The Role of Interstimulus Interval and "Stimulus-Type" in Prepotent Response Inhibition Abilities in People with ASD: A Quantitative and Qualitative Review

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The Role of Interstimulus Interval and "Stimulus-Type" in Prepotent Response Inhibition Abilities in People with ASD: A Quantitative and Qualitative Review

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Autism spectrum disorders (ASD) are associated with prepotent response inhibition difficulties. However, the large variation between studies suggests that understudied factors, such as interstimulus interval (ISI) and "stimulus-type" (both hypothesized proxies of stressors influencing arousal), might influence the inhibitory abilities of people with ASD. Using meta-analysis, we tested whether differences in prepotent response inhibition between people with and without ASD was influenced by ISI. There was not enough variation in "stimulus-type" between the studies to include it as a moderator. Thirty-seven studies met inclusion criteria, with a combined sample size of 950 people with ASD and 966 typically developing controls. Additionally, a qualitative review including studies comparing a neutral and an arousing condition in one experiment was performed to examine whether fast ISI or specific arousing stimuli directly influence prepotent response inhibition. The meta-analysis indicated that ISI was not a relevant moderator. The qualitative review showed that ISI and "stimulus-type" had the same effect for both groups. Although all studies regarding ISI indicated that fast ISI worsened performance, different types of stimuli had either a positive or a negative influence. This could suggest that distinctive stimuli might affect arousal differently. While we replicated the inhibition difficulties in people with ASD (g = .51), our results do not show strong ASD-specific effects of ISI or "stimulus-type" on inhibition. Nonetheless, ISI and "stimulus-type" do seem to influence performance. Future research focusing on potential underlying factors (e.g., baseline physiological arousal) is needed to examine why this is the case. ***Autism Res*** *2016, 9: 1124–1141.* © 2016 International Society for Autism Research, Wiley Periodicals, Inc.

Keywords: autism spectrum disorders; autism; prepotent response inhibition; interstimulus interval; cognitive control; meta-analysis

Introduction

Autism spectrum disorders (ASD) are neurodevelopmental disorders, characterized by social (communicational) difficulties and repetitive behavior (APA, 2013). While both have been hypothesized to be related to inhibitory control difficulties, research has mainly focused on the relationship between repetitive behavior and inhibitory control difficulties (i.e., Langen et al., 2012; Lopez, Lincoln, Ozonoff, & Lai, 2005; Mosconi et al., 2009; Joseph & Tager-Flusberg, 2004). Most research on inhibition and ASD focuses on two types of inhibition, namely on prepotent response inhibition and interference control. Prepotent response inhibition is the ability to inhibit a prepotent response or an ongoing response (a.k.a. active inhibition; Aron, 2011). Interference control is the ability to ignore or suppress irrelevant stimuli (Friedman & Miyake, 2004). In the last two decades, studies on inhibitory control in people with ASD have presented us with conflicting results. However, two recent meta-analyses (Geurts, van den Bergh & Ruzzano, 2014) show that across the lifespan, ASD is indeed associated with medium sized problems in prepotent response inhibition (effect size [ES] 0.55) and interference control (ES 0.31). Yet, after controlling for age and intelligence, a significantly large amount of heterogeneity among the studies was still observed. This suggests that there are unknown factors that influence inhibitory control in people with ASD. In this article, we will explore whether interstimulus interval (ISI) and "stimulus-type" are important factors to take into account when studying prepotent response inhibition in people with ASD.

Besides variations in methodological factors such as age and intelligence quotient (IQ), ISI and "stimulus-type" are two elements of experimental tasks that can vary between studies. For instance, studies all using a Go/NoGo task have reported different ISI's, like an ISI

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of 1150 milliseconds (ms; e.g., Oerlemans et al., 2013), an ISI of 2500 ms (e.g., Lee et al., 2009) or an ISI of 4000 ms (e.g., Ambrosino et al., 2014). Additionally, there are various definitions of ISI (e.g., Metin, Roeyers, Wiersema, van der Meere, & Sonuga-Barke, 2012; Pereira et al., 2014). In this article, ISI is defined as the time between the onset of the first stimulus until the onset of the second stimulus (Metin et al., 2012). Variations in "stimulus-type" include the use of pictures with emotional expressions (e.g., Geurts et al., 2009; Yerys, Kenworthy, Jankowski, Strang, & Wallace, 2013) or the use of familiar and unfamiliar faces (e.g., Pankert, Pankert, Herpertz-Dahlmann, Konrad, & Kohls, 2014), besides the usual arrows, figures and letters (e.g., Sinzig, Burning, Morsch, & Lehmkuhl, 2008; Solomon, Ozonoff, Ursu, Ravizza, & Cummings 2009). The use of emotional stimuli, for example, could lead to reduced performance of people with ASD, as ASD is associated with emotion processing deficits (e.g., Cassidy, Mitchell, Chapman, & Ropar, 2015; Philip et al., 2010). These differences in ISI and "stimulus-type" could be part of the unknown factors that influence inhibitory control in people with ASD (Geurts et al., 2014).

Several researchers suggest that the variations in ISI or "stimulus-type" have different effects on inhibitory control in people with neurodevelopmental disorders (e.g., ASD; ADHD) compared to typically developing individuals based on multiple theoretical motivations (e.g., Geurts et al., 2009; Pankert et al., 2014; Raymaekers, van der Meere, & Roeyers 2004, 2006; Raymaekers, Antrop, van der Meere, Wiersema, & Roeyers, 2007; Sergeant, 2000, 2005; Yerys et al., 2013). One of the most reported theoretical motivations is the cognitive-energetic model of Sanders (1983; e.g., Geurts et al., 2009; Metin et al., 2012; Raymaekers et al., 2004; Sergeant, 2000, 2005). This model suggests that external stimuli, like fast ISI and "arousing" stimuli, could influence physiological arousal levels and negatively influence performance. The model of Sanders (1983) proposes that energetical supply mechanisms (e.g., arousal) together with computational stages (e.g., encoding, searching and decision making) determine information processing efficiency. To prevent a decrease in performance, subjects need to inhibit arousal when they are over-aroused as a consequence of, for example, fast ISI or an "arousing" stimulus. Also, subjects need to increase arousal, when they are under-aroused due to "slow" ISI or a neutral "boring" stimulus, to prevent a decrease in performance. If these energetic supply mechanisms like arousal are not effectively regulated, the person can become over- or under-aroused, which would in turn negatively influence performance (Sanders, 1983). Over-arousal is suggested to lead to faster, inaccurate responses, which in terms of prepotent response inhibition would mean making more commission errors. Under-arousal is suggested to lead to slower responses. Arousal can be defined as a state of being excited or activated. This can either be a physiological or a perceived state (Jeong & Biocca, 2012). Perceived arousal is a subjective state of arousal and is often measured using a questionnaire (e.g., self-assessment manikin, Lang, Bradley, & Cuthbert, 2005; Hirvikoski et al., 2015). Physiological arousal is related to the activation of the nervous system including the central and autonomic nervous systems (e.g., Gomot & Wicker, 2012; Jeong & Biocca, 2012; Thayer & Friedman, 2002; Porges, 2001, 2007; 2013). This is often measured through physiological variables like heart rate variability (HRV), skin conductance, heart rate or brain activity using electroencephalography or magnetic resonance imaging (e.g., Porges et al., 2001; 2007; 2013; Smeekens, Didden, & Verhoeven, 2015; Mathersul, McDonald, & Rushby, 2013b). According to the model of Sanders (1983), the relationship between arousal and performance, including inhibitory control, is visualized as having the shape of an inverted U-curve. The inverted U-curve suggests there is an optimal level of arousal which corresponds with optimal performance. Under- and over-arousal levels would lead to a decrease in performance (Sanders, 1983).

Several studies support the cognitive-energetic model of Sanders (1983). A recent meta-analysis (Metin et al., 2012) examined the influence of different ISIs on prepotent response inhibition in people with attention deficit-hyperactivity disorder (ADHD). ADHD, like ASD, is a neurodevelopmental disorder (APA, 2013) and suggested to be associated with arousal regulation difficulties (van der Meere, Börger & Wiersema, 2010). The meta-analysis shows that people with ADHD make more commission errors than people without ADHD when ISI is fast ($<$2000 ms; Metin et al., 2012). The reaction times of people with ADHD are slower and more variable when ISI is slow ($\geq$6000 ms; Metin et al., 2012). This suggests that both slow and fast ISI decreases performance in people with ADHD. The inverted U-curve hypothesis has also been tested in typically developing individuals. For example, a study by Krypotos Jahfari, van Ast, Kindt, & Forstmann (2011) showed that a stressor (i.e., highly arousing negative pictures of the International Affective Picture System, IAPS, Lang et al., 2005) caused a significant decrease in performance on an emotional stop signal task in people who had high physiological baseline arousal levels compared to people with lower baseline physiological arousal (Krypotos et al., 2011). This study seems to suggest that performance might depend on a subject's baseline physiological arousal level measured before the task. Taken together, there is some behavioral evidence for the model of Sanders (1983), especially within the ADHD literature (e.g., Metin et al., 2012; Sergeant, 2000, 2005).

