Intact Automatic Imitation and Typical Spatial Compatibility in Autism Spectrum Disorder: Challenging the Broken Mirror Theory

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Lay Abstract

An inability to imitate is often described as a core feature of Autism Spectrum Disorder (ASD) and is consistent with the idea that individuals with ASD may possess a ‘broken’ mirror neuron system – the brain areas thought to underlie imitation. Previous research has questioned this assumption however, arguing that when tests of automatic imitation are used (which don’t require lots of other abilities in addition to imitation), imitative behavior is typical or even enhanced in autism. Two experiments are presented which test the theory that individuals with ASD have a specific impairment in imitation. Participants were asked to perform one of two finger movements in response to a number or colored square while observing a hand making either the same, or a different, finger movement. Experiment 1 demonstrated automatic imitation to be typical in individuals with ASD: finger actions were performed faster when the same action was observed, than when a different action was observed. A second experiment was able to distinguish automatic imitation from a phenomenon known as spatial compatibility – in which faster responses are made to events on the same side of space – and demonstrated that increased ASD symptom severity was related to increased, rather than decreased, imitation. These results show that the ability to imitate is intact in ASD, and challenge the ‘Broken Mirror Theory of Autism’.
Scientific Abstract

A lack of imitative behavior is frequently described as a core feature of Autism Spectrum Disorder (ASD), and is consistent with claims of mirror neuron system dysfunction in these individuals. Previous research has questioned this characterization of ASD however, arguing that when tests of automatic imitation are used - which do not require higher-level cognitive processing - imitative behavior is intact or even enhanced in individuals with ASD. In Experiment 1 60 adult individuals with ASD and a matched Control group completed an automatic imitation task in which they were required to perform an index or a middle finger lift while observing a hand making either the same, or the alternate, finger movement. Both groups demonstrated a significant imitation effect whereby actions were executed faster when preceded by observation of the same action, than when preceded by the alternate action. The magnitude of this ‘imitation effect’ was statistically indistinguishable in the ASD and Control groups. Experiment 2 utilized an improved automatic imitation paradigm to demonstrate that, when automatic imitation effects are isolated from those due to spatial compatibility, increasing autism symptom severity is associated with an increased tendency to imitate. Notably, there was no association between autism symptom severity and spatial compatibility, demonstrating the specificity of the link between ASD symptoms and increased imitation. These results provide evidence against claims of a lack of imitative behavior in ASD, and challenge the ‘Broken Mirror Theory of Autism’.

Key words: autism; imitation; mirror neurons; broken mirror theory; individual differences
Introduction

Autism Spectrum Disorder (ASD) is defined by persistent difficulties in social communication and interaction, with accompanying restricted and repetitive patterns of thought and behavior (American Psychiatric Association, 2013). The social and non-social impairments associated with ASD have been argued to be due to different aetiological factors, making a single explanation for the symptoms associated with ASD unlikely (Happé, Ronald, & Plomin, 2006). Within the social domain a great deal of attention has been focussed on the ability of individuals with ASD to imitate, due to the theorized importance of imitation for the development of socio-cognitive abilities including empathy, theory of mind, and language (Rogers & Pennington, 1991). The focus on imitation in ASD has been further increased due to the claim that an atypical or ‘broken’ mirror neuron system (MNS), the neural system subserving imitation (Catmur, Walsh, & Heyes, 2009; Cook, Bird, Catmur, Press, & Heyes, 2014; Heiser, Iacoboni, Maeda, Marcus, & Mazziotta, 2003; Iacoboni et al., 1999), may be responsible for the symptoms of ASD (Ramachandran & Oberman, 2006).

