Age	.11 [-.17, .39]	1.06	.13 [-.18, .45]	1.09
PES x Group x Age	.01 [.01, .02]	.14	-.01 [-.02, -.01]	-.12
$R^2$ (adj)	.05		.08	
AIC	2070.88		1503.42	
BIC	2102.32		1532.16	

Note. PES, Post-error slowing; adj, adjusted; AIC, Akaike Information Criterion; BIC, Bayesian Information Criterion. The subsample consisted of participants whose PES was based on at least eight trials ( $n_{autism} = 78$ ,  $n_{no-autism} = 102$ ). \* =  $p < .05$  \*\* =  $p < .01$ .

focus on autistic adults. Results showed no significant differences between prepotent response inhibition in autistic and non-autistic individuals. Yet, when controlling for response speed, autistic individuals made more inhibitory errors. Difficulties in prepotent response inhibition were related to higher rates of post-error adaptation in non-autistic adults only, providing preliminary evidence for the idea that adaptation plays a different role in prepotent response inhibition in autism. However, autistic and non-autistic adults did not show different rates of adaptation or other features of task strategy (response speed, response variability). Inhibitory performance and adaptation were also not significantly related to ADHD-symptoms in both adults with- and without autism. At older age, people showed a more careful (i.e., slower) and more accurate task strategy. This pattern was not specific to autism, indicating parallel age-related differences between autistic and non-autistic adults in prepotent response inhibition.

The current results provide an accurate reflection of the inconsistent findings on the role of inhibitory difficulties in autism—hinting both at *none*, and *some* inhibitory difficulties in autism. That is, initially no inhibitory difficulties in autism were observed; yet, when controlling for response speed, autistic adults made more inhibitory errors than non-autistic adults. Based on these results, one could argue that autistic adults indeed show inhibitory difficulties. However, one could also argue that overall, the effect sizes were small, and mainly those autistic individuals who responded fastest to the task exhibited inhibitory errors. Hypothetically, autistic people might experience difficulties with inhibitory behavior especially when (feeling) rushed. This would fit with real-life experiences of autistic individuals that certain difficulties increase with time constraints, such as during a higher paced conversation with multiple people instead of one-on-one.

Contrary to what is described in the ADHD literature, we did not observe differences between autistic and non-autistic adults in trial-to-trial adaptive behavior (i.e., PES), overall response speed, or response variability. Therefore, cognitive strategies during inhibitory tasks may be more similar in autistic- and non-autistic adults than previously expected. Consequently, the observed brain-behavior discrepancies during inhibitory tasks (Duerden et al., 2013; Goldberg et al., 2011; Kana et al., 2007) might not be due to the recruitment of additional processes, but the (lack of) sensitivity in our cognitive tasks (Kuiper et al., 2016; Raymaekers et al., 2004; Wessel, 2018). This is in line with larger studies and meta-analyses concluding on inhibitory performance in autism that describe inhibitory differences in autism (Uzefovsky et al., 2016; Van Eylen et al., 2015; Geurts, van den Bergh, & Ruzzano, 2014; Kuiper, et al., 2016). Even though our study was high-powered, our task was generally fast-paced, and trial length fell within the recommended window (1–2s.; Wessel, 2018), inter-individual differences in response speed seemed to have clouded initial inhibitory differences between autistic and non-autistic adults. This further substantiates that inhibitory differences in autism may exist, but are of small extend and unlikely to be a cause of autistic behavior.