Following the model of Sanders (1983), fast ISI or an "arousing" stimulus might lead to a decrease in performance in people with arousal regulation deficiencies. Such deficiencies have been reported in the ASD population. Recent reviews on cardiac autonomic regulation (a.k.a. arousal) in people with ASD suggests that ASD is associated with atypical baseline arousal levels as well as abnormal physiological reactions to a task (Benevidis & Lane, 2015; Klusek, Roberts & Losh, 2015). Although the possible effects of ISI on physiological arousal in people with ASD are still unknown, there is increasing evidence that "stimulus-type" influences physiological arousal in people with ASD (e.g., Klusek et al., 2015; Mathersul, McDonald & Rushby, 2013a). It is suggested that adults do not show an increase in skin conductance and "evoked cardiac deceleration" in response to IAPS pictures whereas controls do show this increase (Mathersul et al., 2013a). Moreover, children with ASD seem to have a higher heart rate (HR) in response to cognitive tasks compared to children without ASD (Klusek et al., 2015). In response to social performance tasks (e.g., public speaking tasks), people with ASD show atypical HR compared with controls as the controls showed an increase in HR in response to the task while people with ASD did not (for review see Klusek et al., 2015). Combining the evidence for arousal regulation difficulties in ASD with the model of Sanders (1983), the heterogeneity found between the studies on prepotent response inhibition in people with ASD in the meta-analysis of Geurts et al. (2014) could be influenced by factors such as presentation rate of stimuli or the type of stimulus used within a task.

The aim of this study is to examine whether it is important to take ISI and "stimulus-type" into account when studying prepotent response inhibition in people with ASD. To our knowledge, no study has been done to date on interference control in people with ASD that manipulated ISI. Combined with the previous finding of Geurts et al. (2014) showing that prepotent response inhibition had the largest ES, leads us to focus solely on prepotent response inhibition and not on interference control as well. We will present both a qualitative and a quantitative review on prepotent response inhibition. The quantitative review will be conducted by means of a meta-analysis and focuses on the effect of arousal on prepotent response inhibition between groups: do ISI and "stimulus-type" explain some of the differences in performance on a prepotent response inhibition task between an ASD and a TD group? The qualitative review focuses on whether ISI and "stimulus-type" influence performance on an inhibitory control task within groups: does a high-arousal condition influence people with, or without, ASD differently than a low-arousal condition? Our hypothesis is that the answer to both questions will be confirmatory.

Methods

Literature search

Pubmed and PsychInfo were used for the literature search. The literature search was done up until July 2015. Search terms included variations of an ASD term (Autism, ASD, Asperger, autistic, pervasive developmental disorder, pervasive developmental disorders-not otherwise specified (PDD-NOS), high functioning autism, HFA) combined with variations of a prepotent response inhibition term (e.g., Inhibition, Response Inhibition, Inhibitory control, active inhibition, cognitive control, Stop task, Go/NoGo task) and variations of an arousal term (i.e., arousal, physiological arousal, cardiac, cardiac autonomic functioning, autonomic*, sympathetic nervous system, parasympathetic nervous system, vagal tone, heart rate (HR), heart rate variability, HRV, HR, skin conductance (SCL), SCL, cardiac vagal control, respiratory sinus arrhythmia, RSA). Search terms were put together using an "AND" and "OR" operator.

Inclusion criteria

To be included in the meta-analysis, articles needed to have the following characteristics: (1) ASD population needs to be studied; (2) ASD participants need to have a clinical diagnosis of ASD (DSM-III; DSM-IV; DSM5; ICD-10; defined by clinical diagnoses, interviews, questionnaires or observation schedules); (3) a typically developing (TD) control group needs to be included; (4) articles should be written in English and published in a peer-reviewed journal. If the inhibition task data is published in two separate articles, the article with the largest sample size will be included; (5) a Go/NoGo, Continuous Performance (CPT) or Stop Signal computerized task is used to indicate prepotent response inhibition. We included only computerized prepotent response inhibition tasks as one of our major goals was to examine whether variations in ISI influence inhibition in people with ASD. Computerized tasks measure ISI precisely and accurately.

To be included in the qualitative review in which we examined the influence of ISI and "stimulus-type" on inhibitory control within a group (ASD or TD), studies needed to have the same characteristics as described for the meta-analysis, except for having a TD group. Additionally, the studies also needed to have two conditions within one prepotent response inhibition task: a neutral condition and an arousing condition.

Data extraction

The articles were screened and evaluated based on inclusion criteria independently by the first two authors (see Fig. 1). If an article was included by one author and not by the other, the third author made the final decision. From each included study, the following data

were extracted: (1) authors and year of publication; (2) population studied (e.g., ASD, TD); (3) sample size per group; (4) mean age per group; (5) mean IQ per group; (6) type of task used (e.g., CPT, Go/NoGo); (7) ISI; (8) type of stimulus used (e.g., neutral or arousing); (9) mean and standard deviation of the dependent measure per task, per group.

Authors were contacted if some of the data was not reported in the article. As studies using similar tasks did not always report the same dependent measure and several studies reported multiple dependent outcome measures, we selected the outcome measure that best reflected prepotent response inhibition (e.g., the stop signal reaction time [SSRT] for the stop signal task) or the measure that was most commonly reported (e.g., commission errors in a Go/NoGo task). See Table 1 for an overview of the characteristics of each included study.

The main purpose of the current meta-analysis is to determine whether ISI or "stimulus-type" explain some of the heterogeneity found in a recent meta-analysis on prepotent response inhibition (Geurts et al., 2014). ISI is the time between the onset of the first stimulus and the onset of the second stimulus (Metin et al., 2012). ISI was calculated based on the information given in the articles. In cases where the ISI depended on the reaction time of participants (e.g., next trial started at the button press of the participant or in case of no response started after a maximum of 3000), we used the maximum time interval between trials (in the example, we would use 3000 ms). If no information regarding ISI was reported, we contacted the authors. As it has been suggested that an ISI of below 2000 ms would lead to more commission errors (e.g., Metin et al., 2012; Raymaekers et al., 2004, 2007). We used this cutoff to analyze the moderator ISI as a dichotomous variable (ISI fast vs. ISI slow). However, we also included the moderator ISI as a continuous moderator to examine whether ISI has an effect on inhibitory control when all the variation in the data is included.

The moderator "stimulus-type" was categorized into "arousing" and "neutral." "Arousing" stimuli were defined as stimuli that are assumed to heighten arousal (e.g., emotions, faces, acoustic noise). "Neutral" stimuli were the more "standard" stimuli, like arrows, crosses and figures.

Analysis

For each study, an ES Hedges'g (Hedges & Olkin, 1985) was calculated (see Table 1). Hedges'g is an unbiased version of the standardized mean difference (*d*). It removes a small positive bias of *d* and it takes the possible difference in standard deviations between groups into account (Hedges & Olkin, 1985). Hedges'g is calculated using the means, standard deviations and sample sizes of each group. Several studies did not report all of these data (Geurts & deWit, 2014; Kana, Keller, Minshew, & Just, 2007; Raymaekers et al., 2004, 2006, 2007; Sebanz, Knoblich, Stumpf, & Prinz, 2005). Hedges'g could be calculated from *t*-statistic (Langen et al., 2012; Raymaekers et al., 2004) and *F*-statistic (Geurts & deWit, 2014) using the formulas of Lipsey & Wilson (2001) for four of these studies. Some studies included multiple prepotent response inhibition tasks. In that case, only the task with the largest ES was included in the analyses. We did this to be in line with the meta-analyses in Geurts, van den Bergh & Ruzzano (2014), even though this might result in an overestimation of the true ES. In case of multiple conditions (e.g., fast vs. slow ISI) in a single study (e.g., Geurts et al., 2009), the ES of the "arousal condition" was included. This was done because we are interested in the effect of this arousal condition on inhibition and because the majority of the studies only has a neutral condition. ES (Hedges'g) can be interpreted as small ($g = 0.20$), medium ($g = 0.50$) or large ($g = 0.80$). A positive ES indicates a better performance of the TD group compared to the ASD group, whereas a negative ES indicates a better performance of the ASD group compared with the TD group.