The Broken Mirror Theory has prompted a great deal of research examining the structural and functional integrity of the MNS in autism (see Hamilton, 2013 for a review of these studies). Empirical evidence examining the Broken Mirror Theory has produced highly mixed results, with as many studies reporting typical MNS structure and function in ASD as those finding impairments (Hamilton, 2013). Of concern is the fact that several studies claiming to support the Broken Mirror Theory have examined ‘mu’ suppression using Electroencephalography (EEG), which has recently been shown to index sensory rather than motor features of observed actions, and therefore to be unsuitable as an index of MNS functioning (Coll, Bird, Catmur, & Press, 2015). Other techniques used to measure MNS functioning in humans have also been criticized (Southgate & Hamilton, 2008; Hamilton, 2013), making imitation perhaps the best current index of MNS function.
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Williams, Whiten and Singh (2004) provided the first systematic review of imitative behavior in ASD, selecting 21 studies judged to be the most methodologically robust of an original 121 investigations. 14 studies found atypical imitative behavior in ASD, while the remaining 7 were judged to have produced uninterpretable results, prompting the authors to the overall conclusion that there is evidence of a specific imitation impairment in autism. It is noteworthy however, that all of the reviewed studies measured intentional or voluntary imitation – where the participant is explicitly asked to copy a model action (e.g. the ‘do-as-I-do’ task, Hayes & Hayes, 1952). These typically involve a number of higher-level cognitive and motivational processes known to be impaired in individuals with ASD, such as executive functioning, pragmatic language understanding, and rapid attentional switching (Courchesne et al., 1994; Happé & Frith, 1995; Russell, 1997; Williams, Goldstein, Carpenter, & Minshew, 2005). Thus, it may be impairments in processes other than imitation which lead to poor performance in tasks of voluntary imitation (Bird, Leighton, Press, & Heyes, 2007). Indeed, Leighton, Bird, Charman and Heyes (2008) demonstrated that individuals with ASD were as impaired on a non-imitative version of a voluntary ‘do-as-I-do’ task as they were on the imitative version. This result challenges the notion that ASD is associated with a specific imitation impairment - suggesting instead that poor performance on tests of voluntary imitation is due to more general cognitive impairments. It has therefore been suggested that in order to obtain a ‘pure’ measure of imitative performance in ASD one must use automatic imitation tasks, due to their reduced reliance on abilities other than imitation (Bird et al., 2007).

Tests of automatic imitation, based on classic Stimulus-Response Compatibility (SRC) paradigms (Kornblum, Hasbroucq, & Osman, 1990; Prinz, 1997), require action responses to be made to action stimuli, where the specific action performed by the stimulus is task-irrelevant. Stürmer, Aschersleben and Prinz (2000) first utilized this paradigm to
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demonstrate that individuals are faster to execute a hand movement whilst observing execution of the same (compatible) rather than a different (incompatible) hand movement. Such tasks are thought to measure automatic rather than intentional imitation because the movement compatibility is formally task-irrelevant yet significantly influences task responses.

Most recent versions of the automatic imitation paradigm (Brass et al., 2000, 2005, 2009; Catmur & Heyes, 2011; Catmur et al., 2009; Gowen, Bradshaw, Galpin, Lawrence, & Poliakoff, 2010; Leighton & Heyes, 2010) utilize a choice reaction-time (RT) task in which a non-action cue (a number or colored shape) specifies both the onset and type of response required for each trial (i.e. the cue acts as discriminative stimulus and imperative cue). For example, Brass and colleagues (Brass et al., 2000, 2005, 2009) instructed participants to perform an index or middle finger lift when presented with the number ‘1’ or ‘2’, respectively. At the same time, a task-irrelevant stimulus hand lifted either the same (imitatively compatible trial), or the alternative (imitatively incompatible trial) finger to that lifted by the participant. RT measures are then used to assess automatic imitation, where imitatively compatible trials result in speeded, and imitatively incompatible trials in slowed, responses when compared with baseline trials on which no action is observed.

