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# Brief report: Inhibition of return in young people with autism and Asperger's disorder

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ABSTRACT The aim of this study was to investigate whether the superior search abilities observed in autism/Asperger's disorder may in part be a consequence of a more pronounced inhibition of return (IOR). Contrary to our prediction, IOR in individuals with autism was comparable to the matched comparison group. However, the autism group committed more false alarm responses than the matched comparison group; this may reflect a possible inhibitory deficit, or suggest that individuals with autism rely more on probabilities to determine their behavioural responses. There was a borderline-significant trend (p = 0.052) to indicate that IOR may be more pronounced in individuals with Asperger's disorder. In contrast to the autism group, the Asperger's disorder group had a pattern of false alarm responses similar to that of the comparison group. The findings further inform Minshew's complex information processing theory which seeks to establish which areas of neuropsychological functioning are preserved and deficit in autism.

ADDRESS Correspondence should be addressed to: NICOLE J. RINEHART, Centre for Developmental Psychiatry and Psychology, School of Psychology, Psychiatry and Psychological Medicine, Monash University, Building 1, 270 Ferntree Gully Road, Nottinghill, Victoria, Australia 3168. e-mail: nicole.rinehart@med.monash.edu.au KEYWORDS Asperger's disorder; autism; inhibition of return; visual search

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Minshew's complex information processing theory of autism (Minshew and Goldstein, 1998; Minshew et al., 1997) emphasizes the equal importance of understanding not only areas of neuropsychological deficit, but also

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areas which are preserved as a means for appreciating the full behavioural impact of the disorder (Minshew et al., 1997). An intact or superior ability to detect unique items in visual search tasks (e.g. 'finding an R target hidden among P and Q distractors') is emerging as a significant aspect of the neuropsychological profile of autism (O'Riordan, 2004, p. 230). O'Riordan's series of experiments manipulating search task parameters has indicated that superior search in individuals with autism is underpinned by an enhanced ability to discriminate between items (O'Riordan, 2004). Plaisted et al. (1998) note that this enhanced ability to visually detect items is 'at odds' with the body of research showing that individuals with autism in fact have significant difficulties with tasks measuring visual orientation and attentional set-shifting. To account for this apparent anomaly in research findings, Plaisted et al. (1998) point to possible differences in domains other than visual attention which may account for enhanced item detection and superior search performance, for example, weak central coherence, reduced generalization of responding.

A possibility that has not yet been explored in the literature is that individuals with autism may also be better at search tasks due to a more pronounced inhibition of return (IOR). IOR is a central cognitive mechanism which supports 'perceptual-motor interactions within complex environments' (Tipper et al., 1996, p. 1289). IOR was demonstrated experimentally by Posner et al. (1984) who showed that reaction time to a target is shorter when the target and cue are presented at the same location, compared to when they are presented in a different location, if the cue-totarget delay is less than 300 ms. However, if the cue and target are separated by more than 300 ms but less than 2000 ms, the reverse pattern occurs, that is, reaction time is slower when the cue and target appear in the same location compared to when they appear in different locations (i.e. IOR). This highly adaptive phenomenon ensures that attention is distributed across the environment to novel locations and does not perseverate on a single, irrelevant location (Tipper et al., 1996). Thus, an important function of IOR is to improve visual search efficiency (Klein and MacInnes, 1999). While a deficient IOR mechanism would make the simplest of search tasks difficult (for example, when searching for a letter 'R' target, the participant might tend to repeatedly look in locations of the visual array which have previously been searched and ruled out), an intact or superior IOR would promote visual search to novel locations, expediting the search task. Klein and MacInnes (1999) have shown that IOR facilitates visual search in normally developing individuals using a Where's Waldo?<sup>TM</sup> paradigm, a paradigm which may be loosely associated with the tasks used to demonstrate superior search ability in individuals affected by autism and Asperger's disorder (e.g. Jolliffe and Baron-Cohen, 1997; O'Riordan, 2004).