For the analyses, we used the Metafor package for R (Viechtbauer, 2010). We conducted a random effects meta-analysis to account for the variation within and between studies. Heterogeneity was tested using *Q* tests and "the I^2 statistic". I^2 indicates the percentage of true heterogeneity (due to between-study variability) in the set of ESs (Thompson & Higgins, 2002). A significant *Q*-test would imply heterogeneity between studies that is not explained by prepotent response inhibition (Huizenga, Vissers & Dolan, 2011). To examine the moderating effect of two study characteristics (ISI and "type of stimuli"), mixed effects analyses were performed. Funnel plots and a regression test for funnel plot asymmetry (Egger, Davey, Schneider, & Minder, 1997) were used to assess for publication biases. If asymmetry was implied then the trim and fill method (Duval and Tweedie, 2000a) was performed. Forest plots and Cook's distance were used to inspect the data for potentially influential studies.

Results

The literature search yielded 41 studies on prepotent response inhibition (from now on referred to as inhibition) in people with ASD compared to TD people (see Table 1). Four of these 41 studies did not report enough data (nor did we receive data from the authors) to calculate an ES, and were therefore excluded from the

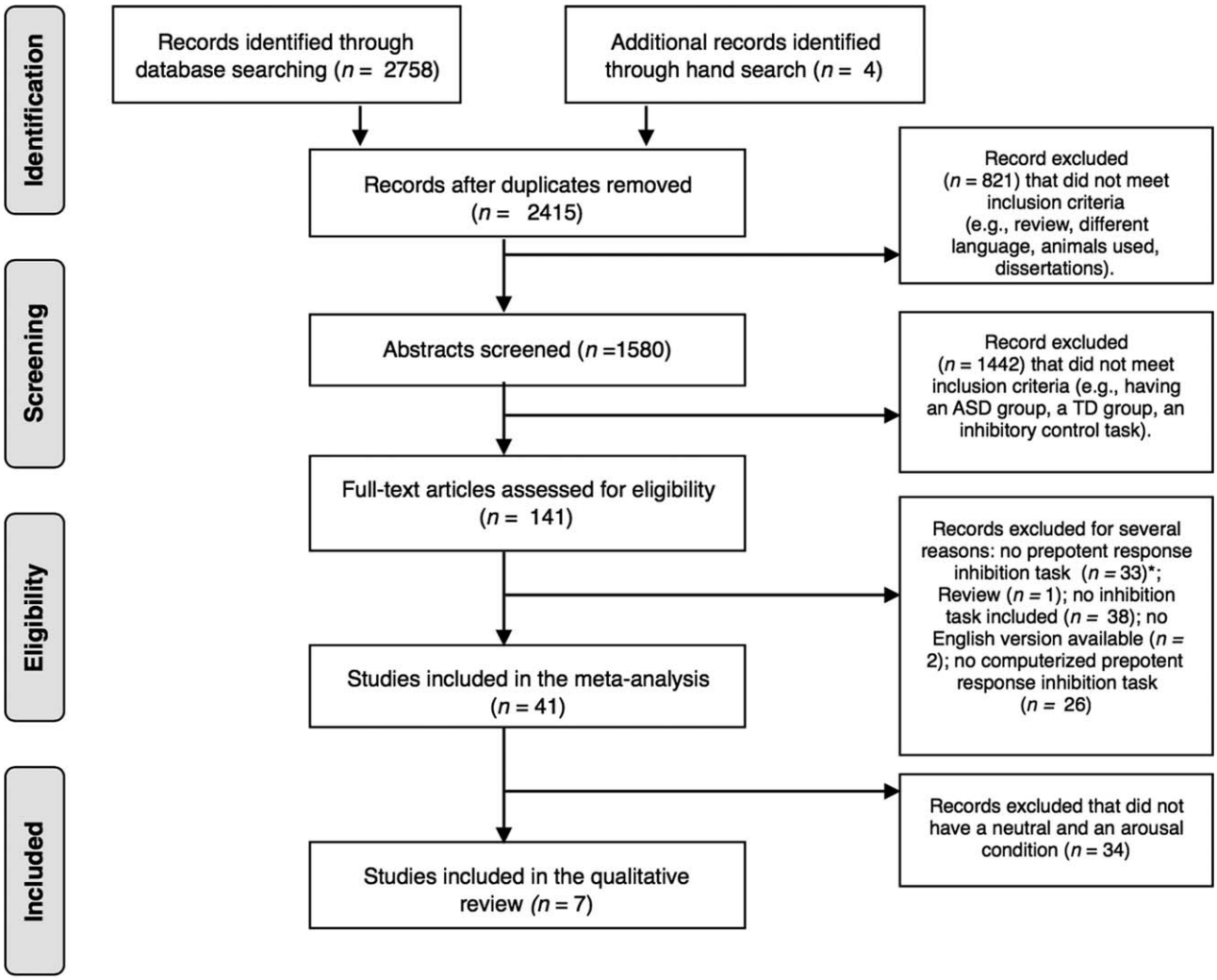

Figure 1. Flowchart of the identification and inclusion or exclusion of studies.

analysis (Kana et al., 2007; Raymaekers et al., 2006, 2007 & Sebanz et al., 2005). The meta-analysis is done with data from 37 inhibition studies, including a total of 950 people with ASD and 966 people without ASD.

Regarding the moderator "ISI," two studies (Chan, Cheung, Han, Sze, & Leung, 2009 & Kilincaslan et al., 2010) did not report enough data to calculate ISI and were excluded from that specific moderator analysis. From the remaining 35 studies, 19 studies were classified as "fast ISI" (<2000ms; mean = 1365.21, SD = 325.62) and 16 as "slow ISI" (≥2000ms; mean = 3021.88, SD = 1983.6).

Regarding the moderator "stimulus-type," five studies (Duerden et al., 2013; Geurts et al., 2009; Ozonoff et al., 1997; Pankert et al., 2014 & Yerys et al., 2013) were categorized as "arousing" and the other 32 studies as "neutral." There were not enough studies that fell into the "arousing" category to include the factor "stimulus-type" as a moderator in the meta-analysis. The five "arousing" studies were, therefore, only included in the qualitative analysis.

Replication Geurts et al. *(2014)*

To determine whether we replicated the findings of the previous meta-analysis (Geurts et al., 2014), we examined the overall effect as well as the influence of age and IQ on inhibitory control. We included an additional 15 studies to the sample size (n= 23) of Geurts et al. (2014). One of the 23 studies included by Geurts et al. (2014) was not included in our meta-analysis as this study did not meet our inclusion criteria of using a computerized task (i.e., Bishop & Norbury, 2005). The overall result of the random effects analysis of