A handful of studies have investigated automatic imitation in this manner in individuals with ASD and found it to be either typical (Hamilton, Brindley, & Frith, 2007; Press, Richardson, & Bird, 2010; Schunke et al., 2015), or even enhanced (Bird et al., 2007; Spengler, Bird, & Brass, 2010). Findings of increased automatic imitation is consistent with clinical symptoms of echolalia and echopraxia (the involuntary copying of the speech and actions of others) in ASD (Grossi, Marcone, Cinquegrana, & Gallucci, 2012). However, research to date has been restricted to small samples, potentially under-powered to detect group differences, and has included a number of studies utilizing facial emotion expressions
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or complex joint action tasks (McIntosh, Reichmann-Decker, Winkielman, & Wilbarger, 2006; Press et al., 2010; Sebanz et al., 2005). Consequently, it could be argued that task performance is more a product of face, emotion or higher level processing abilities than action imitation.

Experiment 1

Experiment 1 sought to provide a robust replication of previous findings of intact automatic imitation in a large sample of individuals with ASD using affect-neutral finger actions. An adapted version of the automatic imitation task (see Cook & Bird, 2012), originally presented by Brass and colleagues (Brass et al., 2000), was used in this study. The task provides two dissociable measures; automatic imitation (AI), the degree to which observation of an action (e.g. lifting of the index finger) prompts performance of the same action, and effector compatibility (EC), the non-imitative tendency for any response made with an effector to be executed faster when cued by the same effector, than when cued by a different effector. On EC trials, the on-screen hand remained stationary, rather than performing a lifting movement as on AI trials, and instead the compatible or incompatible finger (effector) was highlighted with a semi-transparent green mask. EC trials provide a non-imitative control, as they draw attention to the effector and influence response times, without movement of the finger, and therefore without the observed action necessary for imitation (Leighton & Heyes, 2010).

Method

Participants. Sixty high-functioning individuals with a clinical diagnosis of ASD (39 male; $M_{\text{AGE}} = 32.3$ years; $SD = 9.2$) were recruited through the autism outpatient clinic of the Charité University Medicine Berlin or referred by specialized clinicians and centers. An age, gender and IQ-matched sample of 45 healthy control individuals (26 male; $M_{\text{AGE}} = 32.5$
years; SD = 9.7) was recruited via online advertisements and a mailing list for potential participants held at the Freie Universität Berlin. All participants had normal or corrected-to-normal vision and individuals with ASD were diagnosed according to DSM-IV criteria (American Psychiatric Association, 1994), which was confirmed using the Autism Diagnostic Observation Schedule (ADOS; Lord et al., 2000). Twelve individuals in the ASD group, with a clinical diagnosis of ASD, failed to later meet the ADOS Autism Spectrum cut off (score of 7 or above) and a further 5 did not have an available ADOS score. The pattern of results was not altered by the inclusion or exclusion of these individuals, and thus the results reported below include all 60 cases with a clinical diagnosis of ASD.

Autism Spectrum Quotient (AQ; Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001) scores were significantly higher \([t(103) = 15.8, p < .001]\) in the ASD group \((M = 36.1, SD = 8.6)\) than the Control group \((M = 13.7, SD = 4.6)\), and the groups did not differ significantly in age \([t(103) = 0.41, p = .680]\), proportion of females \([\chi^2(1) = .57, p = .45]\), or IQ \([t(103) = 0.33, p = .741]\). Full details of ASD and Control group characteristics are presented in Table 1.

**Stimuli and Procedure.** The task was that used by Cook and Bird (2012) and the stimuli consisted of short video clips (subtending visual angles of 6° vertically and 9° horizontally and of 3000 ms in duration) of a stimulus hand presented on a blue background and rotated around the sagittal and transverse planes with respect to the participant’s hand (Fig. 1A).

Participants were seated approximately 80cm from the computer screen with their right arm supported on the table in front of them, and required throughout the task to use the index and middle fingers of their right hand to depress the ‘V’ and ‘B’ keys on the computer keyboard. On each trial participants were required to lift either their index or their middle finger on the appearance of a ‘1’ or ‘2’, respectively. On 50% of trials participants
simultaneously viewed a five-frame video clip of a human hand lifting the index or middle finger. This lifting action was either compatible or incompatible with the participant’s required finger lift (AI manipulation). On the remaining 50% of trials participants viewed three-frame trials on which the fingers of the observed hand remained static but either the compatible or incompatible finger was covered by a green mask (EC manipulation).