The aim of the present study was to investigate whether individuals with autism and Asperger's disorder show a pronounced IOR which is analogous to the superior visual search ability observed in these populations. This study forms the final part of a series of studies comparing the cognitive-motor profile of children with autism and Asperger's disorder. In addition to making comparisons between the neuropsychological profiles of these disorders, the aim of this series was to parse out areas of impaired and preserved functioning (see most recently Rinehart et al., 2006; also see Rinehart et al., 2002a for a review). On the basis that there is no indication in the literature that visual search ability differs between autism and Asperger's disorder, we hypothesize that a more pronounced IOR will also be evident in both groups. Indeed, IOR is considered to be a measure of automatic inhibition, and our past studies suggest that the dissociation between autism and Asperger's disorder occurs when participants are engaged in tasks which measure controlled inhibitory processes supported by fronto-striatal executive functioning (Rinehart et al., 2002b).

# Method

#### **Participants**

The participants in this study were the same as those in our pseudo-random number generating study (Rinehart et al., 2006). The experiments were conducted over an 18 month period. Twelve individuals with high-functioning autism (HFA) (11 males and one female), as well as 12 comparison participants matched on age, sex, and full-scale IQ, participated in the study. In addition, 12 individuals with Asperger's disorder (AD) (10 males and two females) and another 12 comparison participants were recruited and matched according to age, sex, and full-scale IQ. One-way analysis of variance (ANOVA) confirmed no significant age difference between the HFA and the comparison group (HFA, mean age = 10.6 years, SD = 3.0; comparison group, 10.6 years, SD = 3.3; F(1, 22) < 1, n.s.). A second ANOVA similarly revealed no significant age difference between the AD and the comparison group (AD, mean age = 13.4 years, SD = 4.0; comparison group, 13.0 years, SD = 3.9; F(1, 22) < 1, n.s.).

These participants were recruited in the same way as the HFA and AD participants reported in Rinehart et al. (2006). The participants with high-functioning autism fulfilled DSM-IV (American Psychiatric Association, 1994) criteria for autistic disorder. The participants in the Asperger's disorder group satisfied DSM-IV criteria for Asperger's disorder. Four experienced clinicians were involved, at various times, in diagnosis. Diagnostic information was gathered using the revised Autism Diagnostic Interview

(Lord et al., 1994), structured parent interview, direct child observations, and information from other sources such as teachers and therapists. Inter-rater reliability, calculated on a sample of 107 cases of autism and Asperger's disorder, generated a Cohen kappa of 0.95 for autism and 0.94 for Asperger's disorder, thereby indicating strong agreement.

Participants were included only if their performance and verbal IQ exceeded 70. In addition, participants were excluded if they had previously experienced the following conditions: comorbid medical (e.g. tuberous sclerosis), hearing or visual, neurological (e.g. epilepsy), psychiatric (e.g. Tourette's, attention deficit hyperactivity disorder) or genetic disorders (e.g. fragile X disorder), other than the primary diagnosis of HFA or AD. None of the participants were medicated at the time of this study.

Intellectual functioning in the comparison group was established using a short form of the Wechsler Intelligence Scales (either WPPSI–R or WISC–III–R), consisting of two verbal (information and vocabulary) and two performance (picture completion and block design) subtests. This particular short form loads highly on verbal comprehension and visualperceptual organization skills, and is a reliable estimate of full-scale IQ scores (Sattler, 1992). The comparison groups were matched to clinical participants on the basis of full-scale IQ. One-way ANOVAs uncovered no significant difference in IQ between the HFA (mean IQ = 91.2, SD = 9.1) and their matched comparison group (mean IQ = 102.4, SD = 21.3) and their matched comparison group (mean IQ = 102.0, SD = 10.8), F(1, 22) < 1, n.s.

Behavioural functioning was screened in both comparison groups using the Parent form of the Child Behaviour Checklist (CBC–L: Achenbach, 1991). None of the comparison groups were reported to have behavioural problems.

#### Apparatus and procedure

Participants were positioned 60 cm in front of a Toshiba (440 CDT Satellite Pro) notebook computer with their dominant hand resting on the space bar of the computer keyboard. For each trial, two grey, rectangular boxes were presented and arranged in a horizontal array on the display. Each box spanned 1.8 cm in height and 1.4 cm in width. A + symbol was located in the centre of the screen; the distance between the midpoint of each lateral box and the midpoint of the + was 8.5 cm (Figure 1). After 1500 ms, one of the lateral boxes became white for 50 ms before returning to grey.