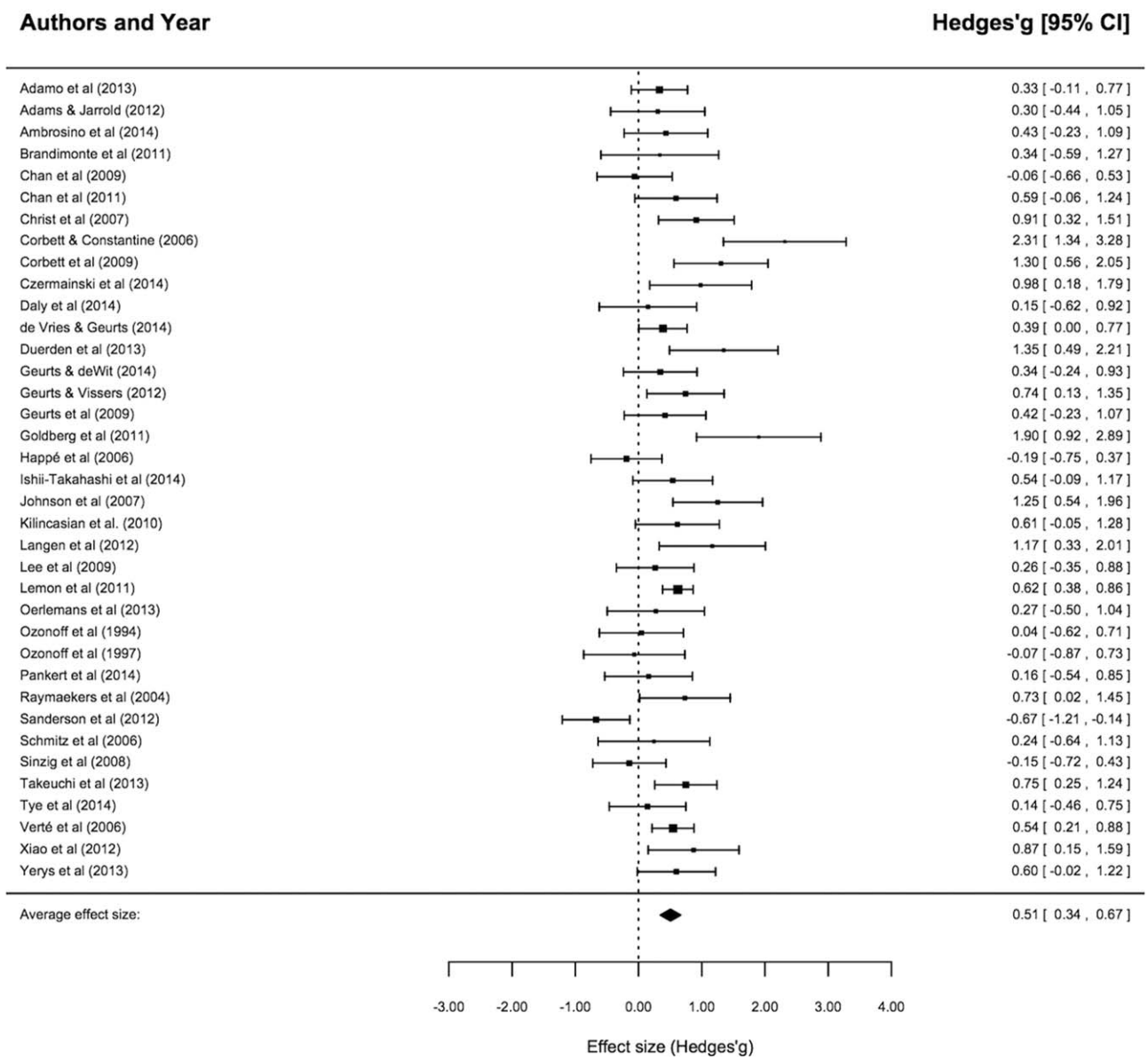

Figure 2. Forest plot of standardized mean difference (Hedges'g) and 95% confidence interval for each study included in the prepotent response inhibition meta-analysis. A positive effect sized indicates a better performance on the inhibition task by the TD group. A negative ESs indicates a better performance by the ASD group.

inhibitory control, showed a similar medium summarized ES ($g = 0.51$, $SE = 0.08$, $P < 0.0001$). Again, heterogeneity between studies was found to be significant, $t^2 = 0.14$, $Q(36) = 85.77$, $P < 0.0001$, $I^2 = 61.2\%$. The forest plot (Fig. 2) showed no potential outliers. Cook's distance was below 0.2 for every study, indicating none of the studies were highly influential. The funnel plot (Fig. 3) did not seem to show a publication bias. A regression test for funnel plot asymmetry did indicate a publication bias ($P = 0.03$). However, a trim and fill analysis did not suggest any missing studies (estimated missing studies: 0; $g = 0.51$; $P < 0.0001$; heterogeneity: $Q = 85.77$, $P < 0.0001$; $I^2 = 61.2\%$). A leave-one-out analysis confirmed that there were no highly influential studies. The effect remained medium and had the same level of significance, regardless of which study was excluded (Hedges'g ranged from 0.47 to 0.53, all $P < 0.001$). The moderator analysis showed that age did not have a significant effect ($QM = 0.12$, $df = 1$, $P = 0.73$) and the heterogeneity remained significant ($QE = 85.4$, $df = 35$, $P < 0.0001$; $I^2 = 62.3\%$). Two studies (Geurts & Vissers, 2012; Schmitz et al., 2006) were

Table 1. Characteristics of the Included Studies and Tasks

Study by	Subjects M/F	Age range/M (SD)	IQM (M/SD)	Group Assignment ASD	Task *(stimulus)*	ISI (ms)	Type of stimulus	Measure	ES
Adamo et al., 2014	ASD 42/4 ADHD 39/7 TD 19/17	7-11.9/ 10 (1) 7-11.9/ 10 (1) 7-11.9/ 10 (1)	109 (17) 106 (12) 112 (14)	Q: P SI: ADOS, ADI-R NSCA: clinical diagnosis CLAS: DSM-IV-TR	Go/NoGo *(SART/ numbers)*	1450	Neutral	Commission errors	.33
Adams & Jarrold, 2012[a]	ASD 12/3 LD 7/8 TD 4/11	12.6-17.4/14.5 (20.8) 12.6-15.9/14.2 (12.6) 8.6-11.9/ 9.1 (12.4)	25.9 (9.9) 24.9 (5.9) 24.5 (3.8)	Q: P SI: - NSCA: clinical diagnosis CLAS: -	Stop signal *(figures)*	4000	Neutral	Commission errors *(signal 3)*	.30
Ambrosino et al., 2014	ASD 19/0 TD 19/0	9.0-12.8/11.5 (1.2) 9.1-14.2/11.1 (1.6)	112.2 (15.3) 120.2 (15.8)	Q:P SI: ADI-R NSCA: clinical diagnosis CLAS: -	Go/NoGo *(figures)*	4000	Neutral	% correct no-go trials	.43
Brandimonte et al., 2011	ASD 10 TD 10	6-12/ 8.3 (2.4) 6-12/ 8.3 (1.9)	87 (7.6) 89 (5.9)	Q: - SI: CARS NSCA: clinical diagnosis CLAS: DSM-IV-TR	Go/NoGo *(figures)*	10000	Neutral	% correct trials	.34
Chan et al., 2009	ASD 14/2 TD 28/10	6-14/ 10.5 (1.7) 6-14/ 9.3 (2.2)	96.8 (18.7) 114.7 (16.6)	Q: - SI: ADOS, CARS NSCA: clinical diagnosis CLAS: DSM-IV	CPT (II) *(letters)*	NA	Neutral	Commission errors	−.06
Chan et al., 2011	ASD 19/1 TD 19/1	7-14/ 10.8 (2.1) 7-14/ 9.8 (1.9)	101.4 (16.8) 110.7 (17.8)	Q: - SI: ADOS, CARS NSCA: clinical diagnosis CLAS: DSM-IV	Go/NoGo *(figure)*	1500	Neutral	Commission errors	.59
Christ et al., 2007	ASD 16/2 TD siblings 12/11 TD 11/14	6-12/ 8.2 (1.6) 6-15/10.2 (2.1) 7-18/11.3 (3.4)	88.4 (16.3) 116.7 (16.5) 107.7 (10.6)	Q: - SI: - NSCA: clinical diagnosis CLAS: DSM-IV	Go/NoGo *(figure)*	2000	Neutral	Commission errors	.91
Corbett & Constantine 2006	ASD 13/2 ADHD 11/4 TD 10/5	7-12/ 10 (2) 7-12/ 9.7 (1.8) 7-12/ 9.6 (1.9)	97.1 (17.5) 106.8 (14.7) 117.4 (15.2)	Q: - SI: ADI-R, ADOS NSCA: clinical diagnosis CLAS: DSM-IV	CPT *(numbers)*	1500	Neutral	Visual response Control Quotient	2.31
Corbett, Constantine, Hendren, Rocke, & Ozonoff, 2009	ASD 17/1 ADHD 12/6 TD 12/6	9.4 (1.9) 9.4 (1.9) 9.6 (1.8)	94.2 (17.8) 105.2 (12.8) 112.2 (14.8)	Q: - SI: ADI-R, ADOS, DISC NSCA: clinical diagnosis CLAS: DSM-IV	CPT *(numbers)*	1500	Neutral	Visual response Control Quotient	1.30
Czermainski et al 2014[a]	ASD 9/2 TD 17/2	11.7 (1.9) 11.4 (1.8)	30.18 (3.81) 31.36 (4.75)	Q: P SI: NSCA: clinical diagnosis CLAS: DSM-IV-TR	Go/NoGo *(numbers)*	1000	Neutral	Errors of commission	.98
Daly et al., 2014	ASD 14/- TD 14/-	31 (13) 31 (11)	115 (13) 123 (20)	Q: - SI: ADI-R, ADOS NSCA: clinical diagnosis CLAS: ICD-10	Go/NoGo *(arrows)*	2300	Neutral	Probability of inhibition	.15