Participants first completed practice trials and were required to make 5 consecutive correct responses for each trial type (AI and EC). The main experiment consisted of 120 trials presented in a pseudo-random order, with an approximate duration of 15 minutes.

Results

Participants who were observed to be significant outliers (>1.5 times the interquartile range of AI or EC effects for each group) were excluded prior to data analysis (1 Control, 2 ASD). As in Cook and Bird (2011, 2012), trials in which an inaccurate finger-lift response was made (5% of trials for Control group; 6% of trials for ASD group; $t(103) = 1.8, p = .08$, or if RTs were less than 150 ms or greater than 2,000 ms, were discarded. AI and EC effects were derived by subtracting the mean RT on compatible trials from that on incompatible trials.

Response time data. RTs on imitatively compatible trials [Control (mean ± standard error of the mean) = 493 ± 10 ms; ASD = 502 ± 9 ms] were faster than those on their respective incompatible trials [Control = 536 ± 12 ms; ASD = 541 ± 9 ms] for both control individuals [$t(44) = 6.4, p < .001, d = 1.0$] and individuals with ASD [$t(59) = 9.2, p < .001, d = 1.2$]. In addition, trials in which the finger highlighted by the green mask was compatible (EC compatible trials) with the required response [Control = 504 ± 9 ms; ASD = 514 ± 9 ms], were faster than their respective EC incompatible trials [Control = 550 ± 13 ms; ASD = 557 ±
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10 ms] for both control individuals \[t(44) = 5.9, p < .001, d = 0.9\] and individuals with ASD \[t(59) = 8.0, p < .001, d = 1.0\].

In order to assess any differences in these effects between groups, a two-way, mixed-model ANOVA was performed on the RT data for both AI (Group × AI) and EC (Group × EC) trials, with Group constituting the between subjects factor (Control, ASD) and AI and EC (compatible, incompatible) the respective within-subjects factors. These data confirmed significant main effects of AI \[F(1,103) = 115.1, p < .001, \eta^2_p = .53\] and EC \[F(1,103) = 93.7, p < .001, \eta^2_p = .48\]. However, there was no interaction between either AI and Group \[F(1,34) < 1, p = .49, \eta^2_p = .002\] or EC and Group \[F(1,34) < 1, p = .74, \eta^2_p = .001\]. Figure 1B shows AI and EC effects for both the Control and ASD groups.

**Interim Discussion and Experiment 2**

Experiment 1 successfully replicates previous findings of intact automatic imitation in a large sample of individuals diagnosed with ASD. However, despite the popularity of automatic imitation paradigms, some forms of these tests have been argued to be unsuitable to test imitation, due to the fact that the RT benefit observed on imitatively compatible trials may not be a product of automatic imitation but rather a product of spatial compatibility (Aicken et al., 2007; Bertenthal, Longo, & Kosobud, 2006; Jiménez et al., 2012). Spatial compatibility effects elicited by the task-irrelevant spatial properties of task-relevant stimuli were first described by Simon and colleagues (Simon, 1968, 1969, Simon & Rudell, 1967) who showed that the spatial location of a stimulus (even if that location is task-irrelevant) facilitates movement responses on the same side of space. Thus, if the spatial location of an observed action matches the spatial location of the required response in an automatic imitation task, then it is possible that the faster responses to matching actions may be due to spatial compatibility rather than automatic imitation. While this criticism cannot explain
some reports of intact automatic imitation in ASD (Bird et al., 2007), it may explain others (Hamilton et al., 2007; Schunke et al., 2015; Press et al., 2010; Spengler et al., 2010), where group differences in automatic imitation may be masked by spatial compatibility.

The paradigm used in Experiment 1 and Cook and Bird (2011, 2012) rotates the stimulus hand with respect to the participant’s hand in order to eliminate a simple spatial compatibility account of the observed automatic imitation effect. However, it is possible that results may be explained by orthogonal spatial compatibility effects whereby stimuli in the upper portion of space facilitate responses on the right side of space, and stimuli in the lower portion of space facilitate responses on the left side of space (Cho & Proctor, 2004).