In the short-delay condition, a red asterisk was laterally presented 100 ms after the lateral cue appeared. In the long-delay condition, the red asterisk was presented 700 ms after the lateral cue appeared. On catch trials, no asterisk was presented. Participants were instructed to press the space bar as soon as they detected the red asterisk.

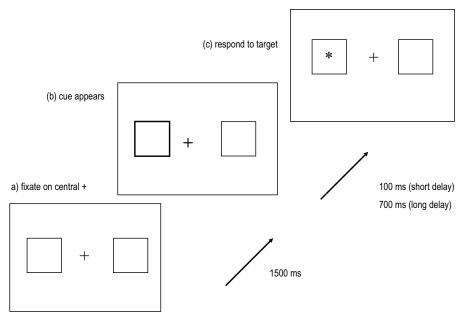


Figure 1 A schematic representation of the inhibition task. This is an example of where the cue and target appear in the same location at either 100 ms or 700 ms cue-to-target delay (+ = central fixation point, illuminated square = cue, \* = target)

IOR is manifested by a retardation of RT when the cue and target are spatially coincident and separated by more than 300 ms. Normal IOR should generate an interaction between delay and relative position of the cue and target.

Each participant received 200 trials, which included 40 catch trials. The position of the lateral cue (left versus right), the position of the red asterisk or target (left versus right), and the delay (short versus long) was counterbalanced in a factorial fashion. The duration from the onset of the target to the depression of the space bar constituted the reaction time (RT); RTs less than 100 ms were deemed to be anticipations; RTs that exceeded 2000 ms were recorded as misses. Participants were given a break after every 25 trials. RT anticipations (e.g. respond after the cue has appeared but before the target), misses (e.g. fail to respond after a target has appeared), and false alarms (e.g. response on a catch trial) were examined separately for the autism and respective comparison group, and for the Asperger's disorder group and their respective comparison group.

# Results

### High-functioning autism (HFA)

**Reaction time** RT data were submitted to a four-way ANOVA with the following factors: group (HFA, comparison group), target side (left or right), relative position (same or different location to cue), and delay (short or long). Target side did not yield any significant interactions or main effects. Hence, the RT data were subjected to a three-way ANOVA after collapsing across target side. Interactions of group by location, F(1, 22) =5.35, p = 0.03, and location by delay, F(1, 22) = 11.31, p = 0.001, were revealed in the subsequent three-way ANOVA. Subsequent two-way ANOVAs revealed that the HFA group were slower at responding to targets when cues and targets appeared in the same location (mean RT = 527 ms, SD =133 ms), relative to when cues and targets were spatially disparate (mean RT = 445 ms, SD = 151 ms), F(1, 11) = 14.01, p = 0.003. In contrast, the comparison group responded similarly to targets regardless of cue-target location (mean RT same location = 452 ms, SD = 79 ms; mean RT different location = 474 ms, SD = 90 ms), F(1, 11) < 1, n.s. The overall location by delay interaction indicates that both groups were exhibiting IOR: for example, all participants were slower to respond to targets which appeared in the same position as their cues, when there was a long cue-to-target delay (mean RT collapsed across groups = 496 ms), but not when there was a short cue-to-target delay (mean RT collapsed across groups = 475 ms). Conversely, all participants were faster to respond to targets which appeared in different positions to their cues, when there was a long cue-to-target delay (mean RT collapsed across groups = 430 ms), but not when there was a short cue-to-target delay (mean RT collapsed across groups = 498 ms).

**Anticipations** A one-way analysis revealed a similar number of anticipation errors for the HFA (mean number of anticipations = 2.63, SD = 4.50) and comparison group (mean number of anticipations = 2.66, SD = 4.20), F(1, 22) < 1, n.s.