Notably, individuals with more observable autistic traits showed more inhibitory difficulties. Exploratory analyses showed that those scoring above the ADOS cut-off produced

about 50% more inhibitory errors than those who scoring below the ADOS cut-off, yet the association between self-reported autistic traits (AQ scores) was practically zero. Thus, it seems that mostly observable autistic traits, not self-reported autistic traits, are associated with higher inhibitory difficulties. Because ADOS-AQ discrepancies are thought to reflect autistic camouflaging (van der Putten et al., 2023), one could hypothesize that this dissociation indicates a role for inhibitory behavior in autistic camouflaging. This is consistent to what is reported on the link between (self-reported) executive functioning and camouflaging (for an overview see: Ai et al., 2022). It also nuances the idea that autistic traits are directly related to inhibitory difficulties. Based on these results, it seems that autistic behavior is not a consequence of an inhibitory deficit, but inhibitory skills might moderate the ability to mask or not to mask one's autistic traits. However, given the known constraints of the discrepancy method to operationalize camouflaging (Fombonne, 2020), additional research on the role of inhibitory performance and camouflaging is vital. Possibly, autistic camouflaging can provide further insight in the inconsistent findings on inhibitory difficulties in autism thus far.

We also observed that adaptation is associated with more inhibitory difficulties in non-autistic adults, but not in autistic adults. This could imply that individuals with autism show less effective adaptive behavior as compared to non-autistic adults. Yet, based on the ADHD literature (Balogh & Czobor, 2016) in which lower PES and higher inhibitory difficulties are observed in individuals with ADHD compared to those without ADHD, one might expect an inverse association between inhibitory difficulties and adaptive behavior in non-autistic adults. It is currently unclear whether non-autistic adults gradually pick up the pace after errors resulting in new errors and thereafter more slowing down (i.e., an adaptive interpretation of PES; Pfister & Foerster, 2022) or perhaps that extensive slowing after an error might make participants more prone to making new errors (i.e., a maladaptive interpretation of PES; Ullsperger et al., 2014). Although these findings complicate the current conclusions on the observed differences between autistic and non-autistic people, they highlight the need to test explicitly whether the assumed association between adaptive and inhibitory behavior is actually different in neurodevelopmental conditions. To date, the association between adaptation and inhibitory difficulties are rarely explicitly tested in both ADHD and autism samples.

Both adaptive behavior and inhibitory performance did not relate to ADHD symptoms in autistic and non-autistic adults. Thus, ADHD symptoms do not seem to play a prominent role in explaining inhibitory performance differences between individuals with autism, and vice versa, inhibitory behavior does not seem to be a (key) mechanism across neurodevelopmental conditions. However, we observed that unique features of timing may indicate differences across neurodevelopmental conditions, extending previous findings in children with ADHD/autism (Karalunas et al., 2018; Raymaekers et al., 2007). First, differences in post error slowing seemed less consistent in autism compared to what is previously observed in ADHD (Balogh & Czobor, 2016). Second, those autistic adults who responded fastest, showed largest difficulties inhibiting their response, whereas in ADHD, largest differences are observed in response variability, not



response speed (Karalunas et al., 2013; Pievsky & McGrath, 2018). The current results seem to confirm such a pattern, as a significant correlation between hyperactivity/impulsivity symptoms and response variability was observed in the autism group. Hence, a key difference between (adult) autism and ADHD might be that individuals with autism need more time, whereas individuals with ADHD show larger variability over time. As the current sample included only a few individuals with an ADHD diagnosis, cross-condition research is needed to further investigate this pattern in neurodevelopmental conditions in adulthood.

Finally, a slower, more cautious response style was observed in older adults with- and without autism, providing further evidence for parallel (similar) age-related effects in autism throughout adulthood (e.g., Davids et al., 2016; Lever & Geurts, 2016; Torenvliet et al., 2023; Tse et al., 2019). As a more cautious response style is an often-observed characteristic of autistic children and young autistic adults when performing experimental tasks, (e.g., Pirrone et al., 2020) it could be that autistic adults diverge from this strategy when growing older and/or that the strategy of the non-autistic adults becomes more cautious, and thus more similar to the “autistic” strategy (“the aging analogy” by Bowler et al., 2014). This would be consistent with research on other cognitive domains showing that initial differences diminish with age (“protective aging” [Lever et al., 2015; Zivrali Yazar et al., 2020]). However, as the age-related patterns in the current study indicate parallel age-related changes from 20 to 80 years, it is unknown if, when, and how such developmental changes occur. Therefore, additional longitudinal research is needed to estimate how these age-related changes in task strategy develop over time.