Experiments with typical individuals have overcome these methodological problems by introducing a spatial compatibility control condition to demonstrate the presence of automatic imitation independently of simple and orthogonal spatial compatibility effects (Catmur & Heyes, 2011; Jiménez et al., 2012; Sowden & Catmur, 2015). Experiment 2 utilizes one of these paradigms in order to re-examine automatic imitation in ASD. Typical, or even enhanced, imitation shown by individuals with ASD in this experiment cannot be due to spatial compatibility.

Method

Participants. Eighteen high-functioning individuals with a clinical diagnosis of ASD (2 female; 1 left-handed; $M_{\text{AGE}} = 37.7$ years, SD = 12.6) and an age, gender and IQ-matched sample of 18 healthy control individuals (3 female; 1 left-handed; $M_{\text{AGE}} = 34.9$ years, SD = 15.2) were recruited from a database held at the Institute of Psychiatry, Psychology and Neuroscience, King’s College London. All participants reported normal or corrected-to-normal vision. Two further individuals with ASD were excluded prior to data analysis as they made errors on more than 15% of trials (Sowden & Catmur, 2015), but all received a small
monetary reward for their participation. Participants with ASD had previously received independent clinical diagnoses (according to DSM-IV criteria; American Psychiatric Association, 1994), and diagnosis was confirmed using the ADOS. AQ scores were significantly higher \([t(34) = 7.0, p < .001]\) in the ASD group \((M = 35.1, SD = 9.0)\) than the Control group \((M = 17.1, SD = 6.3)\), and the two groups did not differ in age \([t(34) = 0.6, p = .55]\), proportion of females \([\chi^2(1) = .23, p = .63]\), or IQ \([t(34) = 0.3, p = .80]\). Full details of ASD and Control group characteristics are presented in Table 1.

**Stimuli and Procedure.** Experimental stimuli were the same as those used by Sowden and Catmur (2015) and presented in color on a black background (See Figure 1C for full trial illustration). Task-irrelevant stimuli were images of a human right or left hand subtending a visual angle of 6.6° horizontally and either 8.5° (static and pixelated control hand), 9.1° (middle finger lifted) or 9.3° (index finger lifted) vertically. Middle and index finger lifts were superimposed onto the resting hand, subtending an angle of 0.6° and 0.7° respectively and left hand stimuli were a direct mirror along the vertical plane of right hand stimuli.

The instantaneous presentation of the movement hand after the static hand produced apparent motion of the finger (lifting of the index or middle finger); previously shown as a robust means of eliciting compatibility effects (Press, Bird, Flach, & Heyes, 2005). Spatial compatibility (SC) was manipulated by using left and right hand stimuli. For example, in a spatially incompatible trial, a participant prompted to lift their right index finger would observe a left hand lifting its index finger. This allowed the spatial location of the observed finger movement to be manipulated independently from finger identity. Task-relevant (discriminative) stimuli comprised squares (occupying a 0.2° visual angle) colored orange or purple. A white square of identical dimensions operated as a fixation point (positioned halfway between the index and middle fingers of the static hand). The allocation of response
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cues (colored squares) to response options (index or middle finger lift) was counterbalanced across participants, with purple and orange squares indicating whether the participant should lift their index finger (from the ‘N’ key) or middle finger (from the ‘M’ key) on each trial.

On baseline trials the static hand was replaced by a pixelated hand which was designed so that it did not elicit SC or AI effects, but matched the transient, alerting visual change of the task-irrelevant movement hand in the standard trials (Wiggett, Downing, & Tipper, 2013).

Participants sat approximately 80cm from the laptop screen, placing their right arm (in the same orientation as the hand stimuli) on the table in front of them, and responses were made with the right hand using an external keyboard. Participants completed 10 practice trials, and were required to repeat these until achieving at least 80% accuracy. The main task consisted of 3 blocks of 36 trials, with each block lasting approximately 4 minutes.