**Misses** A four-way ANOVA similar to that conducted for the RT data revealed a main effect of delay, F(1, 22) = 10.43, p = 0.004, indicating that all participants were more likely to miss a target when the cue-to-target delay was short (mean number of misses = 3.01, SD = 3.79) compared to when the delay was long (mean number of misses = 1.85, SD = 2.75). No other main effects or interactions were found.

**False alarms** One-way ANOVA revealed that the HFA group committed significantly more false alarms (mean number of false alarms = 20.6, SD

= 9.99) than their respective comparison group (mean number of false alarms = 11.7, SD = 10.07), F(1, 22) = 4.75, p = 0.04.

# Asperger's disorder (AD)

**Reaction time** RT data were submitted to a four-way ANOVA. Again target side did not yield any significant interactions or main effects involving group, and so the RT data were collapsed. A trend towards an interaction of group by location by delay was revealed in the subsequent three-way ANOVA, F(1, 22) = 4.22, p = 0.052 (Figure 2a, b). Subsequent two-way ANOVAs confirmed that both groups were exhibiting the IOR effects (AD, F(1, 22) = 44.87, p = 0.001; comparison group, F(1, 22) = 19.52, p = 0.001). Omega square analysis (Keppel, 1991) revealed that 62 percent of the total variance in the Asperger's disorder analysis is accounted for by the IOR effect, while only 42 percent of the total variance in the analysis of comparison group data is accounted for by the IOR effect. Thus, while the same qualitative pattern of results was obtained for the AD and comparison groups, there was a trend towards the IOR effect being more pronounced in the AD group (Figure 2a, 2b).

**Anticipations** A one-way ANOVA revealed a similar number of anticipation errors for the AD (mean number of anticipations = 2.08, SD = 3.95) and comparison group (mean number of anticipations = 1.44, SD = 2.46), F(1, 22) < 1, n.s.

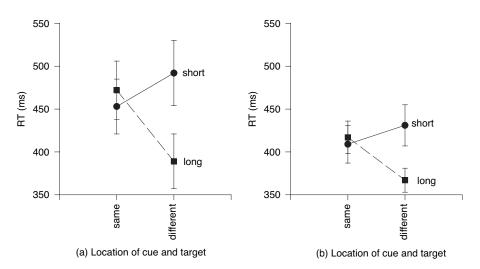


Figure 2 Mean RT (ms) for short (100 ms) and long (700 ms) cue-to-target delays as a function of cue-target location for (a) Asperger's disorder (AD) and (b) comparison groups (SE bars shown)

**Misses** A four-way ANOVA revealed an interaction of location by delay, F(1, 22) = 5.92, p = 0.02, indicating that both groups were more likely to miss a target when there was a short cue-to-target delay (mean number of misses = 2.50, SD = 2.86) versus long cue-to-target delay (1.39, SD = 2.23), or when a target appeared in the same position as the cue after a long delay (1.67, SD = 2.38), versus different position, long cue-to-target delay (1.10, SD = 2.07); this is consistent with the IOR effect.

**False alarms** The AD (mean false alarms = 11.4, SD = 12.50) and comparison groups (mean false alarms = 6.67, SD = 6.47) committed a similar number of false alarms, F(1, 22) < 1, n.s.

# Discussion

Contrary to our hypothesis, we did not find a more pronounced IOR in individuals with autism. While there was a very strong trend (p = 0.052) in favour of a more pronounced IOR in the Asperger's disorder group, this just failed to reach significance. The findings are, however, consistent with the body of research which has demonstrated that basic neuropsychological processes are intact in individuals with autism, particularly those processes which do not involve executive function or working memory (Minshew et al., 1997; Mottron et al., 1999). The finding of intact IOR in autism and Asperger's disorder furthers our knowledge about how these disorders differ from other neurodevelopmental psychiatric disorders which involve repetitive, stereotyped, and rigid symptom patterns. For example, while parallels have been drawn between the repetitive behaviours and frontostriatal neuropathology of autism and obsessive compulsive disorder (OCD) (Bradshaw, 2001; Hollander et al., 2005), unlike autism, the repetitive behaviours of OCD have additionally been linked to a lateralized reduced IOR (Rankins et al., 2004). Anecdotally, this disorder dissociation in IOR might explain why individuals with OCD show symptoms patterns such as perseveratively attending to a single, irrelevant location, e.g. repetitively locking a door, while individuals with autism tend to show different kinds of stereotypical behaviours.