Table 1. Continued

Study by	Subjects M/F	Age range/M (SD)	IQM (M/SD)	Group Assignment ASD	Task *(stimulus)*	ISI (ms)	Type of stimulus	Measure	ES
Duerden et al., 2013[d]	ASD 13 TD 15	20-32/ 25.9 (3.7) 20.8-43.4/ 29 (6.9)	111.89 (13.71) 114.32 (14.8)	Q: SI: ADOS-G, ADI-R NSCA: CLAS: DSM-IV	Go/NoGo (*faces*)	2000	Arousing	Error rate NoGo trial (*faces*)	1.35
Geurts et al., 2009[d]	ASD 16/2 TD 19/3	8-13/ 10.3 (1.6) 8-13/ 10.3 (1.4)	108 (19) 103.2 (24.1)	Q: P, T SI: - NSCA: clinical diagnosis CLAS: DSM-IV-TR	Go/NoGo *(faces)*	2000 *(fast)* 6000 *(slow)*	Arousing	% errors *(fast)*	.42
Geurts & Vissers, 2012	ASD 18/5 TD 18/5	53-83/ 63.6 (7.5) 53-83/ 63.7 (8.1)	109.5 (10.3) 109.8 (7.9)	Q: S SI: - NSCA: clinical diagnosis CLAS: DSM-IV	SART *(numbers)*	1000	Neutral	Commission errors	.74
Geurts & de Wit, 2014c	ASD 20/4 TD 17/7	8-12/ 10.5 (1.1) 8-12/ 10.6 (0.7)	108.5 (17.4) 103.6 (13.9)	Q: P SI: - NSCA: clinical diagnosis CLAS: DSM-IV-TR	Go/NoGo *(figures)*	2000	Neutral	Commission errors	.34
Goldberg et al., 2011	ASD 8/3 TD 12/3	10.4 (1.6) 10.5 (1.2)	104.6 (15.6) 106.3 (13.5)	Q: - SI: ADI-R ADOS-G NSCA: clinical diagnosis CLAS: DSM-IV-R	Go/NoGo *(figures)*	1800	Neutral	% Commission errors	1.90
Happé, Booth, Charlton, & Hughes, 2006	ASD 32/0 ADHD 30/0 TD 32/0	8-16/ 10.9 (2.4) 8-16/ 11.6 (1.7) 8-16/ 11.2 (2)	99.7 (18.7) 99.1 (17.7) 106.8 (13.4)	Q: - SI: - NSCA: clinical diagnosis CLAS: DSM-IV	Go/NoGo *(figures)*	1600	Neutral	% Commission errors	-.19
Ishii-Takahashi et al., 2014	ASD 8/13 ADHD 11/3 TD 13//8	30.8 (7.2) 30.6 (7.4) 28.8 (5.5)	105.1 (14.6) 102.6 (16.6) 109.0 (5.6)	Q: P SI: ADI-R, ADOS NSCA: clinical diagnosis CLAS: DSM-IV	Stop signal task *(figure)*	1200	Neutral	SSRT	.54
Johnson et al., 2007	ASD 20/1 ADHD 20/3 TD 15/3	12.2 (2.4) 10.5 (2.4) 11.1 (1.9)	97.3 (12.3) 98.7 (14.6) 107.7 (11.6)	Q: P SI: ADI-R, ADOS-G NSCA: clinical diagnosis CLAS: -	SART *(numbers)*	1439	Neutral	% Commission errors (Random SART)	1.25
Kana et al., 2007[b]	ASD 11/1 TD 11/1	26.8 (7.7) 22.5 (3.2)	110.1 (12.6) 117 (8.7)	Q: - SI: ADI-R, ADOS-G NSCA: - CLAS: -	Go/NoGo (*letters*)	1000	Neutral	% Commission errors ('false alarms')	-
Kilincaslan, Motavalli, Sozen, & Gurvit, 20120	ASD 18/3 TD 15/3	7-16/ 12.4(2.9) 7-16/ 12(2.4)	105.5 (14.7) 107.3 (13.4)	Q: T SI: - NSCA: clinical diagnosis CLAS: DSM-IV	CPT (*letters*)	-	Neutral	Commission errors	.61
Langen et al., 2011[c]	ASD 21/0 TD 22/0	19-39/ 25.6 (6.1) 19-44/ 28.5 (6.4)	107.5 (15.1) 109.8 (13.7)	Q: - SI: ADI-R, ADOS NSCA: clinical diagnosis CLAS: ICD-10	Go/NoGo *(arrows)*	1300	Neutral	Correct NoGo	1.17

Table 1. Continued

Study by	Subjects M/F	Age range/M (SD)	IQM (M/SD)	Group Assignment ASD	Task *(stimulus)*	ISI (ms)	Type of stimulus	Measure	ES
Lee et al., 2009	ASD 9/3 TD 8/4	8-12/ 10.2 (1.6) 8-12/ 11 (1.8)	113.3 (17.3) 114.9 (10.3)	Q: - SI: ADI-R, ADOS NSCA: clinical diagnosis CLAS: DSM-IV	Go/NoGo *(letters)*	2500	Neutral	Commission errors	.26
Lemon, Gargaro, Enticott, & Rinehart, 2011	ASD 10/13 TD 8/14	M: 6-16/ 11.1 (3.6) F: 6-16/ 11 (3) M: 6-16/ 12.1 (4.2) F: 6-16/ 10.8 (2.3)	M: 91.7 (18.4) F: 97.3 (16.7) M: 108 (11) F: 107 (10.7)	Q: P SI: - NSCA: clinical diagnosis CLAS: DSM-IV	Stop signal *(colors)*	2250	Neutral	SSRT (*average of males and females*)	.62
Oerlemans et al., (2013)	ASD 115/25 Siblings 82/90 TD 54/73	12.4 (3.0) 12.2 (3.9) 11.0 (3.6)	102.0 (13.8) 105.5 (12.9) 107.4 (12.4)	Q: P SI: ADI-R, ADOS NSCA: clinical diagnosis CLAS: DSM-IV	Go/NoGo *(figures)*	1150	Neutral	% commission errors	.27
Ozonoff, Strayer, McMahon, & Filloux, 1994	ASD 13/1 TS 11/3 TD 11/3	8-16/12.4 (2.5) 12.9 (1.73) 12.2 (1.73)	101.9 (17.2) 99.9 (18.6) 100.4 (13.4)	Q: - SI:- NSCA: clinical diagnosis CLAS: DSM-III-R	Go/NoGo *(figures)*	1250	Neutral	Commission errors *(condition 2)*	.05
Ozonoff & Strayer 1997d	ASD 13/0 TD 10/3	9.3-16.9/ 13.9 (2.5) 11.4-15.5/13.1 (1.4)	101 (18.8) 100.1 (11.9)	Q: - SI: - NSCA: autism specialists CLAS: DSM-III	Stop signal *(words)*	2000	Arousing	SSRT (*forbidden nontargets*)	−.07
Pankert et al., 2014[d]	ASD 16/1 TD 12/5	9-14/11.6 (1.5) 9-14/11.7 (1.2)	109.3 (17.5) 109.2 (9.9)	Q: P SI: ADI-R, ADOS-G NSCA: clinical diagnosis CLAS: DSM-IV-TR	Go/NoGo *(letters)*	1400	Arousing	% Commission errors (fam. *social reward*)	.16
Raymaekers et al., 2004[c]	ASD 15/2 TD 15/2	28.4 (8.4) 28.8 (8.9)	111.7 (19.5) 121.0 (9.4)	Q: P,T SI: - NSCA: clinical diagnosis CLAS: DSM-III or DSM-IV	Go/NoGo *(letters)*	1000 *(fast)* 2000 *(medium)* 6000 *(slow)*	Neutral	Commission errors (*fast*)	.73
Raymaekers et al., 2006[b]	ASD 33/6 TD 18/11	7-16/ 11.3 (2.6) 7-16/ 10.5 (2)	107 (13.6) 107 (13)	Q: P,T SI: - NSCA: clinical diagnosis CLAS: DSM-IV-TR	Go/NoGo *(cross)*	3750 *(without signal)* 4833 *(with signal)*	Arousing	% incorrect trials	-
Raymaekers et al., 2007[b]	ASD 27/4 ADHD 15/9 TD 20/8	7-13/ 10.5 (2.2) 7-13/ 9.6 (1.9) 7-13/ 10.5 (2)	107 (12) 99 (11) 107 (8)	Q: P SI: ADI-R NSCA: clinical diagnosis CLAS: DSM-IV	Go/NoGo *(letters)*	2000 *(fast)* 8000 *(slow)*	Neutral	Commission errors	-
Sanderson & Allen 2013[a,c]	ASD 26/5 TD 11/17	13.6 (1.9) 6-11/ 8.8 (1.4)	28.1 (4.99) 29.46 (3.89)	Q: P SI: NSCA: clinical diagnosis CLAS: DSM-IV	Go/NoGo *(figures)*	2500	Neutral	Commission errors	−.67
Schmitz et al., 2006	ASD 10/0 TD 12/0	18-52/ 38 (9) 18-52/ 39 (6)	105 (14) 106 (13)	Q: - SI: ADI NSCA: clinical diagnosis CLAS: ICD-10	Go/NoGo *(arrows)*	1800	Neutral	Nr incorrect trials	.24