Hand stimuli were formally task irrelevant but allowed the independent manipulation of SC and AI. Thus, the on-screen hand performed either imitatively compatible or incompatible actions (AI manipulation) on the same or different side of space (SC manipulation) to the response required by the participant. Hand stimuli in the standard trials were manipulated in a 2 × 2 (AI × SC) design, resulting in four main trial types with a further two baseline trial types for left and right hand stimuli (see Figure 1C). Each of these six trial types were presented 18 times in a randomized order across each block. A fully factorial combination of the six trial types, stimulus onset asynchronies and square color amounted to a total of 36 trials, which made up one full block in the experiment.

Results

Data were processed as in Sowden and Catmur (2015), with trials in which an inaccurate finger-lift response was made (4% of trials for Control group; 5% of trials for
ASD group; \( t(34) = 0.9, p = .40 \), or if RT significantly deviated (±2.5 SD) from each participant's mean RT (< 2% of trials), discarded. Mean RT and number of errors were calculated for each of the six trial types for control and ASD individuals. Compatibility effect data was derived by subtracting the mean RT on compatible trials from that on incompatible trials.

**Response time data.** RTs on imitatively compatible trials were faster than those on their respective incompatible trials for both the Control group [compatible = 517 ± 15 ms; incompatible = 533 ± 19 ms; \( t(17) = 2.7, p = .015, d = 0.6 \)] and the ASD group [compatible = 606 ± 39 ms; incompatible = 637 ± 48 ms; \( t(17) = 2.9, p = .010, d = 0.7 \)]. Similarly, RTs on spatially compatible trials were faster than those on their respective incompatible trials for both the Control group [compatible = 502 ± 17 ms; incompatible = 548 ± 18 ms; \( t(17) = 7.3, p < .001, d = 1.7 \)] and the ASD group [compatible = 591 ± 44 ms; incompatible = 653 ± 43 ms; \( t(17) = 7.0, p < .001, d = 1.7 \)]. Separate two-way, repeated-measures ANOVAs were performed on the RT data for both groups, with within-subjects factors of SC (compatible, incompatible) and AI (compatible, incompatible). These analyses revealed a significant main effect of SC for both the Control group, \( F(1,17) = 53.2, p < .001, \eta^2_p = .76 \), and the ASD group, \( F(1,17) = 49.0, p < .001, \eta^2_p = .74 \), as well as a significant main effect of AI for both the Control, \( F(1,17) = 7.4, p = .015, \eta^2_p = .30 \), and the ASD, \( F(1,17) = 8.5, p = .010, \eta^2_p = .33 \), groups. There was no significant interaction between SC and AI for either the Control, \( F(1,17) < 1, p = .74, \eta^2_p = .007 \), or the ASD, \( F(1,17) < 1, p = .79, \eta^2_p = .004 \), groups. Figure 2D shows SC and AI effects (incompatible RTs – compatible RTs) for both the Control and ASD groups. Both effects are apparent on RTs, with SC effects typically larger than AI effects in both groups. SC and AI effects on RTs are of magnitudes consistent with previous studies using a similar paradigm (Catmur & Heyes, 2011; Sowden & Catmur, 2015).
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In order to assess any differences in these effects between groups, a further three-way, mixed ANOVA (Group × SC × AI) was performed on the RT data, with Group constituting the between subjects factor (control, ASD). These data once again confirmed significant main effects of SC, $F(1,34) = 98.8, p < .001, \eta^2_p = .74$, and AI, $F(1,34) = 14.9, p < .001, \eta^2_p = .30$, as well as no interaction between SC and AI, $F(1,34) < 1, p = .70, \eta^2_p = .004$. Despite SC and AI effects being numerically larger in individuals with ASD, neither the main effect of Group, nor the interaction between Group and SC, $F(1,34) = 2.1, p = .16, \eta^2_p = .06$, or Group and AI, $F(1,34) = 1.6, p = .22, \eta^2_p = .04$, were significant. Finally, no significant bivariate correlation was observed between SC and AI ($r = .04, p = .80$).