It was interesting to find that individuals with autism committed almost twice the number of false alarm errors as the comparison group. A false alarm error means that the participant responded after seeing the cue in a catch trial where there is no subsequent target (i.e. responding somewhere between 100 ms and 2000 ms after a cue appears). The autism group responded to 20/40 catch trials, approximately twice the number as that responded to by the comparison and Asperger's disorder groups. The autism group committed a similar number of anticipation errors (i.e. responding before 100 ms) and misses (i.e. failing to respond after 2000 ms) to the comparison group. This pattern of errors would suggest that individuals with autism were not simply failing to respond to the task instructions, i.e. repetitively hitting the response bar at random time intervals every time a cue appeared. Rather, this pattern may be interpreted as an inhibitory deficit in a similar way as inhibitory deficits are inferred on the basis of increased false alarm errors on the stop-signal task (see Brian et al., 2003). For the stop-signal task ongoing response activity is intermittently interrupted on some trials by a tone that explicitly signals no response is required. In the IOR task the instruction not to respond is more implicit; for example, rather than a tone, participants are instructed to respond only when they see a red asterisk, and it is implied that they are not to respond when only a cue appears. The other point of departure from the stop-signal task is that the IOR task involves 'location-based' inhibition. This account would be consistent with Brian et al.'s (2003) prediction that individuals with autism may show inhibitory deficits in 'location-based' rather than 'identity-based' tasks.

Another possibility is that the increased false alarm rate for the autism group might relate to a response bias in favour of 'signal present' responses, induced by the much greater number of validly cued trials (i.e. 160) than invalidly cued or catch trials (i.e. 40). Thus, unlike the comparison groups and individuals with Asperger's disorder, individuals with autism may rely more on probabilities to determine their response (see also Ristic et al., in press).

It was curious to find that the autism group were disadvantaged at responding when a cue and target appeared in the same location at both the 100 ms and 700 ms delay, but performed similarly to the comparison group when the cue and target appeared in different locations. It is possible that the autism group were making more saccades between the cue and target presentations (see Kemner et al., 1998). This type of eye movement would have the most deleterious effect when cue and target were spatially coincident.

It will be interesting for future research to explore the trend towards a more pronounced IOR in the Asperger's disorder group. This could be achieved by titrating cue–target delay times and exploring the conditions under which an altered IOR anomaly might exist. Such experimentation may, for example, show that individuals with Asperger's disorder exhibit longer IOR effects as SOA is increased, but normal IOR effects when the SOA is less than 700 ms (but greater than 300 ms). Alternatively, IOR may become statistically pronounced in individuals with Asperger's disorder at a critical cue–target location disparity. If a more pronounced IOR is substantiated in individuals with Asperger's disorder, but not autism, then it will also be important for future studies to examine visual search ability separately in these two disorder groups.

The limitations of this research are similar to those in Rinehart et al. (2006) where the same participants were involved. In particular, while it would have been optimal to directly compare the high-functioning autism and Asperger's disorder groups, this was not deemed appropriate because of the developmentally critical age difference between the two groups, and the impact that this would have on the development of inhibitory function and attention. Another limitation of this research is that there was no objective measure of the participant's ability to fixate on the + at the onset of each trial. Notwithstanding, the RTs show differences between same and different is reasonable to assume that participants were correctly fixated in each trial.

In summary, this study indicates that IOR is intact in individuals with autism and Asperger's disorder. The finding that the autism group responded to more catch trials than the other groups may reflect an inhibitory deficit; alternatively it might be a behavioural marker that individuals with autism rely more on probabilities to determine their behavioural responses than non-affected individuals. This latter explanation fits well with the clinical observation that children with autism will often anticipate that if X happens then Y will follow, and become distressed if this logical appraisal of the world does not manifest; in contrast, a non-affected child will have a less rigid conceptualization of their environment.

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