Table 1. Continued

Study by	Subjects M/F	Age range/M (SD)	IQM (M/SD)	Group Assignment ASD	Task *(stimulus)*	ISI (ms)	Type of stimulus	Measure	ES
Sebanz et al., 2005[b, d]	ASD 10/3 TD 10/3	16-29/ 20.9 (4.2) 16-28/ 19.9 (3.0)	109 (10.1) 117.85 (8.7)	Q: - SI: - NSCA: clinical diagnosis CLAS: -	Go/NoGo *(figures)*	2000	Arousing	- (*joint setting*)	-
Sinzig et al., 2008	ASD 16/4 ASD + ADHD 20/1 ADHD 27/3 TD 23/7	8-19/ 14.5 (3) 6-17/ 10.7 (3.1) 7-18/ 12.9 (3.1) 7-18/ 12.8 (2.8)	112 (19) 103 (13) 102 (15.8) 109 (12.8)	Q: - SI: ADOS, ADI-R NSCA: clinical diagnosis CLAS: DSM-IV	Go/NoGo *(figures)*	400	Neutral	Commission errors	−.14
Takeuchi et al., 2013	ASD 24/- ADHD 20/- TD >60/-	6-15/ 10.2 (1.8) 6-15/ 10.3 (2.1) 6-15/10.1 (2.4)	94.3 (8.2) 98.7 (8.7) -	Q: P SI: NSCA: clinical diagnosis CLAS: DSM-IV	Go/NoGo *(pictures)*	2500	Neutral	% commission errors	.75
Tye et al., 2014	ASD 19/- ADHD 18/- ASD + ADHD 29/- TD 26/-	8-13/ 11.7 (1.7) 8-13/ 10.5 (1.9) 8-13/ 10.5 (1.7) 8-13/ 10.6 (1.8)	115.7 (15.7) 104.1 (14.2) 109.7 (13.4) 120.0 (13.4)	Q: P SI: ADI-R, ADOS-G NSCA: clinical diagnosis CLAS: ICD-10	CPT *(letters)*	1650	Neutral	Total commission errors	.14
Verté, Geurts, Roeyers, Oosterlaan, & Sergeant, 2006	ASD 61/5 ADHD 54/11 TS 20/4 TD 67/15	6-13/ 8.7 (2.0) 6-13/ 9.1 (2.0) 6-13/ 10.0 (2.2) 6-13/ 9.2 (1.7)	101.5 (18.2) 99.8 (11.7) 104.8 (13.6) 112.2 (16.0)	Q: P, T SI: ADI-R NSCA: clinical diagnosis CLAS: DSM-IV	Change task *(figures)*	2850	Neutral	SSRT	.54
de Vries & Geurts, 2014	ASD 64/9 TD 26/17	7.9-12.9/ 10.6 (1.4) 8.2-12.0/ 10.4 (1.1)	109.3 (20.9) 107.7 (18.9)	Q: P SI: ADI-R NSCA: clinical diagnosis CLAS: DSM-IV	Stop task *(figure)*	3450	Neutral	SSRT	.38
Xiao et al., 2012	ASD 19/0 ADHD 16/0 TD 16/0	8-14/ 10.1 (2.1) 8-14/ 9.8 (1.2) 8-14/ 9.7 (1.7)	99.3 (9) 103.6 (8.1) 105.6 (13.1)	Q: - SI: ADI-R NSCA: clinical diagnosis CLAS: DSM-IV	Go/NoGo *(letters)*	1500	Neutral	Commission errors	.87
Yerys et al., 2013[d]	ASD 19/3 TD 15/8	7-12.98/10.22 (1.81) 7.3-12.92/10.62 (1.55)	111.43 (12.74) 118.96 (12.86)	Q: P SI: ADOS, ADI-R NSCA: clinical diagnosis CLAS: DSM-IV-TR	Go/NoGo (facial expressions)	1500	Arousing	Commission errors (*emotional*)	.60

Note: A positive ESs indicates a better performance on the inhibitory control task by the TD group compared to the ASD group. A negative ESs indicates a better performance by the ASD group; ADI-R: Autism Diagnostic Interview Revised; ADOS(-G): Autism Diagnostic Observation Schedule(-Generic); ASD: Autism Spectrum Disorders (including Asperger syndrome, autism and PDD-NOS); CARS: Childhood Autism Rating Scale; CLAS: Classification system used; CPT: Continuous Performance Test; DSM-IV: Diagnostic and Statistical Manual of Mental Disorders, fourth edition; F: female; ISI: Interstimulus interval; IQ: Intelligence Quotient; M: male; NSCA: Nonstructural clinical assessment; P: parent; POP: Preparing to Overcome Prepotency; Q: Questionnaire; RT: reaction time; SART: Sustained Attention to Response Test; SI: Structured instrument (e.g., standardized interviews or observation schedules); SSRT: Stop-Signal Reaction Time; T: Teacher, TD: Typically Developing Group; Type of stimulus: the type of stimulus used in the task, this could either be neutral (e.g., arrow, cross) or arousing (e.g., high-arousal pictures, facial expressions).

[a] IQ was assed using the Raven's test. All the other studies used Wechsler Intelligence Scales (WISC, WAIS).

[b] Five studies did not report enough data to calculate an ES. Therefore they were excluded from further analysis.

[c] Three studies did not report all the data needed to calculate an ES, but did report t-statistics or F-statistics, which were used to calculate an ES.

[d] When a study had multiple conditions (i.e., neutral vs. arousing), over the arousal condition an ES was calculated and included, due to this being examined as a moderator.

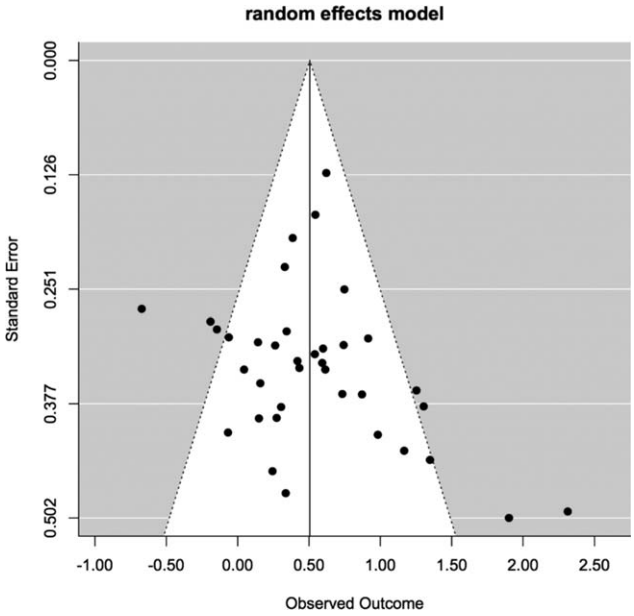

Figure 3. Funnel plot: Prepotent response inhibition

identified as outliers with regards to age (>2 SD from mean). Excluding these two studies did not alter the results ($QM = 0.04$, $df = 1$, $P = 0.84$) and the heterogeneity was still significant ($QE = 84.78$, $df = 33$, $P < 0.0001$; $I^2 = 64.97\%$). This indicates that age did not have a moderating effect. IQ was mostly reported as a Wechsler scaled IQ, with exception of three studies that used Raven's test (Adams & Jarrold, 2012; Czermainski, dos Santos Riesgo, Guimaraes, de Salles, & Bosa, 2014; Sanderson & Allen, 2013). The moderating effect of IQ was examined only on the studies reporting Wechsler scaled IQ. Results showed that IQ was not significant either ($QM = 3.07$, $df = 1$, $P = 0.08$) and the heterogeneity remained significant ($QE = 59.8$, $df = 32$, $P = 0.002$; $I^2 = 42.81\%$). The overall result as well as the results of the IQ moderator analysis were similar to the result of the previous meta-analysis (Geurts et al., 2014). We did not, however, replicate the finding that age was a significant moderator.