**Hand × response effects.** To ensure that trials did not elicit different RTs or response errors depending on whether a left or right task-irrelevant hand was presented or whether an index or middle finger response was required, a further two-way, repeated-measures ANOVA (hand × response) was carried out on the RT data from baseline trials. The within-subject factors were the hand presented (left, right) and the response required (index, middle). This analysis revealed no significant main effects of hand presented or response required, as well as no interaction between the two factors (all $p > .05$). These results suggest that the observed compatibility effects were unlikely to have been generated by general stimulus or response factors.

**Autism severity and imitation effects.** Although the AI effect observed in the RT data from the ASD group was not significantly larger than that observed in the control group (perhaps due to insufficient power to detect group differences), the AI effect in the ASD group was numerically larger in the ASD group than in the Control group. Thus, the relationship between autism severity and AI was assessed. ADOS severity scores showed a significant
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correlation with AI ($r = .56, p = .02$), with higher ASD severity scores correlating with higher AI effects. No correlation was observed between ADOS scores and SC ($r = -.06, p = .82$), with a significant difference observed between these two correlation coefficients using Fisher’s $r$-to-$z$ test ($z$-score $= 1.90, p = .03$).

General Discussion

It has previously been argued that imitative behavior and MNS function are reduced in individuals with ASD (Williams et al., 2006). However, previous investigations of these claims have often tested the performance of individuals with ASD on tests of voluntary imitation, which in addition to imitation require many abilities which have been shown to be impaired in ASD. Experiment 1 utilized a test of automatic imitation in a much larger sample of adults with ASD than obtained previously, and revealed automatic imitation to be intact in individuals with ASD and comparable to that demonstrated by a typical Control group. Due to the possibility that performance on the task used in Experiment 1 may be affected by orthogonal spatial compatibility, potentially masking any group differences in automatic imitation, Experiment 2 used a novel task allowing the independent manipulation and measurement of both automatic imitation and spatial compatibility. This not only allowed the observation that AI effects persist regardless of SC in both individuals with ASD and typical control individuals, but that AI (and not SC) effects are increased with increasing autism symptom severity, as indicated by ADOS scores.

Accordingly, our data demonstrate, contrary to the prevailing view (Williams et al., 2001, 2004), that individuals with autism show intact imitative behavior. In fact, enhanced AI (but not SC) effects were observed in association with higher autism severity. This finding is in line with symptoms of echolalia and echopraxia observed in many individuals with severe ASD, and with previous investigations showing hyper imitative performance in ASD (Bird et
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al., 2007; Grossi et al., 2012; Spengler et al., 2010). As it has been shown that AI effects as described here rely on the MNS (Catmur et al., 2009; Heiser et al., 2003; Heyes, 2011), and that performance on such experimental tasks of AI is associated with mimicry during everyday social interaction (Hogeveen & Obhi, 2012), these data speak to the longstanding debate regarding the integrity of the MNS in ASD. They suggest that the Broken Mirror Theory of autism is unlikely to be a valid explanation of the broad deficits observed in ASD. With the numerous methodological issues associated with neuroscientific investigations of MNS function in humans (Hamilton et al., 2013; Coll et al., 2015), the present study provides some of the most promising evidence to date for intact MNS functioning in ASD.

Rather than characterizing ASD as due to a broken MNS, a more promising approach, in line with our findings of enhanced imitative behavior with increased autism severity, involves conceptualizing neurodevelopmental disorders like ASD as disorders of top-down control or modulation of social behavior (Bird, Catmur, Silani, Frith, & Frith, 2006; Cook, Barbalat, & Blakemore, 2012; Frith, 2003; Schunke et al., 2015; Sowden & Shah, 2014; Wang & Hamilton, 2012). In accord with this notion, Cook and Bird (2012) demonstrated that the modulatory effects of pro-social priming on automatic imitation in typical individuals were absent in those with ASD. Similarly, Spengler et al. (2010) found hyper-imitation to be correlated with impaired theory of mind in these individuals. Hence, a deficiency within a neural network which supports the top-down control of representations of ‘self’ and ‘other’ seems plausible in ASD and would account for hyper-imitation as well as impairments in other socio-cognitive functions in these individuals (Sowden & Shah, 2014). One such neural network suggested to subserve this role in the human brain involves the medial prefrontal cortex and temporoparietal junction (Brass et al., 2005; Santiesteban, Banissy, Catmur, & Bird, 2012; Spengler, von Cramon, & Brass, 2010); a network whose role and function in
self-other processing, and its interplay with the MNS, deserves attention in future autism research.