#### 4.1. Limitations and future directions

A few limitations to the current study should be addressed. Firstly, the Oddball task seemed too easy, as most participants (~79%) made no errors. Although the task was piloted, this unexpected ceiling effect hindered a conclusion on the role of context monitoring in prepotent response inhibition. This left the researchers with a dilemma on whether or not to include their pre-registered analyses in the manuscript. Open science requires researchers to be entirely transparent on the followed procedures (e.g., Munafò et al., 2017), yet the outcomes hindered the interpretation of the data. To retain transparency, we decided to place the results of the pre-registered analyses in the supplementary materials.

Second, the current study focused on prepotent response inhibition, whilst different features of inhibitory behavior, like resistance to distractor interference, exist. Meta-analytic evidence suggest that the effects are larger for prepotent response inhibition (Geurts, van den Bergh, & Ruzzano, 2014), yet studies directly comparing the two types of inhibitory behavior show larger effects for resistance to distractor interference in autism (Christ et al., 2007, 2011). Therefore, extending the current findings to other forms of inhibitory behavior in autistic (and ADHD) adults would be an interesting avenue to pursue.

Third, our age-related results were limited to those who could actively participate and without neurological disorder. Older participants (>70) were difficult to recruit, perhaps because the diagnosis is rarely recognized in older adults and/or because autistic adults might be more at risk for cognitive decline and decreased longevity (e.g., Croen et al., 2015; Hand et al., 2019). Therefore, the current results might not generalize well to the eldest (with autism) and/or those with severe cognitive decline.

## 5. Conclusion

Taken together, autistic adults showed modest inhibitory difficulties, albeit only when controlling for reaction time. Similar rates of post error slowing, response speed, and response variability across the autistic and non-autistic group substantiate that the task was performed in similar fashion, although adaptive behavior less related to inhibitory performance in autism. ADHD symptoms did not relate to inhibitory performance or adaptation in neither autistic nor non-autistic adults. Both autistic and non-autistic adults employed a more cautious response style at older age. Given the extent of the observed effects, similar task strategy, the absent relation with ADHD symptoms, and parallel age-related effects, it is unlikely that differences in inhibitory performance are an explanatory feature of autistic behavior throughout adulthood or an underlying cognitive mechanism across neurodevelopmental conditions.

## CRedit author statement

**Carolien Torenvliet:** Data curation, Formal analyses, Methodology Project administration, Visualization, Writing—original draft, Writing—review & editing. **Annabeth P. Groenman:** Supervision, Methodology, Writing—original draft, Writing—review & editing. **Anne Geeke Lever:** Conceptualization, Data curation, Methodology, Project Administration, Writing—review & editing. **K. Richard Ridderinkhof:** Conceptualization, Methodology, Writing—review & editing. **Hilde M. Geurts:** Conceptualization, Methodology, Funding Acquisition, Supervision, Writing—original draft, Writing—review & editing.

## Resources

Preregistration: Prepotent Response Inhibition in Autism – Not an Inhibition Deficit? <https://osf.io/ayn7e/>

Supplementary materials and R-markdown: <https://osf.io/jg45w/files/osfstorage>

Data: Torenvliet, C., Lever, A. G., Groenman, A., Ridderinkhof, K. R., & Geurts, H. (2023, June). Data prepotent response inhibition in autism. <https://doi.org/10.17605/OSF.IO/GW3QT>

Materials: Lever, A. G., & Torenvliet, C. (2023, June). Materials Prepotent Response Inhibition in Autism. <https://doi.org/10.17605/OSF.IO/3HRZG>

## Declaration of competing interest

We have no conflicts of interest to disclose.

## Open Practices

The study in this article earned Open Data and Preregistered badges for transparent practices. The data for this study and preregistration are available at: <https://doi.org/10.17605/OSF.IO/GW3QT> and <https://osf.io/ayn7e/> respectively.

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## Supplementary materials

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.cortex.2023.05.013>.

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