Results of the moderator meta-analysis: effect of ISI between the groups

The influence of different ISI on inhibition was examined by including it as a moderator in the meta-analysis. Our results show that as a dichotomous moderator, ISI did not have a moderating effect ($QM = 1.19$, $df = 1$, $p = 0.28$; heterogeneity: $QE = 81.87$, $df = 33$; $P < 0.0001$; $I^2 = 62.8\%$). Nor did it have a moderating effect as a continuous moderator ($QM = 0.44$, $df = 1$, $P = 0.51$; heterogeneity: $QE = 81.9$, $df = 33$, $P < 0.0001$; $I^2 = 63.3\%$). However, when exploring the effect of fast and slow ISI separately for inhibition studies, the studies in the 'fast ISI' category showed a significant medium to large ES ($g = 0.61$, $P < 0.0001$). The studies in the "slow ISI" category showed a significant small to medium ES ($g = 0.43$, $P = 0.002$). This could suggest that the differences in inhibition between people with and without ASD are more pronounced in the "fast ISI" studies (ES = .61) than in the "slow ISI" studies (ES = .43). It should be noted that this must be interpreted carefully due to the nonsignificant effect of ISI as a moderator.

Results of qualitative review: effects of ISI and "stimulus-type" within groups

While the meta-analysis informs us about whether ISI might explain some of the differences found between inhibition studies in people with ASD compared to people without ASD, it does not answer the question of whether different ISI or different types of stimuli actually affect performance on an inhibition task. In studies on inhibitory control in people with ASD, tasks or conditions have been adjusted for various reasons. For instance, studies have made adjustments to examine the effect of socially emotional stimuli on inhibitory control (e.g., Yerys et al., 2013), the effect of a possible theory of mind deficiency on inhibitory control in people with ASD (Sebanz et al., 2005) or to examine whether "emotional" stimuli lead to differences in performance due to an arousal modulation deficiency in people with ASD (Geurts et al., 2009). Overall, it is assumed that these stimulus adjustments might alter behavioral or physiological responses in people with ASD and not in people without ASD. In our qualitative review, we have focused on those studies that have incorporated two conditions within one inhibition task: either by having multiple ISI conditions or various "stimulus-type" conditions. This allows us to examine the behavioral influence of a stimulus on performance directly. Due to a relatively small number of such studies (ISI: $N = 3$; "stimulus-type": $N = 5$), we were unable to run a meta-analysis. As shown in Table 2, eight studies met our criteria and we were able to calculate ESs for four of these eight studies (Geurts et al., 2009; Ozonoff & Strayer, 1997; Pankert et al., 2014; Yerys et al., 2013).

Regarding ISI, three studies were included in the qualitative analysis. The study by Geurts et al. (2009) reported that both groups (ASD and TD) made significantly more commission errors in the fast ISI condition (ISI of 2 sec) compared with a slow ISI condition (ISI of 6 seconds). The ESs for both groups were small (ASD: $g = .27$; TD: $g = .02$). Two studies by Raymaekers et al. (2004, 2007) also reported a significant influence of different ISI as they found a better performance on the slow condition compared to the fast ISI condition. The studies do not specifically report whether this effect is

Table 2. Characteristics of the Studies Including a Neutral and an Arousal Condition

Study by	Subjects M/F	Age Mean (SD)	Task/Type of Inhibition	Within group analysis measure	Stimuli	Group	Neutral vs. Arousing based on p-value	ES
Geurts et al., 2009	ASD 16/2 TD 19/3	10.3 (1.6) 10.3 (1.4)	Go/NoGo/PRI	% Errors	Slow ISI vs. Fast ISI	ASD TD	Neutral < Arousing Neutral < Arousing	.27 .02
Ozonoff & Strayer, 1997	ASD 13/0 TD 10/3	13.9 (2.5) 13.1 (1.4)	Stop signal/PRI	SSRT neutral vs. forbidden words	Neutral non-target words vs. Forbidden non-target words	ASD TD	Neutral = Arousing Neutral = Arousing	−.22 −.24
Pankert et al., 2014	ASD 16/1 TD 12/5	9-14/11.6(1.5) 9-14/11.7 (1.2)	Go/NoGo/PRI	% commission errors baseline vs. fam. Social. Reward Visual modality	No reward vs. Familiar social reward	ASD TD	Neutral > Arousing Neutral > Arousing	−.67 −.26
Raymaekers et al., 2004	ASD 15/2 TD 15/2	28.4 (8.4) 28.8 (8.9)	Go/NoGo/PRI	Commission errors main effect of condition	Slow ISI vs. Fast ISI	ASD TD	Neutral < Arousing Neutral < Arousing	- -
Raymaekers et al., 2006	ASD 33/6 TD 18/11	11.3 (2.6) 10.5 (2)	Go/NoGo/PRI	% Inhibition Errors main effect of condition	Without signal vs. With signal	ASD TD	Neutral < Arousing Neutral < Arousing	- -
Raymaekers et al., 2007	ASD 27/4 TD 20/8	10.5 (2.2) 10.5 (2)	Go/NoGo/PRI	% Commission Errors	Slow ISI vs. Fast ISI	ASD TD	Neutral < Arousing Neutral < Arousing	- -
Sebanz et al., 2005	ASD 10/3 TD 10/3	20.9 (4.2) 19.9 (3.0)	Go/NoGo/PRI	Error rate incompatible trials Exp. 2	Alone vs. Joint	ASD TD	Neutral < Arousing Neutral < Arousing	- -
Yerys et al., (2013)	ASD 19/3 TD 15/8	10.22 (1.81) 10.62 (1.55)	Go/NoGo/PRI	Com. Errors	Neutral NoGo vs. Emotional NoGo	ASD TD	Neutral < Arousing Neutral < Arousing	2.17 1.71

Note: = means that the group responded the same in the neutral as in the arousing condition; > means that the group performed better in the arousing condition (e.g., more commission errors or longer reaction time in the neutral condition); < means that the group performed better in the neutral condition (e.g., more commission errors or longer reaction time in the arousing condition); - means this data is not available, described or received.

ASD, Autism Spectrum Disorders (including Asperger syndrome, autism and PDD-NOS); ES, Effect size (Hedges'g); F, female; Group, either ASD or TD; M, male; RT, reaction time; PRI, Prepotent Response Inhibition; SSRT, Stop-Signal Reaction Time; Task, name of the task used; TD, Typically Developing Group; Stimuli, the type of stimulus used in the task.

seen in both the ASD and the TD group. The Raymaekers study from 2004 did, however, report that the ASD group made significantly more commission errors than the TD group in the fast ISI condition. In line with our meta-analysis that showed no group differences, the results of these studies suggest that in both the ASD and TD group a faster ISI leads to more inhibitory control difficulties.

Regarding the effects of different "arousing" stimuli on inhibitory control in people with and without ASD, five studies were found. We were able to calculate the ES of three of those studies (Ozonoff & Strayer, 1997; Pankert et al. 2014 Yerys et al., 2013). Two out of the three studies of which we were able to calculate an ES (ranging from −.22 to −.67), seem to suggest a better performance of both groups on the "arousing" condition compared with the neutral condition, indicated by the negative ESs (Pankert et al., 2014; Ozonoff & Strayer, 1997). Contrary to these studies, the third study by Yerys et al. (2013) reported a better performance on the neutral condition, indicated by large positive ESs (ASD: $g = 2.17$; TD: $g = 1.71$). However, whether the neutral condition in that study can be defined as "neutral" is debatable, as both conditions contained emotional pictures (Yerys et al., 2013). In their Go/NoGo task, the arousal condition included pictures of an emotional face (i.e., happy or sad) as the NoGo stimulus and pictures of a neutral face as the Go stimulus. In the neutral condition it was the other way around, pictures of a neutral face served as the NoGo stimulus and pictures of an emotional face as the Go stimulus. Consequently, the participants were confronted with the same stimuli in both conditions. Yet, the two studies of which we were unable to calculate an ES, did also report a better performance of both groups on the neutral condition compared to the arousing condition (Raymaekers et al., 2006; Sebanz et al., 2005). Notice how all five studies used different "arousing" stimuli: "loaded" words (Ozonoff et al., 1997); familiar social faces (Pankert et al., 2014); an acoustic signal (Raymaekers et al., 2006); performing the inhibition task together with another person (Sebanz et al., 2005) and presenting emotional faces (Yerys et al., 2013). Taken together, these studies seem to suggest that various stimuli have an effect on performance. The effect does not seem to discriminate between people with and without ASD and could lead to either an increase or a decrease in performance.