Conclusion

Imitation, and its neural substrate the MNS, has garnered a great deal of attention and controversy in the study of ASD. In contrast to the Broken Mirror Theory of ASD, this behavioral study demonstrated that automatic imitation is intact or even enhanced in individuals with ASD; indicative of a functional MNS. Experiment 1 established robust automatic imitation in a large sample of individuals with ASD. Experiment 2 isolated automatic imitation from that of spatial compatibility, to show that AI persists in both typical individuals and those with ASD when spatial compatibility is controlled for, and increases with autism symptom severity in autistic individuals. Thus, amongst an abundance of mixed neuroscientific evidence, AI tasks provide a pure and robust measure of imitative performance and offer some of the most promising evidence to date against a broken MNS in ASD.
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Table 1. Participant characteristics for Experiment 1 and 2.

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<td>Mean Age (years)</td>
<td>33.3 (9.2)</td>
<td>32.5 (9.7)</td>
<td>37.7 (12.6)</td>
<td>34.9 (15.2)</td>
</tr>
<tr>
<td>Mean Full-scale IQ</td>
<td>109.2 (15.2)</td>
<td>108.2 (12.5)</td>
<td>113.6 (13.8)</td>
<td>114.7 (10.8)</td>
</tr>
<tr>
<td>Mean AQ</td>
<td>36.1 (8.6)</td>
<td>13.7 (4.6)</td>
<td>35.1 (9.0)</td>
<td>17.1 (6.3)</td>
</tr>
<tr>
<td>ADOS Classification</td>
<td>25 Autism, 18 Autism Spectrum</td>
<td>n/a</td>
<td>11 Autism, 7 Autism Spectrum</td>
<td>n/a</td>
</tr>
<tr>
<td>Mean ADOS-G Score</td>
<td>9.4 (0.5)</td>
<td>n/a</td>
<td>10.61 (2.7)</td>
<td>n/a</td>
</tr>
</tbody>
</table>

NB. ADOS classifications and mean ADOS-G scores apply only to the 48 ASD individuals who met the ADOS classification cut off (≥7).
INTACT AUTOMATIC IMITATION IN AUTISM

A) Intact automatic imitation in autism

B) Experiment 1:

<table>
<thead>
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<th>Control</th>
<th>ASD</th>
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<tbody>
<tr>
<td>AI trial</td>
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<tr>
<td>EC trial</td>
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C) Please replace fingers on the keys

D) Experiment 2:

<table>
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<tr>
<td>CI</td>
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</tbody>
</table>

E) Experiment 2:

Automatic imitation vs ADOS-G score
Figure Legend

**Figure 1.** (A) Examples of automatic imitation (AI) and effector compatibility (EC) trials from Experiment 1 (Cook & Bird, 2012). ‘1’ indicates lift index finger, and thus the examples shown are from imitatively compatible and effector compatible trials. (B) AI and EC effects for both Control and ASD groups. (C) Examples of one trial structure from Experiment 2 and spatial and imitative compatibility of hand stimuli where an orange square indicates a required index finger lift. Movement hands presented at varying stimulus onset asynchronies (SOA: 1600, 2000 or 2400 ms) after the static hand. Hand stimuli from left and right baseline trials are also shown, during which the static hand is replaced by a pixelated hand eliciting no finger movement. (D) Automatic imitation and spatial compatibility effects for both Control and ASD groups (E) Scatterplot of the correlation between autism symptom severity (ADOS-G scores) and AI from Experiment 2. NB. Error bars represent standard error of the mean.