Discussion

The aim of this study was to determine whether ISI and "stimulus-type" should be accounted for when studying prepotent response inhibition in people with ASD. In contrast to our expectations, the meta-analysis did not reveal an effect of ISI on the difference in inhibitory control performance between those with and without ASD. Unfortunately, the "stimulus-type" moderator could not be included in the meta-analysis as there were too few studies using "arousing" stimuli. These studies were included in the qualitative analysis where we focused on the studies that experimentally manipulated ISI or "stimulus-type" within one experiment. Within this analysis, a small effect regarding the influence of ISI on prepotent response inhibition and small to medium effects regarding "stimulus-type" were observed. In line with the results of our meta-analysis, these two types of task manipulation did not differentiate between people with and without ASD. Regardless of group, the findings of the qualitative analysis did indicate more prepotent response inhibition difficulties when ISI was fast. With respect to "stimulus-type," the observed effects were rather contradictory, showing that stimuli could have either a positive or a negative influence on prepotent response inhibition.

While our study included an additional 15 studies, which is more than half of the included studies in the prepotent response inhibition meta-analysis of Geurts et al. (2014), we did replicate the findings of Geurts et al. (2014). This shows that even after including a large amount of recently published studies, the overall finding that people with ASD have more difficulty with prepotent response inhibition than typically developing people remains unchanged. Besides this overall difference in prepotent response inhibition between people with and without ASD, a large amount of heterogeneity between the studies also still remains. Contrary to Geurts et al. (2014), we did not observe an effect of age. Only a few studies focused on adults with ASD in our sample (7 out of 37 studies), while in the meta-analysis of Geurts et al., (2014) 6 out of 23 studies focused on adults. It is possible that there were not enough studies on adults to replicate the age-effect. Although, it has been suggested that older TD adults have more difficulty withholding their response when they are presented with a cue to stop compared to younger TD adults (e.g., Kramer et al., 1994; Williams, Ponesse, Schachar, Logan, & Tannock, 1999). Prepotent response inhibition abilities in adults with ASD are likely to decline similarly with age. The significant difference in prepotent response inhibition compared with TD adults would then still be present. The existing studies on prepotent response inhibition in adults with ASD report too large variations in ESs and the number of studies is too small to draw strong conclusions. More research focusing on prepotent response inhibition in adults with ASD is required.

When combining our results with the cognitive-energetic model of Sanders (1983), our results seem to suggest that both people with and without ASD struggle to regulate their over-arousal levels as both groups showed a decrease in performance in the fast ISI condition and certain "stimulus-type" conditions. A recent review on physiological arousal in people with ASD (Klusek et al., 2015) suggests that people with ASD have atypical baseline physiological arousal levels and abnormal physiological reactions to several experimental tasks, compared to controls (Klusek et al., 2015). Following the model of Sanders (1983) in combination with the review of Klusek et al. (2015), we expected a stronger decrease in performance in the ASD group compared to the TD group in the fast ISI and certain stimuli conditions, as studies show that people with ASD seem to have arousal regulation difficulties compared with a TD group. However, our results show that the ASD and the TD group performed similarly in both the "slow" and "fast" ISI condition as well as in both the neutral and "arousing" stimuli condition. One could argue that the "fast" ISI condition (<2 sec; Metin et al., 2012) might not be fast enough to evoke physiological arousal, and, therefore, had no different impact on people with ASD than on people without ASD. This does not seem to be a plausible explanation as including ISI as a continuous moderator in the meta-analysis did not alter findings. One could also challenge the definition used to determine the role of ISI on inhibitory control performance. The definitions of ISI differed considerably between the included studies. We followed the definition used by Metin et al. (2012) in their meta-analysis. This definition stated that ISI is the time between the onset of the first stimulus and the onset of the second stimulus. Only by applying a similar definition across studies could we statistically examine the role of ISI on prepotent response inhibition. However, we did not test whether another definition (e.g., time between the offset of the first stimulus and the onset of the second stimulus) might alter the results of the analysis as the information on ISI in many reports was limited. Clear terminology (e.g., definition of ISI) is essential for replicating and comparing studies. We suggest standardizing the definition of ISI by following the definition as proposed by Metin et al. (2012) in future studies. For now, we cannot discount the possibility that the way ISI is defined did impact our results. Nonetheless, this seems to be unlikely, as our results regarding the influence of ISI are clearly different from findings among people with ADHD, when using a similar definition of ISI.

Our meta-analysis also showed a large amount of heterogeneity between the studies. Differences in ISI between the studies did not explain some of this heterogeneity, indicating there are other factors that might influence these differences between the studies. Recently, more studies seem to suggest that baseline physiological arousal might be of importance (e.g., Klusek et al., 2015; Krypotos et al., 2011; Neuhaus, Bernier, & Beauchaine, 2014; Patriquin, Scarpa, Friedman, & Porges, 2013; Vaughan Van Hecke et al., 2009). Several studies indicate that people with ASD show heightened arousal when confronted with demanding (cognitive) tasks, but only when they were already highly aroused at baseline before the task (Klusek et al., 2015). This suggests that the stimulus itself might not have triggered a state of over-arousal, but rather that the person was already highly aroused at baseline and when confronted with a stressor performed worse on the cognitive task. The importance of baseline arousal has also been shown in studies with healthy control participants (Krypotos et al., 2011). The results of this study support the idea of an inverted-U curve. Contrary to Sanders (1983) who related general arousal regulations difficulties with the U curve, the results of the Krypotos study (2011) suggest that the baseline arousal position of the subject on the inverted-U curve (either under-, optimally or over-aroused) will determine whether fast ISI or "arousing" stimuli lead to a decrease in performance. As it has been suggested that people with ASD have abnormal baseline physiological arousal levels, including a subgroup that is over-aroused (e.g., Klusek et al., 2015), the observed overall difference in prepotent response inhibition between people with and without ASD might partly be caused by differences in baseline physiological arousal. Unfortunately, none of the included studies has measured physiological arousal at baseline or during the task. Therefore, the possible influence of baseline physiological arousal remains a hypothesis. Various other factors could influence the heterogeneity between the prepotent response inhibition studies in people with ASD, such as processing speed or attentional load. Processing speed, for instance, has been suggested to differ between individuals with ASD and has a large discrepancy with IQ score in people with ASD (e.g., Scheuffgen, Happe, Anderson, & Frith, 2000; Wallace, Anderson, Happa, 2009). This suggests that even when people are matched on IQ score, the processing speed might still differ between the groups and could lead to a difference in performance. Therefore, future research on prepotent response inhibition in people with ASD should include physiological measures and focus on exploring multiple possibly influential factors to try to explain the large amount of heterogeneity between the studies on prepotent response inhibition in people with ASD.

Our qualitative review suggested an influence of both ISI and "stimulus-type" on prepotent response inhibition, although this was similar for both people with and without ASD. In both groups, fast ISI resulted in a decrease in prepotent response inhibition performance. ISI determines the presentation rate of stimuli. Faster ISI is suggested to require faster information processing abilities as the stimuli need to be processed more quickly (e.g., Hansen & Hillyard, 1984; Gomes, Barrett, Duff, Barnhardt, & Ritter, 2008). This makes the task more demanding. In our sample this would suggest that both groups struggled similarly with the processing of the stimuli in the fast condition, as both groups performed similarly. However, a too slow ISI could also increase task difficulty, as it relies on an increase in focused attention duration and in maintaining stimulus representations in working memory (Gomes et al., 2008). In the meta-analysis of Metin et al. (2012) that focused on people with ADHD, slow and more variable responses were observed in people with ADHD when ISI exceeded 6 sec. The authors argue that this can be related to factors like sustained attention and motor timing (Metin et al., 2012). Only one of the studies included in our meta-analysis exceeded the 6 seconds (i.e., Brandimonte, Filippello, Coluccia, Altgassen, & Kliegel, 2011). Excluding this study did not alter the results from the ISI moderator analysis. Factors like attention and motor time could still drive the slow versus fast ISI effect in our sample as we did not control for these factors. As both groups performed similarly, these factors might also have similar influences on people with ASD as without ASD. Regarding "stimulus-type," several studies have shown that certain tasks (e.g., cognitive task or social performance task) lead to heightened arousal in people with ASD (Klusek et al., 2015) but also that certain tasks do not (e.g., social interaction task; Klusek et al., 2015). Future research should include physiological measures to examine whether certain types of stimuli might alter physiological arousal levels and examine if this influences prepotent response inhibition in people with ASD. Taken together, if we want to understand the underlying mechanisms of the influence of a fast ISI or an "arousing" stimulus on inhibitory control in ASD, future research should focus on different kinds of possible influential factors and include physiological measures to be able to account for possibly influences on arousal.

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